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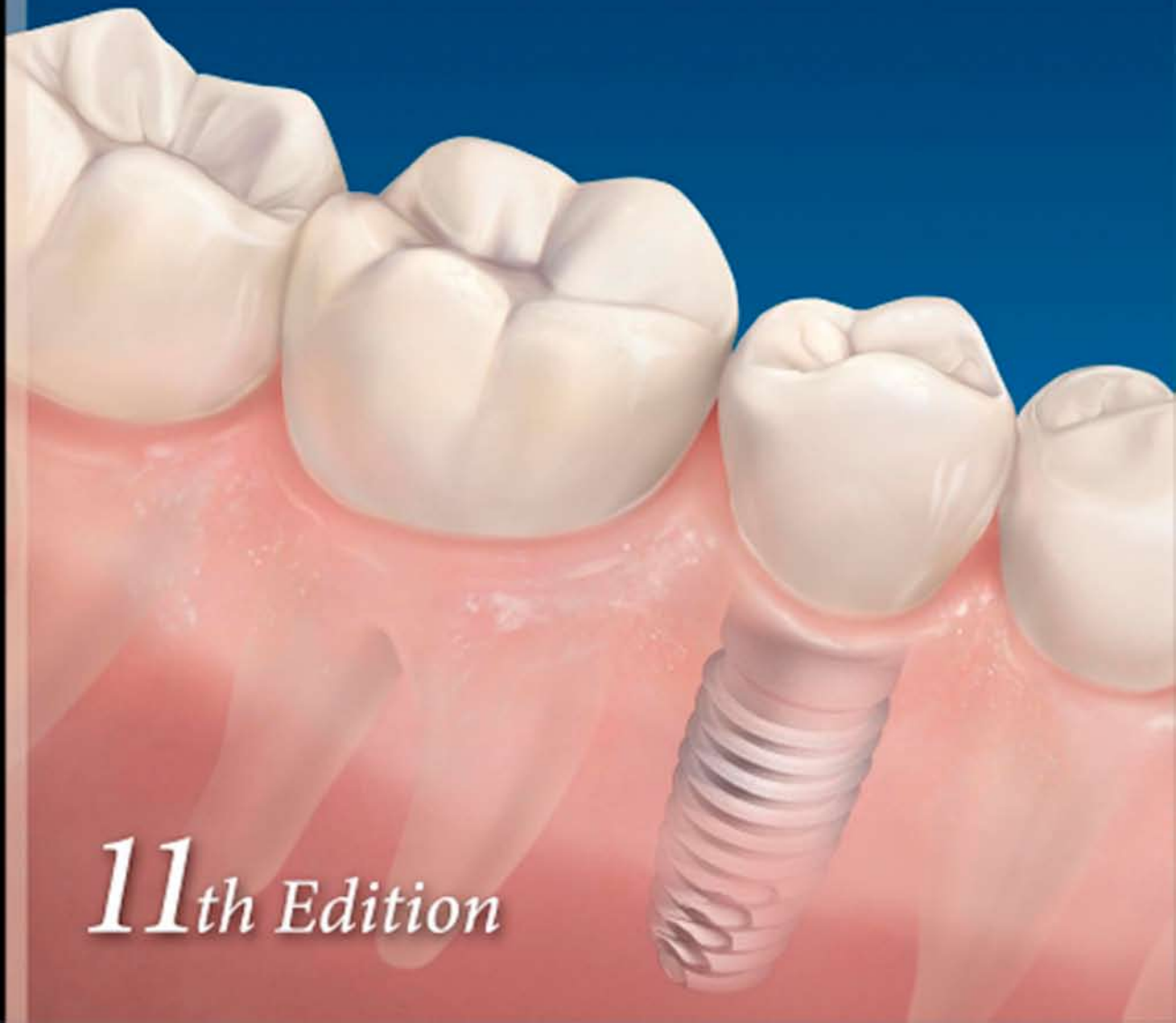

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Dr. Michael G. Newman graduated from the University of California, Los Angeles (UCLA) College of Letters and Sciences with a degree in psychology. He completed his dental training at the UCLA School of Dentistry in 1972. Dr. Newman received a Certificate in Periodontics and Oral Medicine at the Harvard School of Dental Medicine and a Certificate in Oral Microbiology from the Forsyth Dental Institute under the mentorship of Dr. Sigmund Socransky. He is a diplomate of the American Board of Periodontology and is Professor Emeritus of periodontics at the UCLA School of Dentistry. Dr. Newman is a fellow and past president of the American Academy of Periodontology. In 1975, he won the Balint Orban Memorial Prize from the American Academy of Periodontology. He had been in private practice of periodontics for more than 25 years. In 2007, Dr. Newman received the Gold Medal, the highest honor bestowed by The American Academy of Periodontology.

Dr. Newman has published more than 250 abstracts, journal articles, and book chapters and has co-edited eight textbooks. He has served as an ad hoc reviewer for the National Institute of Dental and Craniofacial Research, is a consultant to the Council on Scientific Affairs of the American Dental Association, and is a reviewer for numerous scientific and professional journals and governmental research organizations.

Dr. Newman has lectured throughout the world on microbiology, antimicrobials, evidence-based methodology, risk factors, and diagnostic strategies for periodontal disease. Dr. Newman has a strong interest in applied science and transfer of new technology for practical use. He has served as a consultant to many major dental and pharmaceutical companies throughout the world. Dr. Newman is the founding editor in chief of the *Journal of Evidence-Based Dental Practice* and was the associate editor of the *International Journal of Oral and Maxillofacial Implants*.

Henry H. Takei, DDS, MS, FACD

Dr. Henry H. Takei graduated in 1965 from the Marquette University School of Dentistry in Milwaukee, Wisconsin. He completed his Periodontics Certificate and Master of Science degree in 1967 at the Marquette University and the Veterans Administration Hospital in Wood, Wisconsin.

Dr. Takei is a Distinguished Clinical Professor of periodontics at the UCLA School of Dentistry and a consultant in periodontics at the Veterans Administration Hospital in Los Angeles. He maintains a private practice limited to periodontics and implant surgery.

Dr. Takei has published numerous articles on periodontal surgery and has contributed chapters to five textbooks. He has been actively involved in continuing education and has lectured throughout the world on clinical periodontology and implant surgery.

Dr. Takei has been honored nationally and internationally with awards from numerous periodontal organizations, universities, and study clubs for his contributions to education.

He received the Distinguished Alumnus Award by Marquette University in 2001 and the Honorary Distinguished Alumnus Award by the University of California, Los Angeles in 1998. The American Academy of Periodontology has honored Dr. Takei with the prestigious Master Clinician Award in 2006.

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Perry R. Klokkevold, DDS, MS

Dr. Perry R. Klokkevold graduated from the University of California, San Francisco School of Dentistry in 1986. His postdoctoral clinical training includes a general practice residency in hospital dentistry completed in 1987, a postgraduate periodontal residency completed in 1994, and a surgical implant fellowship completed in 1995. All of his postgraduate training was completed at the UCLA School of Dentistry. He completed a Master of Science degree program in oral biology at UCLA in 1995.

Dr. Klokkevold is a diplomate of the American Board of Periodontology. He is an associate professor in the Division of Associated Specialties, Section of Periodontics, at the UCLA School of Dentistry and director of the UCLA Postgraduate Periodontics Residency program. Dr. Klokkevold previously served as clinical director and director of the general practice residency program in hospital dentistry at UCLA School of Dentistry from 1987 to 1992. Dr. Klokkevold has maintained a faculty practice limited to the specialty of periodontics and dental implant surgery at UCLA for more than 15 years.

Dr. Klokkevold has published numerous articles for international journals and has written several book chapters on topics that include juvenile periodontitis, periodontal medicine, influence of systemic disease and risk factors on periodontitis, bone regeneration, and dental implants. He is a reviewer for several journals, including the *Journal of Periodontology* and the *International Journal of Oral and Maxillofacial Implants*. Dr. Klokkevold has lectured nationally and internationally on many periodontal and implant-related topics.

Fermin A. Carranza, Dr Odont, FACD

Dr. Fermin A. Carranza graduated from the University of Buenos Aires School of Dentistry in Argentina in 1948 and completed his postdoctoral training in periodontics at Tufts University School of Dental Medicine in 1952 under the mentorship of Dr. Irving Glickman.

Dr. Carranza is professor emeritus of periodontology at the UCLA School of Dentistry. He was head of the Department of Periodontics at the University of Buenos Aires from 1966 to 1974, and at UCLA from 1974 until his retirement in 1994.

Dr. Carranza has published more than 218 scientific papers and abstracts on basic and applied aspects of periodontics and 18 books, including the past five editions of Clinical Periodontology. He has received numerous awards and recognition for his work, including the IADR Science Award in Periodontal Disease and the Gies Award of the American Academy of Periodontology.

Dr. Carranza has lectured throughout the world on clinical periodontology, pathology, and therapy.

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PREFACE

This eleventh edition is the most comprehensive textbook and information resource about periodontology available anywhere. Using Elsevier's advanced technology and high standards of quality, the international team of editors and contributors have developed a major periodontal resource intended to be used by everyone with an interest in periodontology.

Our goal in completely revising this classic textbook was to preserve the same tradition of comprehensiveness and organization that has made the book the number one choice among educators, students, clinicians, researchers, and policy makers. And, for the first time, the book will become part of Elsevier's Expert Consult series in medicine and dentistry.

Since publication of the first edition of this book 58 years ago, periodontology has made tremendous progress. Analysis of periodontal tissues and elucidation of the mechanisms and causes of disease have extended far beyond histology and physiology and into the realm of cellular and molecular biological understanding.

New therapeutic goals and clinical techniques, based on an improved understanding of disease, have facilitated better outcomes and brought us closer to achieving the ultimate goal of optimal periodontal health and function. Today, reconstruction and regeneration of lost periodontal structures, replacement of compromised teeth with implants, and creation of esthetic results are integral parts of clinical practice. This book is written for all individuals—from students to educators, clinicians to researchers, and specialists to generalists. It is our belief that periodontal care of the public is primarily the role of general dentists and dental hygienists. The responsibility to examine, diagnose, and either treat or refer all periodontal problems is unquestionable. Likewise, the role of periodontitis in systemic disease makes diagnosis essential and treatment imperative.

The multifaceted, complex task of producing the eleventh edition required the collaboration of numerous experts from various fields, and their contributions are invaluable. We know that this new edition will continue to be as useful to dentists, hygienists, periodontists, students, educators, and researchers as the previous editions and that it will contribute to continuous progress of our profession.

Michael G. Newman
Henry H. Takei
Perry R. Klokkevold
Fermin A. Carranza

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ACKNOWLEDGMENTS

The originator of this book and author of its first four editions, published in 1953, 1958, 1964, and 1972, was Dr. Irving Glickman, professor and chairman of the Department of Periodontology at Tufts University School of Dental Medicine, in Boston, Massachusetts.

After Dr. Glickman's death in 1972 at age 58, responsibility for continuing this book moved to Dr. Fermin A. Carranza, once a student and collaborator of Dr. Glickman. At the time Dr. Carranza was professor and chairman of periodontics at the School of Dentistry, University of California, Los Angeles (UCLA). The following four editions were published in 1979, 1984, 1990, and 1996 under the guidance of Dr. Carranza, who is now professor emeritus at UCLA.

In 2002, the task of maintaining the book's tradition of almost half a century changed hands once again. Dr. Michael G. Newman and Dr. Henry H. Takei joined Dr. Carranza to take major responsibility for the ninth edition.

For the tenth edition, Dr. Perry R. Klokkevold from UCLA joined as a co-editor.

Advances in basic science and clinical techniques have increased the knowledge base so dramatically that it is virtually impossible for a single individual to memorize and retain all of the information needed to practice excellently. It is also a certainty that the enormity of providing the information of a discipline must be borne by many experts willing to share their experience and knowledge.

The editors are indebted to everyone who has worked tirelessly to produce this milestone print and electronic edition of *Carranza's Clinical Periodontology*.

Many scientists and clinicians have shared their knowledge and expertise from previous editions of *Carranza's Clinical Periodontology*, but their names no longer appear as contributors to the present one. We express our deep gratitude to all of them for their many valuable concepts and ideas.

Our appreciation is given to Elsevier and particularly to John Dolan and Brian Loehr. Their expertise and detailed attention to every word and every concept contributed greatly to producing a quality book and a truly useful website.

We express gratitude to our parents, colleagues, friends, and mentors who have always been so tolerant, encouraging, and understanding, and who guided our first steps in our profession and helped us develop our ideas in the field.

Dr. Newman: my family: Susan, Andrea, Natalie, Scott and Kara my parents Paul, Rose, John, and Inez. Sigmund S. Socransky, Stephen Stone, J.D. Murray, Fermin A. Carranza, Jr., and Henry H. Takei: My gratitude to my co-editors and contributors whose expertise and willingness to participate in this work have made this book an excellent educational standard.

Dr. Takei: My wife, June; my children Scott and Akemi; their spouses, Kozue and David; my grandchildren, Hana, Markus, Carter, and Arden. My graditude to my mentors Dr. Fermin A. Carranza, Jr., Dr. Donald Van Scotter, Dr. Delbert Nachazel and Dr. John Pfeiffer. Thank you to my three co-editors and friends Michael G. Newman, Fermin A. Carranza, Jr., and Perry R. Klokkevold. Special gratitude to Laura Miyabe for her professional support. I would like to acknowledge and thank all of my periodontal postdoctoral students at UCLA for their help and support throughout the preparation of this classic textbook.

Dr. Klokkevold: my wife, Angie; my daughters Ashley and Brianna; my parents Carl and Loretta; my mentors Henry H. Takei, John Beumer III, Bradley G. Seto, Charles N. Bertolami, Peter K. Moy, and E. Barrie Kenney. I thank all of the residents with whom I have had the pleasure to work over the years, for the passion and inspiration they bring to me as an educator and clinician. Special thanks to my co-editors, Michael G. Newman, Henry H. Takei, and Fermin A. Carranza, Jr., for their friendship, support, and encouragement.

Dr. Carranza: my wife, Rita; my children Fermin, Patricia, and Laura; and my grandchildren. Irving Glickman, Fermin Carranza, Sr., and Romulo L. Cabrini. My gratitude also to my co-editors, who will continue the tradition of this book.

Michael G. Newman
Henry H. Takei
Perry R. Klokkevold
Fermin A. Carranza

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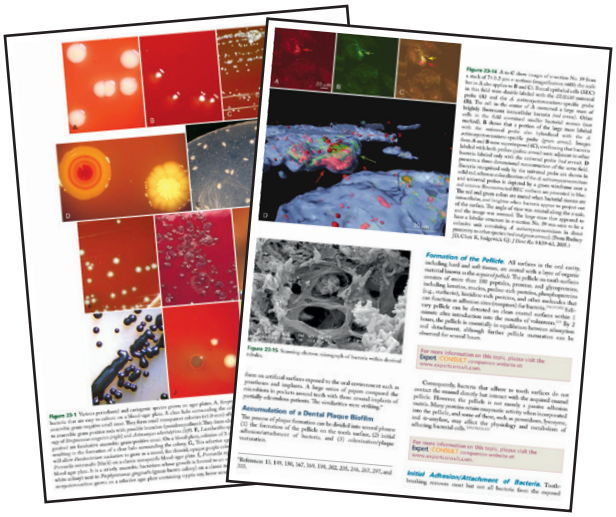
Part IV: Etiology of Periodontal Diseases focuses on dental students' comprehensive education in periodontal anatomy, physiology, and pathogenesis.



COMPLETELY REVISED

Chapter 24 Genetic Factors and Periodontal Disease, is written by world-renowned experts in their fields.

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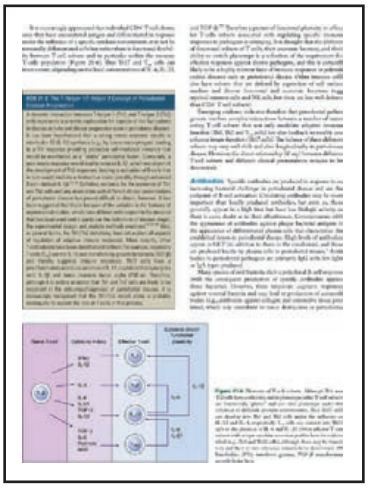
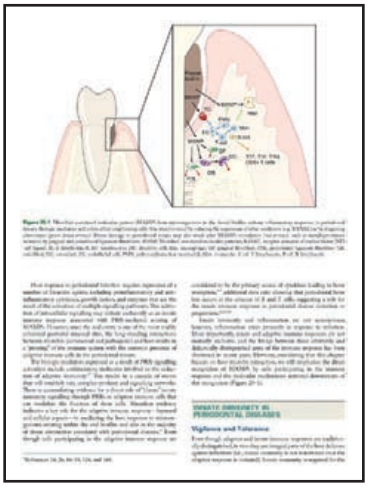


Chapter 25 Molecular Biology of Host-Bacterial Interactions



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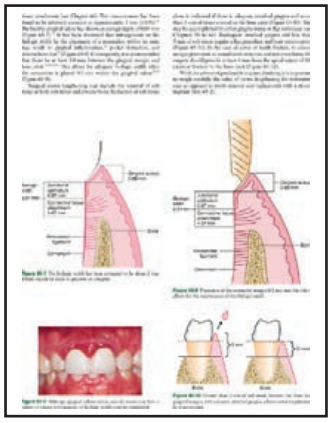
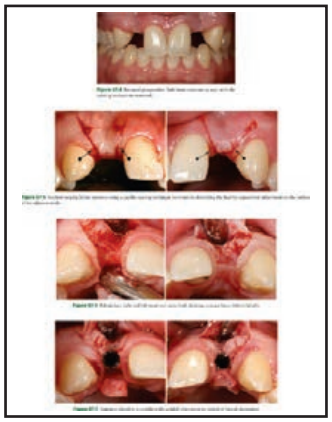
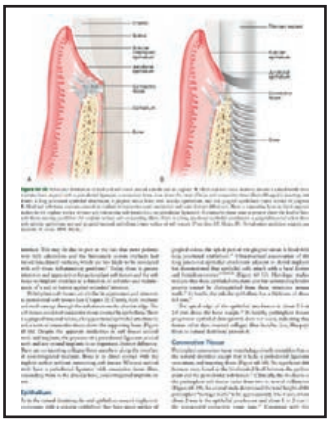
Chapter 65 Preparation of the Periodontium for Restorative Dentistry



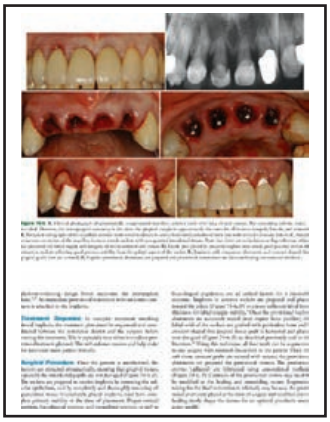
Chapter 67 Multidisciplinary Approach to Dental and Periodontal Problems



Chapter 68 Periimplant Anatomy, Biology, and Function



Chapter 74 Esthetic Management of Difficult Cases



ATLAS OF PERIODONTAL PATHOLOGY (ONLINE ONLY)

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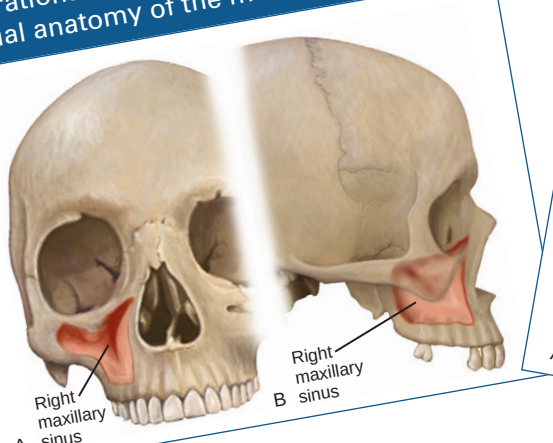
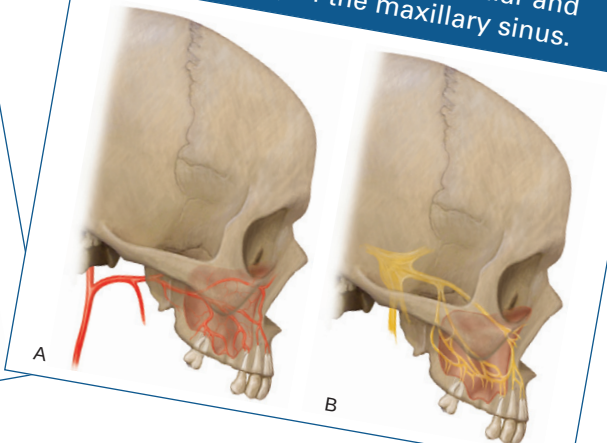
NEW ART

New illustrations identify clinically important anatomy

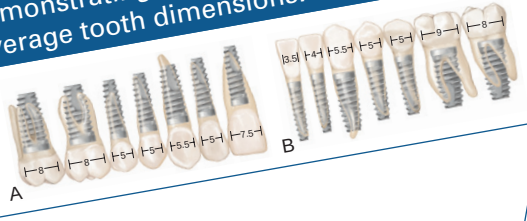
Illustrations showing the location and spacial anatomy of the maxillary sinus.



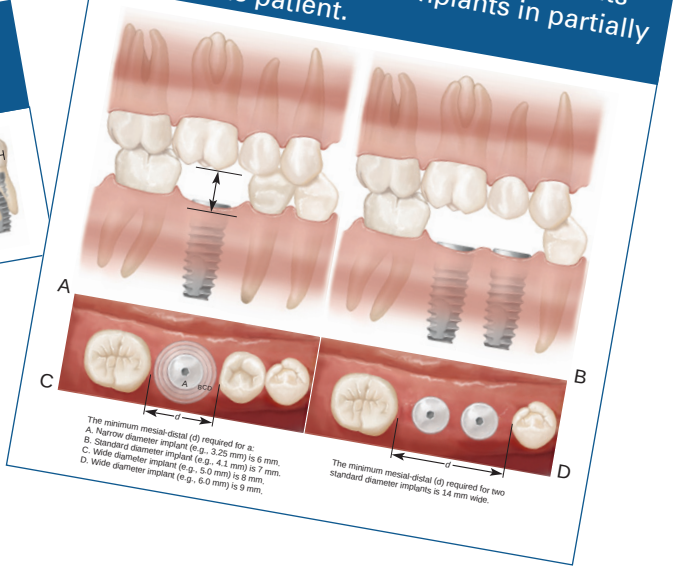
Illustrations depicting the vascular and neural anatomy of the maxillary sinus.



Illustrations with superimposed implants demonstrating various sizes relative to average tooth dimensions.



Illustrations defining space requirements for single and multiple implants in partially edentulous patient.



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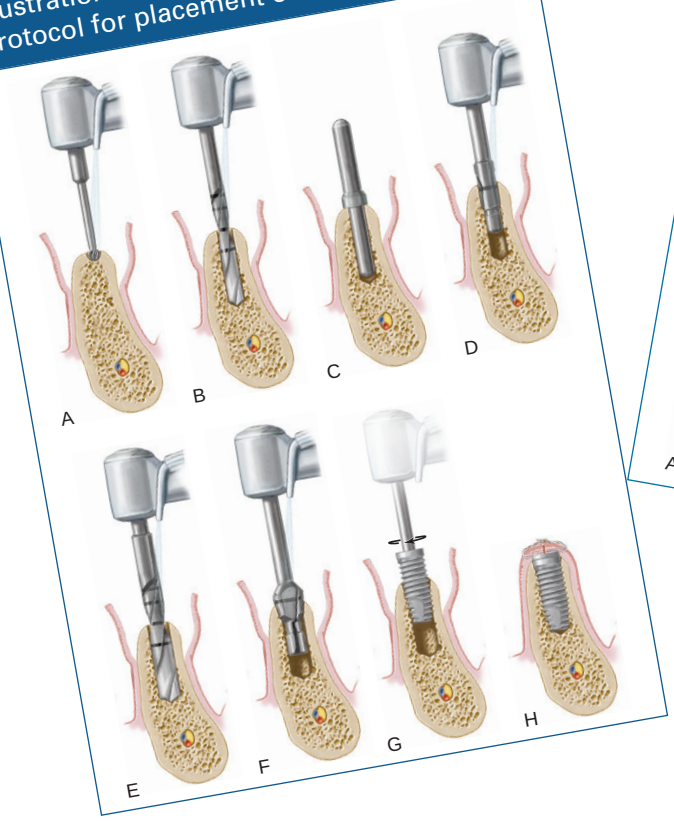
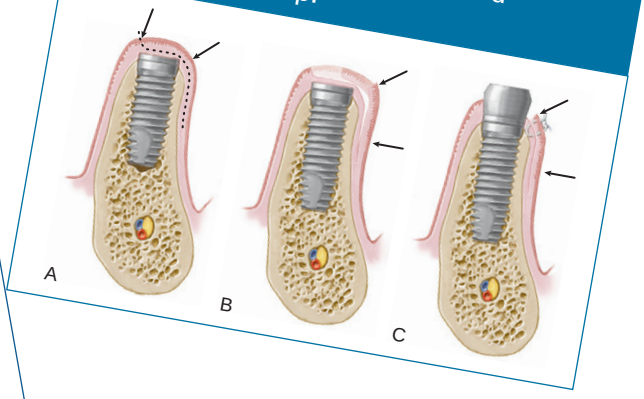
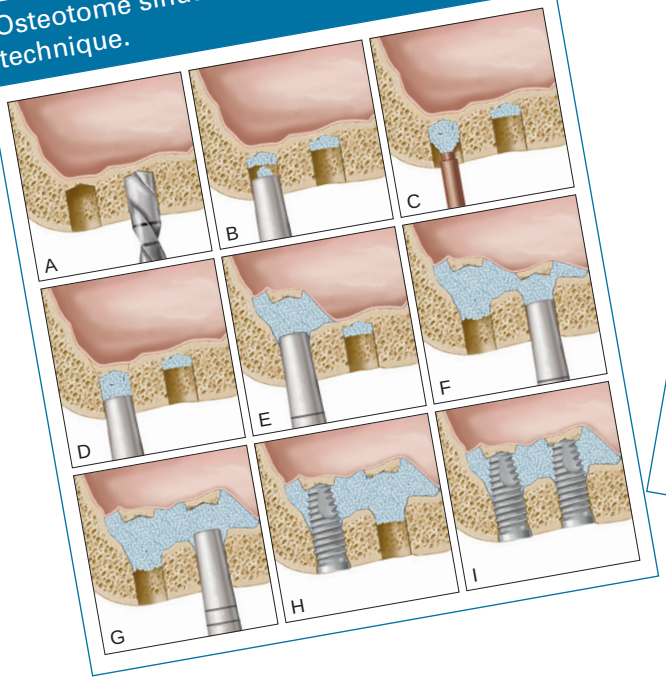
Illustrations depicting the standard drilling protocol for placement of a dental implant.



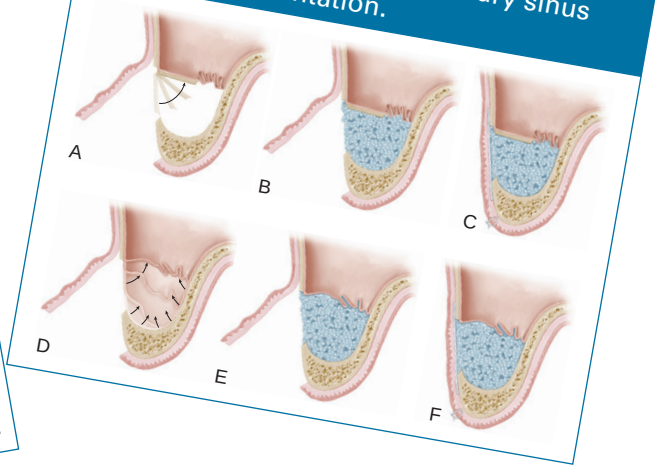
Illustrations depicting the use of a split-thickness flap.



Osteotome sinus floor elevation (OSFE) technique.



Two techniques for the lateral window procedure to access the maxillary sinus for bone augmentation.



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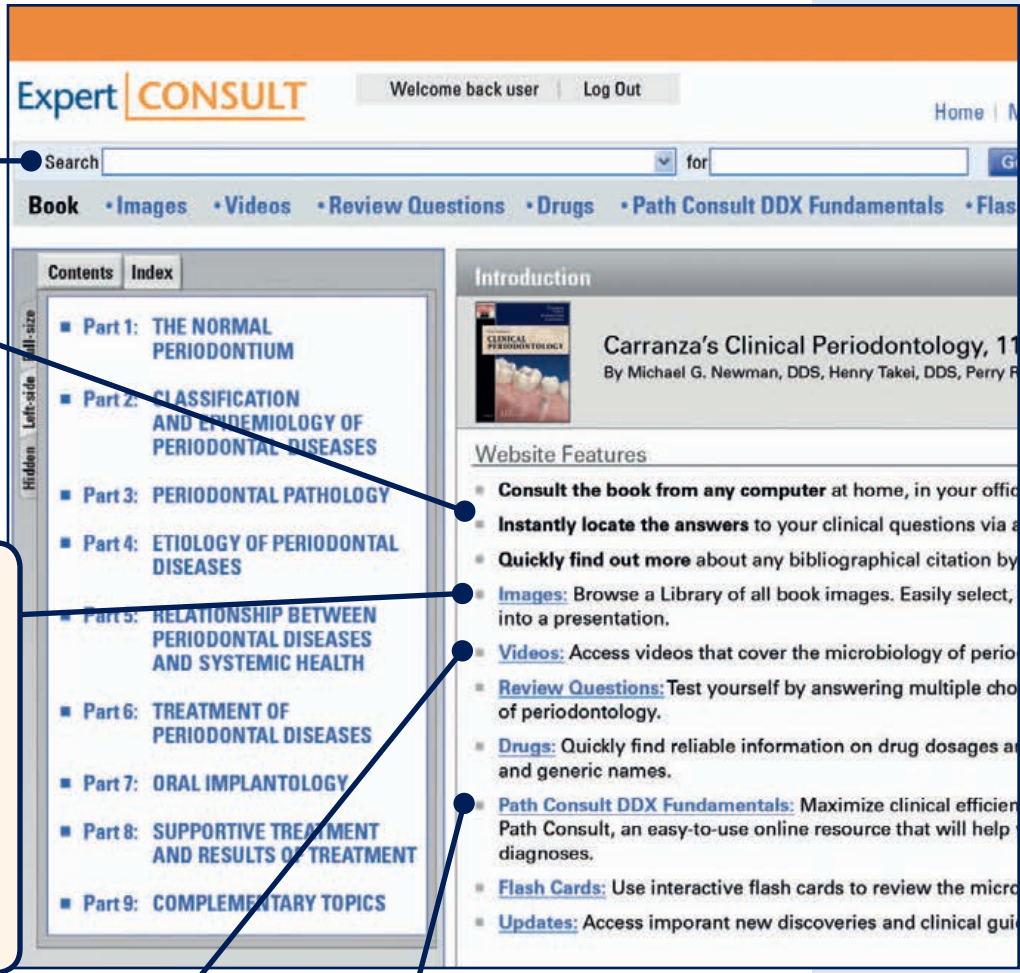
Expert CONSULT

The study and practice of periodontology are ever changing, with new concepts, technology, and information emerging every day. The eleventh edition of *Carranza's Clinical Periodontology* is designed to evolve and grow continuously with each of these new developments through the Expert Consult platform website. *Carranza's Clinical Periodontology* ExpertConsult will be a "living" online resource that not only contains the complete textbook but also much more. This information hub is readily available to all purchasers of the *Carranza's Clinical Periodontology* eleventh edition.

Below is an introduction to each of the supplemental features found on the *Carranza's Clinical Periodontology* Expert Consult website at www.expertconsult.com.

COMPLETE ONLINE, SEARCHABLE TEXT

- The *Carranza's Clinical Periodontology* online component allows the entire textbook to be searchable.
- The accompanying search engine enables users to instantly locate all content related to a given topic.



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- Every image from the eleventh edition of *Carranza's Clinical Periodontology* is viewable in the electronic image collection. Students can review key points from the text, and instructors are able to customize lectures and lesson plans with supporting material taken directly from the eleventh edition.

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INTERACTIVE REFERENCES

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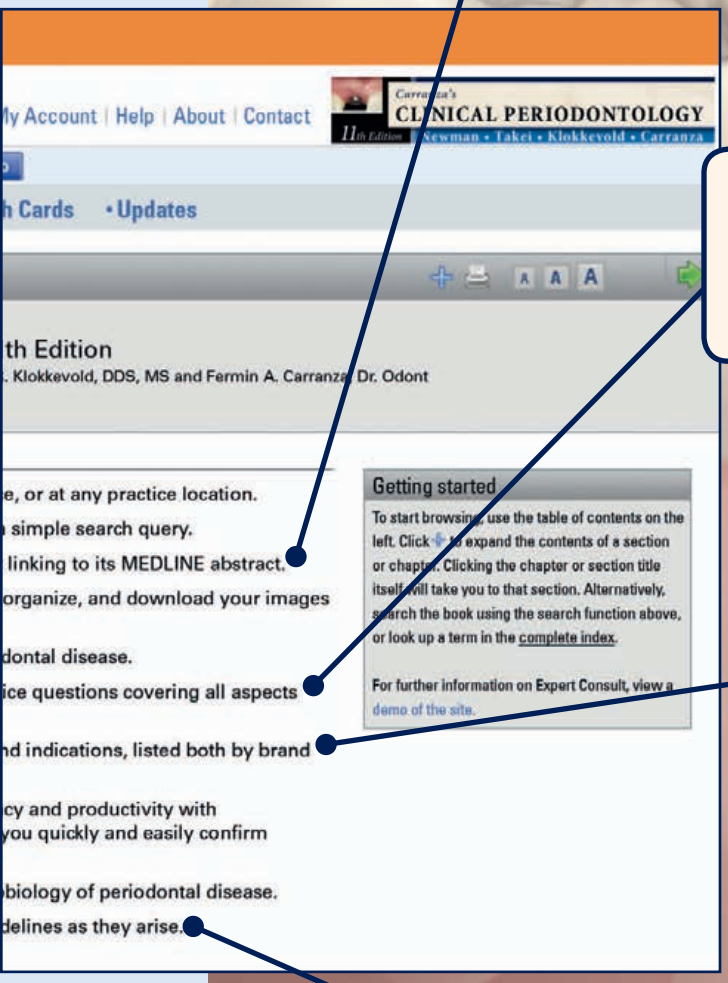

TESTING CENTER

The Clinical Periodontology online Testing Center challenges users' knowledge of the text with online examinations accompanied by instant scoring and answer links to relevant text material.

INSTANT ACCESS TO DRUG DATABASE

- Drug names throughout the online text are linked directly to corresponding monographs from Mosby's Drug Consult, consistently updated with the most current data available for hundreds of dental drugs.

CONTINUOUS CONTENT UPDATES

- Changes in periodontology are addressed through regular amendments to the *Carranza's Clinical Periodontology* text. These updates provide new information and multimedia on developments throughout the entire spectrum of periodontics and reflect the same quality coverage found in the textbook.

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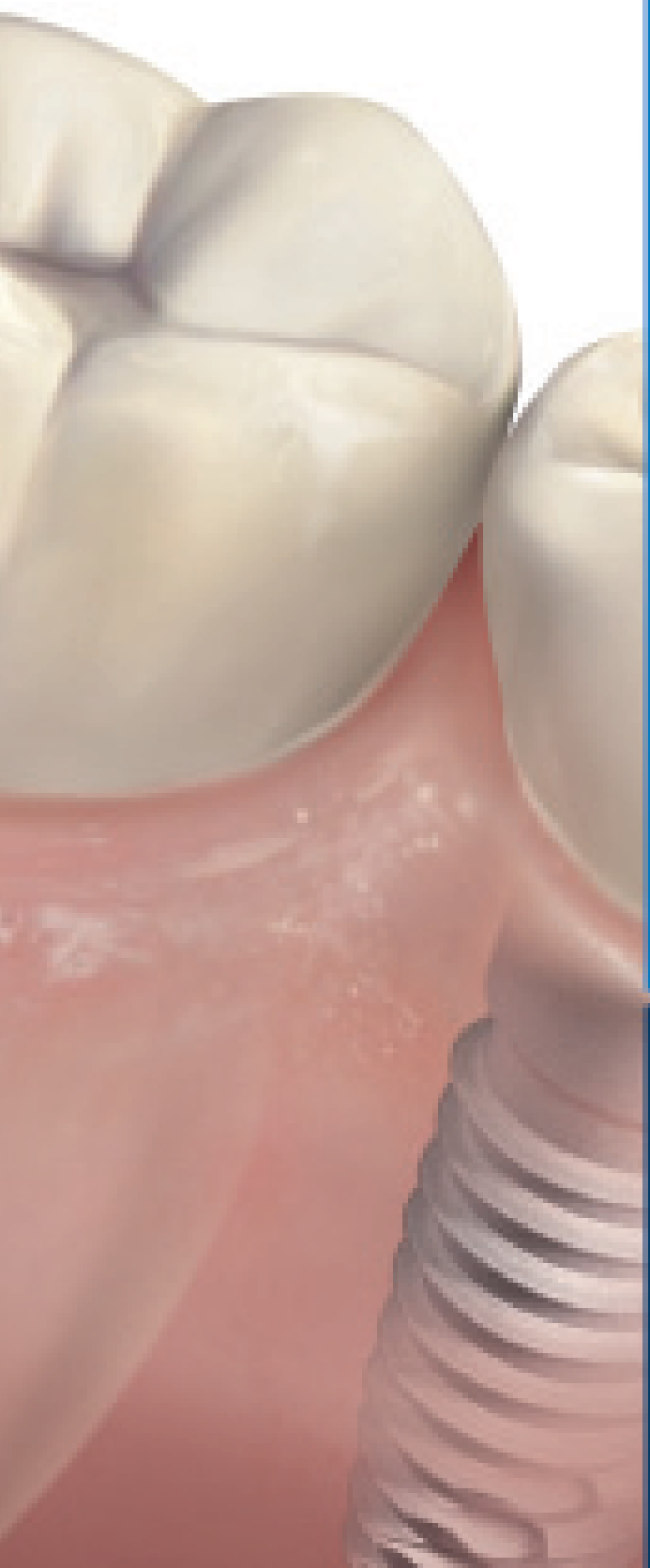
Introduction

Knowing the historical past of our specialty and the role played by our forefathers in reaching our present status provides a realization that our current understanding is but a fleeting phase in the development of science and technology and gives us an appreciation of the origin and evolution of our knowledge that helps to more intelligently understand the present and plan for the future.



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CHAPTER 1

The Historical Background of Periodontology

Gerald Shklar and Fermin A. Carranza

CHAPTER OUTLINE

EARLY CIVILIZATIONS
THE CLASSICAL WORLD
THE MIDDLE AGES
THE RENAISSANCE
EIGHTEENTH CENTURY
NINETEENTH CENTURY
Necrotizing Ulcerative Gingivitis
TWENTIETH CENTURY
Vienna
Berlin
United States and Other Countries
Focal Infection
Dental Implants
After World War II
HISTORY OF THIS BOOK

Gingival and periodontal diseases have afflicted humans since the dawn of history. Studies in paleopathology have indicated that destructive periodontal disease, as evidenced by bone loss, affected early humans in such diverse cultures as ancient Egypt and early pre-Columbian America. The earliest historical records dealing with medical topics reveal an awareness of periodontal disease and the need for treatment. Almost all early writings that have been preserved have sections or chapters dealing with oral diseases, and periodontal problems comprise a significant amount of space in these writings. Calculus and systemic disease were frequently postulated as causes of periodontal disorders.

However, methodic, carefully reasoned therapeutic discussions did not exist until the Arabic surgical treatises of the Middle Ages, and modern treatment, with illustrated text and sophisticated instrumentation, did not develop until the time of Pierre Fauchard in the eighteenth century.

EARLY CIVILIZATIONS

Oral hygiene was practiced by the Sumerians, the Babylonians, and the Assyrians, including gingival massage combined with various herbal medications.[25,33]

Periodontal disease was the most common of all diseases found in the embalmed bodies of the ancient Egyptians.[7,44] The Ebers papyrus contains many references to gingival disease and offers a number of prescriptions for strengthening the teeth and gums.[14]

The medical works of ancient India and China devote significant space to oral and periodontal problems and oral hygiene[47] and describe gingival inflammations, periodontal abscesses, and gingival ulcerations.[12,21] The early Hebrews also recognized the importance of oral hygiene. Many pathologic conditions of the teeth and surrounding structures are described in the Talmudic writings.

THE CLASSICAL WORLD

Among the ancient Greeks, Hippocrates of Cos (460–377 BC), the father of modern medicine, discussed the function and eruption of the teeth and the etiology of periodontal disease. He believed that inflammation of the gums could be caused by accumulations of "pituita" or calculus, with gingival hemorrhage occurring in cases of persistent splenic maladies.[10,27]

Among the Romans, Aulus Cornelius Celsus (25 BC–50 AD) referred to diseases that affect the soft parts of the mouth and their treatment, including oral hygiene. Paul of Aegina (625–690 AD) wrote that tartar deposits must be removed with either scrapers or a small file and that the teeth should be carefully cleaned after the last meal of the day.[41]

THE MIDDLE AGES

The decline and fall of the Roman Empire that plunged Europe into an age of darkness was accompanied by the rise of Islam and

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the golden age of Arabic science and medicine. The Arabic treatises derived their information from Greek medical treatises but added many refinements and novel approaches, particularly in surgical specialties.[45]

Albucasis (936–1013) was born and lived in Moorish Spain, and his medical encyclopedia in 30 volumes, called *al-Tasrif,* was translated into Latin in the twelfth century and was the medical text used in European universities until the seventeenth century. The contributions of Albucasis to dentistry and periodontology were outstanding achievements.[1] He had a clear understanding of the major etiologic role of calculus deposits and described the techniques of scaling the teeth, using a set of instruments that he developed (Figure 1-1), splinting loose teeth with gold wire, and filing gross occlusal abnormalities.

Avicenna (980–1037) was possibly the greatest of the Arabic physicians. His *Canon,* a comprehensive treatise on medicine, was in continuous use for almost 600 years. Avicenna used an extensive "materia medica" for oral and periodontal diseases and rarely resorted to surgery.[3]

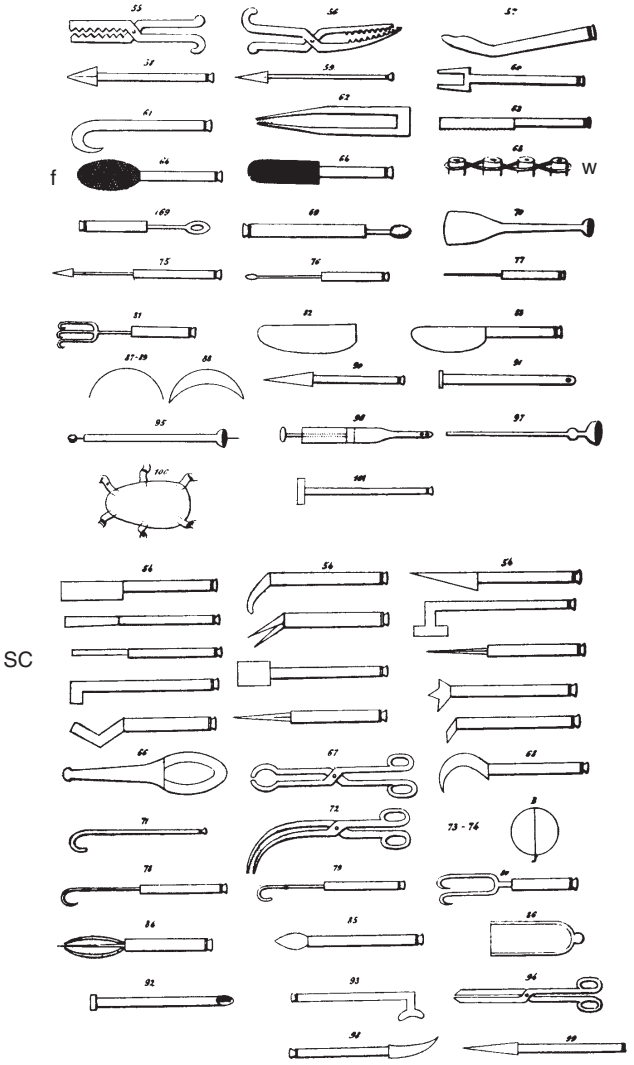

Figure 1-1 Illustration of Albucasis' periodontal instruments, showing scalers *(sc),* files *(f),* and the wiring of loose teeth *(w).*

THE RENAISSANCE

During the Renaissance, with the rebirth of classical scholarship and the development of scientific thought and medical knowledge, as well as the flowering of art, music, and literature, significant contributions were made to anatomy and surgery.

Albucasis' work was expanded in the fifteenth century by the Turkish author Serefeddin Sabuncuoglu (1385–1468), who included illustrations of surgical removal of hypertrophic and swollen gingiva and lingual frenum (Figure 1-2). Drug treatment should be initiated if there are swollen gums, mobile teeth, and pus formation. If there is no response, surgical treatment should be done. A tube is placed on the gums. A hot cautery is inserted into the cannula, and the gingival tissue is cauterized. If correctly applied, the adjacent teeth will be warm.

Paracelsus (1493–1541) developed an interesting and unusual theory of disease: the doctrine of calculus. Paracelsus recognized the extensive formation of tartar on the teeth and related this to toothache. He considered toothache comparable to the pain produced by calculus in other organs such as the kidneys.[39]

Andreas Vesalius (1514–1564), born in Brussels, taught at the University of Padua, and wrote a magnificent book on anatomy with many excellent illustrations.[48] Bartholomeus Eustachius (1520–1574) of Rome was another outstanding anatomist and wrote a small book on dentistry, *Libellus de Dentibus* ("A Little Treatise on the Teeth"), in 30 chapters.[16] This was the first original book on the teeth and included a description of the periodontal tissues, diseases of the mouth, and their treatment modalities and rationale for treatment. For the treatment of periodontitis, Eustachius advised both scaling of calculus and curettage of granulation tissue so that actual reattachment of the gingival and periodontal tissues could take place.

The Frenchman Ambroise Paré (1509–1590), was the outstanding surgeon of the Renaissance, and his contributions to dental

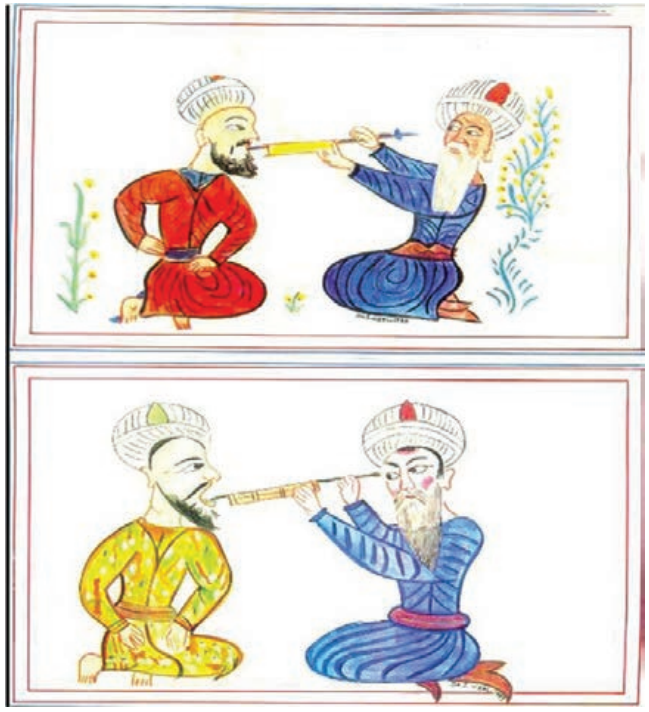

Figure 1-2 Illustration by Serefeddin Sabuncuoglu showing gingival cauterization, from Abulcasis and redrawn by Professor Ilter Uzel, Turkey.

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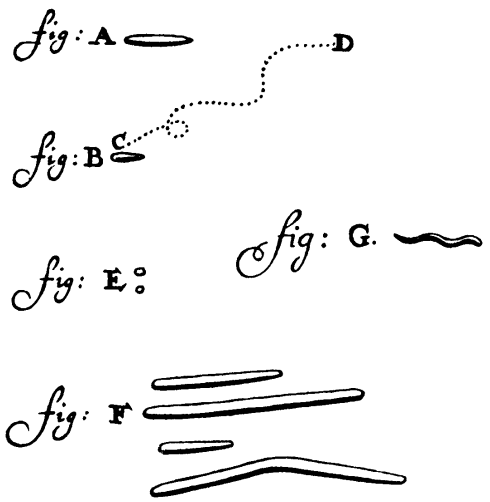

Figure 1-3 Leeuwenhoek's drawing of oral spirochetes, bacilli, and other microorganisms.

Figure 1-4 Frontispiece of Fauchard's book *The Surgeon Dentist* (1746 edition).

surgery included gingivectomy for hyperplastic gingival tissues.[40] He also understood the etiologic significance of calculus and used a set of scalers to remove the hard deposits on the teeth.

The first book in a common language (German) and specifically devoted to dental practice, entitled *Artzney Buchlein* or *Zene Artzney* ("Medicine of the Teeth"), was published in Leipzig in 1530.[2] It contains three chapters devoted to periodontal problems, including a crude concept of systemic and local factors in the etiology of periodontal disease. The presence of local infective agents, or "worms," also is mentioned.

A variety of ointments, often astringent in nature, is suggested, and the binding of loose teeth to sound ones with silk or gold thread is recommended. Cauterizing the gingiva with a hot iron is mentioned.

The Italian physician, mathematician, and philosopher Girolamo Cardano (1501–1576) appears to have been the first to differentiate types of periodontal disease. In a publication dated 1562, he mentions one type of disease that occurs with advancing age and leads to progressive loosening and loss of teeth, as well as a second, very aggressive type that occurs in younger patients.[26] It was not until late in the twentieth century that this classification was rediscovered and became widely accepted.

Anton van Leeuwenhoek (1632–1723) of Delft, Holland, was a layman, but he had an inquisitive mind and a hobby of grinding lenses that allowed him to develop the microscope. He used it to discover microorganisms, cellular structure, blood cells, sperm, and various other microscopic structures, including the tubular structure of dentin.[9,13] Using material from his own mouth, Leeuwenhoek first described oral bacterial flora, and his drawings offered a reasonably good presentation of oral spirochetes and bacilli (Figure 1-3). He even performed antiplaque experiments using strong vinegar in his own mouth and in vitro on bacteria in a dish.[13]

EIGHTEENTH CENTURY

Modern dentistry essentially developed in eighteenth century Europe, particularly France and England. Pierre Fauchard, born in Brittany in 1678, is rightly regarded as the father of the dental profession as we know it. His book, *The Surgeon Dentist,* published in 1728, covered all aspects of dental practice, including restorative dentistry, prosthodontics, oral surgery, periodontics, and orthodontics[17] (Figure 1-4). Fauchard described in detail his periodontal instruments and the scaling technique to use them (Figure 1-5).

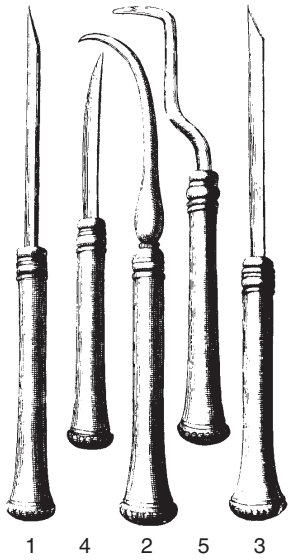

Figure 1-5 The five types of instruments used by Fauchard for detaching tartar from the teeth: *1,* chisel; *2,* parrot beak; *3,* graver; *4,* convex blade; and *5,* Z-shaped hook.

John Hunter (1728–1793), the most distinguished anatomist, surgeon, and pathologist of eighteenth century England, wrote an excellent treatise on dentistry entitled *The Natural History of the Human Teeth.*[30] He offered remarkably clear illustrations of the anatomy of the teeth and their supporting structures and described the features of periodontal diseases.

A contemporary of Hunter, Thomas Berdmore (1740–1785), was considered the outstanding dentist in England and in 1770 published a book, with several chapters devoted to periodontal problems.[4]

NINETEENTH CENTURY

Leonard Koecker (1785–1850) was a German-born dentist who practiced in Baltimore. In a paper in 1821, he mentioned the

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careful removal of tartar and the need for oral hygiene by the patient, recommending that it be performed in the morning and after every meal using an astringent powder and a toothbrush, placing "the bristles … into the spaces of the teeth." Koecker was an early advocate of the "odontogenic focal infection" theory and recommended the extraction of all severely involved teeth and roots, including all unopposed molars, to prevent systemic infections.[35]

Levi Spear Parmly (1790–1859) was a New Orleans dentist who is considered the father of oral hygiene and the inventor of dental floss.[11,18]

In the mid-nineteenth century, John W. Riggs (1811–1885) was the leading authority on periodontal disease and its treatment in the United States, to the point that periodontitis was known as "Riggs' disease" (Figure 1-6). He graduated from the Baltimore College of Dental Surgery in 1854 and practiced in Hartford, Connecticut, where he died on November 11, 1885. Riggs seems to have been the first individual to limit his practice to periodontics and therefore can be considered the first specialist in this field. Riggs' publications, however, are limited. In an 1876 paper, Riggs was a strong proponent of the so-called conservative approach to periodontal therapy, developing the concept of oral prophylaxis and prevention, advocating cleanliness of the mouth, and opposing surgery, which at the time consisted of gingival resection.[43]

Riggs and his disciples had great influence in the dental profession. Among Riggs' followers were L. Taylor, D.D. Smith, R.B. Adair, and W.J. Younger. The instruments designed by Younger,[57] and later modified by his student Robert Good, were used widely until well beyond the middle of the twentieth century.

Several major developments in medical science occurred in the second half of the nineteenth century and started the era that can be called *modern medicine,* which includes dentistry.[9,36] The first was the discovery of anesthesia by Horace Wells (1813–1848) of Hartford, Connecticut, in 1845 and by William Morton (1819–1868) of Boston in 1846, who discovered the general anesthetic effects of nitrous oxide and ether, respectively. *Local anesthesia* was developed by the Vienna ophthalmologist Carl Köller (1857–1944), who produced anesthesia of the eye with drops of cocaine. Procaine (Novocaine) was developed in 1905 by the Munich chemists Alfred Einhorn and Richard Willstädter. Later, with the addition of adrenaline, discovered separately in the United States by Jokichi Takamine and Thomas Bell Aldrich, local anesthesia was born.[29]

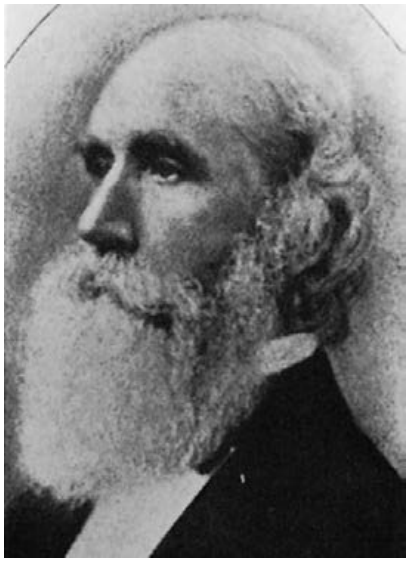

Figure 1-6 John W. Riggs (1811–1885). *(From Hoffman-Axthelm W:* History of dentistry, *Chicago, 1981, Quintessence.)*

The second scientific breakthrough was made by the French chemist Louis Pasteur (1822–1895), who established the *germ theory of disease.* Subsequently, the German physician Robert Koch (1843–1910), in a series of brilliant investigations, discovered the microorganism that causes the cattle disease anthrax and the bacterial etiology of tuberculosis and cholera.

The concepts of Pasteur were transferred to clinical and surgical practice by Joseph Lister (1827–1912) of England, and thus the era of antisepsis (and later, asepsis) in surgery was born. Anesthesia and antisepsis made possible the extraordinary advances in surgical techniques.

Pasteur, Koch, and their collaborators and followers (Elie Metchnikoff, Emile Roux, Paul Ehrlich, Emil von Behring, Shibasaburo Kitasato, and many others) discovered the bacterial etiology of numerous diseases (e.g., pneumonia, puerperal fever, diphtheria, meningitis, plague, dysentery, syphilis) and gave birth to two sciences that became basic to periodontics: bacteriology and immunology.

A third scientific finding that changed the practice of dentistry in general and periodontics in particular was the *discovery of radiographs* by the German physicist Wilhelm Röntgen (1845–1923; also Roentgen). Röntgen's discovery was made in 1895 at the University of Würzburg and was purely a basic science finding, but it was immediately taken up by physicians and dentists and proved to be a crucial development in periodontics and many other areas of medicine and dentistry.

Also in the late nineteenth century, studies by Rudolph Virchow (1821–1902), Julius Cohnhein (1839–1884), Elie Metchnikoff (1845–1916), and others had started to reveal the microscopic changes occurring in inflammation.[8,9] This resulted in an understanding of the pathogenesis of periodontal disease based on histopathologic studies. The Russian N.N. Znamensky described the complex interaction of local and systemic factors in the etiology of periodontal disease. His observations and concepts were summarized in 1902 in a classic paper in which he described the presence in inflamed gingivae of a cellular infiltrate that extends deeper as the disease progresses, causing bone resorption associated with multinucleated cells (osteoclasts) and Howship's lacunae[58] (Figure 1-7).

The first individual to identify bacteria as the cause of periodontal disease appears to have been the German dentist Adolph Witzel (1847–1906).[23,56] The first true oral microbiologist, however, was the American Willoughby D. Miller (1853–1907), whose professional activities took place in Berlin, where he embarked on a research career that introduced modern bacteriology principles to dentistry. Although his greatest accomplishments were in caries research, in his classic book, *The Microorganisms of the Human Mouth,* published in 1890, he described the features of periodontal disease and considered the role of predisposing factors, irritational factors, and bacteria in its etiology. He believed that the disease was not caused by a specific bacterium but by a complex array of various bacteria normally present in the oral cavity. This constitutes what was later known as the *nonspecific plaque hypothesis,* which went unchallenged for seven decades.[23,37]

Bacterial plaque was described by J. Leon Williams (1852–1932), an American dentist who practiced in London and in 1897 described a gelatinous accumulation of bacteria adherent to the enamel surface in relation to caries.[55] In 1899, G.V. Black (1836–1915) coined the term *gelatinous microbic plaque.*[5]

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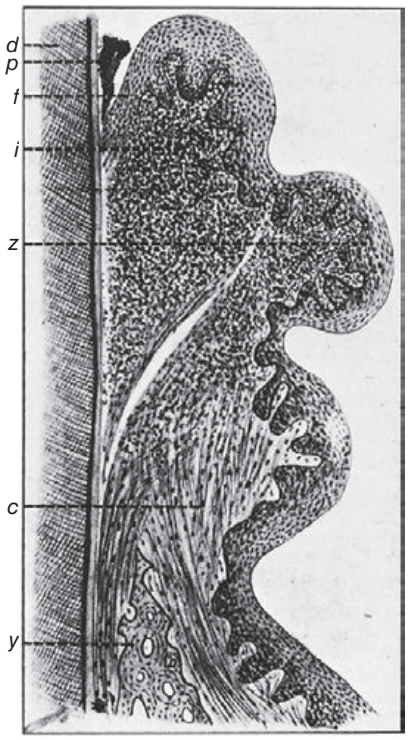

Figure 1-7 Microscopic features of periodontal disease as presented by Znamensky.

Salomon Robicsek (1845–1928) was born in Hungary and practiced dentistry in Vienna. He developed a surgical technique consisting of a scalloped, continuous gingivectomy excision, exposing the marginal bone for subsequent curettage and remodeling.[46]

The first description (1901) of a possible role of trauma from occlusion and bruxism in periodontal disease is generally attributed to the Austrian dentist Moritz Karolyi (1865–1945), who also recommended its correction by grinding occlusal surfaces and the preparation of bite plates.[34]

Necrotizing Ulcerative Gingivitis

Necrotizing ulcerative gingivitis (NUG) had been recognized in the fourth century BC by Xenophon, who mentioned that Greek soldiers were affected with "sore mouth and foul-smelling breath." In 1778, Hunter described the clinical features of this disease and differentiated it from scurvy and chronic periodontitis.

Hyacinthe Jean Vincent (1862–1950),[23,49] a French physician working at the Pasteur Institute in Paris, and Hugo Carl Plaut (1858–1928),[42] in Germany, described the spirillum and fusiform bacilli associated with what later became known as *Vincent's angina,* and in 1904 Vincent described these organisms in ulceronecrotic gingivitis.[50]

TWENTIETH CENTURY

In the first third of the twentieth century, periodontics flourished in central Europe, with two major centers of excellence: Vienna and Berlin.[22]

Vienna

The Vienna school developed the basic histopathologic concepts on which modern periodontics was built. The major representative from this group was Bernhard Gottlieb (1885–1950), who published extensive microscopic studies of periodontal disease in human autopsy specimens (Figure 1-8).[19] His major contributions appeared in the German literature in the 1920s and described the attachment of the gingival epithelium to the tooth, the histopathology of inflammatory and degenerative periodontal disease, the biology of the cementum, active and passive tooth eruption, and traumatic occlusion. A book published in 1938 by Gottlieb and Orban presented a complete review in English of the concepts developed by Gottlieb and his co-workers in Vienna.[24]

Figure 1-8 Bernhard Gottlieb (1885–1950). *(From Gold SI:* J Clin Periodontol *12:171, 1985.)*

Figure 1-9 Balint J. Orban (1899–1960). *(From* J Periodontol *31:266, 1960.)*

A younger contemporary of Gottlieb's in Vienna was Balint J. Orban (1899–1960) (Figure 1-9), who carried out extensive histologic studies on periodontal tissues that serve as the basis for much of current therapy. Other members of the Viennese school were Rudolph Kronfeld (1901–1940), Joseph P. Weinmann (1889–1960), and Harry Sicher (1889–1974). All these scientists emigrated to the United States in the 1930s and contributed greatly to the progress of American dentistry.

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Figure 1-10 Oskar Weski (1879–1952). *(From Hoffman-Axthelm W:* History of dentistry, *Chicago, 1981, Quintessence.)*

Figure 1-11 Robert Neumann (1882–1958). *(Courtesy Dr. Steven I. Gold, New York.)*

Berlin

The Berlin group consisted mostly of clinical scientists who developed and refined the surgical approach to periodontal therapy. Prominent in this group were Oskar Weski (Figure 1-10) and Robert Neumann (Figure 1-11).

Weski (1879–1952) carried out pioneering studies correlating radiographic and histopathologic changes in periodontal disease.[53] He also conceptualized the periodontium as formed by cementum, gingiva, periodontal ligament, and bone and gave it the name *paradentium,* which was later changed (for etymologic reasons) to *parodontium,* a term still used in Europe.

Neumann (1882–1958), in a book published in 1912[38] (with new editions in 1915, 1920, and 1924), described the principles of periodontal flap surgery, including osseous recontouring as it is currently known[20] (Figure 1-12). Other clinicians who described flap surgery at the beginning of the twentieth century were Leonard Widman of Sweden (1871–1956)[54] and A. Cieszynski of Poland. A bitter controversy developed among Widman, Cieszynski, and Neumann in the 1920s over the priority in describing the periodontal flap.

United States and Other Countries

In the United States, before World War II, important contributions to periodontal surgery were made by A. Zentler, J. Zemsky, G.V. Black, O. Kirkland, A.W. Ward, A.B. Crane, H. Kaplan, and others. In 1923, Ward introduced the surgical pack under the trade name Wondr-Pak.[51]

The nonsurgical approach was championed by Isadore Hirschfeld (1882–1965) of New York, who wrote classic papers on oral hygiene,[28] local factors, and other topics. In 1913, Alfred Fones (1869–1938) opened the first school for dental hygienists in Bridgeport, Connecticut.[9]

In other countries, H.K. Box (Canada); M. Roy and R. Vincent (France); R. Jaccard and A.-J. Held (Switzerland); F.A. Carranza, Sr., and R. Erausquin (Argentina); W.W. James, A. Counsell, and E.W. Fish (Great Britain); and A. Leng (Chile) are well known for their important contributions. Probably the most comprehensive

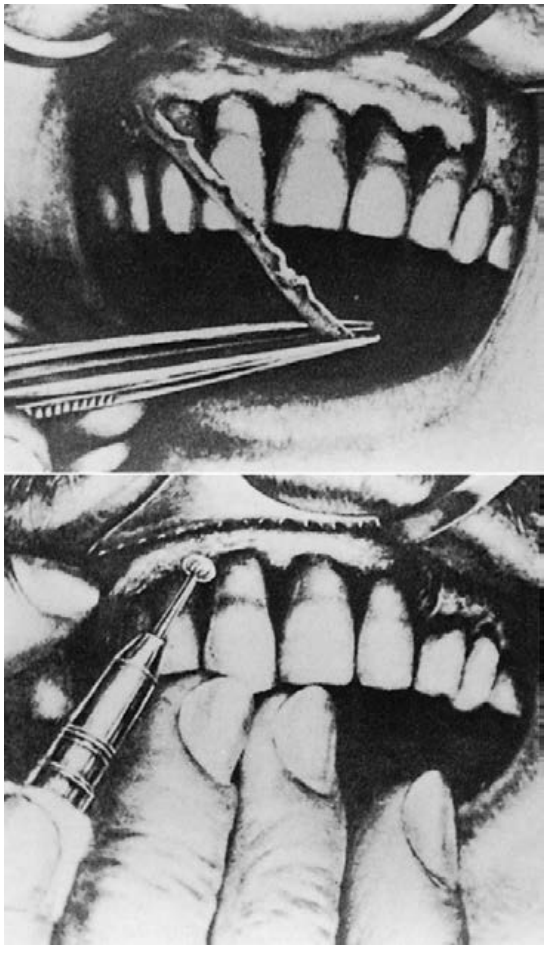

Figure 1-12 Surgical procedure advocated by Robert Neumann in the early part of the twentieth century. *Top,* After raising a mucoperiosteal flap, its edge is trimmed with scissors, leaving a scalloped outline. *Bottom,* Osseous recontouring with burs. *(From Gold SI:* J Periodontol *53:456, 1982.)*

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book on periodontics published in the first half of the twentieth century was "El Paradencio, Su Patologia y Tratamiento," written by the Uruguayan F.M. Pucci, which appeared in 1939.

Focal Infection

The concept of systemic diseases originating in dental and oral infections had been mentioned in the Assyrian clay tablets (seventh century BC), by Hippocrates (460–370 BC), in the Babylonian Talmud (third century AD), and by Girolamo Cardano and the German Walter Hermann Ryff in the sixteenth century.[29,52] In the nineteenth century, Benjamin Rush (famous physician and one of the signers of the American Declaration of Independence) in 1818 and Leonard Koecker in 1828 recognized the role of oral sepsis in rheumatic and other diseases. Later in the nineteenth century, W.D. Miller also mentioned oral infections as the cause of many diseases.[37]

In a paper published in 1900[31] and a decade later in a lecture at McGill University in Montreal,[32] William Hunter (1861–1937), a British physician, indicted dentistry as being the cause of oral sepsis, which in turn caused rheumatic and other chronic diseases. This idea was taken up by Billings, Rosenow, and many others, who advocated extractions of all teeth with periodontal or periapical infections to prevent systemic diseases. This led to wholesale extractions of teeth (and removal of tonsils).

The focal infection theory fell into disrepute when it was found that extractions failed to eliminate or reduce the systemic diseases to which the infected teeth were supposed to be linked.[15] However, the concept has been revisited in the 1990s, this time with a more solid research foundation.

Dental Implants

The replacement of human teeth by implants has been attempted for centuries. Skulls with metal or stone implants have been found in a Gallo-Roman necropolis in France from the second century AD and in a mandible of Mayan origin dated about 600 AD.[9]

In 1806 the Italian M. Maggiolo attempted to place solid-gold roots in human jaws, and later in the nineteenth century, several other investigators used porcelain and metallic implants. In the first half of the twentieth century, several attempts were made using elaborate surgical techniques and complicated constructs of gold and other precious metals, and microscopic investigations were begun on the tissue response to various metals.

In 1939, A.E. Strock of Harvard University started implanting cobalt-chromium (Vitallium) screws into tooth sockets. After World War II, numerous attempts were made with different materials and shapes of implants, including tantalum twisted spiral (Formiggini), Vitallium tree shaped (Lee), acrylic tooth root replica (Hodosh), Vitallium double helical spiral (Chércheve), tantalum tripodal pins (Scialom), tantalum vent-plant and titanium blade (Linkow), and vitreous carbon.[9]

In the 1950s the Swedish orthopedic surgeon Per-Ingvar Brånemark developed a technique using titanium, screw-shaped intraosseous implants. This proved to be quite successful and was gradually adopted by the dental profession after the 1982 international conference in Toronto. The success and predictability of Brånemark's technique are attributed to the achievement of direct contact between vital bone and the implant surface without intervening soft tissue, a phenomenon later termed *osseointegration*.[6] Numerous variations of the Brånemark concept were presented by A. Kirsch, G.A. Niznick, A. Schroeder, and others and are widely used at present.

After World War II

The United States and Scandinavia took a leading role in basic and clinical periodontal research during and after the 1950s, with major advances in the fields of experimental pathology, microbiology, immunology, and therapy.

In the United States, five individuals led in the efforts to advance our understanding of the disease processes and the technical approaches to solve them: Irving Glickman (1914–1972) (Figure 1-13), Henry M. Goldman (1911–1991), Balint J. Orban (1899–1960) (see Figure 1-8), Sigurd P. Ramfjord (1911–1997), and Helmut A. Zander (1912–1991). In the clinical area, the influence of John Prichard (1907–1990) and Saul Schluger (1908–1990) led to new concepts and new directions in the pursuit of clinical success and excellence.

The leading figure of the Scandinavian group was Jens Waerhaug (1907–1980) (Figure 1-14) of Oslo, whose dissertation, *The Gingival Pocket* (1952), and his lifetime of research opened a new

Figure 1-13 Irving Glickman (1914–1972).

Figure 1-14 Jens Waerhaug (1907–1980). *(From* J Clin Periodontol *7:534, 1980.)*

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era in the understanding of the biology of the periodontium and management of periodontal problems.

The next generations centered their attention more on the role of microorganisms and the host response, including its defensive and its destructive aspects. Their contributions, as well as those of their predecessors, are documented in this book.

Several workshops and international conferences have summarized the existing knowledge on the biologic and clinical aspects of periodontology. Worthy of mention are those that were conducted in 1951, 1966, 1977, 1989, 1996, 1999, and 2008, cosponsored and published by the American Academy of Periodontology (AAP).

The AAP, founded in 1914 by two female periodontists, Grace Rogers Spalding (1881–1953) and Gillette Hayden (1880–1929), has become the leader in organized periodontics. Its monthly scientific publication, *The Journal of Periodontology,* presents all the advances in this discipline. In Europe the periodontal societies have joined to form the European Federation of Periodontology, which meets regularly at the Europerio meeting. Their official publication is the *Journal of Clinical Periodontology.* Other scientific periodontal journals in English include *Journal of Periodontal Research, Periodontology 2000,* and *International Journal of Periodontics and Restorative Dentistry.* In other languages, *Journal de Parodontologie* (France*), Periodoncia* (Spain), and *Journal of the Japanese Association of Periodontology* deserve mention.

Periodontal education in the United States also has grown in the second half of the twentieth century, and most dental schools have separate and independent units for teaching and research in this discipline. Periodontics was recognized as a specialty of dentistry by the American Dental Association in 1947. The first university-based programs for the training of specialists in periodontics were begun in several universities (Columbia, Michigan, Tufts) in the late 1940s; these 1-year programs expanded to 2-year programs about 10 years later. In 1995 the AAP mandated that all postgraduate periodontal programs increase to a 3-year curriculum because of the increased knowledge in periodontics and the expansion of the scope of periodontics to include placement of dental implants and administration of conscious sedation. Currently in the United States, more than 50 periodontal graduate programs are based in universities and hospitals.

HISTORY OF THIS BOOK

The originator of this book and author of its first four editions, published in 1953, 1958, 1964, and 1972, was Dr. Irving Glickman (see Figure 1-13), professor and chairman of the Department of Periodontology at Tufts University School of Dental Medicine, in Boston, Massachusetts.

Dr. Glickman was an outstanding researcher, superb educator, and a gifted speaker and writer, whose concepts shaped periodontal thinking for many years. His style of writing, his ideas, and his philosophy of dental practice can still be found in many areas of this book.

After Dr. Glickman's death in 1972 at age 58, responsibility for continuing this book moved to Dr. Fermin A. Carranza, who had been a student and collaborator of Dr. Glickman. At the time, Dr. Carranza was professor and chairman of periodontics at the School of Dentistry of the University of California, Los Angeles (UCLA). The following four editions were published in 1979, 1984, 1990, and 1996 under the guidance of Dr. Carranza, who is now professor emeritus at UCLA.

In 2002 the task of maintaining the book's tradition of almost half a century changed hands once again. Drs. Michael G. Newman and Henry H. Takei joined Dr. Carranza to take major responsibility for the ninth edition. For the tenth (2006) and eleventh (2011) editions, they were joined by Dr. Perry Klokkevold.

References can be found on the companion
Expert CONSULT website at www.expertconsult.com

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PART 1

The Normal Periodontium

Michael G. Newman

The periodontium consists of the investing and supporting tissues of the tooth: gingiva, periodontal ligament, cementum, and alveolar bone. It has been divided into two parts: the *gingiva,* the main function of which is protecting the underlying tissues, and the *attachment apparatus,* composed of the periodontal ligament, cementum, and alveolar bone. The cementum is considered a part of the periodontium because, with the bone, it serves as the support for the fibers of the periodontal ligament.

The periodontium is subject to morphologic and functional variations, as well as changes associated with age. This section deals with the normal features of the tissues of the periodontium, since this knowledge is necessary for an understanding of periodontal diseases.

The soft and hard tissues surrounding dental implants have many similar features and some important differences with the periodontal tissues, as discussed in Chapter 68.

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CHAPTER 2

Anatomy of the Periodontium

Joseph P. Fiorellini, David M. Kim, and N. Guzin Uzel

CHAPTER OUTLINE

CLINICAL FEATURES
- Marginal Gingiva
- Gingival Sulcus
- Attached Gingiva
- Interdental Gingiva

MICROSCOPIC FEATURES
- Gingival Epithelium
- Gingival Connective Tissue

CORRELATION OF CLINICAL AND MICROSCOPIC FEATURES
- Color
- Size
- Contour
- Shape
- Consistency
- Surface Texture
- Position

Refer to Expert CONSULT companion website at www.expertconsult.com for additional content.

The *oral mucosa* consists of the following three zones:

1. The gingiva and the covering of the hard palate, termed the *masticatory mucosa.*
2. The dorsum of the tongue, covered by *specialized mucosa.*
3. The oral mucous membrane lining the remainder of the oral cavity. The *gingiva* is the part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the necks of the teeth.

CLINICAL FEATURES

In an adult, normal gingiva covers the alveolar bone and tooth root to a level just coronal to the cementoenamel junction. The gingiva is divided anatomically into *marginal, attached,* and *interdental areas.* Although each type of gingiva exhibits considerable variation in differentiation, histology, and thickness according to its functional demands, all types are specifically structured to function appropriately against mechanical and microbial damage.[4] That is, the specific structure of different gingiva reflects its effectiveness as a barrier to the penetration by microbes and noxious agents into the deeper tissue.

Marginal Gingiva

The marginal, or *unattached,* gingiva is the terminal edge or border of the gingiva surrounding the teeth in collarlike fashion (Figures 2-1 and 2-2). In about 50% of cases, it is demarcated from the adjacent attached gingiva by a shallow linear depression, the *free gingival groove.*[3] Usually about 1 mm wide, the marginal gingiva forms the soft tissue wall of the gingival sulcus. It may be separated from the tooth surface with a periodontal probe.

For more information on general aspects of gingival epithelium biology, please visit the companion Expert CONSULT website at www.expertconsult.com.

Gingival Sulcus

The gingival sulcus is the shallow crevice or space around the tooth bounded by the surface of the tooth on one side and the epithelium lining the free margin of the gingiva on the other side. It is V-shaped and barely permits the entrance of a periodontal probe. The clinical determination of the depth of the gingival sulcus is an important diagnostic parameter. Under absolutely normal or ideal conditions, the depth of the gingival sulcus is 0 mm or close to 0 mm.[50] These strict conditions of normalcy can be produced experimentally only in germ-free animals or after intense, prolonged plaque control.[8,23]

In clinically healthy human gingiva, a sulcus of some depth can be found. The depth of this sulcus, as determined in histologic sections, has been reported as 1.8 mm, with variations from 0 to 6 mm[90]; other studies have reported 1.5 mm[136] and 0.69 mm.[47] The clinical evaluation used to determine the depth of the sulcus involves the introduction of a metallic instrument, the periodontal probe, and the estimation of the distance it penetrates. The histologic depth of a sulcus does not need to be exactly equal to the depth of penetration of the probe. The so-called probing depth

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Figure 2-1 Normal gingiva in a young adult. Note the demarcation (mucogingival line) *(arrows)* between the attached gingiva and the darker alveolar mucosa.

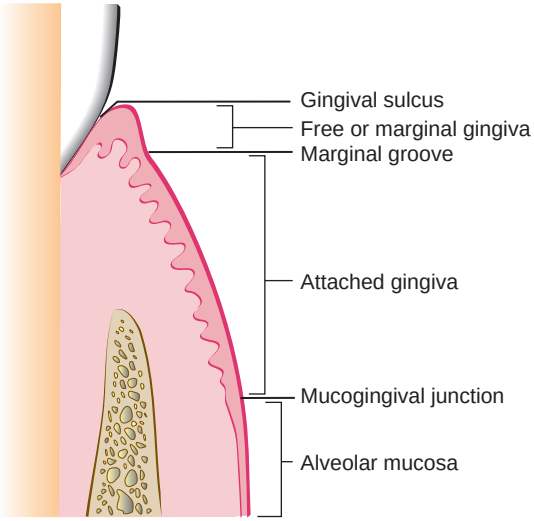

Figure 2-2 Diagram showing anatomic landmarks of the gingiva.

of a clinically normal gingival sulcus in humans is 2 to 3 mm (see Chapter 30).

Attached Gingiva

The attached gingiva is continuous with the marginal gingiva. It is firm, resilient, and tightly bound to the underlying periosteum of alveolar bone. The facial aspect of the attached gingiva extends to the relatively loose and movable alveolar mucosa and is demarcated by the *mucogingival junction* (see Figure 2-2).

The *width of the attached gingiva* is another important clinical parameter. It is the distance between the mucogingival junction and the projection on the external surface of the bottom of the gingival sulcus or the periodontal pocket. It should not be confused with the *width of the keratinized gingiva* because the latter also includes the marginal gingiva (see Figure 2-2).

The width of the attached gingiva on the facial aspect differs in different areas of the mouth.[16] It is generally greatest in the incisor region (3.5 to 4.5 mm in maxilla, 3.3 to 3.9 mm in mandible), and narrower in the posterior segments (1.9 mm in maxillary and 1.8 mm in mandibular first premolars)[3] (Figure 2-3).

Because the mucogingival junction remains stationary throughout adult life,[1] changes in the width of the attached gingiva are caused by modifications in the position of its coronal portion. The width of the attached gingiva increases with age[4] and in supra-

Figure 2-3 Mean width of attached gingiva in human permanent dentition.

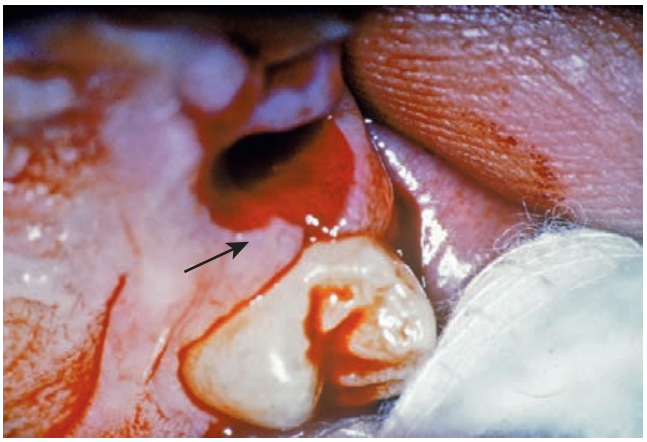

Figure 2-4 Site of extraction showing the facial and palatal interdental papillae and the intervening col *(arrow)*.

erupted teeth.[2] On the lingual aspect of the mandible, the attached gingiva terminates at the junction of the lingual alveolar mucosa, which is continuous with the mucous membrane lining the floor of the mouth. The palatal surface of the attached gingiva in the maxilla blends imperceptibly with the equally firm and resilient palatal mucosa.

Interdental Gingiva

The interdental gingiva occupies the gingival embrasure, which is the interproximal space beneath the area of tooth contact. The interdental gingiva can be pyramidal or can have a "col" shape. In the former the tip of one papilla is located immediately beneath the contact point; the latter presents a valleylike depression that connects a facial and lingual papilla and conforms to the shape of the interproximal contact[31] (Figures 2-4 and 2-5).

The shape of the gingiva in a given interdental space depends on the contact point between the two adjoining teeth and the presence or absence of some degree of recession. Figure 2-6 depicts the variations in normal interdental gingiva.

The facial and lingual surfaces are tapered toward the interproximal contact area, whereas the mesial and distal surfaces are slightly concave. The lateral borders and tips of the interdental papillae are formed by the marginal gingiva of the adjoining teeth. The intervening portion consists of attached gingiva (Figure 2-7).

If a diastema is present, the gingiva is firmly bound over the interdental bone and forms a smooth, rounded surface without interdental papillae (Figure 2-8).

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MICROSCOPIC FEATURES

Microscopic examination reveals that gingiva is composed of the overlying stratified squamous epithelium and the underlying central core of connective tissue. Although the epithelium is predominantly cellular in nature, the connective tissue is less cellular and composed primarily of collagen fibers and ground substance. These two tissues are considered separately.*

Gingival Epithelium

General Aspects of Gingival Epithelium Biology. Historically, the epithelial compartment was thought to provide only a physical barrier to infection and the underlying gingival attachment. However, we now believe that epithelial cells play an active role in innate host defense by responding to bacteria in an interactive manner,[33] which means the epithelium participates actively in responding to infection, in signaling further host reactions, and in integrating innate and acquired immune responses. For example, epithelial cells may respond to bacteria by increased proliferation, alteration of cell-signaling events, changes in differentiation and cell death, and ultimately, alteration of tissue homeostasis.[33] To understand this new perspective of the epithelial innate defense responses and the role of epithelium in gingival health and disease, it is important to understand its basic structure and function (Box 2-1).

The gingival epithelium consists of a continuous lining of stratified squamous epithelium, and the three different areas can be defined from the morphologic and functional points of view: the oral or outer epithelium, sulcular epithelium, and junctional epithelium.

The principal cell type of the gingival epithelium, as well as of other stratified squamous epithelia, is the *keratinocyte.*

*A detailed description of gingival histology can be found in Schroeder HE: *The periodontium,* New York, 1986, Springer-Verlag; and in Biological structure of the normal and diseased periodontium, *Periodontology 2000* 13, 1997.

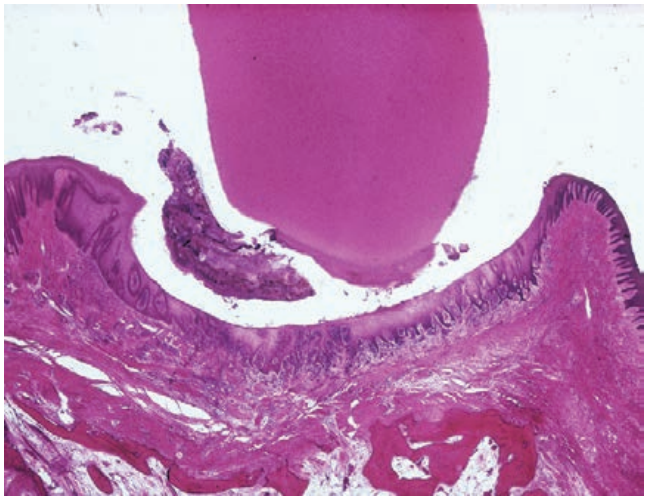

Figure 2-5 Faciolingual section (monkey) showing col between the facial and lingual interdental papillae. The col is covered with nonkeratinized stratified squamous epithelium.

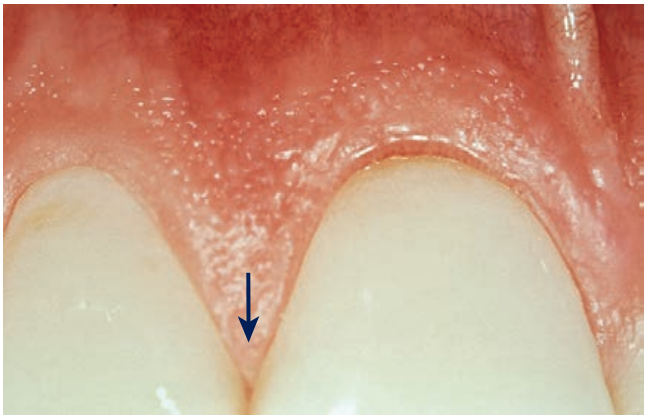

Figure 2-7 Interdental papillae *(arrow)* with central portion formed by attached gingiva. The shape of the papillae varies according to the dimension of the gingival embrasure. *(Courtesy Dr. Osvaldo Costa.)*

Figure 2-8 Absence of interdental papillae and col where proximal tooth contact is missing. *(Courtesy Dr. Osvaldo Costa.)*

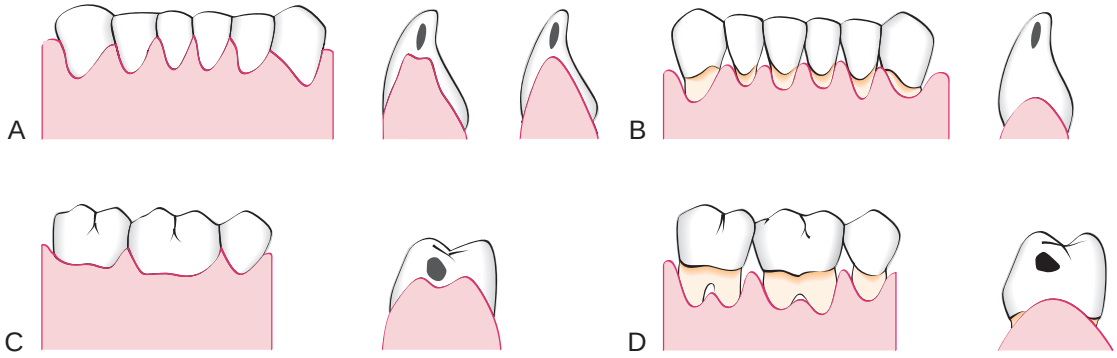

Figure 2-6 Diagram comparing anatomic variations of the interdental col in the normal gingiva *(left side)* and after gingival recession *(right side).* **A** and **B,** Mandibular anterior segment, facial and buccolingual views, respectively. **C** and **D,** Mandibular posterior region, facial and buccolingual views, respectively. Tooth contact points are shown by black marks in lower individual teeth.

Other cells found in the epithelium are the clear cells or nonkeratinocytes, which include the Langerhans cells, Merkel cells, and melanocytes.

The main function of the gingival epithelium is to protect the deep structures, while allowing a selective interchange with the oral environment. This is achieved by proliferation and differentiation of the keratinocytes.

BOX 2-1 Functions and Features of Gingival Epithelium

Functions
Mechanical, chemical, water, and microbial barrier
Signaling functions

Architectural Integrity
Cell-cell attachments
Basal lamina
Keratin cytoskeleton

Major Cell Type
Keratinocyte

Other Cell Types
Langerhans cells
Melanocytes, Merkel cells

Constant Renewal
Replacement of damaged cells

Cell-Cell Attachments
Desmosomes, adherens junctions
Tight junctions, gap junctions

Cell–Basal Lamina
Synthesis of basal lamina components
Hemidesmosome

Modified from Dale BA: *Periodontol 2000* 30:71, 2002.

Proliferation of keratinocytes takes place by mitosis in the basal layer and less frequently in the suprabasal layers, in which a small proportion of cells remain as a proliferative compartment while a larger number begin to migrate to the surface.

Differentiation involves the process of keratinization, which consists of progressions of biochemical and morphologic events that occur in the cell as they migrate from the basal layer (Figure 2-9). The main morphologic changes are (1) progressive flattening of the cell with an increasing prevalence of tonofilaments, (2) intercellular junctions coupled to the production of keratohyaline granules, and (3) disappearance of the nucleus. (See Schroeder[105] for further details.)

A complete keratinization process leads to the production of an *orthokeratinized* superficial horny layer similar to that of the skin, with no nuclei in the stratum corneum and a well-defined stratum granulosum (Figure 2-10). Only some areas of the outer gingival epithelium are orthokeratinized; the other gingival areas are covered by parakeratinized or nonkeratinized epithelium,[20] considered to be at intermediate stages of keratinization. These areas can progress to maturity or dedifferentiate under different physiologic or pathologic conditions.

In *parakeratinized epithelia* the stratum corneum retains pyknotic nuclei, and the keratohyalin granules are dispersed, not giving rise to a stratum granulosum. The *nonkeratinized epithelium* (although cytokeratins are the major component, as in all epithelia) has neither granulosum nor corneum strata, whereas superficial cells have viable nuclei.

Immunohistochemistry, gel electrophoresis, and immunoblot techniques have made identification of the characteristic pattern of cytokeratins possible in each epithelial type. The keratin proteins are composed of different polypeptide subunits characterized by their isoelectric points and molecular weights. They are numbered in a sequence contrary to their molecular weight. Generally, basal cells begin synthesizing lower-molecular-weight keratins, such as K19 (40 kD), and express other higher-molecular-weight keratins as they migrate to the surface. K1 keratin polypeptide (68 kD) is the main component of the stratum corneum.[29]

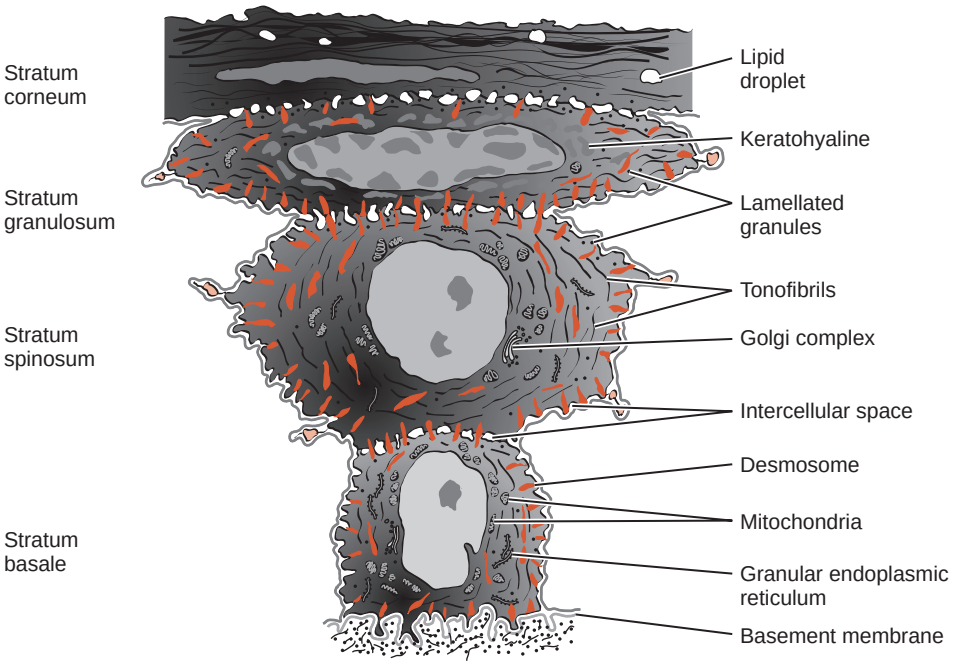

Figure 2-9 Diagram showing representative cells from the various layers of stratified squamous epithelium as seen by electron microscopy. *(Modified from Weinstock A: In Ham AW:* Histology, *ed 7, Philadelphia, 1974, Lippincott.)*

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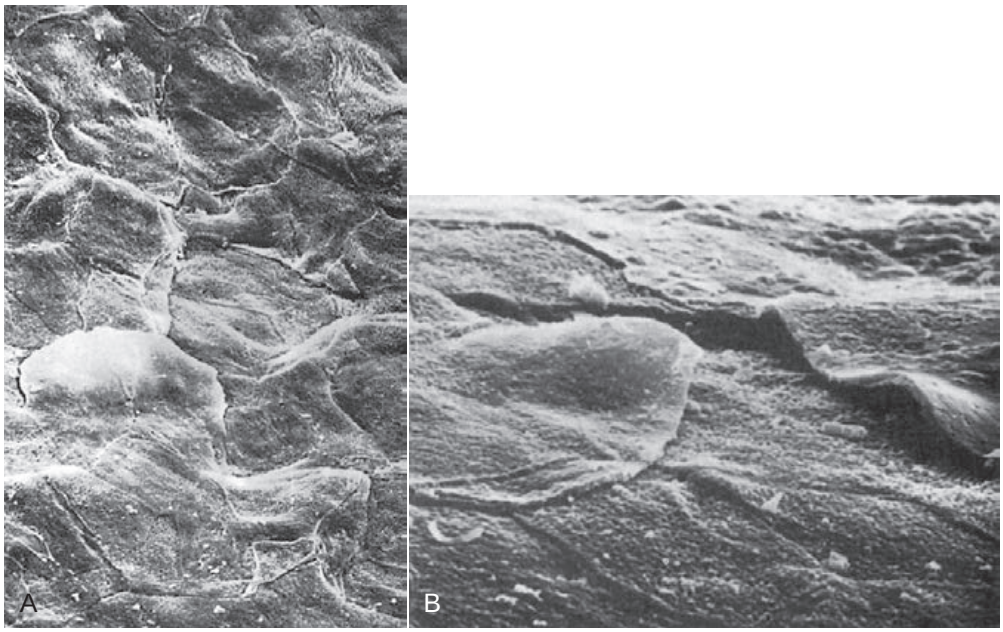

Figure 2-10 **A,** Scanning electron micrograph of keratinized gingiva showing the flattened keratinocytes and their boundaries on the surface of the gingiva. (×1000.) **B,** Scanning electron micrograph of gingival margin at edge of gingival sulcus showing several keratinocytes about to be exfoliated. (×3000.) *(From Kaplan GB, Pameijer CH, Ruben MP:* J Periodontol *48:446, 1977.)*

For more information on general aspects of gingival epithelium biology, please visit the companion Expert CONSULT website at www.expertconsult.com.

Electron microscopy reveals that keratinocytes are interconnected by structures on the cell periphery called *desmosomes.*[70] These desmosomes have a typical structure consisting of two dense attachment plaques into which tonofibrils insert and an intermediate, electron-dense line in the extracellular compartment. Tonofilaments, which are the morphologic expression of the cytoskeleton of keratin proteins, radiate in brushlike fashion from the attachment plaques into the cytoplasm of the cells. The space between the cells shows cytoplasmic projections resembling microvilli that extend into the intercellular space and often interdigitate.

Less frequently observed forms of epithelial cell connections are tight junctions *(zonae occludens),* in which the membranes of the adjoining cells are believed to be fused.[127,134] Evidence suggests that these structures allow ions and small molecules to pass from one cell to another.

Cytoplasmic organelle concentration varies among different epithelial strata. Mitochondria are more numerous in deeper strata and decrease toward the surface of the cell.

For more information on general aspects of gingival epithelium biology, please visit the companion Expert CONSULT website at www.expertconsult.com.

The uppermost cells of the stratum spinosum contain numerous dense granules, *keratinosomes* or *Odland bodies,* which are modified lysosomes. They contain a large amount of acid phosphatase, an enzyme involved in the destruction of organelle membranes, which occurs suddenly between the granulosum and corneum strata and during the intercellular cementation of cornified cells. Thus acid phosphatase is another enzyme closely related to the degree of keratinization.[18,54,132]

Nonkeratinocyte cells are present in gingival epithelium as in other malpighian epithelia. *Melanocytes* are dendritic cells located in the basal and spinous layers of the gingival epithelium. They synthesize melanin in organelles called *premelanosomes* or *melanosomes*[30,102,120] (Figure 2-11). These contain tyrosinase, which hydroxylates tyrosine to dihydroxyphenylalanine (dopa), which in turn is progressively converted to melanin. Melanin granules are phagocytosed and contained within other cells of the epithelium and connective tissue called *melanophages* or *melanophores.*

Langerhans cells are dendritic cells located among keratinocytes at all suprabasal levels (Figure 2-12). They belong to the mononuclear phagocyte system (reticuloendothelial system) as modified monocytes derived from the bone marrow. They contain elongated granules and are considered macrophages with possible antigenic properties.[36] Langerhans cells have an important role in the immune reaction as antigen-presenting cells for lymphocytes. They contain g-specific granules (Birbeck's granules) and have marked adenosine triphosphatase activity. They are found in oral epithelium of normal gingiva and in smaller amounts in the sulcular epithelium; they are probably absent from the junctional epithelium of normal gingiva.

Merkel cells are located in the deeper layers of the epithelium, harbor nerve endings, and are connected to adjacent cells by desmosomes. They have been identified as tactile perceptors.[87]

The epithelium is joined to the underlying connective tissue by a *basal lamina* 300 to 400 Å thick, lying approximately 400 Å beneath the epithelial basal layer.[66,110,121] The basal lamina consists of lamina lucida and lamina densa. Hemidesmosomes of the basal epithelial cells abut the lamina lucida, which is mainly composed of the glycoprotein laminin. The lamina densa is composed of type IV collagen. The basal lamina, clearly distinguishable at the ultrastructural level, is connected to a reticular condensation of the underlying connective tissue fibrils (mainly collagen type IV) by the anchoring fibrils.[86,95,124] Anchoring fibrils have been measured

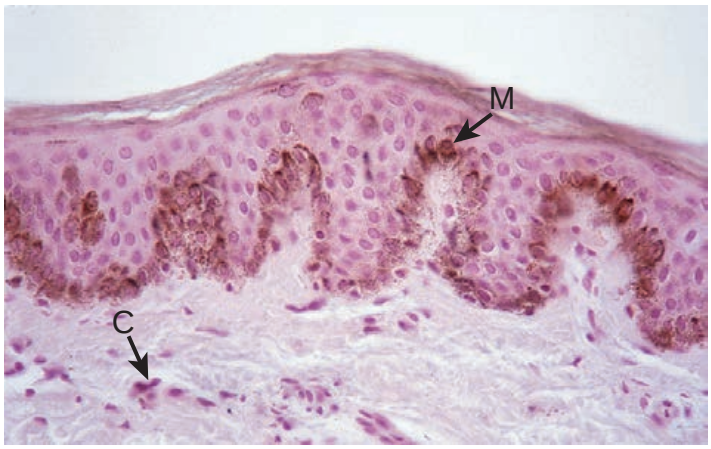

Figure 2-11 Pigmented gingiva of dog showing melanocytes *(M)* in the basal epithelial layer and melanophores *(C)* in the connective tissue (Glucksman technique).

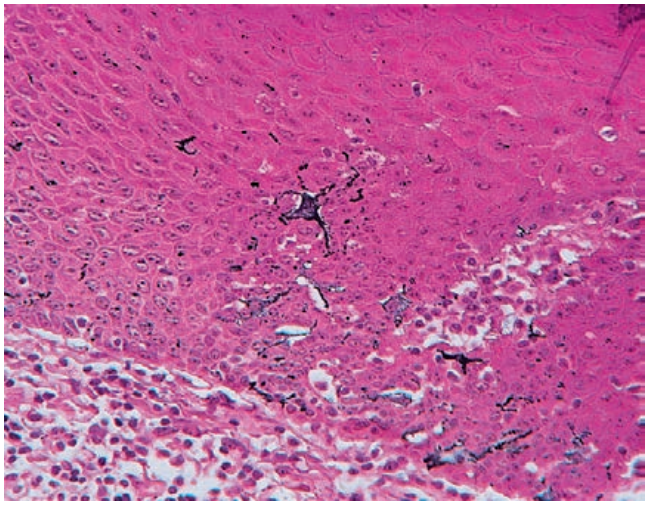

Figure 2-12 Human gingival epithelium, oral aspect. Immunoperoxidase technique showing Langerhans cells.

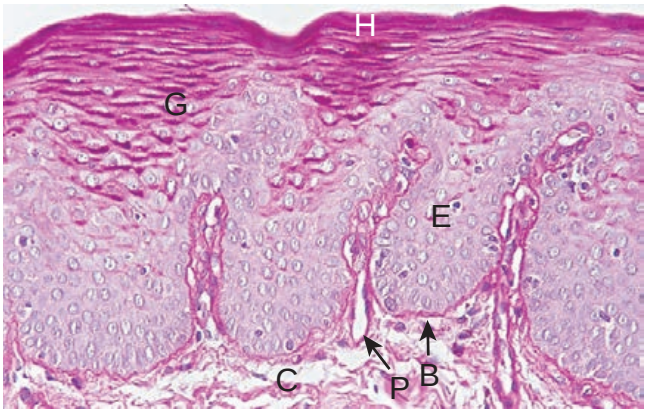

Figure 2-13 Normal human gingiva stained with the periodic acid–Schiff (PAS) histochemical method. The basement membrane *(B)* is seen between the epithelium *(E)* and the underlying connective tissue *(C)*. In the epithelium, glycoprotein material occurs in cells and cell membranes of the superficial hornified *(H)* and underlying granular layers *(G)*. The connective tissue presents a diffuse, amorphous ground substance and collagen fibers. The blood vessel walls stand out clearly in the papillary projections of the connective tissue *(P)*.

at 750 nm in length from their epithelial end to their connective tissue end, in which they appear to form loops around collagen fibers. The complex of basal lamina and fibrils is the periodic acid–Schiff (PAS)–positive and argyrophilic line observed at the optical level[112,125] (Figure 2-13). The basal lamina is permeable to fluids but acts as a barrier to particulate matter.

Structural and Metabolic Characteristics of Different Areas of Gingival Epithelium. The epithelial component of the gingiva shows regional morphologic variations that reflect tissue adaptation to the tooth and alveolar bone.[106] These variations include the oral epithelium, sulcular epithelium, and junctional epithelium. Whereas the oral epithelium and sulcular epithelium are largely protective in function, the junctional epithelium serves many more roles and is of considerable importance in regulating tissue health.[11] It is now recognized that epithelial cells are not "passive bystanders" in the gingival tissues; rather, they are metabolically active and capable of reacting to external stimuli by synthesizing a number of cytokines, adhesion molecules, growth factors, and enzymes.[11]

Oral (Outer) Epithelium. The oral, or outer, epithelium covers the crest and outer surface of the marginal gingiva and the surface of the attached gingiva. On average, the oral epithelium is 0.2 to 0.3 mm in thickness. It is keratinized or parakeratinized or presents various combinations of these conditions (Figure 2-14). The prevalent surface, however, is parakeratinized.[14,20,133]

The oral epithelium is composed of four layers: stratum basale (basal layer), stratum spinosum (prickle cell layer), stratum granulosum (granular layer), and stratum corneum (cornified layer).

The degree of gingival keratinization diminishes with age and the onset of menopause[92] but is not necessarily related to the different phases of the menstrual cycle.[60] Keratinization of the oral mucosa varies in different areas in the following order: palate (most keratinized), gingiva, ventral aspect of the tongue, and cheek (least keratinized).[84]

For more information on oral (outer) epithelium, please visit the companion Expert CONSULT website at www.expertconsult.com.

Sulcular Epithelium. The sulcular epithelium lines the gingival sulcus (Figure 2-15). It is a thin, nonkeratinized stratified squamous epithelium without rete pegs, and it extends from the coronal limit of the junctional epithelium to the crest of the

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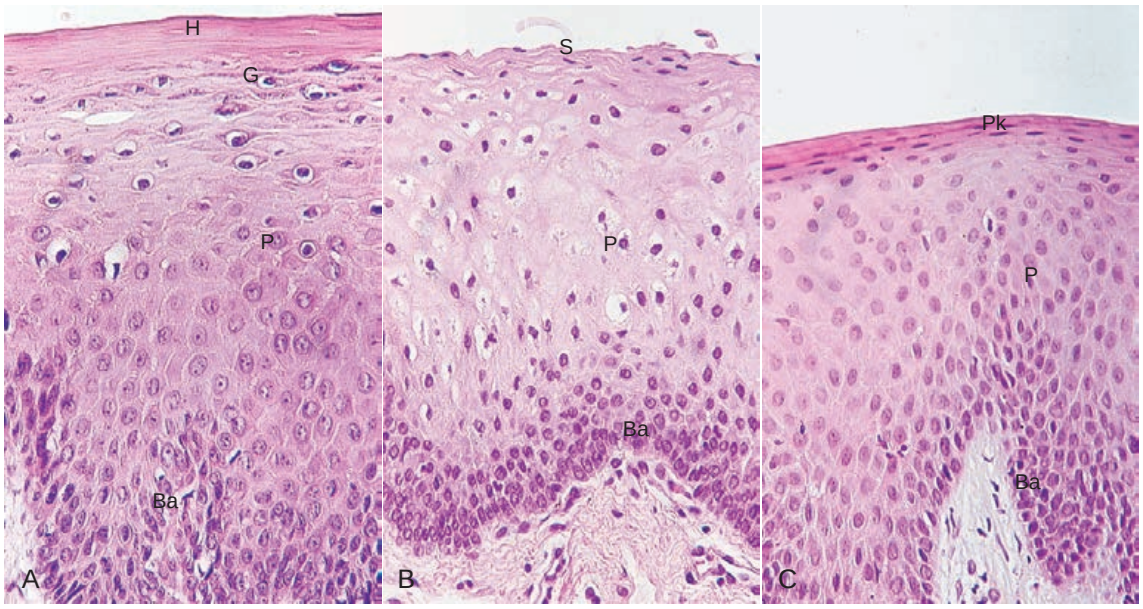

Figure 2-14 Variations in gingival epithelium. **A,** Keratinized. **B,** Nonkeratinized. **C,** Parakeratinized. Horny layer *(H),* granular layer *(G),* prickle cell layer *(P),* basal cell layer *(Ba),* flattened surface cells *(S),* parakeratotic layer *(Pk).*

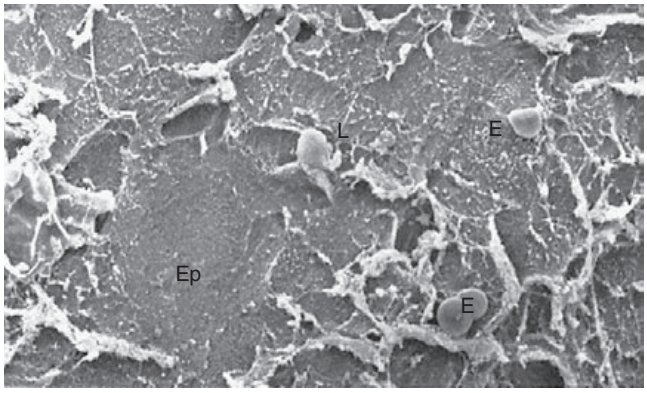

Figure 2-15 Scanning electron microscopic view of epithelial surface facing the tooth in a normal human gingival sulcus. The epithelium *(Ep)* shows desquamating cells, some scattered erythrocytes *(E),* and a few emerging leukocytes *(L).* (×1000.)

gingival margin (Figure 2-16). It usually shows many cells with hydropic degeneration.[14]

As with other nonkeratinized epithelia, the sulcular epithelium lacks granulosum and corneum strata and K1, K2, and K10 to K12 cytokeratins, but it contains K4 and K13, the so-called esophageal-type cytokeratins. It also expresses K19 and normally does not contain Merkel cells.

For more information on sulcular epithelium, please visit the companion Expert CONSULT website at www.expertconsult.com.

The sulcular epithelium is extremely important because it may act as a semipermeable membrane through which injurious bacterial products pass into the gingival and tissue fluid from the gingiva seeps into the sulcus.[126] Unlike the junctional epithelium, however, the sulcular epithelium is not heavily infiltrated by

Figure 2-16 Epon-embedded human biopsy specimen showing a relatively normal gingival sulcus. The soft tissue wall of the gingival sulcus is made up of the oral sulcular epithelium *(ose)* and its underlying connective tissue *(ct),* whereas the base of the gingival sulcus is formed by the sloughing surface of the junctional epithelium *(je).* The enamel space is delineated by a dense cuticular structure *(dc).* A relatively sharp line of demarcation exists between the junctional epithelium and the oral sulcular epithelium *(arrow),* and several polymorphonuclear leukocytes *(pmn)* can be seen traversing the junctional epithelium. The sulcus contains red blood cells resulting from the hemorrhage occurring at the time of biopsy. (×391; inset ×55.) *(From Schluger S, Youdelis R, Page RC:* Periodontal disease, *Philadelphia, 1977, Lea & Febiger.)*

polymorphonuclear neutrophil leukocytes (PMNs), and it appears to be less permeable.[11]

Junctional Epithelium. The junctional epithelium consists of a collarlike band of stratified squamous nonkeratinizing epithelium. It is 3 to 4 layers thick in early life, but the number of layers increases with age to 10 or even 20 layers. Also, the junctional epithelium tapers from its coronal end, which may be 10 to 29 cells wide to 1 or 2 cells at its apical termination, located at the cementoenamel junction in healthy tissue. These cells can be grouped in two strata: the basal layer facing the connective tissue and the suprabasal layer extending to the tooth surface. The length of the junctional epithelium ranges from 0.25 to 1.35 mm.

The junctional epithelium is formed by the confluence of the oral epithelium and the reduced enamel epithelium during tooth eruption (Figure 2-17). However, the reduced enamel epithelium is not essential for its formation; in fact, the junctional epithelium is completely restored after pocket instrumentation or surgery, and it forms around an implant.[69]

Cell layers not juxtaposed to the tooth exhibit numerous free ribosomes and prominent membrane-bound structures, such as Golgi complexes, and cytoplasmic vacuoles, presumably phagocytic. Lysosome-like bodies also are present, but the absence of keratinosomes (Odland bodies) and histochemically demonstrable acid phosphatase, correlated with the low degree of differentiation, may reflect a low defense power against microbial plaque accumulation in the gingival sulcus. Similar morphologic findings have been described in the gingiva of germ-free rats. PMNs are found routinely in the junctional epithelium of both conventional rats and germ-free rats.[137] Research has shown that although numerous migrating PMNs are evident and present around healthy junctional epithelium, a considerable increase in PMN numbers can be expected with the accumulation of dental plaque and gingival inflammation.[11]

For more information on junctional epithelium, please visit the companion Expert CONSULT website at www.expertconsult.com.

The junctional epithelium is attached to the tooth surface (epithelial attachment) by means of an internal basal lamina. It is attached to the gingival connective tissue by an external basal lamina that has the same structure as other epithelial–connective tissue attachments elsewhere in the body.[71,76] The internal basal lamina consists of a lamina densa (adjacent to the enamel) and a lamina lucida to which hemidesmosomes are attached. Hemidesmosomes have a decisive role in the firm attachment of the cells to the internal basal lamina on the tooth surface. Recent data suggest that the hemidesmosomes may also act as specific sites of signal transduction and thus may participate in regulation of gene expression, cell proliferation, and cell differentiation.[61] Organic strands from the enamel appear to extend into the lamina densa.[123] The junctional epithelium attaches to afibrillar cementum present on the crown (usually restricted to an area within 1 mm of the cementoenamel junction)[108] and root cementum in a similar manner.

Histochemical evidence for the presence of neutral polysaccharides in the zone of the epithelial attachment has been reported.[128] Data also have shown that the basal lamina of the junctional epithelium resembles that of endothelial and epithelial cells in its laminin content but differs in its internal basal lamina, which has no type IV collagen.[63,99] These findings indicate that the cells of the junctional epithelium are involved in the production of laminin and play a key role in the adhesion mechanism.

The attachment of the junctional epithelium to the tooth is reinforced by the gingival fibers, which brace the marginal gingiva against the tooth surface. For this reason, the junctional epithelium and the gingival fibers are considered a functional unit, referred to as the *dentogingival unit.*[73]

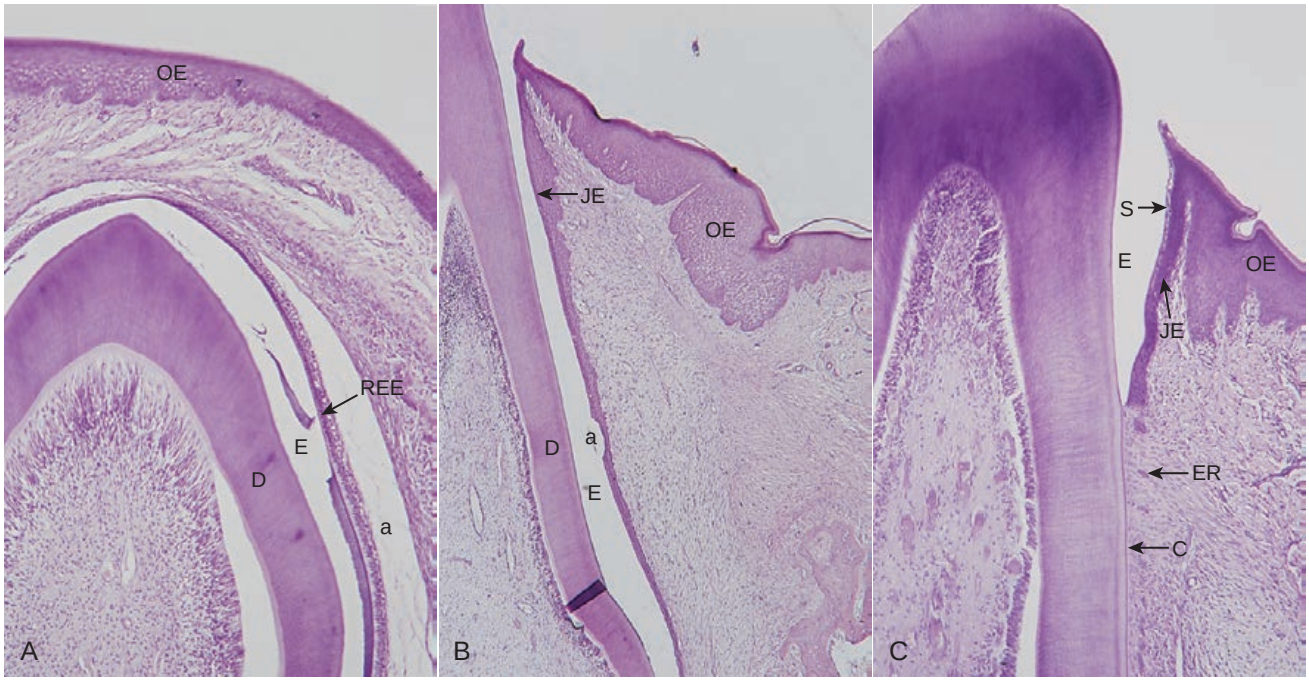

Figure 2-17 Eruption process in cat's tooth. **A,** Unerupted tooth. Dentin *(D),* remnants of enamel matrix *(E),* reduced enamel epithelium *(REE),* oral epithelium *(OE),* artifact *(a).* **B,** Erupting tooth forming junctional epithelium *(JE).* **C,** Completely erupted tooth. Sulcus with epithelial debris *(S),* cementum *(C),* and epithelial rests *(ER).*

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In conclusion, it is usually accepted that the junctional epithelium exhibits several unique structural and functional features that contribute to preventing pathogenic bacterial flora from colonizing the subgingival tooth surface.[94] First, junctional epithelium is firmly attached to the tooth surface, forming an epithelial barrier against plaque bacteria. Second, it allows access of gingival fluid, inflammatory cells, and components of the immunologic host defense to the gingival margin. Third, junctional epithelial cells exhibit rapid turnover, which contributes to the host-parasite equilibrium and rapid repair of damaged tissue. Also, some investigators indicate that the cells of the junctional epithelium have an endocytic capacity equal to that of macrophages and neutrophils and that this activity might be protective in nature.[27]

Development of Gingival Sulcus. After enamel formation is complete, the enamel is covered with *reduced enamel epithelium* (REE), which is attached to the tooth by a basal lamina and hemidesmosomes.[72,122] When the tooth penetrates the oral mucosa, the REE unites with the oral epithelium and transforms into the junctional epithelium. As the tooth erupts, this united epithelium condenses along the crown, and the ameloblasts, which form the inner layer of the REE (see Figure 2-17), gradually become squamous epithelial cells. The transformation of the REE into a junctional epithelium proceeds in an apical direction without interrupting the attachment to the tooth. According to Schroeder and Listgarten,[108] this process takes between 1 and 2 years.

The junctional epithelium is a continually self-renewing structure, with mitotic activity occurring in all cell layers.[72,122] The regenerating epithelial cells move toward the tooth surface and along it in a coronal direction to the gingival sulcus, where they are shed[12] (Figure 2-18). The migrating daughter cells provide a continuous attachment to the tooth surface. The strength of the epithelial attachment to the tooth has not been measured.

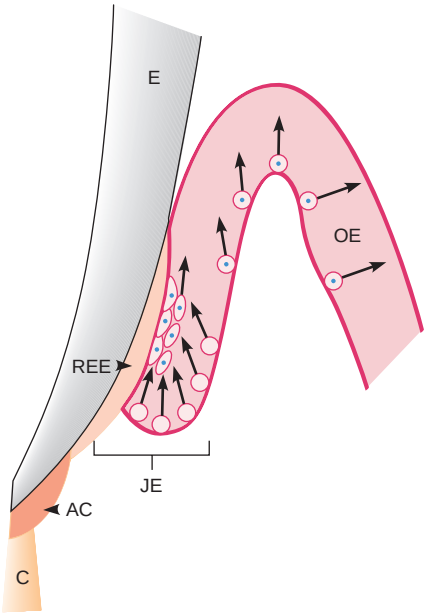

Figure 2-18 Junctional epithelium on an erupting tooth. The junctional epithelium *(JE)* is formed by the joining of the oral epithelium *(OE)* and the reduced enamel epithelium *(REE)*. Afibrillar cementum *(AC)* is sometimes formed on enamel after degeneration of the REE. The arrows indicate the coronal movement of the regenerating epithelial cells, which multiply more rapidly in the JE than in the OE. *E,* Enamel; *C,* root cementum. A similar cell turnover pattern exists in the fully erupted tooth. *(Modified from Listgarten MA:* J Can Dent Assoc *36:70, 1970.)*

The gingival sulcus is formed when the tooth erupts into the oral cavity. At that time, the junctional epithelium and REE form a broad band attached to the tooth surface from near the tip of the crown to the cementoenamel junction.

The gingival sulcus is the shallow V-shaped space or groove between the tooth and gingiva that encircles the newly erupted tip of the crown. In the fully erupted tooth, only the junctional epithelium persists. *The sulcus consists of the shallow space that is coronal to the attachment of the junctional epithelium and bounded by the tooth on one side and the sulcular epithelium on the other. The coronal extent of the gingival sulcus is the gingival margin.*

Renewal of Gingival Epithelium. The oral epithelium undergoes continuous renewal. Its thickness is maintained by a balance between new cell formation in the basal and spinous layers and the shedding of old cells at the surface. The mitotic activity exhibits a 24-hour periodicity, with the highest and lowest rates occurring in the morning and evening, respectively.[123] The mitotic rate is higher in nonkeratinized areas and is increased in gingivitis, without significant gender differences. Opinions differ as to whether the mitotic rate is increased[75,76,83] or decreased[10] with age.

For more information on renewal of gingival epithelium, please visit the companion Expert CONSULT website at www.expertconsult.com.

Regarding junctional epithelium, it was previously thought that only epithelial cells facing the external basal lamina were rapidly dividing. However, evidence indicates that a significant number of the cells, such as the basal cells along the connective tissue, are capable of synthesizing deoxyribonucleic acid (DNA), demonstrating their mitotic activity.[97,98] Rapid shedding of cells effectively removes bacteria adhering to the epithelial cells and therefore is an important part of the antimicrobial defense mechanisms at the dentogingival junction.[92]

Cuticular Structures on the Tooth. The term *cuticle* describes a thin, acellular structure with a homogeneous matrix, sometimes enclosed within clearly demarcated, linear borders.

Listgarten[74] has classified cuticular structures into coatings of developmental origin and acquired coatings. *Acquired coatings* include those of exogenous origin such as saliva, bacteria, calculus, and surface stains (see Chapters 22 and 23). *Coatings of developmental origin* are those normally formed as part of tooth development. They include the REE, coronal cementum, and dental cuticle.

After enamel formation is completed, the ameloblastic epithelium is reduced to one or two layers of cells that remain attached to the enamel surface by hemidesmosomes and a basal lamina. This REE consists of postsecretory ameloblasts and cells from the stratum intermedium of the enamel organ.

In some animal species the REE disappears entirely and very rapidly, thereby placing the enamel surface in contact with the connective tissue. Connective tissue cells then deposit a thin layer of cementum known as *coronal cementum* on the enamel. In humans, thin patches of afibrillar cementum sometimes may be seen in the cervical half of the crown.

Electron microscopy has shown a *dental cuticle* consisting of a layer of homogeneous organic material of variable thickness (approximately 0.25 μm) overlying the enamel surface. It is nonmineralized and not always present. In some cases near the

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cementoenamel junction, it is deposited over a layer of afibrillar cementum, which in turn overlies enamel. The cuticle may be present between the junctional epithelium and the tooth. Ultrastructural histochemical studies have shown that the dental cuticle is proteinaceous,[64] and it may be an accumulation of tissue fluid components.[46,107]

Gingival Fluid (Sulcular Fluid). The value of the gingival fluid is that it can be represented as either a transudate or an exudate. The gingival fluid contains a vast array of biochemical factors, offering potential use as a diagnostic or prognostic biomarker of the biologic state of the periodontium in health and disease.[42] The gingival fluid contains components of connective tissue, epithelium, inflammatory cells, serum, and microbial flora inhabiting the gingival margin or the sulcus (pocket).[41] In the healthy sulcus the amount of the gingival fluid is very small. During inflammation, however, the gingival fluid flow increases, and its composition start to resemble that of an inflammatory exudate.[28]

The main route of the gingival fluid diffusion is through the basement membrane, through the relatively wide intracellular spaces of the junctional epithelium, and then into the sulcus.[94]

The gingival fluid is believed to (1) cleanse material from the sulcus, (2) contain plasma proteins that may improve adhesion of the epithelium to the tooth, (3) possess antimicrobial properties, and (4) exert antibody activity to defend the gingiva (see Chapter 6).

Gingival Connective Tissue

The major components of the gingival connective tissue are collagen fibers (about 60% by volume), fibroblasts (5%), vessels, nerves, and matrix (about 35%).

The connective tissue of the gingiva is known as the *lamina propria* and consists of two layers: (1) a *papillary layer* subjacent to the epithelium, which consists of papillary projections between the epithelial rete pegs, and (2) a *reticular layer* contiguous with the periosteum of the alveolar bone. Connective tissue has a cellular and an extracellular compartment composed of fibers and ground substance. Thus the gingival connective tissue is largely a fibrous connective tissue that has elements originating directly from the oral mucosal connective tissue, as well as some fibers (dentogingival) that originate from the developing dental follicle.[11]

The *ground substance* fills the space between fibers and cells, is amorphous, and has a high content of water. It is composed of proteoglycans, mainly hyaluronic acid and chondroitin sulfate, and glycoproteins, mainly fibronectin. Glycoproteins account for the faint PAS-positive reaction of the ground substance.[43] Fibronectin binds fibroblasts to the fibers and many other components of the intercellular matrix, helping mediate cell adhesion and migration. Laminin, another glycoprotein found in the basal lamina, serves to attach it to epithelial cells.

The three types of connective tissue fibers are collagen, reticular, and elastic. Collagen type I forms the bulk of the lamina propria and provides the tensile strength to the gingival tissue. Type IV collagen (argyrophilic reticulum fiber) branches between the collagen type I bundles and is continuous with fibers of the basement membrane and blood vessel walls.[76]

The elastic fiber system is composed of oxytalan, elaunin, and elastin fibers distributed among collagen fibers.[26]

Therefore densely packed collagen bundles that are anchored into the acellular extrinsic fiber cementum just below the terminal point of the junctional epithelium form the connective tissue attachment. The stability of this attachment is a key factor in limiting the migration of junctional epithelium.[27]

Gingival Fibers. The connective tissue of the marginal gingiva is densely collagenous, containing a prominent system of collagen fiber bundles called the *gingival fibers.* They consist of type I collagen.[95] The gingival fibers have the following functions:

1. To brace the marginal gingiva firmly against the tooth.
2. To provide the rigidity necessary to withstand the forces of mastication without being deflected away from the tooth surface.
3. To unite the free marginal gingiva with the cementum of the root and the adjacent attached gingiva.

The gingival fibers are arranged in three groups: gingivodental, circular, and transseptal.[65]

Gingivodental Group. The gingivodental fibers are those on the facial, lingual, and interproximal surfaces. They are embedded in the cementum just beneath the epithelium at the base of the gingival sulcus. On the facial and lingual surfaces, they project from the cementum in fanlike conformation toward the crest and outer surface of the marginal gingiva, terminating short of the epithelium (Figures 2-19 and 2-20). They also extend externally to the periosteum of the facial and lingual alveolar bones, terminating in the attached gingiva or blending with the periosteum of the bone. Interproximally, the gingivodental fibers extend toward the crest of the interdental gingiva.

Circular Group. The circular fibers course through the connective tissue of the marginal and interdental gingivae and encircle the tooth in ringlike fashion.

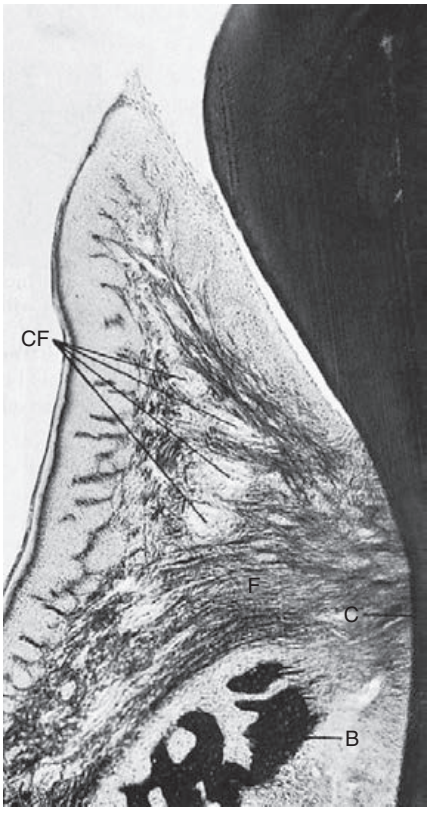

Figure 2-19 Faciolingual section of marginal gingiva showing gingival fibers *(F)* extending from the cementum *(C)* to the crest of the gingiva, to the outer gingival surface, and external to the periosteum of the bone *(B).* Circular fibers *(CF)* are shown in cross-section between the other groups. *(Courtesy Sol Bernick.)*

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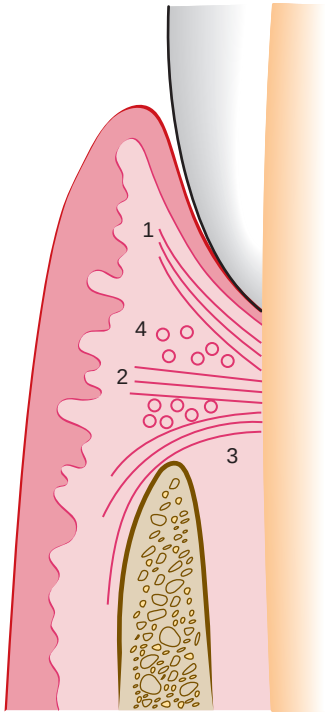

Figure 2-20 Diagram of the gingivodental fibers extending from the cementum *(1)* to the crest of the gingiva, *(2)* to the outer surface, and *(3)* external to the periosteum of the labial plate. Circular fibers *(4)* are shown in cross-section.

Transseptal Group. Located interproximally, the transseptal fibers form horizontal bundles that extend between the cementum of approximating teeth into which they are embedded. They lie in the area between the epithelium at the base of the gingival sulcus and the crest of the interdental bone and are sometimes classified with the principal fibers of the periodontal ligament.

Page et al[91] also have described (1) a group of *semicircular fibers* that attach at the proximal surface of a tooth, immediately below the cementoenamel junction, go around the facial or lingual marginal gingiva of the tooth, and attach on the other proximal surface of the same tooth and (2) a group of *transgingival fibers* that attach in the proximal surface of one tooth, traverse the interdental space diagonally, go around the facial or lingual surface of the adjacent tooth, again traverse diagonally the interdental space, and attach in the proximal surface of the next tooth.

Tractional forces in the extracellular matrix produced by fibroblasts are believed to be the forces responsible for generating tension in the collagen. This keeps the teeth tightly bound to each other and to the alveolar bone.

Cellular Elements.

The preponderant cellular element in the gingival connective tissue is the *fibroblast.* Numerous fibroblasts are found between the fiber bundles. Fibroblasts are of mesenchymal origin and play a major role in the development, maintenance, and repair of gingival connective tissue. As with connective tissue elsewhere in the body, fibroblasts synthesize collagen and elastic fibers, as well as the glycoproteins and glycosaminoglycans of the amorphous intercellular substance. Fibroblasts also regulate collagen degradation through phagocytosis and secretion of collagenases.

Fibroblast heterogeneity is now a well-established feature of fibroblasts in the periodontium.[101] Although the biologic and clinical significance of such heterogeneity is not yet clear, it seems that this is necessary for the normal functioning of tissues in health, disease, and repair.[11]

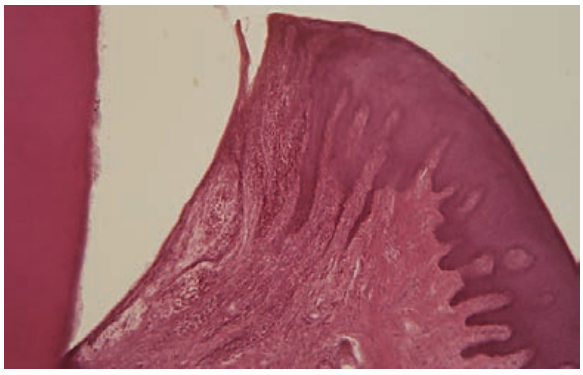

Figure 2-21 Section of clinically normal gingiva showing some degree of inflammation, which is almost always present near the base of the sulcus.

Mast cells, which are distributed throughout the body, are numerous in the connective tissue of the oral mucosa and the gingiva.[24,116,117,135] *Fixed macrophages* and *histiocytes* are present in the gingival connective tissue as components of the mononuclear phagocyte system (reticuloendothelial system) and are derived from blood monocytes. *Adipose cells* and *eosinophils,* although scarce, also are present in the lamina propria.

In clinically normal gingiva, small foci of plasma cells and lymphocytes are found in the connective tissue near the base of the sulcus (Figure 2-21). Neutrophils can be seen in relatively high numbers in both the gingival connective tissue and the sulcus. These inflammatory cells usually are present in small amounts in clinically normal gingiva.

Speculations about whether small amounts of leukocytes should be considered a normal component of the gingiva or an incipient inflammatory infiltrate without clinical expression are of theoretic rather than practical importance. Lymphocytes are absent when gingival normalcy is judged by strict clinical criteria or under special experimental conditions,[8,89] but they are practically constant in healthy, normal gingiva, even before the complete tooth eruption.[68,78,104]

For more information on cellular elements, please visit the companion Expert CONSULT website at www.expertconsult.com.

Repair of Gingival Connective Tissue.

Because of the high turnover rate, the connective tissue of the gingiva has remarkably good healing and regenerative capacity. Indeed, it may be one of the best healing tissues in the body and generally shows little evidence of scarring after surgical procedures. This is likely caused by rapid reconstruction of the fibrous architecture of the tissues.[82] However, the reparative capacity of gingival connective tissue is not as great as that of the periodontal ligament or the epithelial tissue.

Blood Supply, Lymphatics, and Nerves.

Microcirculatory tracts, blood vessels, and lymphatic vessels play an important role in drainage of tissue fluid and in the spread of inflammation. In gingivitis and periodontitis, the microcirculation and vascular formation change greatly in the vascular network directly under the gingival sulcular epithelium and junctional epithelium.[80]

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Understanding normal gingival structure and function provides clinicians with the basis for making clinical decisions that will maintain gingival health. The thick keratinized surface epithelium coupled with the dense type I collagenous gingival connective tissues must be protected from damage during periodontal and restorative therapy. The junctional epithelium and the sulcular epithelium of the gingival sulcus are frequently disrupted by periodontal procedures, such as root planing, and by restorative techniques involving the gingival margins. These epithelial layers have great reparative power; however, their recuperative protective function of providing a barrier and a seal against the tooth depends on an intact dense collagen substructure is provided by the gingival fibers. The clinician has the obligation to minimize trauma to the gingival fibers during therapy so that normal gingival form and function can be maintained.

Gingivitis is initiated by subgingival bacteria causing disruption of the barrier function of sulcular epithelium and subsequent inflammation. Patients need to be able to use oral hygiene techniques that reduce the subgingival bacterial load without causing significant damage to the epithelial structure lining the gingival sulcus. Therapists have to tailor oral hygiene instruction to each patient's oral gingival anatomy with due regard to the underlying cellular dynamics.

Patients with a thin biotype of gingival architecture are particularly vulnerable to connective tissue loss and epithelial damage, thus they need special atraumatic treatment and oral hygiene techniques. Thick biotypes are more resilient to therapy and may be better able to be treated with more aggressive procedures without risking loss of normal gingival function.

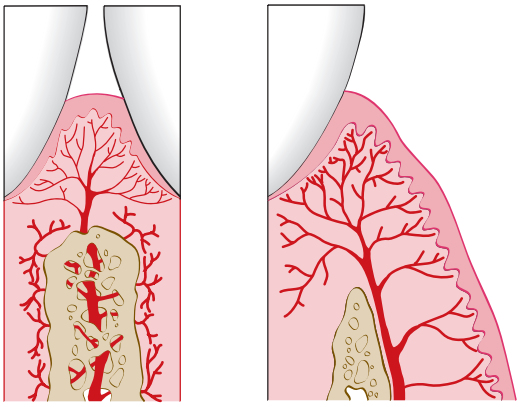

Figure 2-22 Diagram of arteriole penetrating the interdental alveolar bone to supply the interdental tissues *(left)* and a supraperiosteal arteriole overlying the facial alveolar bone, sending branches to the surrounding tissue *(right)*.

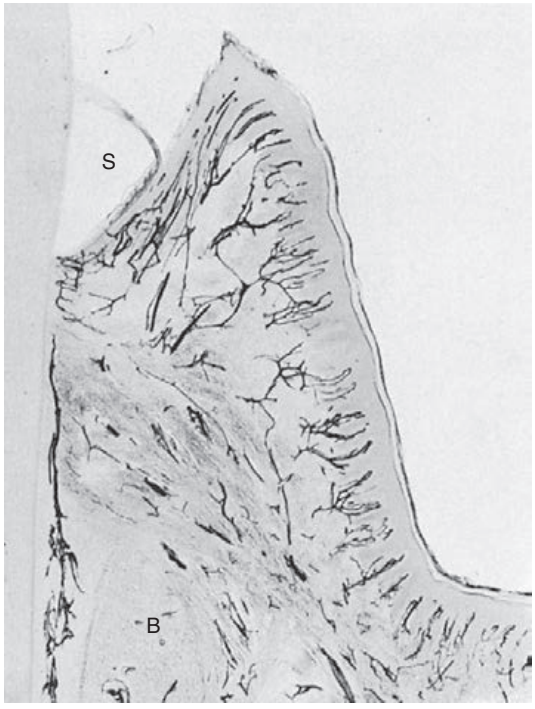

Figure 2-23 Blood supply and peripheral circulation of the gingiva. Tissues perfused with India ink. Note the capillary plexus parallel to the sulcus *(S)* and the capillary loops in the outer papillary layer. Note also the supraperiosteal vessels external to the bone *(B)*, which supply the gingiva, and a periodontal ligament vessel anastomosing with the sulcus plexus. *(Courtesy Sol Bernick.)*

Blood vessels are easily evidenced in tissue sections by means of immunohistochemical reactions against proteins of endothelial cells (factor VIII and adhesion molecules). Before these techniques were developed, vascularization patterns of periodontal tissues had been described using histoenzymatic reactions for alkaline phosphatase and adenosine triphosphatase because of the great activity of these enzymes in endothelial cells.[25,138]

For more information on blood supply, lymphatics, and nerves, please visit the companion Expert CONSULT website at www.expertconsult.com.

Three sources of blood supply to the gingiva are as follows (Figures 2-22 and 2-23):

1. *Supraperiosteal arterioles* along the facial and lingual surfaces of the alveolar bone, from which capillaries extend along the sulcular epithelium and between the rete pegs of the external gingival surface.[39,53] Occasional branches of the arterioles pass through the alveolar bone to the periodontal ligament or run over the crest of the alveolar bone.
2. *Vessels of the periodontal ligament,* which extend into the gingiva and anastomose with capillaries in the sulcus area.

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3. *Arterioles,* which emerge from the crest of the interdental septa[44] and extend parallel to the crest of the bone to anastomose with vessels of the periodontal ligament, with capillaries in the gingival crevicular areas and vessels that run over the alveolar crest.

Beneath the epithelium on the outer gingival surface, capillaries extend into the papillary connective tissue between the epithelial rete pegs in the form of terminal hairpin loops with efferent and afferent branches, spirals, and varices[25,53] (Figures 2-23 and 2-24). The loops are sometimes linked by cross-communications,[44] and flattened capillaries serve as reserve vessels when the circulation is increased in response to irritation.[49]

Along the sulcular epithelium, capillaries are arranged in a flat, anastomosing plexus that extends parallel to the enamel from the base of the sulcus to the gingival margin.[25] In the col area, a mixed pattern of anastomosing capillaries and loops occurs.

As mentioned, anatomic and histologic changes have been shown to occur in the gingival microcirculation with gingivitis. Prospective studies of the gingival vasculature in animals have demonstrated that in the absence of inflammation, the vascular network is arranged in a regular, repetitive, and layered pattern.[25,96] In contrast, the inflamed gingival vasculature exhibits an irregular vascular plexus pattern, with the microvessels exhibiting a looped, dilated, and convoluted appearance.[96]

The role of the lymphatic system in removing excess fluids, cellular and protein debris, microorganisms, and other elements is important in controlling diffusion and the resolution of inflammatory processes.[79] The *lymphatic drainage of the gingiva* brings in the lymphatics of the connective tissue papillae.[113] It progresses into the collecting network external to the periosteum of the alveolar process, then to the regional lymph nodes, particularly the submaxillary group. In addition, lymphatics just beneath the junctional epithelium extend into the periodontal ligament and accompany the blood vessels.

Neural elements are extensively distributed throughout the gingival tissues. Within the gingival connective tissues, most nerve fibers are myelinated and are closely associated with the blood vessels.[77] *Gingival innervation* is derived from fibers arising from nerves in the periodontal ligament and from the labial, buccal, and palatal nerves.[13] The following nerve structures are present in the connective tissue: a meshwork of terminal argyrophilic fibers, some of which extend into the epithelium; Meissner-type tactile corpuscles; Krause-type end bulbs, which are temperature receptors; and encapsulated spindles.[9]

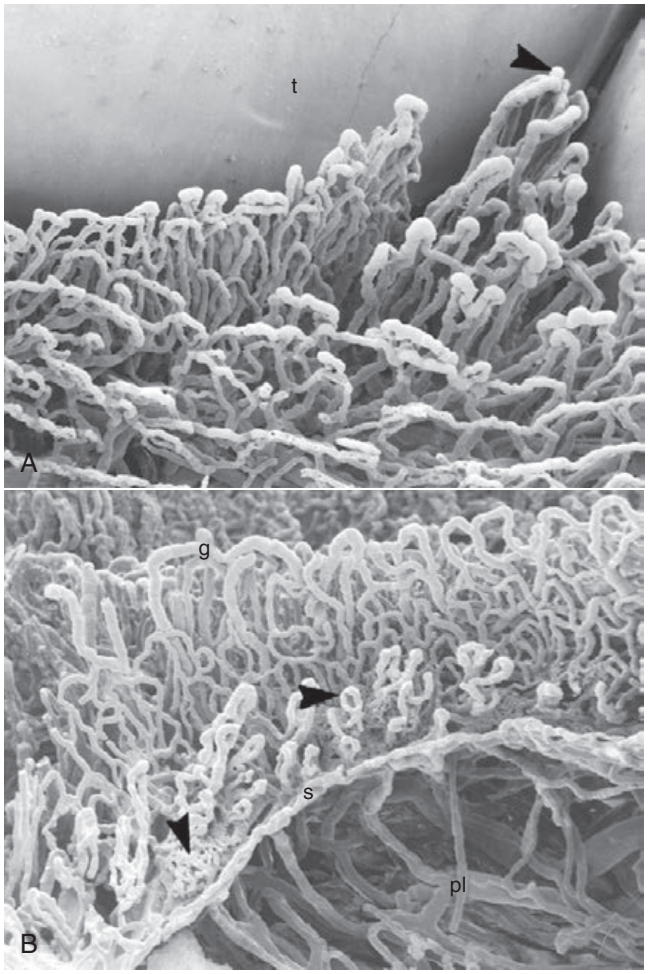

Figure 2-24 Scanning electron microscopic view of gingival tissues of rat molar palatal gingiva after vascular perfusion of plastic and corrosion of soft tissue. **A,** Oral view of gingival capillaries: *t,* tooth; interdental papilla *(arrowhead).* (×180.) **B,** View from the tooth side. Note the vessels of the plexus next to the sulcular and junctional epithelium. The arrowheads point to vessels in sulcus area with mild inflammatory changes. *g,* Crest of marginal gingiva; *s,* bottom of gingival sulcus; *pl,* periodontal ligament vessels. (×150.) *(Courtesy N.J. Selliseth and K. Selvig, University of Bergen, Norway.)*

CORRELATION OF CLINICAL AND MICROSCOPIC FEATURES

An understanding of the normal clinical features of the gingiva requires the ability to interpret them in terms of the microscopic structures they represent.

Color

The color of the attached and marginal gingiva is generally described as "coral pink" and is produced by the vascular supply, the thickness and degree of keratinization of the epithelium, and the presence of pigment-containing cells. The color varies among different persons and appears to be correlated with the cutaneous pigmentation. It is lighter in blond individuals with fair complexion than in swarthy, dark-haired individuals (Figure 2-25).

The attached gingiva is demarcated from the adjacent alveolar mucosa on the buccal aspect by a clearly defined mucogingival line. The alveolar mucosa is red, smooth, and shiny rather than pink and stippled. Comparison of the microscopic structure of the attached gingiva with that of the alveolar mucosa provides an explanation for the difference in appearance. The epithelium of the alveolar mucosa is thinner, is nonkeratinized, and contains no rete pegs (Figure 2-26). The connective tissue of the alveolar mucosa is loosely arranged, and the blood vessels are more numerous.

Physiologic Pigmentation (Melanin). Melanin, a non–hemoglobin-derived brown pigment, is responsible for the normal pigmentation of the skin, gingiva, and remainder of the oral mucous membrane. It is present in all normal individuals, often not in sufficient quantities to be detected clinically, but is absent or severely diminished in albinos. Melanin pigmentation in the oral cavity is prominent in black individuals (see Figure 2-25). Ascorbic acid directly downregulates melanin pigmentation in gingival tissues.[118]

According to Dummett,[37] the distribution of oral pigmentation in black individuals is as follows: gingiva, 60%; hard palate, 61%; mucous membrane, 22%; and tongue, 15%. Gingival pigmentation

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Figure 2-25 A, Clinically normal gingiva in a young adult. **B,** Heavily pigmented (melanotic) gingiva in a middle-aged adult. *(From Glickman I, Smulow JB:* Periodontal disease: clinical, radiographic, and histopathologic features, *Philadelphia, 1974, Saunders.)*

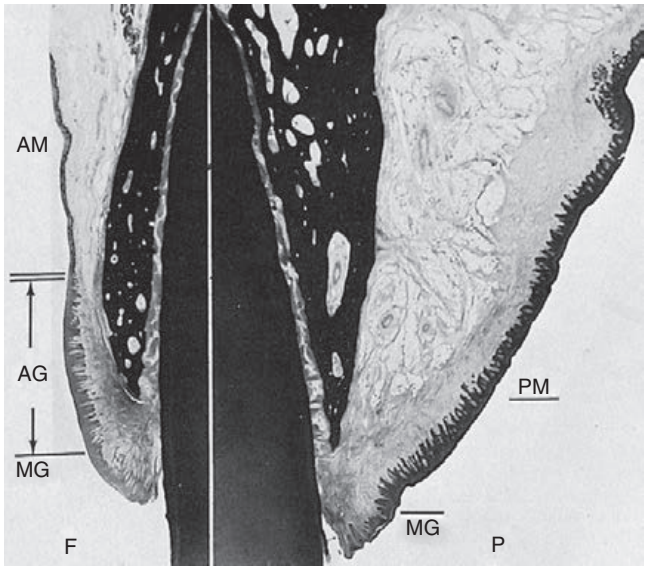

Figure 2-26 Oral mucosa, facial and palatal surfaces. The facial surface *(F)* shows the marginal gingiva *(MG),* attached gingiva *(AG),* and alveolar mucosa *(AM).* The double line marks the mucogingival junction. Note the differences in the epithelium and connective tissue in the attached gingiva and alveolar mucosa. The palatal surface *(P)* shows the marginal gingiva *(MG)* and thick, keratinized palatal mucosa *(PM).*

Figure 2-27 Thickened shelflike contour of gingiva on tooth in lingual version aggravated by local irritation caused by plaque accumulation.

occurs as a diffuse, deep-purplish discoloration or as irregularly shaped, brown and light-brown patches. It may appear in the gingiva as early as 3 hours after birth and often is the only evidence of pigmentation.[37]

Oral *repigmentation* refers to the clinical reappearance of melanin pigment after a period of clinical *depigmentation* of the oral mucosa resulting from chemical, thermal, surgical, pharmacologic, or idiopathic factors.[38] Information on the repigmentation of oral tissues after surgical procedures is extremely limited, and no definitive treatment is offered at this time.

Size

The size of the gingiva corresponds with the sum total of the bulk of cellular and intercellular elements and their vascular supply. Alteration in size is a common feature of gingival disease.

Contour

The contour or shape of the gingiva varies considerably and depends on the shape of the teeth and their alignment in the arch, the location and size of the area of proximal contact, and the dimensions of the facial and lingual gingival embrasures. The marginal gingiva envelops the teeth in collarlike fashion and follows a scalloped outline on the facial and lingual surfaces. It forms a straight line along teeth with relatively flat surfaces. On teeth with pronounced mesiodistal convexity (e.g., maxillary canines) or teeth in labial version, the normal arcuate contour is accentuated, and the gingiva is located farther apically. On teeth in lingual version, the gingiva is horizontal and thickened (Figure 2-27). In addition, gingival tissue biotype varies significantly. A thin and clear gingiva is found in one-third of the population, primarily in females with slender teeth with a narrow zone of keratinized tissue, whereas a clear thick gingiva with a broad zone of keratinized tissue is present in two-thirds of the population, primarily in males.[34]

Shape

The shape of the interdental gingiva is governed by the contour of the proximal tooth surfaces and the location and shape of gingival embrasures. When the proximal surfaces of the crowns are relatively flat faciolingually, the roots are close together, the interdental bone is thin mesiodistally, and the gingival embrasures and interdental gingiva are narrow mesiodistally. Conversely, with proximal surfaces that flare away from the area of contact, the mesiodistal diameter of the interdental gingiva is broad (Figure 2-28). The height of the interdental gingiva varies with the location of the proximal contact. Thus, in the anterior region of the dentition, the interdental papilla is pyramidal in form, whereas the papilla is more flattened in a buccolingual direction in the molar region.

Consistency

The gingiva is firm and resilient and with the exception of the movable free margin, tightly bound to the underlying bone. The collagenous nature of the lamina propria and its contiguity with the mucoperiosteum of the alveolar bone determine the firmness of the attached gingiva. The gingival fibers contribute to the firmness of the gingival margin.

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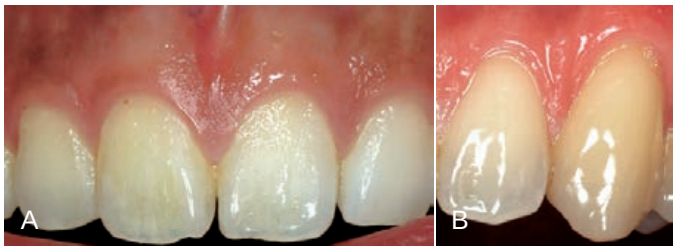

Figure 2-28 Shape of interdental gingival papillae correlated with shape of teeth and embrasures. **A,** Broad interdental papillae. **B,** Narrow interdental papillae.

Surface Texture

The gingiva presents a textured surface similar to an orange peel and is referred to as being *stippled* (see Figure 2-25). Stippling is best viewed by drying the gingiva.

The attached gingiva is stippled; the marginal gingiva is not. The central portion of the interdental papillae is usually stippled, but the marginal borders are smooth. The pattern and extent of stippling vary among individuals and different areas of the same mouth.[51,96] Stippling is less prominent on lingual than facial surfaces and may be absent in some persons.

Stippling varies with age. It is absent in infancy, appears in some children at about 5 years of age, increases until adulthood, and frequently begins to disappear in old age.

Microscopically, stippling is produced by alternate rounded protuberances and depressions in the gingival surface. The papillary layer of the connective tissue projects into the elevations, and the elevated and depressed areas are covered by stratified squamous epithelium (Figure 2-29). The degree of keratinization and the prominence of stippling appear to be related.

Scanning electron microscopy has shown considerable variation in shape, but a relatively constant depth of stippling. At low magnification, a rippled surface is seen, interrupted by irregular depressions 50 µm in diameter. At higher magnification, cell micropits are seen.[30]

Stippling is a form of adaptive specialization or reinforcement for function. It is a feature of healthy gingiva, and reduction or loss of stippling is a common sign of gingival disease. When the gingiva is restored to health after treatment, the stippled appearance returns.

The surface texture of the gingiva is also related to the presence and degree of epithelial keratinization. Keratinization is considered a protective adaptation to function. It increases when the gingiva is stimulated by toothbrushing. However, research on free gingival grafts (see Chapter 63) has shown that when connective tissue is transplanted from a keratinized area to a nonkeratinized area, it becomes covered by a keratinized epithelium.[62] This finding suggests a connective tissue–based genetic determination of the type of epithelial surface.

Position

The *position* of the gingiva refers to the level at which the gingival margin is attached to the tooth. When the tooth erupts into the oral cavity, the margin and sulcus are at the tip of the crown; as eruption progresses, they are seen closer to the root. During this eruption process, as described earlier, the junctional epithelium, oral epithelium, and reduced enamel epithelium undergo extensive alterations and remodeling while maintaining the shallow physiologic depth of the sulcus. Without this remodeling of the epithelia, an abnormal anatomic relationship between the gingiva and the tooth would result.

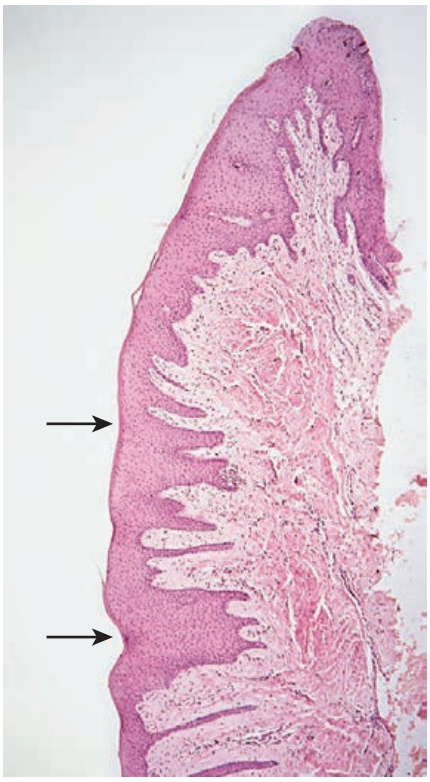

Figure 2-29 Gingival biopsy of patient shown in Figure 2-7, demonstrating alternate elevations and depressions *(arrows)* in the attached gingiva responsible for stippled appearance.

Continuous Tooth Eruption. According to the concept of continuous eruption,[50] eruption does not cease when teeth meet their functional antagonists but continues throughout life. Eruption consists of an active and a passive phase. *Active eruption* is the movement of the teeth in the direction of the occlusal plane, whereas *passive eruption* is the exposure of the teeth by apical migration of the gingiva.

This concept distinguishes between the *anatomic crown* (portion of the tooth covered by enamel) and the *anatomic root* (portion of the tooth covered by cementum) and between the *clinical crown* (part of the tooth that has been denuded of its gingiva and projects into the oral cavity) and *clinical root* (portion of the tooth covered by periodontal tissues). When the teeth reach their functional antagonists, the gingival sulcus and junctional epithelium are still on the enamel, and the clinical crown is approximately two-thirds of the anatomic crown.

For more information on continuous tooth eruption, please visit the companion Expert CONSULT website at www.expertconsult.com.

As teeth erupt, cementum is deposited at the apices and furcations of the roots and bone is formed along the fundus of the alveolus and at the crest of the alveolar bone. In this way, part of the tooth substance lost by attrition is replaced by lengthening of the root, and socket depth is maintained to support the root.

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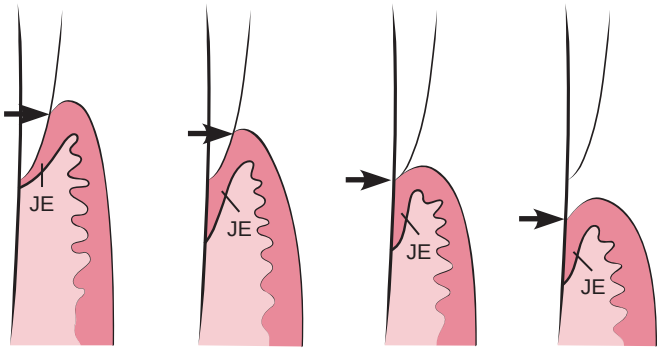

Figure 2-30 Diagrammatic representation of the four steps in passive eruption according to Gottlieb and Orban.[50] *1,* Base of the gingival sulcus *(arrow)* and the junctional epithelium *(JE)* are on the enamel. *2,* Base of the gingival sulcus *(arrow)* is on the enamel, and part of the junctional epithelium is on the root. *3,* Base of the gingival sulcus *(arrow)* is at the cementoenamel line, and the entire junctional epithelium is on the root. *4,* Base of the gingival sulcus *(arrow)* and the junctional epithelium are on the root.

Although originally thought to be a normal physiologic process, passive eruption is now considered a pathologic process. Passive eruption is divided into the following four stages (Figure 2-30):

Stage 1: The teeth reach the line of occlusion. The junctional epithelium and base of the gingival sulcus are on the enamel.

Stage 2: The junctional epithelium proliferates so that part is on the cementum and part is on the enamel. The base of the sulcus is still on the enamel.

Stage 3: The entire junctional epithelium is on the cementum, and the base of the sulcus is at the cementoenamel junction. As the junctional epithelium proliferates from the crown onto the root, it does not remain at the cementoenamel junction any longer than at any other area of the tooth.

Stage 4: The junctional epithelium has proliferated farther on the cementum. The base of the sulcus is on the cementum, a portion of which is exposed. Proliferation of the junctional epithelium onto the root is accompanied by degeneration of gingival and periodontal ligament fibers and their detachment from the tooth. The cause of this degeneration is not understood. At present, it is believed to be the result of chronic inflammation and therefore a pathologic process.

As noted, apposition of bone accompanies active eruption. The distance between the apical end of the junctional epithelium and the crest of the alveolus remains constant throughout continuous tooth eruption (1.07 mm).[47]

Exposure of the tooth by the apical migration of the gingiva is called *gingival recession,* or *atrophy.* According to the concept of continuous eruption, the gingival sulcus may be located on the crown, cementoenamel junction, or root, depending on the age of the patient and stage of eruption. Therefore some root exposure with age would be considered normal and referred to as *physiologic recession.* Again, this concept is not accepted at present. Excessive exposure is termed *pathologic recession* (see Chapter 22).

References can be found on the companion Expert CONSULT website at www.expertconsult.com

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CHAPTER 3

Aging and the Periodontium

Ian Needleman

CHAPTER OUTLINE

EFFECTS OF AGING ON THE PERIODONTIUM
- Gingival Epithelium
- Gingival Connective Tissue
- Periodontal Ligament
- Cementum
- Alveolar Bone
- Bacterial Plaque
- Immune and inflammatory responses

EFFECTS OF AGING ON THE PROGRESSION OF PERIODONTAL DISEASES

AGING AND THE RESPONSE TO TREATMENT OF THE PERIODONTIUM

Increased health awareness and improvements in preventive dentistry have led to decreasing tooth loss for all age groups. The effects of this shift in tooth retention need to be considered carefully. In particular, increased life expectancy and greater health expectations may lead to changes in demand from older individuals for periodontal treatment and potentially a substantial increase for supportive periodontal therapy. Therefore an understanding of the impact of aging on the periodontium is critical. This chapter first reviews the literature concerning the fundamental aspects of aging on the periodontal tissues. Following this, broader aspects of aging are examined, as well as the possible effects on treatment outcomes.

The evidence base is not without problems, many of which make it difficult to draw conclusions on the effects of aging. Some of these problems include inconsistency in the definition of a true "older" group, inadequate exclusion of adults with systemic diseases that can modify study findings, and attempts to extrapolate results from animal research. For the purposes of this chapter, the effects of aging are limited to a narrow review of possible biologic and microbiologic changes.* The reader should be fully aware that this excludes many important age-associated phenomena, including the reduction in an individual's cognitive or motor function skills, which may have a direct impact on periodontal management. These will be discussed more fully in Chapter 39.

Since this chapter was first written 10 years ago, the volume of new research on aging and the periodontium has not increased substantially. Newer molecular techniques are being employed in related laboratory research, and these techniques are starting to provide insight into age-associated effects. These approaches are likely to be prove fruitful in the future. Despite the limited data directly investigating the periodontium, much effort and resource is being used in researching questions partially related to this topic. These questions include the effect of periodontal infection on general health (see Chapter 28) and the impact of osteoporosis on periodontal status (see Chapter 38).

*For further reading on the effects of aging on the dental and periodontal patient, the reader should consult Lamster IB, Northridge ME: *Improving oral health for the elderly: an interdisciplinary approach,* New York, 2008, Springer.

EFFECTS OF AGING ON THE PERIODONTIUM

Gingival Epithelium

Thinning and decreased keratinization of the gingival epithelium have been reported with age.[43] The significance of these findings could mean an increase in epithelial permeability to bacterial antigens, a decreased resistance to functional trauma, or both. If so, such changes might influence long-term periodontal outcomes. However, other studies have found no age-related differences in the gingival epithelium of humans or dogs.[8,19] Other reported changes with aging include the flattening of rete pegs and altered cell density. Conflicting data regarding surgical regeneration times for gingival epithelium have been ascribed to problems in research methodology.[48]

The effect of aging on the location of the junctional epithelium has been the subject of much speculation. Some reports show migration of the junctional epithelium from its position in healthy individuals (i.e., on enamel) to a more apical position on the root surface with accompanying gingival recession.[8] However, in other animal studies, no apical migration has been noted.[22] With continuing gingival recession, the width of the attached gingiva would be expected to decrease with age, but the opposite appears to be true.[2,3] Alternatively, the migration of the junctional epithelium to

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Periodontal tissues are programmed to provide healthy support for the dentition for the life of the patient. Although there are some concerns that older patients have increased susceptibility to periodontal disease, the role of age in disease progression is minimal. With appropriate dental therapy and maintenance of low plaque levels, clinicians can assure older patients that they can keep a functioning dentition even though there may be a slightly lowered resistance to bacterial-induced periodontal inflammation and an increased risk of higher levels of putative periodontal pathogenic bacteria such as *Treponema forsythia, Treponema denticola, Porphyromonas gingivalis, and Aggregatibacter actinomycetemcomitans*.

In response to treatment of periodontitis, older patients generally have all the needed cellular and molecular mechanisms to ensure healing and repair, but these mechanisms may be slower and somewhat lower than those in younger patients. However, with the more complex cellular interactions that occur in association with the healing of advanced regenerative periodontal surgical operations involving bone grafts, guided tissue regeneration, and use of bioactive agents, older patients cannot be expected to have as favorable treatment outcomes as younger ones. Thus clinicians may decide to use simpler approaches to treating advanced periodontal lesions in these patients. Nonsurgical procedures may be favored in cases in which the patients age is thought to be significant.

The distinction between actual age in years and biologic status is a clinical skill that clinicians need to appreciate. With the historic improvements in the health status of older patients, more demanding treatment plans can be implemented for patients with advanced age than were appropriate in previous decades. Patients' motor skills and cognitive abilities can be eroded by age, thus dentists need to modify teaching oral hygiene techniques based on the assessment of each mature patients' potential to learn and apply personal plaque removal techniques. The use of antibacterial mouthwashes and mechanical oral hygiene devices have a special role in these patients.

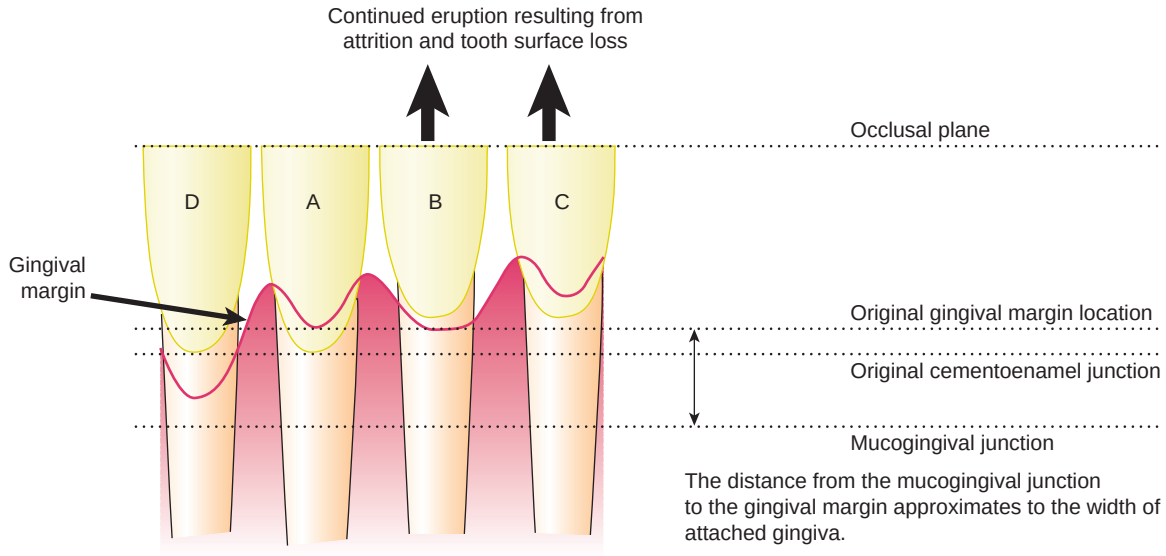

Figure 3-1 Diagram showing the relationship of the gingival margin with the crown and root surface. **A,** Normal relationship with the gingival margin 1 to 2 mm above the cementoenamel junction. **B,** Wear of the incisal edge and continued tooth eruption. The gingival margin remains in the same position as in **A,** therefore the root surface is exposed and clinical recession is evident. The width of the attached gingiva has not changed. **C,** Wear of the incisal edge and continued tooth eruption. The gingival margin has moved with the tooth, therefore the entire dentogingival complex has moved coronally with a resulting increase in the width of the attached gingiva. **D,** No wear of incisal edge is evident. Gingiva has moved apically, and clinical recession is evident. The width of attached gingiva is reduced.

the root surface could be caused by the tooth erupting through the gingiva in an attempt to maintain occlusal contact with its opposing tooth (passive eruption) as a result of tooth surface loss from attrition (Figure 3-1). The consensus is that gingival recession is not an inevitable physiologic process of aging but is explained by cumulative effects of inflammation or trauma on the periodontium[6,8] (see later discussion) (Figure 3-2).

Gingival Connective Tissue

Increasing age results in coarser and denser gingival connective tissues.[51] Qualitative and quantitative changes to collagen have been reported. These include an increased rate of conversion of soluble to insoluble collagen, increased mechanical strength, and increased denaturing temperature. These results indicate increased collagen stabilization caused by changes in the macromolecular conformation.[26] Not surprisingly, greater collagen content has been found in the gingivae of older animals despite a lower rate of collagen synthesis decreasing with age.[8,26,48]

Periodontal Ligament

Changes in the periodontal ligament that have been reported with aging include decreased numbers of fibroblasts and a more irregular structure, paralleling the changes in the gingival connective tissues.[8,26,48] Other findings include decreased organic matrix

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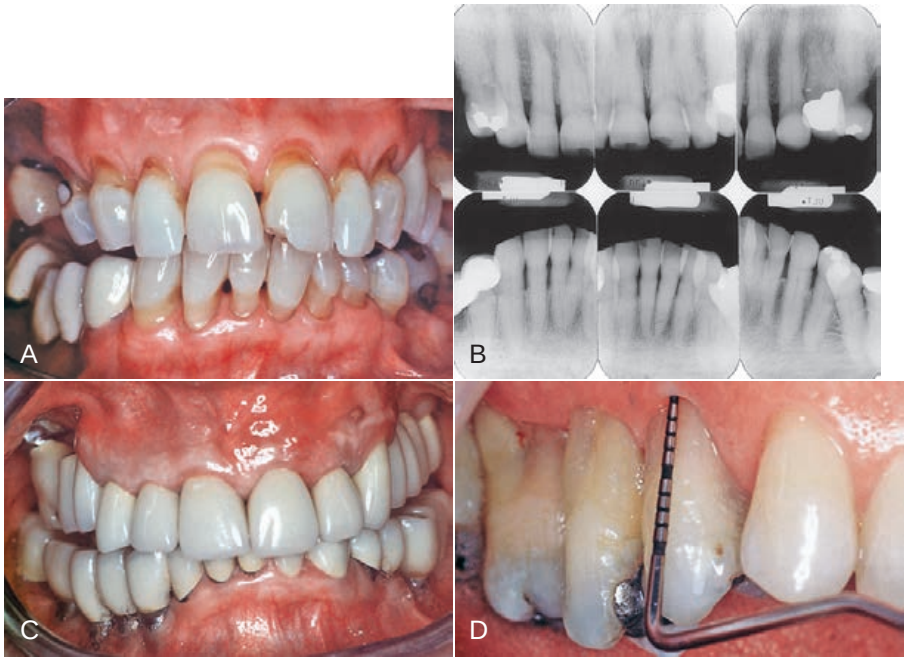

Figure 3-2 Three scenarios illustrating the variation of the position of the gingival margin with age. **A,** Overeruption with recession in older individual (68-year-old female) with generalized recession and previous (treated) history of periodontitis. Note some overeruption of lower anterior teeth and wear of teeth related to oral hygiene measures. **B,** Radiographs of the patient in **A. C,** Overeruption without recession in an older individual (72-year-old female) with no periodontitis but marked lower incisor tooth wear and overeruption. Note how the gingival margin has migrated coronally with the erupting teeth. **D,** Extensive recession in young individual (32-year-old male) with marked recession and no history of periodontitis. The recession has resulted from a combination of anatomically thin tissues and toothbrush-related trauma.

production and epithelial cell rests and increased amounts of elastic fiber.[48] Conflicting results have been reported for changes in the width of periodontal ligament in human and animal models. Although true variation might exist, this finding probably reflects the functional status of the teeth in the studies because the width of the space will decrease if the tooth is unopposed (hypofunction) or will increase with excessive occlusal loading.[26,48] Both scenarios might be anticipated as a result of tooth loss in this population. These effects also might explain the variability in studies reporting qualitative changes within the periodontal ligament.

Recognition that the periodontal ligament has an important role in alveolar bone metabolism has led to an increased interest in investigating this role in relation to maintenance of periodontal health and pathogenesis of periodontitis. One approach to investigating this role is to examine mediators of bone homeostasis such as receptor activator of nuclear factor-κB ligand (RANKL) and osteoprotegerin (OPG). RANKL is widely recognized for its role in activating osteoclasts, whereas OPG antagonizes RANKL binding, thus helping to maintain balance. The interplay of cytokines, in particular the interleukin family, has also been extensively researched in periodontal pathogenesis. Some of the interleukins are potent mediators of inflammation (e.g., interleukin-1 [IL-1]), whereas others have been shown to downregulate this process (e.g., Il-4 and IL-10). Therefore evaluating the balance between these homeostatic processes would seem to be of great merit in evaluating possible negative changes in the periodontium. Such a strategy appears predictive of hard tissue destruction in rheumatoid arthritis.[15] Recent findings comparing periodontal ligament cells in older (>60 years of age) versus younger (15 to 20 years of age) individuals have suggested greater gene expression for proinflammatory cytokines.[7] This effect has been reported more widely as an age-associated change, although it is unclear whether as cause or effect.[35] However, in addition to increased Il-1 and IL-6 expression, OPG was also increased, suggesting that increased OPG might be a homeostatic response to the upregulation of inflammation.[7] If this homeostatic mechanism was effective, it might explain why an increase in inflammation did not result in greater tissue damage with age. Periodontal ligament cell proliferation was decreased with age, suggesting an impairment of repair potential, although such an impact does not appear to be manifested clinically.[50] Further research investigating periodontal ligament cells from individuals diagnosed with periodontitis will be important in understanding the potentially important relationship between aging and periodontal ligament cells.

Cementum

Some consensus regarding aging effects on cementum exists. An increase in cemental width is a common finding; this increase may be 5 to 10 times with increasing age.[8] This finding is not surprising because deposition continues after tooth eruption. The increase in width is greater apically and lingually.[48] Although cementum has limited capacity for remodelling, an accumulation of resorption bays explains the finding of increasing surface irregularity.[16]

Alveolar Bone

Reports of morphologic changes in alveolar bone mirror age-related changes in other bony sites. Specific to the periodontium are findings of a more irregular periodontal surface of bone and less regular insertion of collagen fibers.[48] Although age is a risk factor for the bone mass reductions in osteoporosis, it is not causative and therefore should be distinguished from physiologic aging processes.[18] Overriding the diverse observations of bony changes with age is the important finding that the healing rate of bone in extraction sockets appears to be unaffected by increasing age.[4] Indeed, the success of osseointegrated dental implants, which relies on intact bone healing responses, does not appear to be age related.[9] However, balancing this view is the recent observation that bone graft preparations (decalcified freeze-dried bone) from donors older than 50 years of age possessed significantly less osteogenic potential than graft material from younger donors.[42] The possible significance of this phenomenon on normal healing responses needs to be investigated.

Bacterial Plaque

Dentogingival plaque accumulation has been suggested to increase with age.[17] This might be explained by the increase in hard tissue

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surface area as a result of gingival recession and the surface characteristics of the exposed root surface as a substrate for plaque formation compared with enamel. Other studies have shown no difference in plaque quantity with age. This contradiction might reflect the different age ranges of experimental groups as variable degrees of gingival recession and root surface exposure. For supragingival plaque, no real qualitative differences have been shown for plaque composition.[17] For subgingival plaque, one study has shown similar subgingival flora to a normal flora, whereas another study reported increased numbers of enteric rods and pseudomonads in older adults.[31,46] Mombelli suggests caution in the interpretation of this finding because of increased oral carriage of these species among older adults.[29] It has been speculated that a shift occurs in the importance of certain periodontal pathogens with age, specifically including an increased role for *Porphyromonas gingivalis* and a decreased role for *Aggregatibacter actinomycetemcomitans.* However, differentiating true age effects from the changes in ecologic determinants for periodontal bacteria will be difficult. This topic is considered in more detail in a recent review.[29]

Another approach to investigating microbiology is to conduct intervention studies and to examine their impact on the microflora. In individuals 60 to 75 years of age, the prevalence of *P. gingivalis, Treponema forsythia, T. denticola, A. actinomycetemcomitans, and Prevotella intermedia* were high and not clearly related to probing depth.[39] The finding of high levels of the organisms could have been related to the sample of individuals, since they were mostly low income and had no recent dental care. Long-term use (5 years) of a 0.12% chlorhexidine mouth rinse did not promote reductions in the proportions of the organisms in individuals experiencing alveolar bone loss compared with the placebo mouth rinse, possibly a result of the mouth rinse routine being less frequent than daily.[39]

Immune and Inflammatory Responses

Recent advances in the study of the effects of aging on the immune response *(immunosenescence)* have altered the understanding of this phenomenon. In particular, more recent studies have set tighter controls on excluding individuals with systemic conditions known to affect the immune response. As a result, age has been recognized as having much less effect in altering the host response than previously thought.[20,27] Differences between young and older individuals can be demonstrated for T and B cells, cytokines, and natural killer cells but not for polymorphonuclear cells and macrophage activity. McArthur concluded "Measurement of indicators of immune and inflammatory competency suggested that, within the parameters tested, there was no evidence for age-related changes in host defences correlating with periodontitis in an elderly (65 to 75 years) group of individuals, with and without disease."[27] Age-related differences in the inflammatory response in gingivitis have been clearly demonstrated and are discussed later in this textbook.

In relation to systemic inflammatory responses, C-reactive protein (CRP) is an acute phase protein and widely regarded as a marker of inflammatory burden and response to bacterial infection.[44] When investigating serum CRP levels in individuals 60 to 75 years of age comparing those with progressive periodontitis against stable disease, CRP levels were increased in those with progressive periodontitis and no other systemic condition.[47] This might indicate that inflammatory burden can be investigated using CRP, although there is growing evidence that CRP alone may not be as reliable a marker in older persons.[40]

A further modulator of the immune and inflammatory responses is nutrition. Interest in this field is increasing rapidly in medicine and periodontology.[11] Nutrition has been studied extensively in geriatric medicine because of the nutritional intake changes with age. The impact of this as a potential risk factor for periodontal diseases and their progression is therefore of interest. Data have started to appear that suggest a negative association of serum folate with periodontitis (i.e., after controlling for major known confounders, lower serum folate in dentate adults (>60 years of age) is associated with greater levels of periodontitis).[53] These cross-sectional data cannot demonstrate causality alone, but the recognized relationship between folate protection for chronic inflammatory diseases, such as cardiovascular disease,[28] offers biologic plausibility to a potential relationship and merits further investigation with prospective studies.

In summary, although many contradictions exist, a survey of the literature demonstrates that some age-related changes are evident in the periodontium and host response. Whether these changes are significant in altering the progression of periodontal diseases or the response of an older adult to periodontal treatment is examined next.

EFFECTS OF AGING ON THE PROGRESSION OF PERIODONTAL DISEASES

In a classic experimental gingivitis study, subjects were rendered plaque and inflammation free through frequent professional cleaning. Once this was achieved, the subjects abstained from oral hygiene measures for periods of 3 weeks to allow gingivitis to develop.[25] In this experimental model, a comparison of developing gingivitis between young and older individuals demonstrated a greater inflammatory response in older subjects, both in humans and dogs.[8,12,13,17] In the older age group (65 to 80 years of age), the findings included a greater size of infiltrated connective tissue, increased gingival crevicular fluid flow, and increased gingival index.[12,13] Other studies have not demonstrated differences between subjects; this may be related to smaller differences between the ages of the younger and older experimental groups.[52] Intriguingly, even at the baseline level of excellent gingival health before commencing plaque accumulation, differences may exist between groups, with older individuals demonstrating more inflammation.[12,13]

The phrase "getting long in the tooth" expresses a widespread belief that age is inevitably associated with an increased loss of connective tissue attachment. However, this observation might equally well reflect a cumulative exposure to a number of potentially destructive processes. These exposures might include plaque-associated periodontitis, chronic mechanical trauma from tooth brushing, and iatrogenic damage from unfavorable restorative dentistry or repeated scaling and root planing. The effects of these exposures act in one direction only (i.e., increased loss of attachment).[49]

In an attempt to differentiate the effects of age from these other processes, several studies have been designed to eliminate confounding issues and address more clearly the question of age as a risk factor for periodontitis. A *risk factor* is defined as "any characteristic, behaviour, or exposure with an association to a particular disease. The relationship is not necessarily causal in nature.… Some risk factors, if causal, can be modified to reduce one's risk of initiation or progression of disease, such as smoking or improved oral hygiene … while other factors cannot be modified, such as genetic factors."[14] The conclusions from these studies are strikingly consistent and show that the effect of age is either nonexistent or provides a small and clinically insignificant increased risk of loss of periodontal support.[10,24,32,36,37] Indeed, in comparison with the odds ratio of 20.52 for poor oral hygiene status and periodontitis, the

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odds ratio for age was only 1.24[1] and smoking is much more influential than age.[32] Therefore age has been suggested to be not a true risk factor but a background or an associated factor for periodontitis.[36] In addition, clarification of a genetic basis for susceptibility to severe forms of periodontitis underline the overriding importance of plaque, smoking, and susceptibility in explaining most of the variation in periodontal disease severity between individuals.[21] Nevertheless, a longitudinal study of essentially untreated periodontitis in a Japanese elderly population (at least 70 years of age) indicated that 296/394 (75%) of individuals had a least one site with 3 mm or more loss of attachment over a 2-year period.[33] Smoking and baseline attachment level of 6 mm or more were significantly associated with the disease progression.

AGING AND THE RESPONSE TO TREATMENT OF THE PERIODONTIUM

The successful treatment of periodontitis requires both meticulous home-care plaque control by the patient and meticulous supragingival and subgingival debridement by the therapist.[30] Unfortunately, only a few studies have directly compared such an approach among patients of different age groups. The few studies that have done so clearly demonstrate that despite the histologic changes in the periodontium with aging, no differences in response to nonsurgical or surgical treatment have been shown for periodontitis.[5,23,50] However, if plaque control is not ideal, continued loss of attachment is inevitable. Furthermore, without effective periodontal therapy, progression of diseases might be faster with increasing age.[38] Attempts to aid plaque control by chemical means have also been reported.[45]

A purely biologic or physiologic review indicates that aging has some impact on the structure and function of the periodontium, as well as the immune response, and nature of either supragingival or subgingival plaque. However, these changes have a negligible impact on an individual's responsiveness to treatment. Aging might affect other aspects of managing periodontal health such as the risk of root caries[41] (see Chapter 39), and the resulting difficulties should not be underestimated. Interestingly, a recent study has identified greater compliance with supportive maintenance among older individuals than younger patients.[34]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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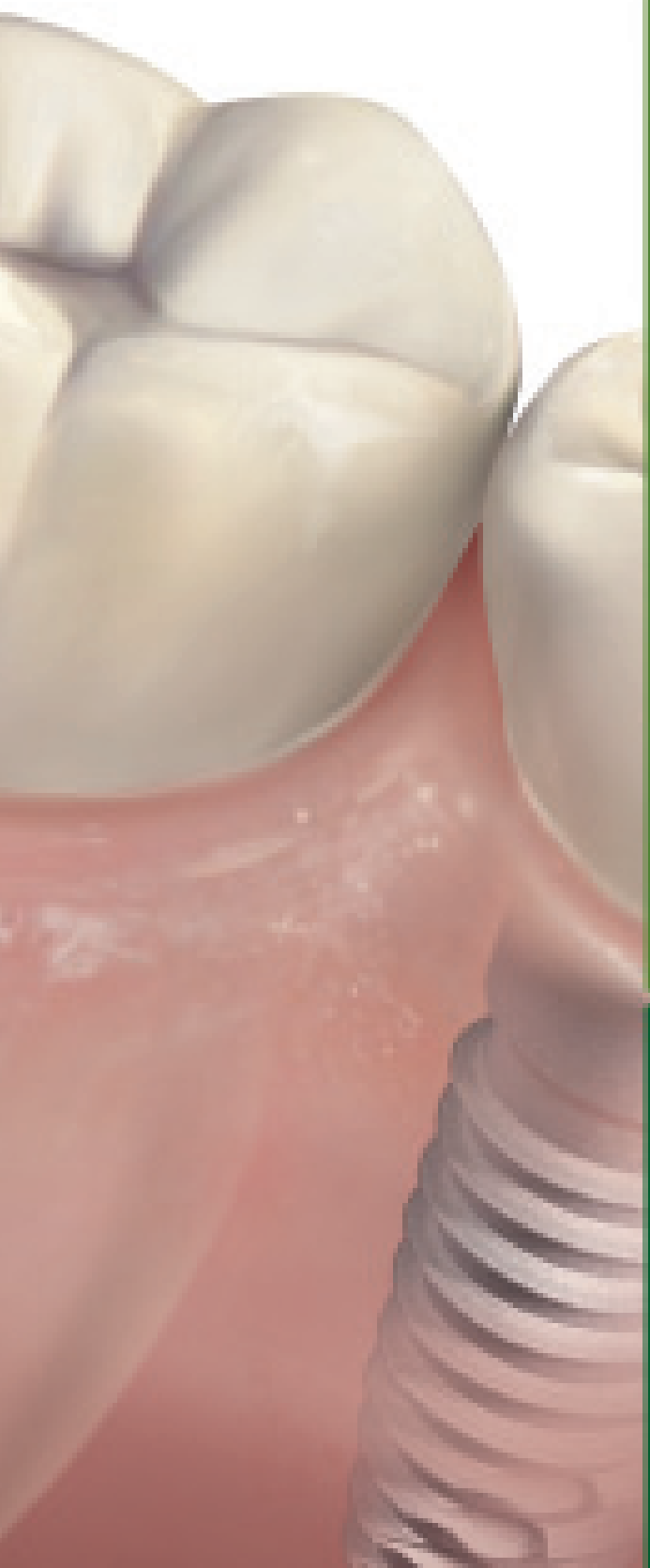
PART 2

Classification and Epidemiology of Periodontal Diseases

Michael G. Newman



Periodontal disease classifications are useful to help establish diagnosis, determine prognosis, and facilitate treatment planning. Different classifications of periodontal diseases have been used over the years and have been replaced as new knowledge has improved our understanding of the etiology and pathology of the diseases of the periodontium, as discussed in Chapter 4.

Also, as covered in Chapter 5, Part 2 includes information about the epidemiology of periodontal diseases that will help students and clinicians analyze a disease and base their diagnostic and therapeutic decisions on its prevalence, incidence, and distribution in large populations or groups. Knowledge of the epidemiology of a disease improves our understanding and sharpens our decisions in individual cases.

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CHAPTER 4

Classfication of Diseases and Conditions Affecting the Periodontium

James E. Hinrichs and M. John Novak

CHAPTER OUTLINE

GINGIVAL DISEASES
- Dental Plaque–Induced Gingival Diseases
- Non–Plaque-Induced Gingival Lesions

PERIODONTITIS
- Chronic Periodontitis
- Aggressive Periodontitis
- Periodontitis as a Manifestation of Systemic Diseases

NECROTIZING PERIODONTAL DISEASES
- Necrotizing Ulcerative Gingivitis
- Necrotizing Ulcerative Periodontitis

ABSCESSES OF THE PERIODONTIUM

PERIODONTITIS ASSOCIATED WITH ENDODONTIC LESIONS
- Endodontic–Periodontal Lesions
- Periodontal–Endodontic Lesions
- Combined Lesions

DEVELOPMENTAL OR ACQUIRED DEFORMITIES AND CONDITIONS
- Localized Tooth-Related Factors That Modify or Predispose to Plaque-Induced Gingival Diseases or Periodontitis
- Mucogingival Deformities and Conditions around Teeth
- Mucogingival Deformities and Conditions of Edentulous Ridges
- Occlusal Trauma

Our understanding of the etiology and pathogenesis of oral diseases and conditions is continually changing with increased scientific knowledge. In light of this, a classification can be most consistently defined by the differences in the clinical manifestations of diseases and conditions because they are clinically consistent and require little, if any, clarification by scientific laboratory testing. The classification presented in this chapter is based on the most recent, internationally accepted, consensus opinion of the diseases and conditions affecting the tissues of the periodontium and was presented and discussed at the 1999 International Workshop for the Classification of the Periodontal Diseases organized by the American Academy of Periodontology (AAP).[3] Box 4-1 presents the overall classification system, and each of the diseases or conditions are discussed where clarification is needed. In each case, the reader is referred to pertinent reviews on the subject and specific chapters within this book that discuss the topics in more detail.

GINGIVAL DISEASES

Dental Plaque–Induced Gingival Diseases

Gingivitis that is associated with dental plaque formation is the most common form of gingival disease (Figure 4-1). Box 4-2 outlines the classifications of gingival diseases. The epidemiology of gingival disease is reviewed in Chapter 5, its etiology is detailed in Chapters 21 through 28, and clinical characteristics are discussed in Chapters 8 through 18 and elsewhere in this textbook.[9,13,14,24,25] Gingivitis has been previously characterized by the presence of clinical signs of inflammation that are confined to the gingiva and associated with teeth showing no attachment loss. Gingivitis also has been observed to affect the gingiva of periodontitis-affected teeth that have previously lost attachment but have received periodontal therapy to stabilize any further attachment loss. In these treated cases, plaque-induced gingival

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BOX 4-1 Classification of Periodontal Diseases and Conditions

Gingival Diseases
Plaque-induced gingival diseases*
Non–plaque-induced gingival lesions

Chronic Periodontitis†
Localized
Generalized

Aggressive Periodontitis
Localized
Generalized

Periodontitis as a Manifestation of Systemic Diseases
Necrotizing Periodontal Diseases
Necrotizing ulcerative gingivitis (NUG)
Necrotizing ulcerative periodontitis (NUP)

Abscesses of the Periodontium
Gingival abscess
Periodontal abscess
Pericoronal abscess

Periodontitis Associated with Endodontic Lesions
Endodontic–periodontal lesion
Periodontal–endodontic lesion
Combined lesion

Developmental or Acquired Deformities and Conditions
Localized tooth-related factors that predispose to plaque-induced gingival diseases or periodontitis
Mucogingival deformities and conditions around teeth
Mucogingival deformities and conditions on edentulous ridges
Occlusal trauma

* These diseases may occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is stable and not progressing.
† Chronic periodontitis can be further classified based on extent and severity. As a general guide, extent can be characterized as localized (<30% of sites involved) or generalized (>30% of sites involved). Severity can be characterized based on the amount of clinical attachment loss (CAL) as follows: *slight* = 1 or 2 mm CAL; *moderate* = 3 or 4 mm CAL; and *severe* ≥5 mm CAL. Data from Armitage GC: *Ann Periodontol* 4:1, 1999.

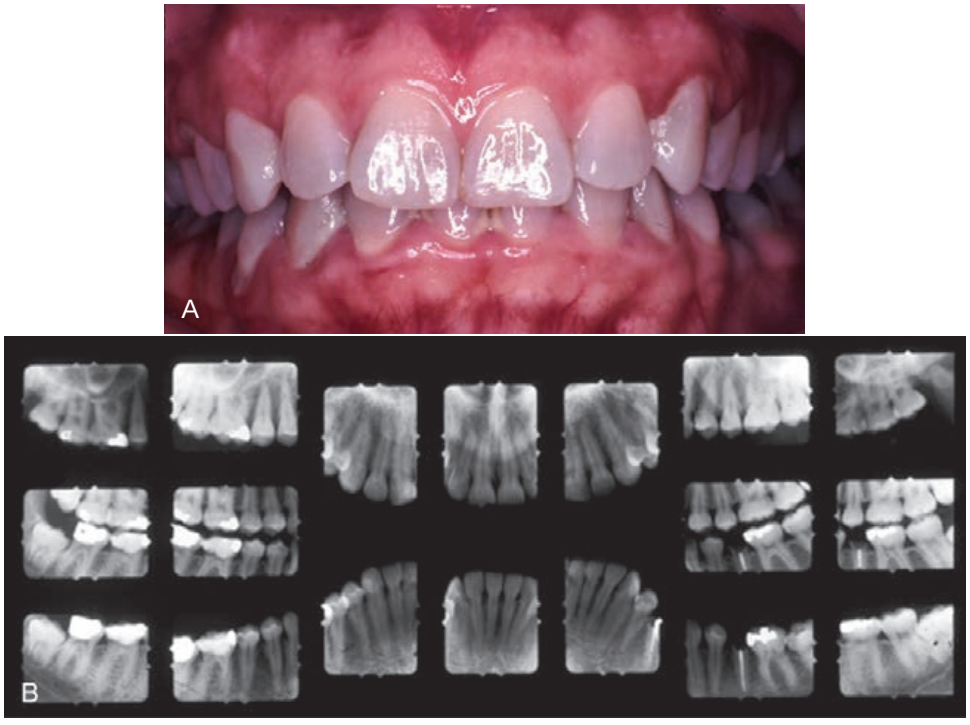

Figure 4-1 **A,** Plaque-related gingivitis depicts marginal and papillary inflammation, with 1 to 4 mm probing depths and generalized zero clinical attachment loss, except recession in tooth #28. **B,** Radiographic images of the patient.

inflammation may recur but without any evidence of further attachment loss.

From this evidence, it has been concluded that plaque-induced gingivitis may occur on a periodontium with no attachment loss or on a periodontium with previous attachment loss that is stable and not progressing. This implies that gingivitis may be the diagnosis for inflamed gingival tissues associated with a tooth with no previous attachment loss or with a tooth that has previously undergone attachment and bone loss (reduced periodontal support) but is not currently losing attachment or bone, even though gingival inflammation is present (Figure 4-2). For this diagnosis to be made, longitudinal records of periodontal status, including clinical attachment levels, should be available.

Gingivitis Associated with Dental Plaque Only. Plaque-induced gingival disease is the result of an interaction between the microorganisms found in the dental plaque biofilm and the tissues and inflammatory cells of the host. The plaque-host

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BOX 4-2 Gingival Diseases

Dental Plaque–Induced Gingival Diseases

These diseases may occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is stable and not progressing.

I. Gingivitis associated with dental plaque only
   A. Without local contributing factors
   B. With local contributing factors (see Box 4-4)
II. Gingival diseases modified by systemic factors
   A. Associated with endocrine system
      1. Puberty-associated gingivitis
      2. Menstrual cycle–associated gingivitis
      3. Pregnancy associated
         a. Gingivitis
         b. Pyogenic granuloma
      4. Diabetes mellitus–associated gingivitis
   B. Associated with blood dyscrasias
      1. Leukemia-associated gingivitis
      2. Other
III. Gingival diseases modified by medications
   A. Drug-influenced gingival diseases
      1. Drug-influenced gingival enlargements
      2. Drug-influenced gingivitis
         a. Oral contraceptive–associated gingivitis
         b. Other
IV. Gingival diseases modified by malnutrition
   A. Ascorbic acid deficiency gingivitis
   B. Other

Non–Plaque-Induced Gingival Lesions

I. Gingival diseases of specific bacterial origin
   A. *Neisseria gonorrhoeae*
   B. *Treponema pallidum*
   C. *Streptococcus* species
   D. Other
II. Gingival diseases of viral origin
   A. Herpesvirus infections
      1. Primary herpetic gingivostomatitis
      2. Recurrent oral herpes
      3. Varicella zoster
   B. Other
III. Gingival diseases of fungal origin
   A. *Candida* species infections: generalized gingival candidiasis
   B. Linear gingival erythema
   C. Histoplasmosis
   D. Other
IV. Gingival lesions of genetic origin
   A. Hereditary gingival fibromatosis
   B. Other
V. Gingival manifestations of systemic conditions
   A. Mucocutaneous lesions
      1. Lichen planus
      2. Pemphigoid
      3. Pemphigus vulgaris
      4. Erythema multiforme
      5. Lupus erythematosus
      6. Drug induced
      7. Other
   B. Allergic reactions
      1. Dental restorative materials
         a. Mercury
         b. Nickel
         c. Acrylic
         d. Other
      2. Reactions attributable to:
         a. Toothpastes or dentifrices
         b. Mouth rinses or mouthwashes
         c. Chewing gum additives
         d. Foods and additives
      3. Other
VI. Traumatic lesions (factitious, iatrogenic, or accidental)
   A. Chemical injury
   B. Physical injury
   C. Thermal injury
VII. Foreign body reactions
VIII. Not otherwise specified

Data from Holmstrup P: *Ann Periodontol* 4:20, 1999; and Mariotti A: *Ann Periodontol* 4:7, 1999.

SCIENCE TRANSFER

Although plaque-induced gingivitis and periodontitis are the *most common diseases* of the periodontium, there are many other pathologic processes that are manifested in periodontal tissues. Clinicians need to be cognizant that tissues of the oral cavity can be subjected to a wide range of locally specific diseases and deformities, as well as being an indicator of systemic pathologic entities. Some diseases of the periodontium produce specific diagnostic changes, and others modify the response to the dental bacterial biofilms. These complexities require dentists to develop a broad knowledge and understanding of a wide range of dental and systemic diseases so that their diagnostic skills can give significant benefit to their patients.

The most widely accepted classification of periodontal diseases need to be routinely modified as new diagnostic modalities are developed. In the future there will be an increased utilization of genetic and biotech markets for more accurate and objective diagnoses of periodontal pathology. One group of diseases that are underrepresented in many classifications are the benign and malignant tumors that affect periodontal tissues.

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interaction can be altered by the effects of local factors, systemic factors, medications, and malnutrition, all of which can influence the severity and duration of the response. Local factors that may contribute to gingivitis, in addition to plaque-retentive calculus formation on crown and root surfaces (see Chapter 22). These factors are contributory because of their ability to retain plaque microorganisms and inhibit their removal by patient-initiated plaque control techniques.

Gingival Diseases Modified by Systemic Factors. Systemic factors contributing to gingivitis, such as the endocrine changes associated with puberty (Figure 4-3), the menstrual cycle, pregnancy (Figure 4-4, *A*), and diabetes, may be exacerbated because of alterations in the gingival inflammatory response to plaque.[13,14,24] This altered response appears to result from the effects of systemic conditions on the host's cellular and immunologic functions. These changes are most apparent during pregnancy, when the prevalence and severity of gingival inflammation may increase, even in the presence of low levels of plaque. Blood dyscrasias, such as leukemia, may alter immune function by disturbing the normal balance of immunologically competent white blood cells supplying the periodontium. Gingival enlargement and bleeding are common findings and may be associated with swollen, spongy gingival tissues caused by excessive infiltration of blood cells (Figure 4-5).

Gingival Diseases Modified by Medications. Gingival diseases modified by medications are increasingly prevalent because of the increased use of drugs known to induce gingival enlargement (e.g., anticonvulsant drugs such as phenytoin, immunosuppressive drugs such as cyclosporine (Figure 4-6), and calcium channel blockers such as nifedipine (Figure 4-7), verapamil, diltiazem, and sodium valproate.[9,14,24] The development and severity of gingival enlargement in response to medications are patient specific and may be influenced by uncontrolled plaque accumulation, as well as elevated hormonal levels. The increased use of oral contraceptives by premenopausal women has been associated with a higher incidence of gingival inflammation and development of gingival

Figure 4-2 Maxillary second molar exhibits mild inflammation at mesial-palatal surface. However, the clinical attachment loss has been stable for 15 years after apical positioned flap and periodontal maintenance. Diagnosis is a history of moderate periodontitis, but the case is in remission.

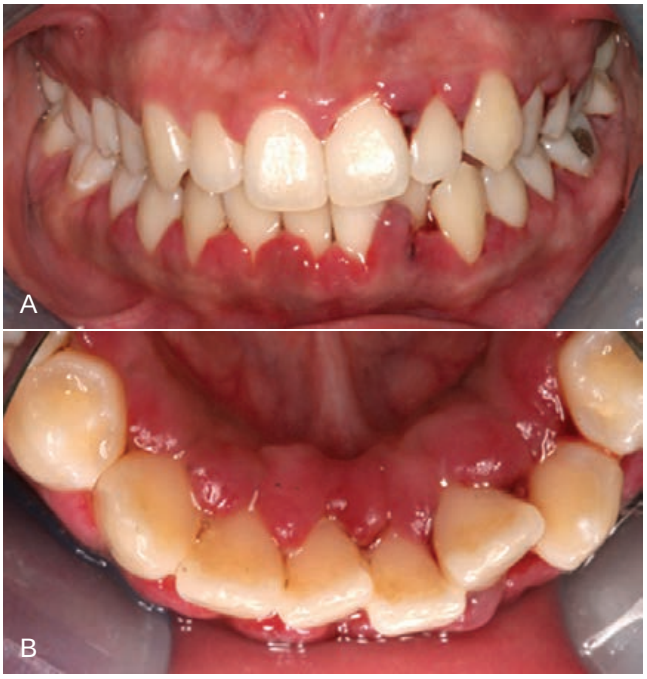

Figure 4-3 Thirteen-year-old female with hormone exaggerated marginal and papillary inflammation, with 1 to 4 mm probing depths yet minimal clinical attachment loss. **A,** Facial view. **B,** Lingual view.

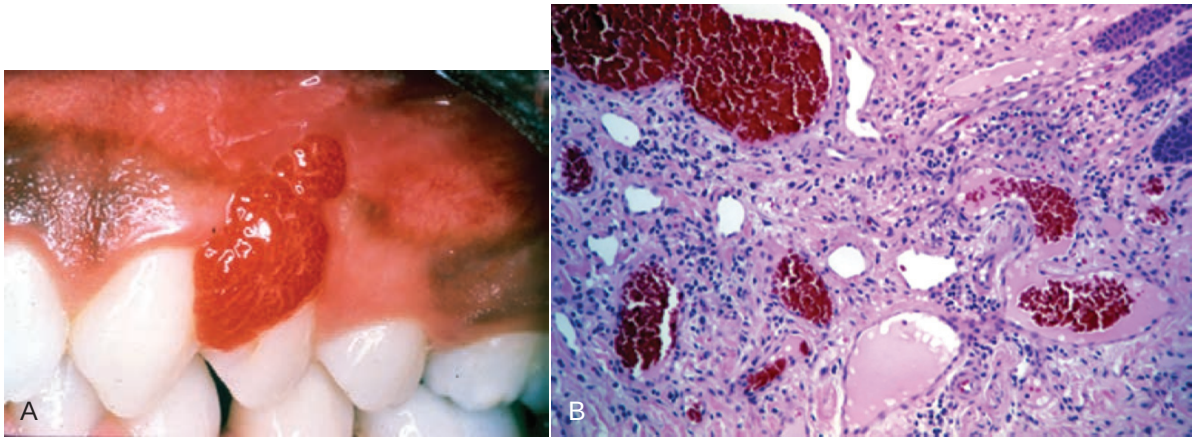

Figure 4-4 **A,** Clinical image of pyogenic granuloma in 27-year-old pregnant female. **B,** Histologic image depicts dense inflammatory infiltrate and prominent vessels.

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enlargement, which may be reversed by discontinuation of the oral contraceptive.

Gingival Diseases Modified by Malnutrition. Gingival diseases modified by malnutrition have received attention because of clinical descriptions of bright red, swollen, and bleeding gingiva associated with severe ascorbic acid (vitamin C) deficiency or scurvy.[14] Nutritional deficiencies are known to affect immune function and may alter the host's ability to protect itself against some of the detrimental effects of cellular products such as oxygen radicals. Unfortunately, little scientific evidence is available to support a role for specific nutritional deficiencies in the development or severity of gingival inflammation or periodontitis in humans.

Figure 4-5 Twelve-year-old female with a primary medical diagnosis of leukemia that exhibits swollen/spongy gingiva.

Non–Plaque-Induced Gingival Lesions

Oral manifestations of systemic conditions that produce lesions in the tissues of the periodontium are rare. These effects are observed more frequently in lower socioeconomic groups, developing countries, and immunocompromised individuals.[10] Benign mucous membrane pemphigoid is an example of a non–plaque-induced lesion without socioeconomic implications. Sloughing gingival tissues leave painful ulcerations of the gingiva (Figure 4-8, *A* and *B*). Autoimmune antibodies are targeted at the basement membrane and cleave it from the underlying connective tissue. (Figure 4-8, *C* and *D*).

Gingival Diseases of Specific Bacterial Origin. Gingival diseases of specific bacterial origin are increasing in prevalence, especially as a result of sexually transmitted diseases, such as gonorrhea *(Neisseria gonorrhoeae),* and to a lesser degree, syphilis *(Treponema pallidum).*[26,29] Oral lesions may be secondary to systemic infection or may occur through direct infection. Streptococcal gingivitis or gingivostomatitis is a rare entity that may

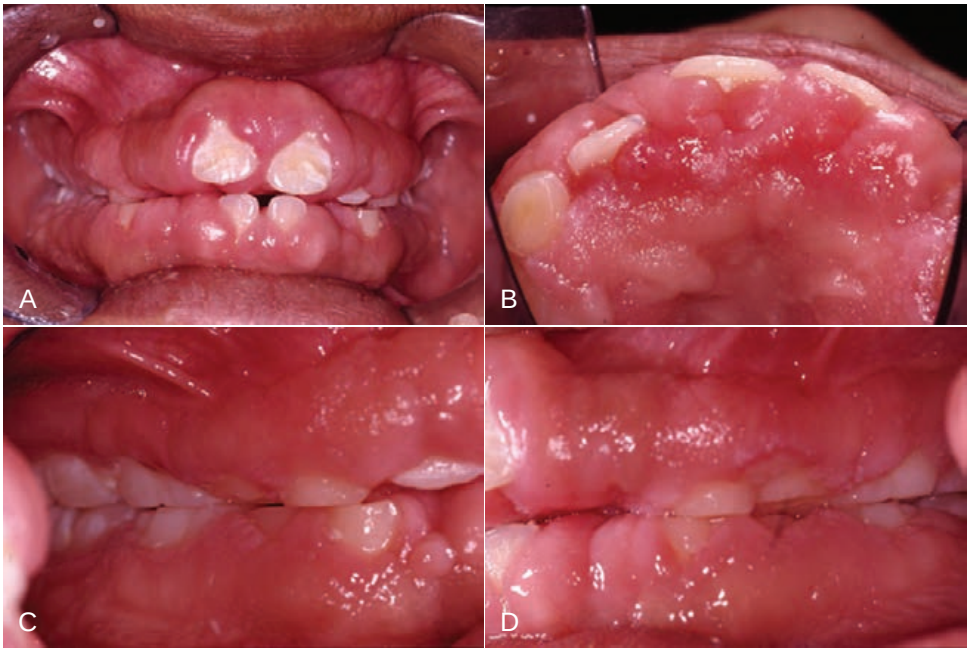

Figure 4-6 Clinical images of 9-year-old male with severe gingival overgrowth secondary to heart transplant and cyclosporine therapy.

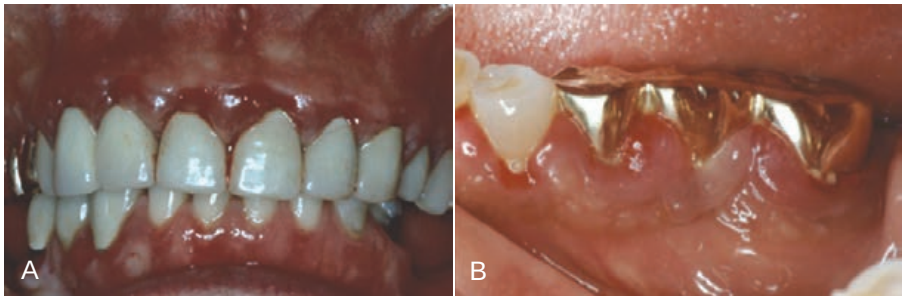

Figure 4-7 Clinical images of gingival overgrowth following use a of calcium channel blocker to control hypertension.

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present as an acute condition with fever, malaise, and pain associated with acutely inflamed, diffuse, red, and swollen gingiva with increased bleeding and occasional gingival abscess formation. The gingival infections usually are preceded by tonsillitis and have been associated with group A β-hemolytic streptococcal infections.

Gingival Diseases of Viral Origin. Gingival diseases of viral origin may be caused by a variety of deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) viruses, the most common being the herpesviruses (Figure 4-9, *A* and *B*). Lesions are frequently related to reactivation of latent viruses, especially as a result of reduced immune function. The oral manifestations of viral infection have been comprehensively reviewed.[10,28] Viral gingival diseases are treated with topical and/or systemic antiviral drugs (Figure 4-9, *C* and *D*).

Gingival Diseases of Fungal Origin. Gingival diseases of fungal origin are relatively uncommon in immunocompetent individuals but occur more frequently in immunocompromised individuals and those with normal oral flora disturbed by the long-term use of broad-spectrum antibiotics.[10,29,30] The most common oral fungal infection is candidiasis, caused by infection with *Candida albicans,* which also can be seen under prosthetic devices, in individuals using topical steroids, and in individuals with decreased salivary flow, increased salivary glucose, or decreased salivary pH. A generalized candidal infection may manifest as white patches on the gingiva, tongue, or oral mucous membrane that can be removed with gauze, leaving a red, bleeding surface. In individuals infected with human immunodeficiency virus (HIV), candidal infection may present as erythema of the attached gingiva and has been referred to as *linear gingival erythema or HIV-associated gingivitis* (see Chapter 19).

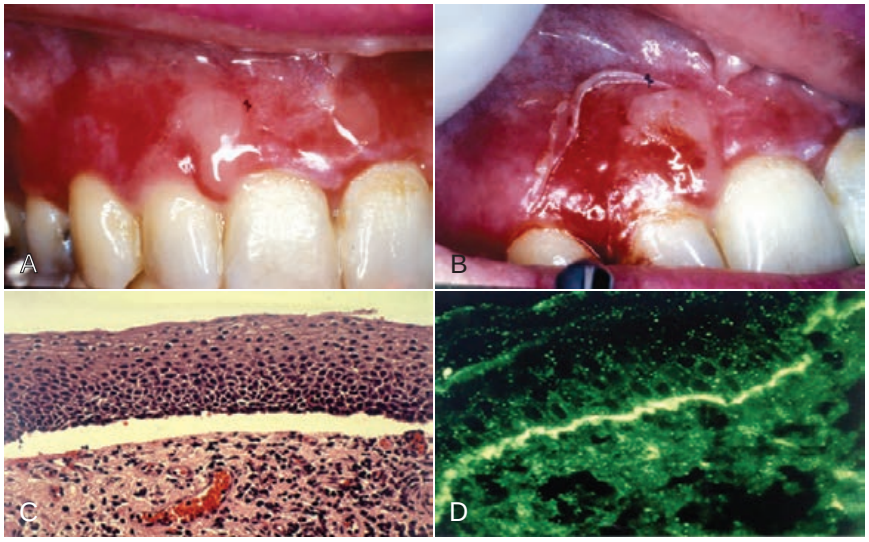

Figure 4-8 Sixty-two year-old female with benign mucous membrane pemphigoid. **A** and **B,** Clinical image with sloughing epithelial surface. **C,** Hematoxylin and eosin (H&E) stain depicting separation of epithelium from connective tissue. **D,** Immunofluorescent-labeled antibodies to basement membrane.

Figure 4-9 **A** and **B,** Twenty-nine year-old male with primary herpetic infection and severe gingival inflammation. **C** and **D,** Six weeks postsystemic acyclovir.

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Diagnosis of candidal infection can be made by culture, smear, and biopsy. Less common fungal infections have also been described.[29,30]

Gingival Diseases of Genetic Origin. Gingival diseases of genetic origin may involve the tissues of the periodontium and have been described in detail.[1] One of the most clinically evident conditions is *hereditary gingival fibromatosis,* which exhibits autosomal dominant or (rarely) autosomal recessive modes of inheritance. The gingival enlargement may completely cover the teeth, delay eruption, and present as an isolated finding or may be associated with several more generalized syndromes.

Gingival Manifestations of Systemic Conditions. Gingival manifestations of systemic conditions may appear as desquamative lesions, ulceration of the gingiva, or both.[10,23,29] Allergic reactions that manifest with gingival changes are uncommon but have been observed in association with several restorative materials (Figure 4-10, *A*), toothpastes, mouthwashes, chewing gum (Figure 4-11), and foods (see Box 4-2). The diagnosis of these conditions may prove difficult and may require an extensive history and selective elimination of potential causes. Histologic traits of biopsies from gingival allergic reactions include dense infiltrate of eosinophilic cells (Figure 4-10, *B* and *C*).

Traumatic Lesions. Traumatic lesions may be self-inflicted and *factitious* in origin, produced by intentional or unintentional artificial means (Figure 4-12). Other examples of traumatic lesions include toothbrush trauma resulting in gingival ulceration, recession, or both. *Iatrogenic trauma* (induced by the dentist or health

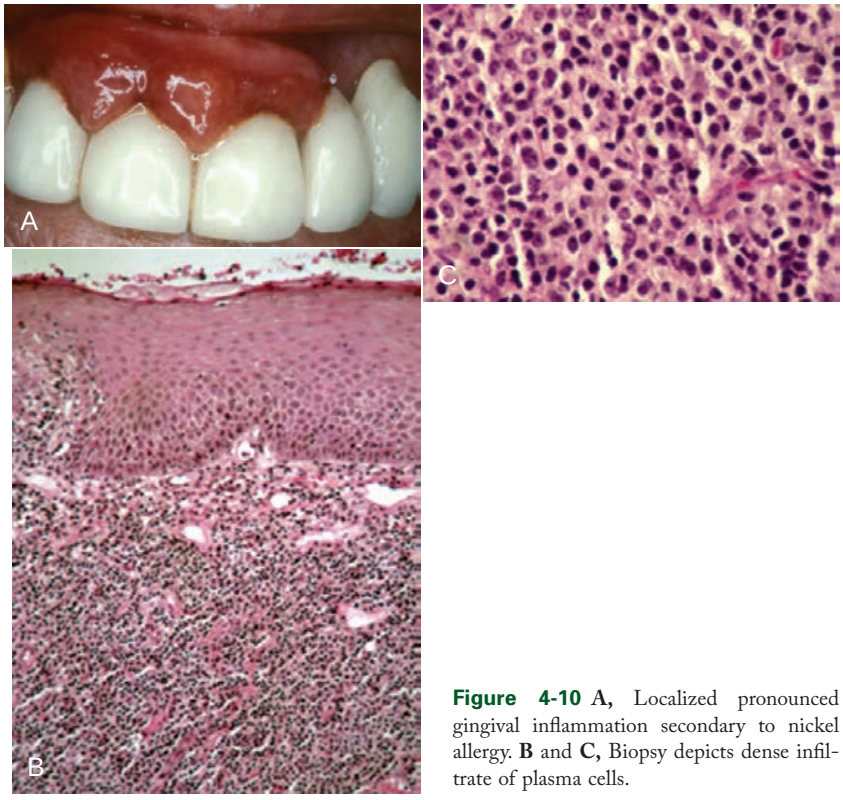

Figure 4-10 A, Localized pronounced gingival inflammation secondary to nickel allergy. **B** and **C,** Biopsy depicts dense infiltrate of plasma cells.

Figure 4-11 Generalized severe allergic response of gingiva as a result of additive in chewing gum.

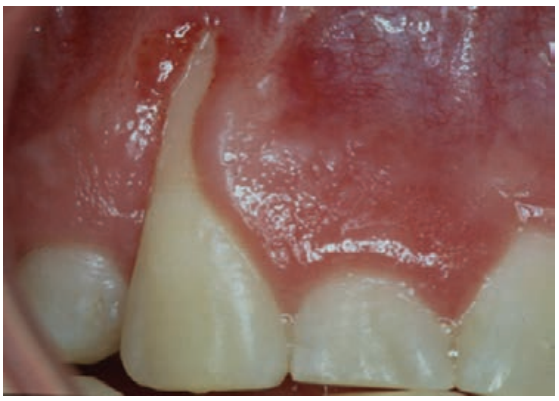

Figure 4-12 Self-inflicted gingival dehiscence induced via patient's fingernail.

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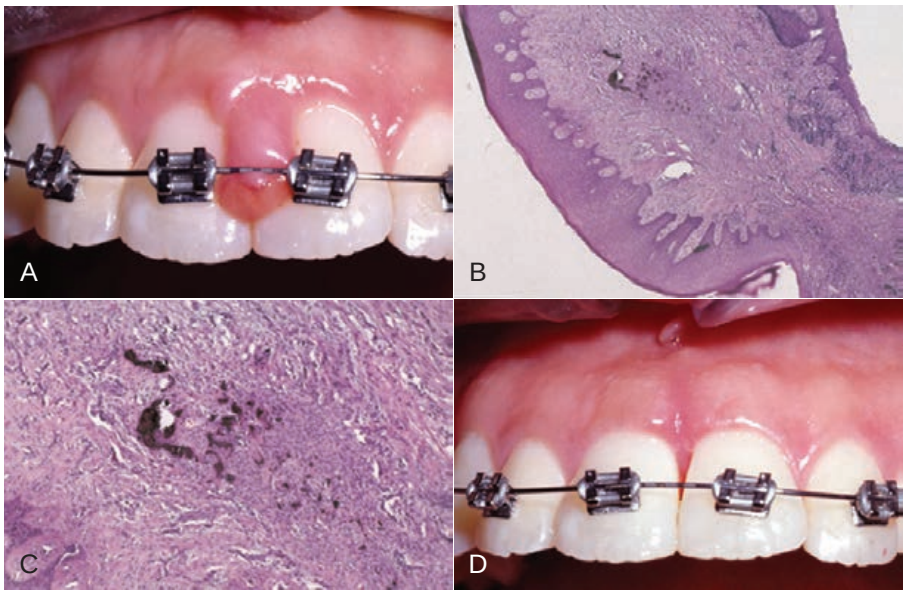

Figure 4-13 **A,** Proliferative gingival overgrowth secondary to impaction of foreign body. **B,** Histology of peripheral ossifying fibroma. **C,** Higher magnification of image in **B**. **D,** Four weeks postexcisional biopsy.

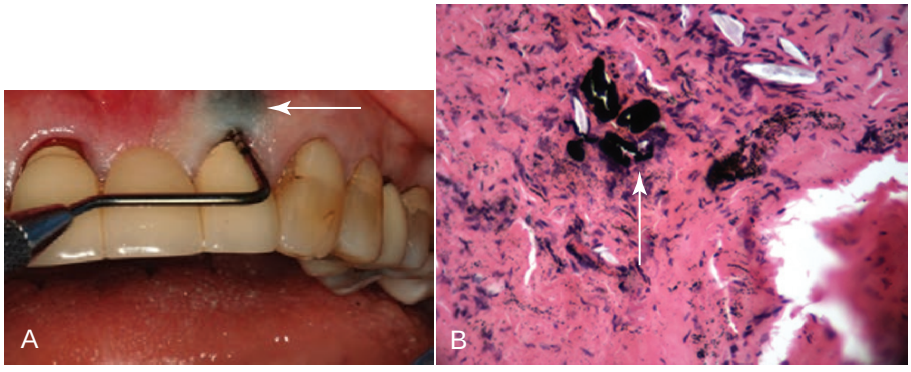

Figure 4-14 **A,** Gingival pigmentation associated with previous apicoectomy and amalgam retro-fill. **B,** Biopsy depicts metal fragments.

professional) to the gingiva can be caused by induction of orthodontic cement or preventive or restorative materials (Figure 4-13, *A*). Peripheral ossifying fibroma may develop in response to embedment of a foreign body (Figure 4-13, *B* and *C*). Alternatively, *accidental damage* to the gingiva may occur through minor burns from hot foods and drinks.[10]

Foreign Body Reactions. Foreign body reactions lead to localized inflammatory conditions of the gingiva and are caused by the introduction of foreign material into the gingival connective tissues through breaks in the epithelium.[10] Common examples are the introduction of amalgam into the gingiva during the placement of a restoration, extraction of a tooth, or endodontic apicoectomy with retrofill leaving an amalgam tattoo (Figure 4-14, *A*) with resultant metal fragments observed in biopsies (Figure 4-14, *B*), or the introduction of abrasives during polishing procedures.

PERIODONTITIS

Periodontitis is defined as "an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with increased probing depth formation, recession, or both." The clinical feature that distinguishes periodontitis from gingivitis is the presence of clinically detectable attachment loss. This loss often is accompanied by periodontal pocket formation and changes in the density and height of subjacent alveolar bone. In some cases, recession of the marginal gingiva may accompany attachment loss, thus masking ongoing disease progression if only probing depth measurements are taken without measurements of clinical attachment levels. Clinical signs of inflammation, such as changes in color, contour, and consistency, as well as bleeding on probing, may not always be positive indicators of ongoing attachment loss. However, the presence of continued bleeding on probing at sequential visits has proved to be a reliable indicator of the presence of inflammation and the potential for subsequent attachment loss at the bleeding site. The attachment loss associated with periodontitis has been shown to progress either continuously or in episodic bursts of disease activity.

Although many classifications of the different clinical manifestations of periodontitis have been presented over the past 20 years, consensus workshops in North America in 1989[6] and Europe in 1993[4] identified that periodontitis may present in early-onset, adult-onset, and necrotizing forms (Table 4-1). In addition, the AAP consensus concluded that periodontitis may be associated

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TABLE 4-1 Classification of the Various Forms of Periodontitis

Classification	Forms of Periodontitis	Disease Characteristics
AAP World Workshop in Clinical Periodontics, 1989[5]	Adult periodontitis	Age of onset >35 years Slow rate of disease progression No defects in host defenses
	Early-onset periodontitis (may be prepubertal, juvenile, or rapidly progressive)	Age of onset <35 years Rapid rate of disease progression Defects in host defenses Associated with specific microflora
	Periodontitis associated with systemic disease	Systemic diseases that predispose to rapid rates of periodontitis Diseases: diabetes, Down syndrome, HIV infection, Papillon-Lefèvre syndrome
	Necrotizing ulcerative periodontitis	Similar to acute necrotizing ulcerative gingivitis but with associated clinical attachment loss
	Refractory periodontitis	Recurrent periodontitis that does not respond to treatment
European Workshop in Periodontology, 1993[3]	Adult periodontitis	Age of onset: fourth decade of life Slow rate of disease progression No defects in host response
	Early-onset periodontitis	Age of onset: before fourth decade of life Rapid rate of disease progression Defects in host defense
	Necrotizing periodontitis	Tissue necrosis with attachment and bone loss
AAP International Workshop for Classification of Periodontal Diseases, 1999[2]	Chronic periodontitis Aggressive periodontitis Periodontitis as a manifestation of systemic diseases	See Box 4-3

AAP, American Academy of Periodontology; *HIV,* human immunodeficiency virus.

with systemic conditions, such as diabetes and HIV infection, and that some forms of periodontitis may be refractory to conventional therapy. Early-onset disease was distinguished from adult-onset disease by the age of onset (35 years of age was set as an arbitrary separation of diseases), the rate of disease progression, and the presence of alterations in host defenses. The early-onset diseases were more aggressive, occurred in individuals younger than 35 years of age, and were associated with defects in host defenses, whereas adult forms of disease were slowly progressive, became clinically more evident in the fourth decade of life, and were not associated with defects in host defenses. In addition, early-onset periodontitis was subclassified into prepubertal, juvenile, and rapidly progressive forms with localized or generalized disease distributions.

Extensive clinical and basic scientific research of these disease entities has been performed in many countries, and some disease characteristics outlined 10 years ago no longer stand up to rigid scientific scrutiny.[7,13,31] In particular, supporting evidence was lacking for the distinct classifications of adult periodontitis, refractory periodontitis, and the various different forms of early-onset periodontitis as outlined by the AAP Workshop for the International Classification of Periodontal Diseases in 1999[3] (see Table 4-1).

It has been observed that chronic periodontal destruction, caused by the accumulation of local factors, such as plaque and calculus, can occur before age 35 years and that the aggressive disease seen in young patients may be independent of age but has a familial (genetic) association. With respect to refractory periodontitis, little evidence supports that this is indeed a distinct clinical entity because the causes of continued loss of clinical attachment and alveolar bone after periodontal therapy are currently poorly defined and apply to many disease entities. In addition, the clinical and etiologic manifestations of the different diseases outlined in North America in 1989 and in Europe in 1993 were not consistently observed in different countries around the world and did not always fit the models presented. As a result, the AAP held an International Workshop for the Classification of Periodontal Diseases in 1999 to further refine the classification system based on current clinical and scientific data.[3] The resulting classification of the different forms of periodontitis was simplified to describe three general clinical manifestations of periodontitis: chronic periodontitis, aggressive periodontitis, and periodontitis as a manifestation of systemic diseases (see Table 4-1 and Box 4-3).

Chronic Periodontitis

Chronic periodontitis is the most common form of periodontitis[7]; Box 4-3 includes the characteristics of this form of periodontitis. Chronic periodontitis is most prevalent in adults but can be observed in children; therefore the age range of older than 35 years previously designated for the classification of this disease has been discarded. Chronic periodontitis is associated with the accumulation of plaque and calculus and generally has a slow-to-moderate rate of disease progression, but periods of more rapid destruction may be observed. Increases in the rate of disease progression may be caused by the impact of local, systemic, or environmental factors that may influence the normal host-bacteria interaction. Local factors may influence plaque accumulation (Box 4-4); systemic diseases, such as diabetes mellitus and HIV infection, may influence

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BOX 4-3 Periodontitis

The disease periodontitis can be subclassified into the following three major types based on clinical, radiographic, historical, and laboratory characteristics.

Chronic Periodontitis

The following characteristics are common to patients with chronic periodontitis:

- Prevalent in adults but can occur in children.
- Amount of destruction consistent with local factors.
- Associated with a variable microbial pattern.
- Subgingival calculus frequently found.
- Slow-to-moderate rate of progression with possible periods of rapid progression.
- Possibly modified by or associated with the following:
  - Systemic diseases such as diabetes mellitus and human immunodeficiency virus (HIV) infection.
  - Local factors predisposing to periodontitis.
  - Environmental factors such as cigarette smoking and emotional stress.

Chronic periodontitis may be further subclassified into localized and generalized forms and characterized as slight, moderate, or severe based on the common features described above and the following specific features:

- Localized form: <30% of sites involved.
- Generalized form: >30% of sites involved.
- Slight: 1 to 2 mm clinical attachment loss (CAL).
- Moderate: 3 to 4 mm CAL.
- Severe: ≥5 mm CAL.

Aggressive Periodontitis

The following characteristics are common to patients with aggressive periodontitis:

- Otherwise clinically healthy patient.
- Rapid attachment loss and bone destruction.
- Amount of microbial deposits inconsistent with disease severity.
- Familial aggregation of diseased individuals.

The following characteristics are common but not universal:

- Diseased sites infected with *Actinobacillus actinomycetemcomitans*.
- Abnormalities in phagocyte function.
- Hyperresponsive macrophages, producing increased prostaglandin E_2 (PGE_2) and interleukin-1β (IL-1β).
- In some cases, self-arresting disease progression.

Aggressive periodontitis may be further classified into localized and generalized forms based on the common features described here and the following specific features:

Localized form

- Circumpubertal onset of disease.
- Localized first molar or incisor disease with proximal attachment loss on at least two permanent teeth, one of which is a first molar.
- Robust serum antibody response to infecting agents.

Generalized form

- Usually affecting persons under 30 years of age (however, may be older).
- Generalized proximal attachment loss affecting at least three teeth other than first molars and incisors.
- Pronounced episodic nature of periodontal destruction.
- Poor serum antibody response to infecting agents.

Periodontitis as a Manifestation of Systemic Diseases

Periodontitis may be observed as a manifestation of the following systemic diseases:

1. Hematologic disorders
   a. Acquired neutropenia
   b. Leukemias
   c. Other
2. Genetic disorders
   a. Familial and cyclic neutropenia
   b. Down syndrome
   c. Leukocyte adhesion deficiency syndromes
   d. Papillon-Lefèvre syndrome
   e. Chédiak-Higashi syndrome
   f. Histiocytosis syndromes
   g. Glycogen storage disease
   h. Infantile genetic agranulocytosis
   i. Cohen syndrome
   j. Ehlers-Danlos syndrome (types IV and VIII autosomal dominant [AD])
   k. Hypophosphatasia
   l. Other
3. Not otherwise specified

Data from Flemmig TF: *Ann Periodontol* 4:32, 1999; Kinane DF: *Ann Periodontol* 4:54, 1999; and Tonetti MS, Mombelli A: *Ann Periodontol* 4:39, 1999.

the host defenses, and environmental factors, such as cigarette smoking and stress, also may influence the response of the host to plaque accumulation. Chronic periodontitis may occur as a localized disease in which less than 30% of evaluated sites demonstrate attachment and bone loss, or it may occur as a more generalized disease in which greater than 30% of sites are affected. The disease also may be described by the severity of disease as slight: 1 to 2 mm (Figure 4-15), moderate: 3 to 4 mm (Figure 4-16), or severe: ≥5 mm (Figure 4-17) based on the amount of clinical attachment loss (see Box 4-3).

Aggressive Periodontitis

Aggressive periodontitis differs from the chronic form primarily by (1) the rapid rate of disease progression seen in an otherwise healthy individual (Figures 4-18 and 4-19), (2) an absence of large accumulations of plaque and calculus, and (3) a family history of aggressive disease suggestive of a genetic trait[20,31] (see Box 4-3). This form of periodontitis was previously classified as early-onset periodontitis (see Table 4-1) and therefore still includes many of the characteristics previously identified with the localized and generalized forms of early-onset periodontitis. Although the clinical presentation of aggressive disease appears to be universal, the etiologic factors involved are not always consistent. Box 4-3 outlines additional clinical, microbiologic, and immunologic characteristics of aggressive disease that may be present. As was previously described for early-onset disease, aggressive forms of periodontitis usually affect young individuals at or shortly after puberty and may be observed during the second and third decades of life (i.e., 10 to

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BOX 4-4 Developmental or Acquired Deformities and Conditions

Localized Tooth-Related Factors That Modify or Predispose to Plaque-Induced Gingival Diseases or Periodontitis

1. Tooth anatomic factors
2. Dental restorations or appliances
3. Root fractures
4. Cervical root resorption and cemental tears

Mucogingival Deformities and Conditions around Teeth

1. Gingival or soft tissue recession
   a. Facial or lingual surfaces
   b. Interproximal (papillary)
2. Lack of keratinized gingiva
3. Decreased vestibular depth
4. Aberrant frenum or muscle position
5. Gingival excess
   a. Pseudopocket
   b. Inconsistent gingival margin
   c. Excessive gingival display
   d. Gingival enlargement (see Box 4-2)
   e. Abnormal color

Mucogingival Deformities and Conditions on Edentulous Edges

1. Vertical and/or horizontal ridge deficiency
2. Lack of gingiva or keratinized tissue
3. Gingival or soft tissue enlargements
4. Aberrant frenum or muscle position
5. Decreased vestibular depth
6. Abnormal color

Occlusal Trauma

1. Primary occlusal trauma
2. Secondary occlusal trauma

Data from Blieden TM: *Ann Periodontol* 4:91, 1999; Halmon WW: *Ann Periodontol* 4:102, 1999; and Pini Prato GP: *Ann Periodontol* 4:98, 1999.

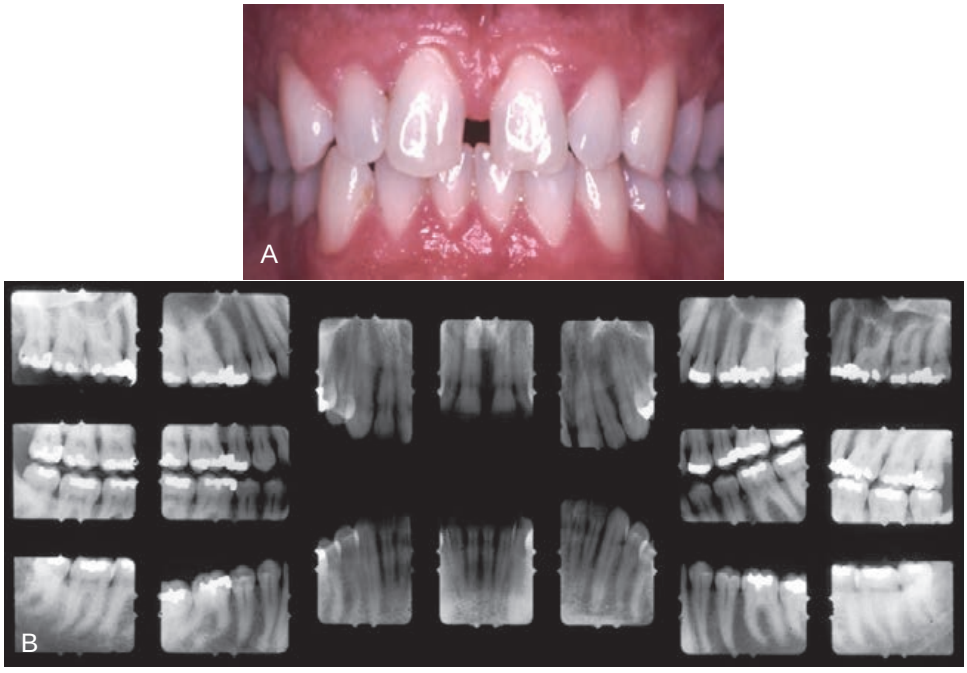

Figure 4-15 **A,** Clinical image of plaque-related slight/early chronic periodontitis with 1 to 2 mm clinical attachment loss in 40-year-old female. **B,** Radiographic images of the patient.

30 years of age). The disease may be localized as previously described for localized juvenile periodontitis (LJP) or generalized as previously described for generalized juvenile periodontitis (GJP) and rapidly progressive periodontitis (RPP) (see Table 4-1). Box 4-3 provides the common features of the localized and generalized forms of aggressive periodontitis.

Periodontitis as a Manifestation of Systemic Diseases

Several hematologic and genetic disorders have been associated with the development of periodontitis in affected individuals[12,13] (see Box 4-3). The majority of these observations of effects on the periodontium are the result of case reports, and few research studies have been performed to investigate the exact nature of the effect of the specific condition on the tissues of the periodontium. It is speculated that the major effect of these disorders is through alterations in host defense mechanisms that have been clearly described for disorders, such as neutropenia and leukocyte adhesion deficiencies, but are less well understood for multifaceted syndromes. The clinical manifestation of many of these disorders appears at an early age and may be confused with aggressive forms of periodontitis with rapid attachment loss and the potential for early tooth loss. With the introduction of this form of periodontitis in this and previous classification systems (see Table 4-1), the potential exists

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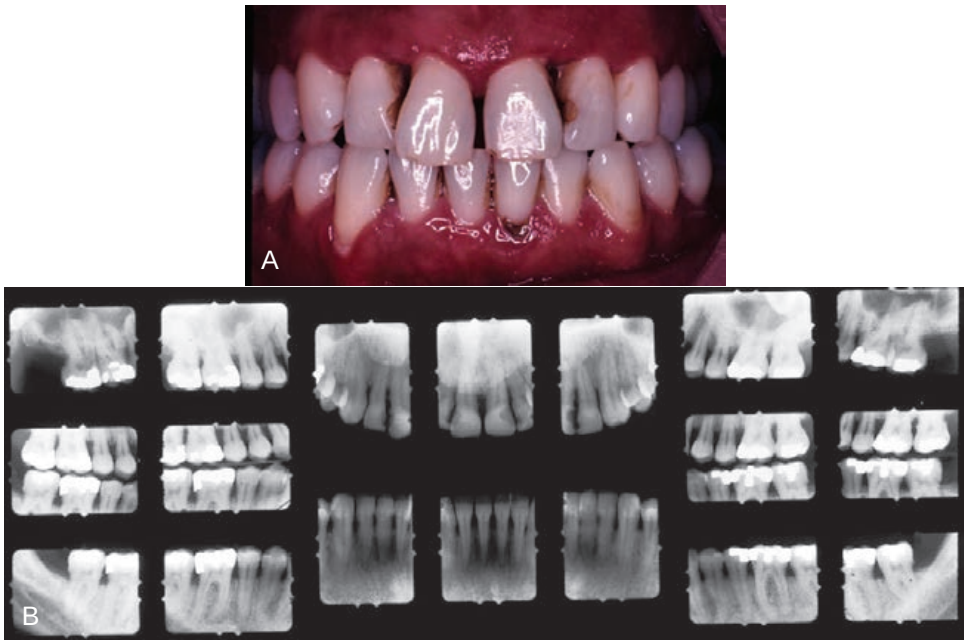

Figure 4-16 **A,** Clinical image of plaque-related moderate chronic periodontitis with 3 to 4 mm clinical attachment loss in 53-year-old male smoker. **B,** Radiographic images of the patient.

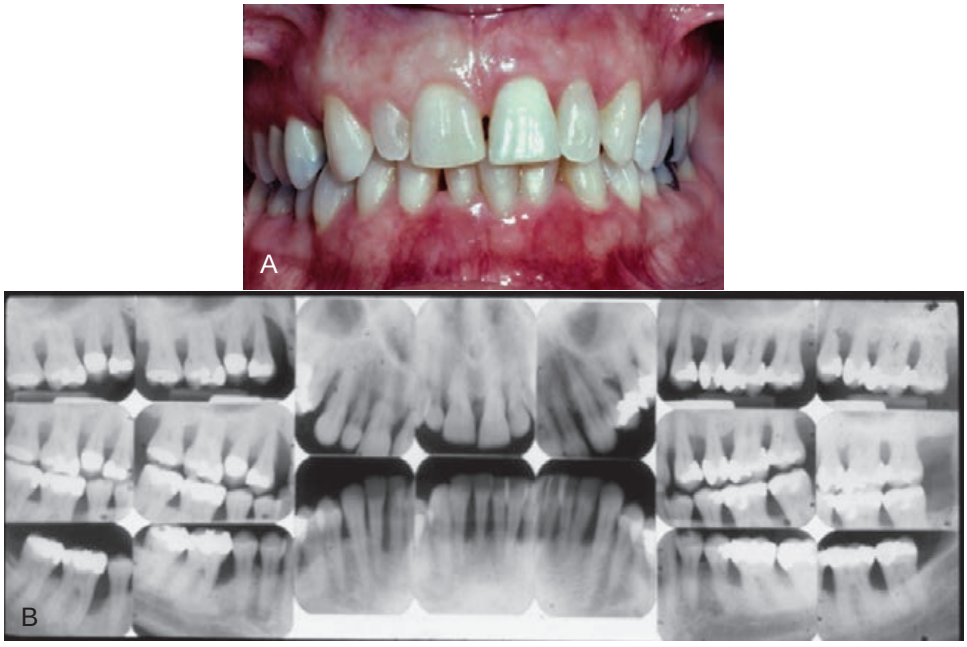

Figure 4-17 **A,** Clinical image of plaque-related severe/advanced chronic periodontitis with ≧5 mm clinical attachment loss in 47-year-old female. **B,** Radiographic images of the patient.

for overlap and confusion between periodontitis as a manifestation of systemic disease and both the aggressive and the chronic form of disease when a systemic component is suspected. At present, *periodontitis as a manifestation of systemic disease* is the diagnosis to be used when the systemic condition is the major predisposing factor and local factors, such as large quantities of plaque and calculus, are not clearly evident. In the case in which periodontal destruction is clearly the result of local factors but has been exacerbated by the onset of such conditions as diabetes mellitus (Figures 4-20 and 4-21) or HIV infection, the diagnosis should be *chronic periodontitis modified by the systemic condition.*

Sarcoidosis is a chronic disease expressed as a cell-mediated delayed-type hypersensitivity that primarily affects the lungs, lymph nodes, skin, eyes, liver, spleen, and small bones of the hands and feet, etc.[21] Sarcoidosis rarely affects the oral cavity, with incidence of occurrence in descending order noted in the lymph nodes,

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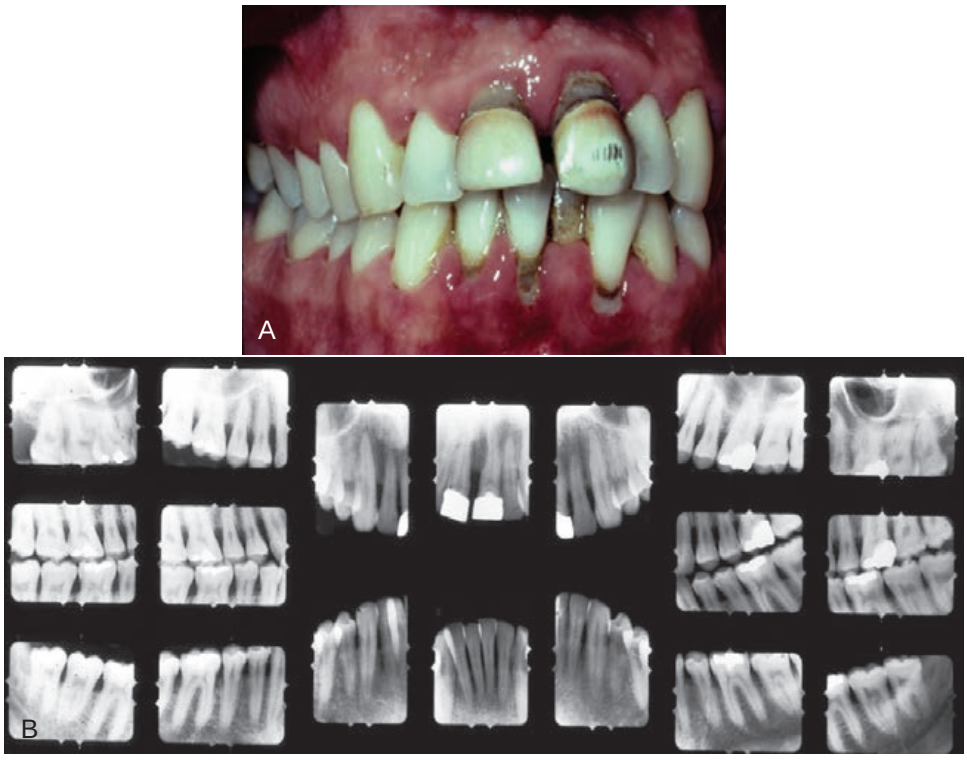

Figure 4-18 **A,** Clinical image of plaque-related aggressive moderate periodontitis with 1 to 7 mm PD and 3 to 4 mm clinical attachment loss in 31-year-old male. **B,** Radiographic images of the patient.

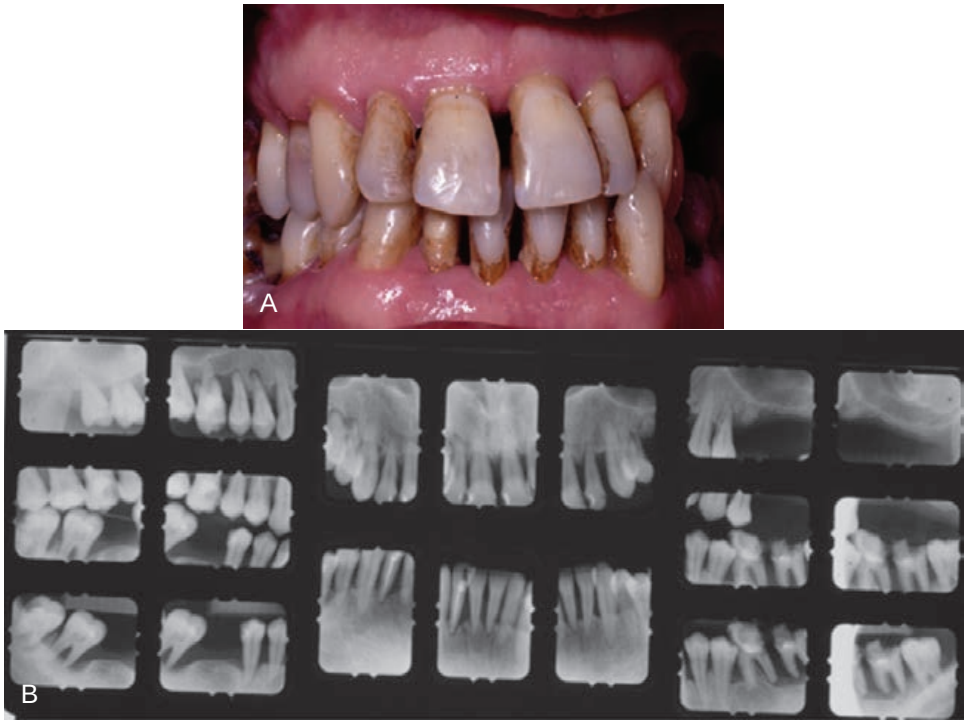

Figure 4-19 **A,** Clinical image of plaque-related aggressive severe periodontitis with 3 to 13 mm PD and 7 to 15 mm clinical attachment loss in 32-year-old male. **B,** Radiographic images of the patient.

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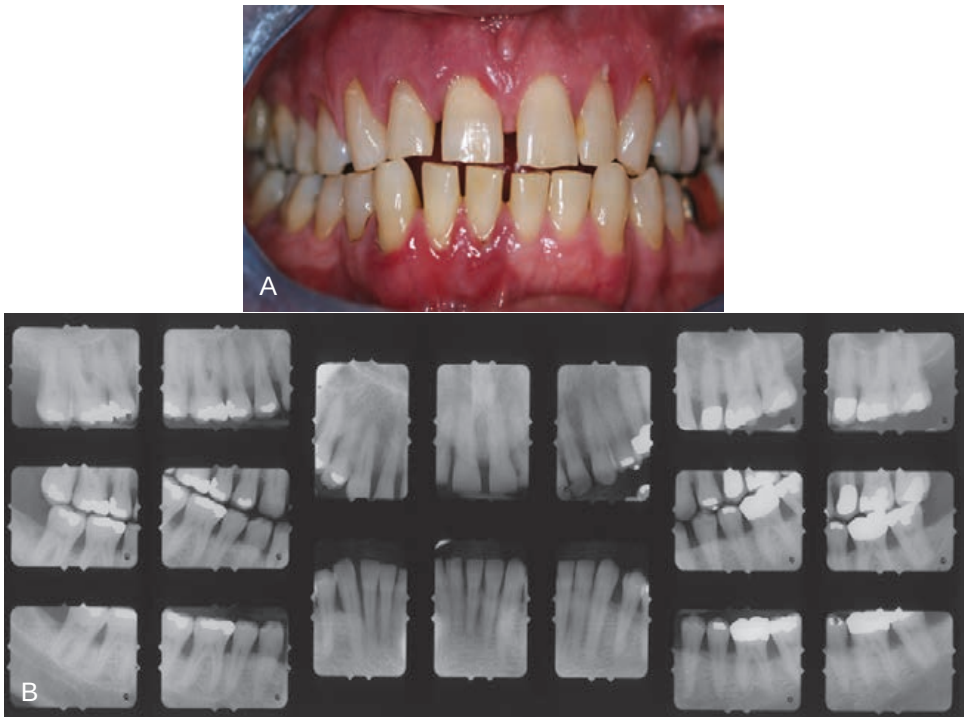

Figure 4-20 **A,** Clinical image of plaque-related severe aggressive periodontitis in 53-year-old male, smoker, with diabetes and hemoglobin A_{1c} (HbA_{1c}) = 10.7. **B,** Radiographic images of the patient.

Figure 4-21 Selective probing depths of same 53-year-old diabetic patient shown in Figure 4-20 with severe aggressive periodontitis.

lips, soft palate, buccal mucosa, gingiva, tongue, and bone.[21] Figure 4-22 depicts the pretreatment pattern of bone loss and recession associated with sarcoidosis, as well as pulmonary parenchymal fibrous infiltrate noted in the lungs as depicted by a white lacy pattern on the chest x-ray (Figure 4-22, *C*). Histologic features of sarcoidosis include the presence of an intense chronic inflammatory infiltrate with focal areas of noncaseating granulomas and a positive Kveim test[2] (Figure 4-23, *C*). Remineralization of alveolar bone is noted on radiographs obtained 1-year after systemic administration of steroids (Prednisone)[18] (Figure 4-23, *A*).

NECROTIZING PERIODONTAL DISEASES

The clinical characteristics of necrotizing periodontal diseases may include but are not limited to ulcerated and necrotic papillary and marginal gingiva covered by a yellowish white or grayish slough or pseudomembrane, blunting and cratering of papillae, bleeding on provocation or spontaneous bleeding, pain, and fetid breath. These diseases may be accompanied by fever, malaise, and lymphadenopathy, although these characteristics are not consistent. Two forms of necrotizing periodontal disease have been described: *necrotizing*

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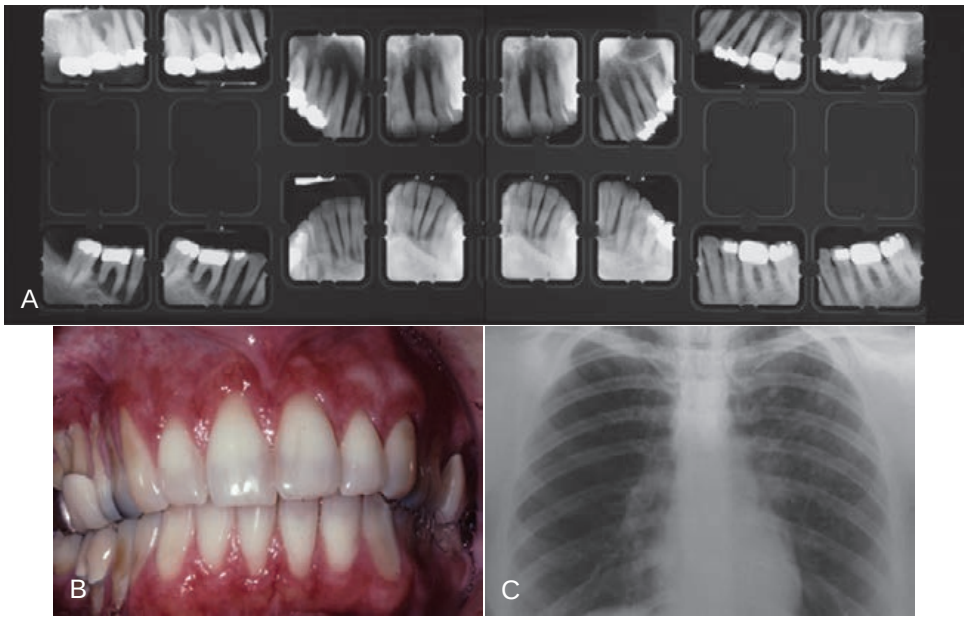

Figure 4-22 Sarcoidosis pretreatment. **A,** Intraoral x-rays depict more extensive bone loss for anterior teeth than clinical attachment loss. **B,** Extensive recession plus clinical attachment loss. **C,** Pulmonary parenchymal fibrous infiltrate.

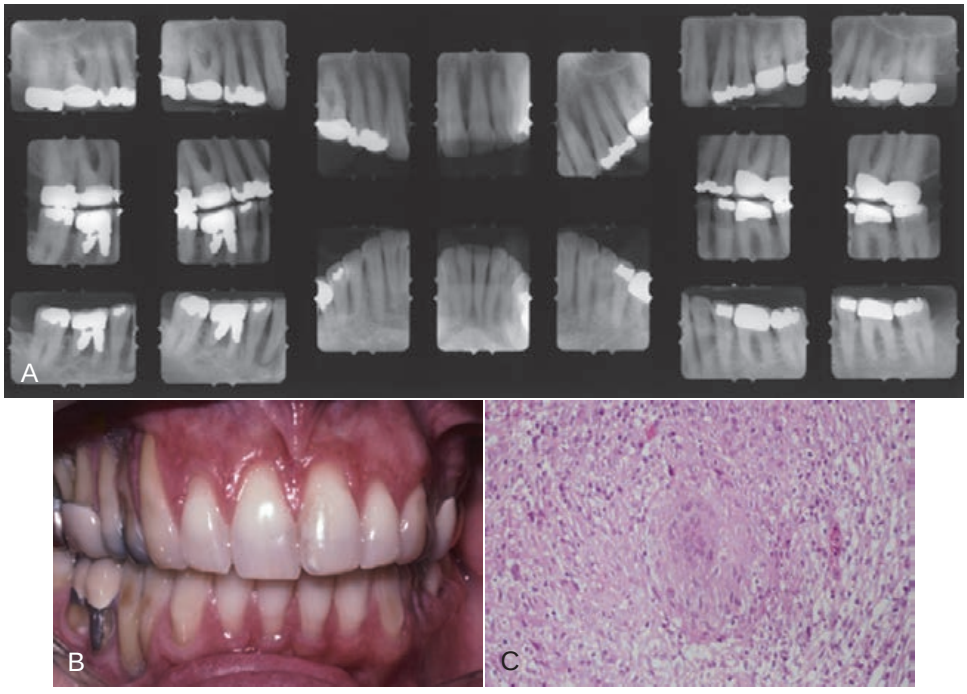

Figure 4-23 Sarcoidosis after treatment with prednisone. **A,** Intraoral x-rays depict remineralization of bone. **B,** Reduction in gingival inflammation while extensive recession and clinical attachment loss persist. **C,** Pretreatment biopsy.

ulcerative gingivitis (NUG) (Figure 4-24) and *necrotizing ulcerative periodontitis (NUP)* (Figure 4-25). NUG has been previously classified under "gingival diseases" or "gingivitis" because clinical attachment loss is not a consistent feature, whereas NUP has been classified as a form of "periodontitis" because attachment loss is present. Recent reviews of the etiologic and clinical characteristics of NUG and NUP have suggested that the two diseases represent clinical manifestations of the same disease, except that distinct features of NUP are clinical attachment and bone loss. As a result, both NUG and NUP have been determined as a separate group of diseases that have *tissue necrosis* as a primary clinical feature (see Box 4-1).

Necrotizing Ulcerative Gingivitis

The clinical and etiologic characteristics of NUG[27] are described in detail in Chapter 10. The defining characteristics of NUG are its bacterial etiology, its necrotic lesion, and predisposing factors such as psychologic stress, smoking, and immunosuppression. In addition, malnutrition may be a contributing factor in developing countries. NUG is usually seen as an acute lesion that responds well to

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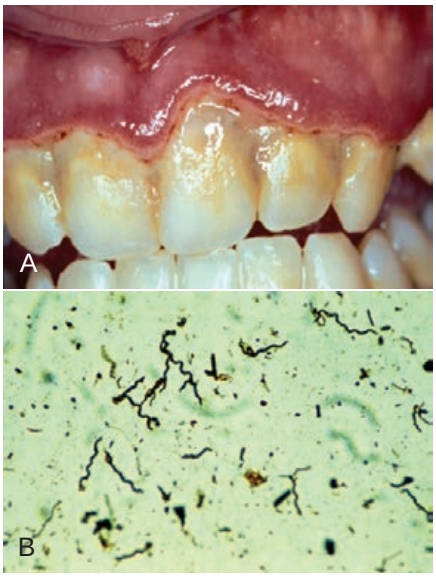

Figure 4-24 **A,** Ulcerative gingivitis illustrating sloughing of marginal gingiva. **B,** Phase-contrast microscopy reveals spirochetes in subgingival plaque sample.

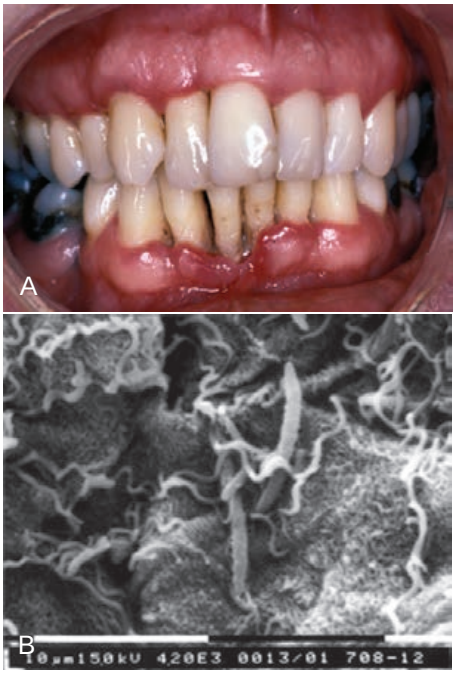

Figure 4-25 **A,** Necrotizing ulcerative periodontitis with severe clinical attachment loss in 28-year-old male infected with human immunodeficiency virus (HIV). **B,** Spirochetes noted on surface of sloughed epithelial cells.

antimicrobial therapy combined with professional plaque and calculus removal and improved oral hygiene.

Necrotizing Ulcerative Periodontitis

NUP differs from NUG in that loss of clinical attachment and alveolar bone is a consistent feature.[19] All other characteristics appear to be the same between the two forms of necrotizing disease. The characteristics of NUP are described in detail in Chapter 17. NUP may be observed among patients with HIV infection and manifests as local ulceration and necrosis of gingival tissue with exposure and rapid destruction of underlying bone, spontaneous bleeding, and severe pain. HIV-infected patients with NUP are 20.8 times more likely to have CD4+ cell counts below 200 cells/mm^3 of peripheral blood than HIV-infected patients without NUP, suggesting that immunosuppression is a major contributing factor. In addition, the predictive value of NUP for HIV-infected patients with CD4+ cell counts below 200 cells/mm^3 was 95.1%, and the cumulative probability of death within 24 months of a NUP diagnosis in HIV-infected individuals was 72.9%. In developing countries, NUP also has been associated with severe malnutrition, which may lead to immunosuppression in some patients.

ABSCESSES OF THE PERIODONTIUM

A periodontal abscess is a localized purulent infection of periodontal tissues and is classified by its tissue of origin.[16] The clinical, microbiologic, immunologic, and predisposing characteristics are discussed in Chapters 5 and 23.

PERIODONTITIS ASSOCIATED WITH ENDODONTIC LESIONS

Classification of lesions affecting the periodontium and pulp is based on the sequence of the disease process.

Endodontic–Periodontal Lesions

In endodontic–periodontal lesions, pulpal necrosis precedes periodontal changes. A periapical lesion originating in pulpal infection and necrosis may drain to the oral cavity through the periodontal ligament, resulting in destruction of the periodontal ligament and adjacent alveolar bone. This may present clinically as a localized, deep, periodontal probing depth extending to the apex of the tooth (Figure 4-26, *A*). A resultant extensive alveolar ridge defect (Figure 4-26, *B* and *C*) may necessitate reconstructive surgery (Figure 4-26, *D*) before placement of implants and prosthesis (Figure 4-27) to reestablish a functional and esthetic outcome. Pulpal infection also may drain through accessory canals, especially in the area of the furcation, and may lead to furcal involvement through loss of clinical attachment and alveolar bone.

Periodontal–Endodontic Lesions

In periodontal–endodontic lesions, bacterial infection from a periodontal pocket associated with loss of attachment and root exposure may spread through accessory canals to the pulp, resulting in pulpal necrosis. In the case of advanced periodontal disease, the infection may reach the pulp through the apical foramen. Scaling and root planing removes cementum and underlying dentin and may lead to chronic pulpitis through bacterial penetration of dentinal tubules. However, many periodontitis-affected teeth that have been scaled and root planed show no evidence of pulpal involvement.

Combined Lesions

Combined lesions occur when pulpal necrosis and a periapical lesion occur on a tooth that is also periodontally involved. An angular intrabony defect that communicates with a periapical lesion of pulpal origin results in a combined periodontal-endodontic lesion.

In all cases of periodontitis associated with endodontic lesions, the endodontic infection should be controlled before beginning definitive management of the periodontal lesion, especially when regenerative or bone-grafting techniques are planned.

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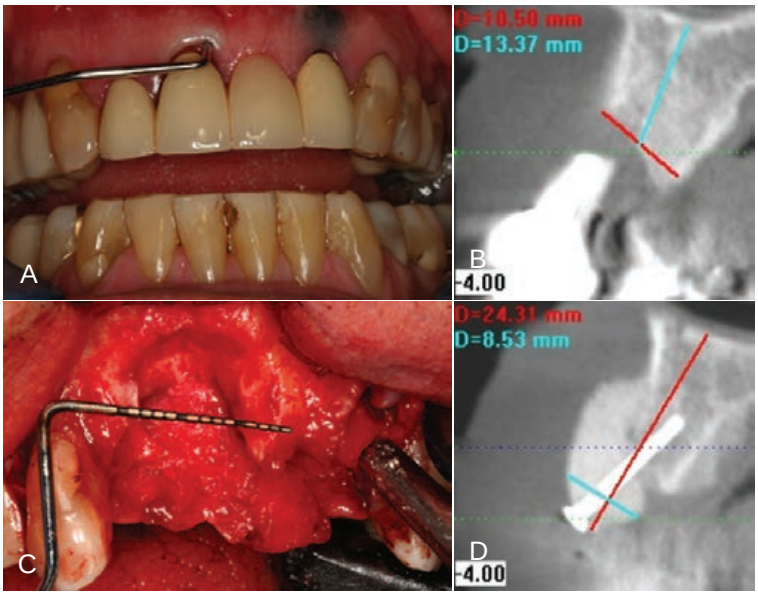

Figure 4-26 **A** and **C,** Clinical image of extensive loss of alveolar ridge secondary to periapical endodontic lesion. **B,** CT scan depicts alveolar bone loss. **D,** CT image of regenerated ridge via allogenic bone graft, tenting screw and membrane.

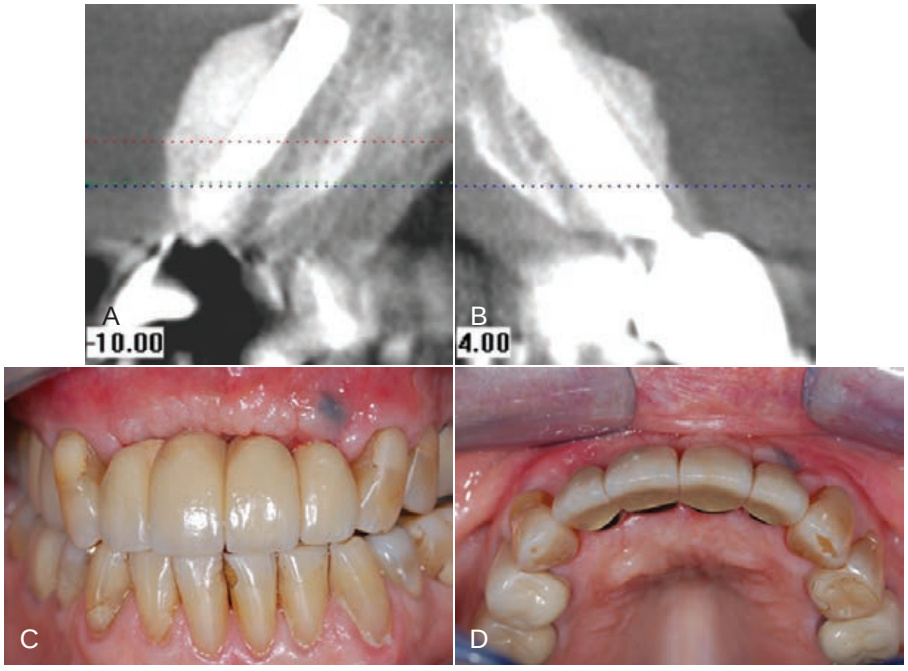

Figure 4-27 Same patient depicted in Figure 4-26. **A** and **B,** CT scan of regenerated ridge with implants placed in area 7, 9, and 10. **C** and **D,** Clinical image of implant-supported bridge.

DEVELOPMENTAL OR ACQUIRED DEFORMITIES AND CONDITIONS

Localized Tooth-Related Factors That Modify or Predispose to Plaque-Induced Gingival Diseases or Periodontitis

In general, these localized tooth-related factors contribute to the initiation and progression of periodontal disease through an enhancement of plaque accumulation or the prevention of effective plaque removal by normal oral hygiene measures.[5] These factors fall into four subgroups as outlined in Box 4-4.

Tooth Anatomic Factors. Tooth anatomic factors are associated with malformations of tooth development or tooth location. Anatomic factors, such as cervical enamel projections and enamel pearls, have been associated with clinical attachment loss, especially in furcation areas. Cervical enamel projections are found on 15% to 24% of mandibular molars and 9% to 25% of maxillary molars, and strong associations have been observed with furcation involvement.[15] Palatogingival grooves, found primarily on maxillary incisors, are observed in 8.5% of individuals and are associated with increased plaque accumulation, clinical attachment, and bone loss. Proximal root grooves on incisors and maxillary premolars also

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predispose to plaque accumulation, inflammation, and loss of clinical attachment and bone.

Tooth location is considered important in the initiation and development of disease. Malaligned teeth predispose individuals to plaque accumulation with resultant inflammation in children and may predispose to clinical attachment loss in adults, especially when associated with poor oral hygiene habits. In addition, open contacts have been associated with increased loss of alveolar bone, most probably through food impaction.[11]

Dental Restorations or Appliances. Dental restorations or appliances are frequently associated with the development of gingival inflammation, especially when they are located subgingivally. This may apply to subgingivally placed onlays, crowns, fillings, and orthodontic bands. Restorations may impinge on the biologic width by being placed deep in the sulcus or within the junctional epithelium. This may promote inflammation and loss of clinical attachment and bone, with apical migration of the junctional epithelium and reestablishment of the attachment apparatus at a more apical level.

Root Fractures. Root fractures caused by traumatic forces, restorative or endodontic procedures (Figure 4-28, *A* to *C*) may lead to periodontal involvement through an apical migration of plaque along the fracture when the fracture originates coronal to the clinical attachment and is exposed to the oral environment with resultant alveolar ridge defect (Figure 4-28, *D*).

Cervical Root Resorption and Cemental Tears. Cervical root resorption, as noted on the computed tomography (CT) scans in Figure 4-29, *A* and *B,* and cemental tears may lead to periodontal destruction when the lesion communicates with the oral cavity and allows bacteria to migrate subgingivally. Avulsed teeth that are re-implants frequently develop ankylosis and cervical resorption many years after re-implantation. Atraumatic removal of such ankylosed teeth and reconstruction of resultant ridge defects with bone grafts, dental implants, and prostheses are viable solutions for such defects (Figure 4-30).

Mucogingival Deformities and Conditions around Teeth

Mucogingival deformity is a generic term used to describe the mucogingival junction and its relationship to the gingiva (Figure 4-31), alveolar mucosa, and frenula muscle attachments. A *mucogingival deformity* is a significant departure from the normal shape of gingiva and alveolar mucosa and may involve the underlying alveolar bone. *Mucogingival surgery* corrects defects in the morphology, position, and/or amount of gingiva and is described in detail in Chapter 63. The surgical correction of mucogingival deformities may be performed for esthetic reasons, to enhance function, or to facilitate oral hygiene.[22]

Mucogingival Deformities and Conditions of Edentulous Ridges

Mucogingival deformities such as lack of stable keratinized gingiva between the vestibular fornices and the floor of the mouth (Figure 4-32, *A*) may require soft tissue grafting and vestibular deepening before prosthodontic reconstruction (Figure 4-32, *B* to *D*). Alveolar bone defects in edentulous ridges (Figure 4-33, *A* and *B*) usually require corrective surgery (Figure 4-33, *C* and *D*) to restore form and function before placement of implants and prosthesis to replace missing teeth (Figure 4-34).[22]

Occlusal Trauma

The etiology of trauma from occlusion and its effects on the periodontium[8] is discussed in detail in Chapters 20 and 49.

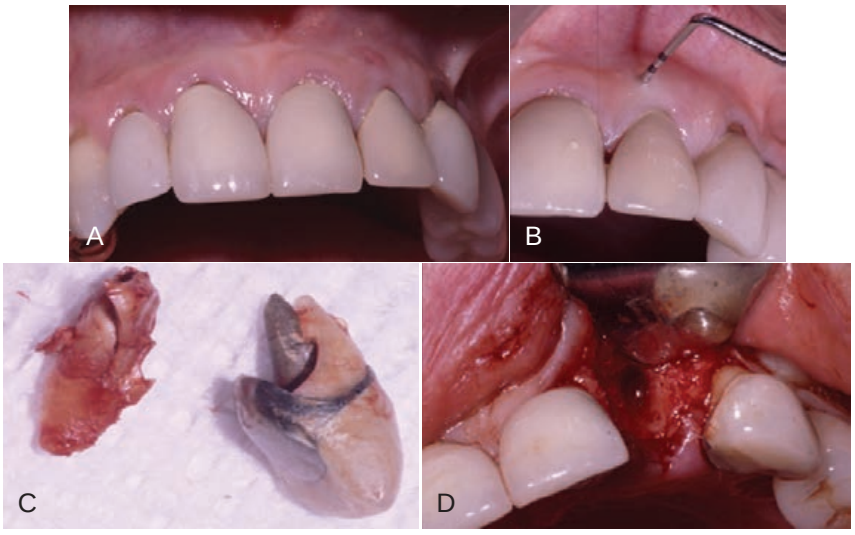

Figure 4-28 **A** and **B,** Clinical image of fistula tract. **C,** Root fracture. **D,** Resultant alveolar ridge defect.

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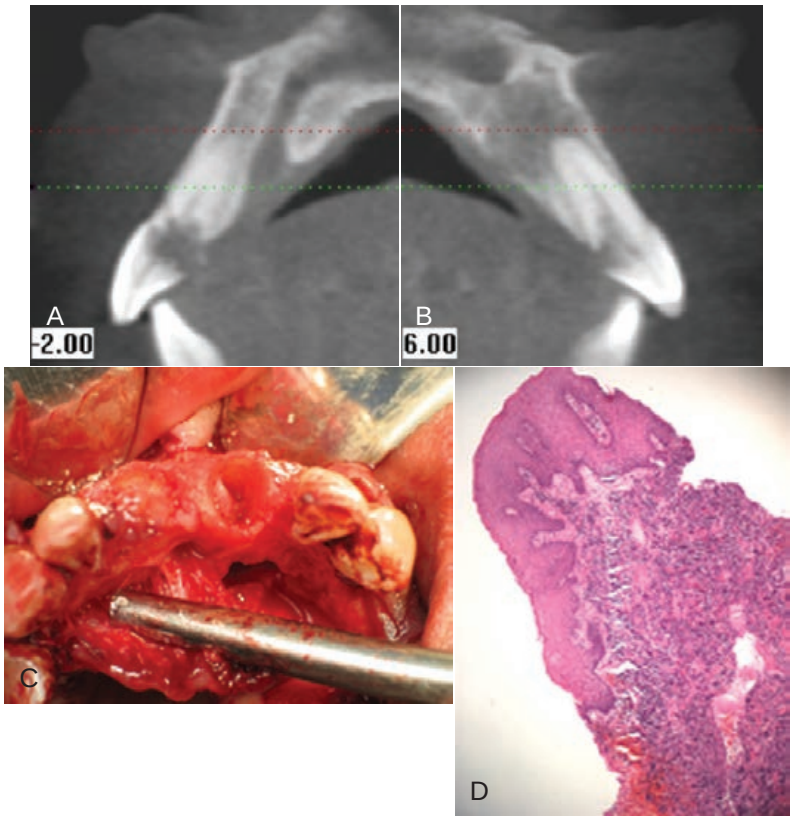

Figure 4-29 **A** and **B,** CT scan reveals severe cervical root resorption of maxillary central incisors and periapical abscess. **C,** Crowns fractured because of resorption. **D,** Biopsy of soft tissue from resorption.

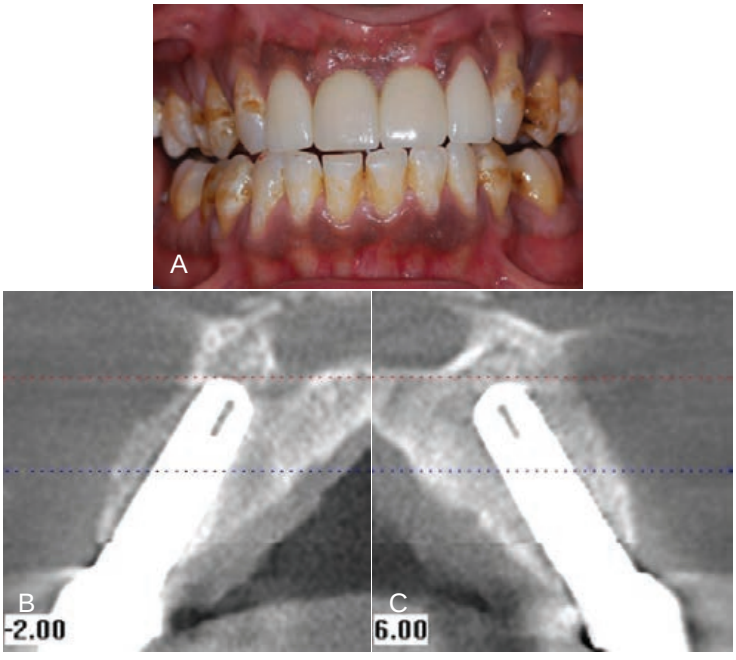

Figure 4-30 **A,** Posttreatment clinical image of same patient depicted in Figure 4-29 with implant-supported crowns and veneers on laterals. **B** and **C,** CT scan of bone grafts and implants replacing central incisors lost as the result of severe cervical root resorption.

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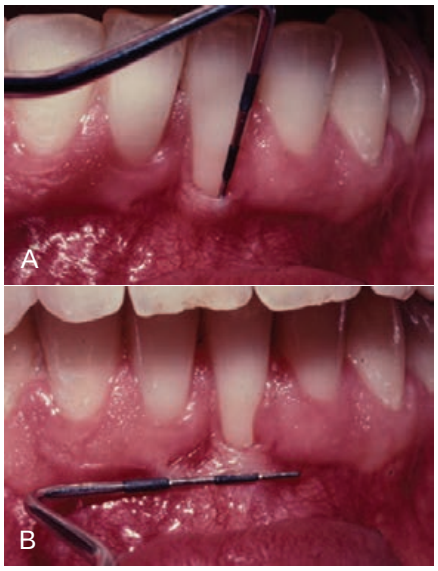

Figure 4-31 **A,** Mucogingival defect depicted by recession. **B,** Defects extend into alveolar mucosa and lacks keratinized gingiva.

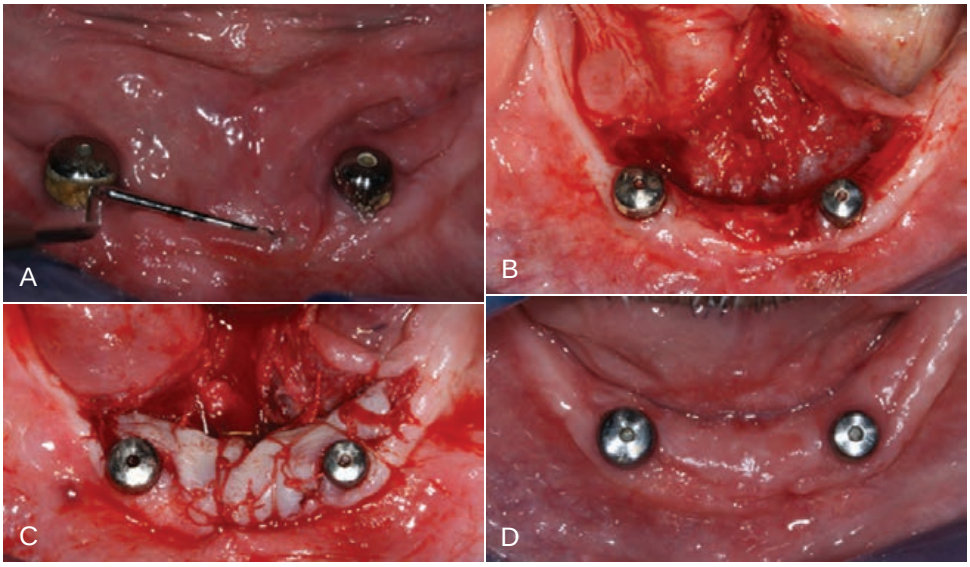

Figure 4-32 **A,** Mucogingival ridge defect from floor of mouth to vestibular fornix. **B,** Partial thickness flap with vestibular deepening. **C,** Placement of free gingival graft. **D,** Reestablishment of vestibular depth and keratinized attached gingiva.

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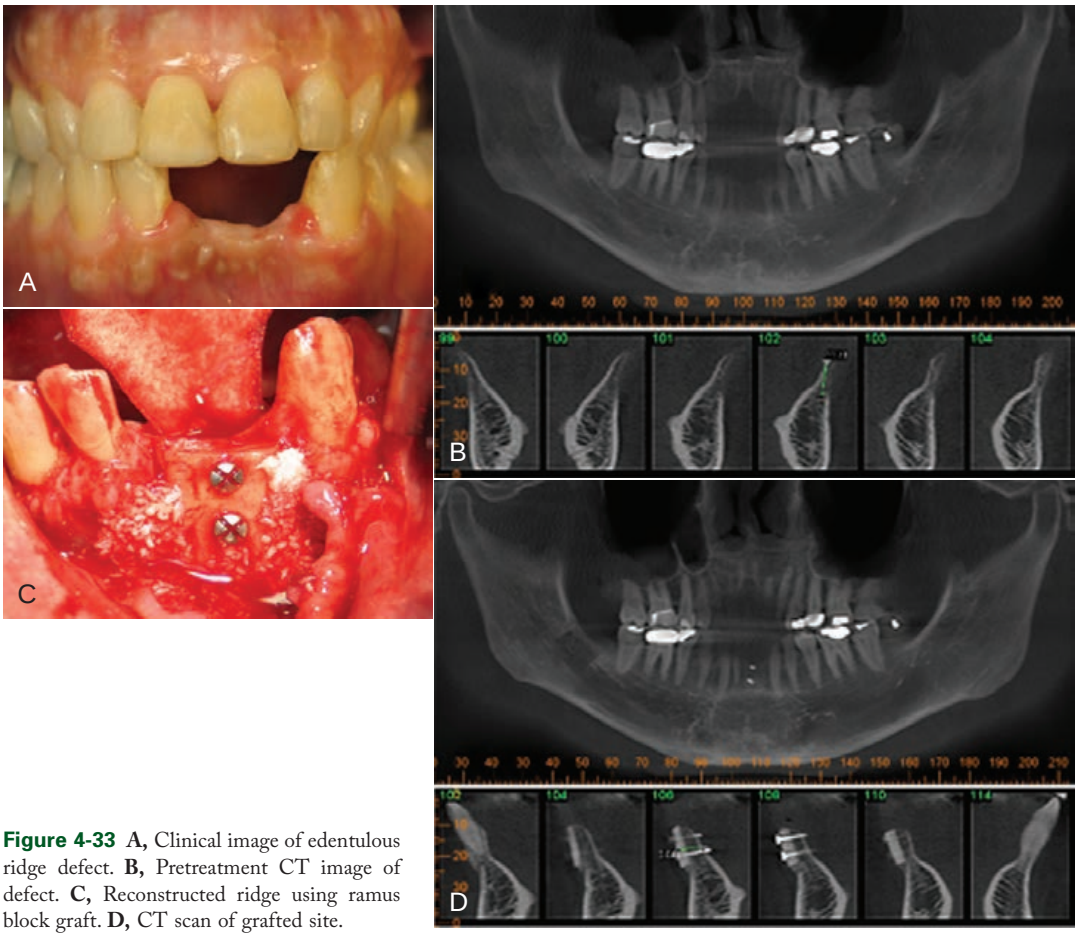

Figure 4-33 **A,** Clinical image of edentulous ridge defect. **B,** Pretreatment CT image of defect. **C,** Reconstructed ridge using ramus block graft. **D,** CT scan of grafted site.

Figure 4-34 **A,** Clinical image of ridge 6 months after grafting. **B,** Placement of implants in area of teeth #23 and 25. **C,** Porcelain fused to metal (PFM) crowns for maxillary incisors and implant-supported bridge from teeth #23 to #25.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 5

Fundamentals in the Methods of Periodontal Disease Epidemiology

Philippe Hujoel

CHAPTER OUTLINE

THE NEED FOR EPIDEMIOLOGY
- Measuring the Occurrence of Conditions or Diseases
- Periodontal Conditions Typically Measured Clinically
- Translating Periodontal Conditions into Traditional Epidemiologic Measures of Disease Occurrence
- True and Surrogate Measures of the Periodontal Condition
- Challenges in Obtaining Epidemiologic Measures of Periodontal Conditions and Diseases

EPIDEMIOLOGIC STUDY DESIGNS
- Randomized Controlled Trials
- Cohort Studies
- Case-Control Studies

ETIOLOGY
- Suspected Modifiable Etiologic Factors for Periodontal Disease
- The Cause of Periodontal Disease for the Patient Sitting in Your Chair

DIAGNOSIS
- Periodontal Conditions Versus Periodontal Diseases
- Diagnostic Tests Available to Assess Periodontal Conditions
- Translating Periodontal Diagnostic Test Results into Periodontal Disease Diagnosis
- Periodontal Disease Diagnoses

UNITED STATES PREVALENCE DATA OF PERIODONTAL DISEASE

THE NEED FOR EPIDEMIOLOGY

Beliefs on the cause of a disease impact how diagnosis, treatment, prevention, systemic consequences, and research agendas are considered. In mainstream periodontics, theories on the causes of periodontal disease have typically been specific and strong over the last half century. "Periodontal (gum) diseases, including gingivitis and destructive periodontal disease, are serious infections." This statement[69] from a professional dental organization in 2009 reflects the common and relatively recent belief that periodontal conditions are caused by bacteria.[71] Such a tenet on infection led to the following corollaries:

- Diagnoses of noninfectious periodontal diseases that were in clinical use for 200+ years were by decree eliminated from the periodontal lexicon.[53]
- The "cause-related phase of periodontal therapy" became focused on antimicrobials, antibiotics, and other plaque control measures.[41]
- Respected institutions claimed that destructive periodontal disease was "largely preventable" through a program of good oral hygiene.[47]
- Since medical infections were once assumed to cause chronic noncommunicable systemic diseases,[52] so could similarly periodontal "infections" be assumed to cause systemic diseases.
- Research agendas focused on topics such as periodontal vaccines and microbial diagnostics[12] and systemic consequences of periodontal infections.

However, additional evidence is required to be reasonably confident in the belief that periodontal disease indeed is an infectious disease. In a modern evidence-based world, case-control studies, cohort studies, and randomized controlled trials are the study designs that can obtain high-level evidence on the causes and management of a disease. These three epidemiologic study designs provide reliable inference on etiology, diagnosis, treatment, and prognosis. **This chapter focuses on the role of epidemiology in providing evidence on etiology and on clinical epidemiology and how it relates to diagnosis.**

Case-control studies and *cohort studies* are the two study designs most commonly employed to reliably identify the etiologic factors that cause disease. Smoking, ionizing radiation, hepatitis B, and high blood pressure are examples of causes of common human chronic disease that were identified with these research designs.

Randomized controlled trials offer the most reliable methods to evaluate the diagnosis, management, and prognosis of diseases. Prostate surface antigen (PSA) screening, polio vaccination, and

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SCIENCE TRANSFER

At present, it is difficult to accurately assess epidemiologic data on periodontal disease because of the wide variety of indices and measurements used, as well as the great differences in experience and reliability of the clinical examiners. For example, some epidemiologic surveys evaluated periodontal pocket depth with probes that have a sphere on the tip; in contrast, practicing clinicians measure pocket depths with probes that have a fine, tapered tip. These epidemiologic data may be very different from the detailed day-to-day measurements that are used for diagnosis and treatment planning of individual patients. Thus the reports that periodontal disease prevalence in adults in the United States has decreased over the last decade may or may not reflect an accurate picture.

One common mistake of interpretation of epidemiologic data is to assume a cause and effect relationship when two measurements show a high statistical connection. These statistical connections may or may not be related to cause and effect, and inductive controlled clinical trails are needed to prove if such a relationship exists.

hormone replacement therapy are examples of diagnoses and treatments for chronic diseases that were evaluated by randomized controlled trials.

Suggestions that periodontal diseases are infectious diseases are often justified by studies with a modern evidence-based approach that ranks below these study designs. Some scientific works, such as the experimental gingivitis study or an experimental infection study in nonhuman primates, have been cited frequently to support the "periodontal disease is an infectious disease" assertion. But there are reasons that these studies are ranked as a lower level of evidence than controlled clinical studies. Is the transition from an unnatural inflammation-free condition referred to as "Aarhus superhealthy gingiva"[70] to experimental gingivitis[44] (which is different from clinical gingivitis) indeed "didactic proof of the essential role of dental plaque bacteria in periodontal diseases"[70] or is it a reflection of the unhealthy effects of the typical western diet? Is a "burst" of bone loss[22] subsequent to the injection of *Bacteroides gingivalis* proof that "this microorganism (is) of great importance to the control of destructive periodontal disease"? In a modern evidence-based world, data from these small, uncontrolled case-series studies and experimental animal studies would be considered insufficient evidence to make "periodontal-disease-as-an-infectious-disease" a cornerstone of clinical thinking.

Epidemiologic studies—higher level than case-series or animal studies—have had a powerful impact on the incidence of chronic diseases by reliably identifying the primary causes. Reliable evidence on what causes disease allows laboratory research to focus on interventions on the causal pathways, which can then lead to clinical trials. "Medical science continually passes the baton of discovery from (epidemiologic) observation to laboratory studies to human clinical trials."[40] For example, epidemiologic observations identified hepatitis B as the main cause of liver carcinoma, one of the most common cancers in the world.[5] Subsequently, the baton of discovery was passed to basic science in which a recombinant engineered vaccine for hepatitis B was developed. Then, the baton of discovery was passed to clinical epidemiologists who assessed the effectiveness of vaccinations and documented dramatic declines of mortality rates due to liver cancer.[39] Similar success stories in the management of chronic diseases in which epidemiology played a critical role include coronary heart disease and blood pressure medication, dental caries and fluoride, and lung cancer and smoking intervention programs.

The emerging high-level epidemiologic evidence on the etiology of periodontal diseases suggests that systemic factors may be the true drivers of the epidemic. Factors, such as cigarette smoking and diabetes, are increasingly recognized as causes of periodontal disease, and their effect on the periodontium appears unrelated to infectious factors.[7,61] Organizations, such as the World Health Organization, suggest that periodontal disease prevention be made an integral part of programs that focus on tobacco control, diet, and physical activity.[54] The consequences of such a shift from a belief of periodontal disease as an infectious disease to a belief that periodontal disease is a lifestyle disease may impact on several aspects of clinical care. Regardless of your current beliefs on the etiology of periodontal disease, it will become increasingly important to become familiar with epidemiologic methodology to be able to judge this emerging evidence independently and critically.

Measuring the Occurrence of Conditions or Diseases

The fundamental tools of epidemiology are simple sums and divisions that reflect how many individuals or sites have or develop a particular condition or disease.

The *prevalence* is the number of individuals or sites that have a particular condition or disease within a defined population. The prevalence is the sum of the number of individuals or sites divided by the sum of all examined individuals or sites that exhibit the condition or disease of interest. The prevalence of a condition can range from 0% (no one has the condition or disease of interest) to 100% (everyone has the condition or disease of interest).

As an example of prevalence, the Centers for Disease Control (CDC) reported on the prevalence of individuals with at least one periodontal pocket depth of 4 mm or deeper. The CDC reported that in 1988–1994, a little over 1 in 5 Americans had such a condition (a prevalence of a little over 20%), whereas in 1999–2004, only 1 in 10 fell in this category[17] (or a prevalence of around 10%). These findings suggests a more than 50% decline in the prevalence of pocket depth greater than or equal to 4 mm for adults 20 to 64 years of age in approximately a decade. These epidemiologic data confirm another report of declining destructive periodontal prevalence in the United States (US).[9] Such information on prevalence measures of periodontal conditions may have implications for manpower needs in the US and may provide clues with respect to etiologic factors that drive such changes. The vast majority of countries do not have such prevalence surveillance systems,[54] which makes it difficult to determine whether these trends observed in the US are isolated or part of a more general trend.

The *risk* is the probability than an individual or a site will develop a particular condition or disease during follow-up. The risk for a condition or a disease is (similar to prevalence) a number that ranges between 0% and 100%. The simplest way to estimate risk is to have a fixed number of persons or teeth at risk at some defining

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moment (time zero [t_0]). Individuals or teeth within individuals are followed up over time subsequent to this defining moment. After a follow-up period (from time t_0 to time t_n), the risk can be calculated as the proportion of persons, or teeth, who develop the clinical outcome of interest during the follow-up.

Since the risk is estimated as a proportion, it is without dimension and ranges between 0 and 1. When a risk is reported, it should be accompanied by a specific time period to which it is applied. A 5% risk for death may be considered small when it refers to a 20-year period but quite high when it refers to a 3-month period.

As an example of the clinical utility of the concept of risk, consider concerns on occupational human immunodeficiency virus (HIV) infection for dentists. It has been reported that the risk for developing an HIV infection subsequent to an accidental needlestick with HIV-contaminated blood is 0.3%. Such a statistic has an intuitive appeal and can be related to patients or colleagues. A risk of 0.003 (0.3%) indicates that for every 1000 individuals that have an accidental HIV-contaminated needlestick, 3 are expected to develop an HIV infection.

The *odds* for an event is the probability that an event happened divided by the probability that an event did not happen. Whereas probability is a value that has to range between 0 and 1, odds values range from 0 to infinity. If the probability for observing an event is small, then the odds and the probability are almost identical. For example, if the probability for a vertical root fracture after an endodontic procedure is 0.001, the odds are 0.001/0.999 or 0.001001.

Odds are commonly reported in studies because they are often easier to estimate with statistical models than probabilities. For example, the odds for developing an HIV infection subsequent to an accidental needlestick with HIV-contaminated blood is 0.003 (0.003/0.997). The odds for developing oral cancer when smoking for 15 years is 0.098 (0.089/0.911).

Incidence rates are an alternative intuitive measure to describe disease occurrence. One of the best examples of an incidence rate is given by the speedometer in a car that displays at any given time the number of miles or kilometers being traveled per hour. In clinical trials or epidemiology, the rate reflects the number of disease occurrences per time unit per person or site. The most commonly used estimator for a disease rate is a ratio in which the numerator is the number of subjects or sites diagnosed with the disease of interest and the denominator is the sum of the time at risk over all subjects or sites in the population.

Incidence rates, as opposed to any of the previously introduced measures of disease occurrence, imply an element of time. The denominator in the incidence rate has time as the dimension. Therefore the dimension of incidence rate is 1/time. This dimension is often referred to as "person-time' or "site-time" to distinguish the time summation from ordinary clock-time. The magnitude of the incidence rate can vary between 0 and infinity. When there are no new disease onsets during the study period, the incidence rate is 0. When every person observed dies instantaneously at the start of the study, (and thus the sum of the time periods is 0), the incidence rate is infinity.

An example of the application of rates is provided in Figure 5-1, in which the number of teeth lost per 1000 tooth-years is plotted as a function of the maximum probing depth at the start of follow-up. The plot suggests a nonlinear relationship between maximum pocket and tooth loss with a substantial increase in tooth loss rate for teeth that have periodontal pockets of 7 mm or deeper.

For the study of risk, the population studied is usually limited to those individuals at risk for the outcome of interest. Thus, if the outcome of interest is a disease, the following subjects are excluded from the cohort: persons who already have the disease, persons who have immunity to the disease, or persons who are biologically incapable of developing the disease.

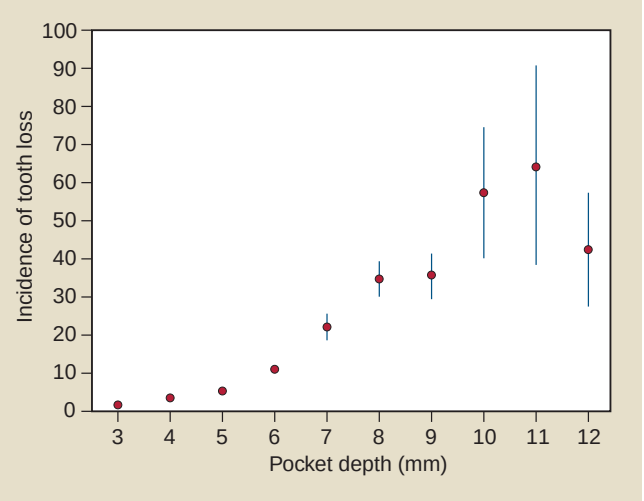

Figure 5-1 Rate of tooth loss (per 1000 tooth/year) as a function of maximum probing depth per tooth in a cohort of 1021 patients (ages 40–65 years) under periodontal specialist care for destructive periodontal disease. *(Data from Hujoel PP, Cunha-Cruz J, Selipsky H, et al:* Periodontol 2000 *39:22-29, 2005.)*

Periodontal Conditions Typically Measured Clinically

A clinical periodontal examination typically can include a variety of measures that reflect on characteristics of the periodontium. Most dental records of periodontal patients contain information on teeth that are present, missing, or impacted, as well as information on the periodontal status of those teeth that are measurable. Typically, a periodontal probe is used and information such as clinical probing depth, bleeding on probing, gingival recession, mobility of teeth, and the presence of furcation involvements are charted. In addition, some clinicians may collect information on the presence of gingivitis by evaluating the color and form of the gingival tissues. These measures may or may not be complemented with radiographic examinations that may provide information on marginal bone levels.

In research settings or in some selected private practices, additional periodontal measures may be collected such as clinical attachment levels, microbiologic measures, gingival crevicular fluid volume, the presence of certain biomarkers in the crevicular fluid, and a variety of indices that measure the amount of gingival inflammation or dental plaque or debris accumulation.

In one survey of clinical studies, it was observed that the gingival index (GI) and bleeding on probing are the two most common methods to assess gingival inflammation.[23] The GI was proposed in 1963 as a method for assessing the severity and quantity of gingival inflammation.[42,43] With this particular index, only gingival tissues are assessed. Each of the four gingival areas of the tooth (facial, mesial, distal, and lingual) are assessed for inflammation and rated as normal gingiva (a score of 0) to severely inflamed gingiva with a tendency to spontaneously bleed (score of 3). Gingiva that is mildly inflamed but without bleeding on probing is given a GI of 1, whereas moderately inflamed gingiva with bleeding is given a score of 2. The scores can be averaged by patient to provide patient means. Alternative, site-specific analyses can relate both local

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factors and patient-specific factors to the GI. The cumbersome nature of charting a GI makes it unlikely that it is in common use in clinical practice.

Bleeding on probing is a measure of periodontal inflammation that may be more commonly used by researchers and clinicians. The specific approach to obtain a bleedings measure can vary from one study to the next, as well as from one clinician to another. For example, in the Third National Health and Nutrition Examination Survey (NHANES III),[1] bleeding measures were obtained as follows. First, the facial and mesiofacial sites of teeth in two randomly selected quadrants, one maxillary and one mandibular, were selected. A special probe known as the *National Institute of Dental Research (NIDR) probe* was used in these assessments. This color-coded probe is marked at 2, 4, 6, 8, 10, and 12 mm. To begin the assessment, the examiner dried a quadrant of teeth with air. Then, starting with the most posterior tooth in the quadrant (excluding the third molar), the examiner placed a periodontal probe 2 mm into the gingival sulcus at the facial site and carefully swept the probe from the mesiofacial to the mesial interproximal area. After probing the sites in the quadrant, the examiner assesses the presence or absence of bleeding at each probed site. The same procedure was repeated for the remaining quadrant. As opposed to the NIDR protocol in which gingival bleeding is assessed by sweeping the periodontal probe while inserted only 2 mm into the periodontal sulci or pockets, clinicians may go to the depth of the periodontal sulci or pocket and measure pocket depth in conjunction with bleeding on probing.

Commonly used measures of periodontal tissue destruction in clinical studies—as opposed to periodontal inflammation—are mean probing depth, mean attachment loss, and mean recession level.[31] Again, the clinical protocol on how such mean values are collected and calculated can once again vary considerably from one study to the next. One example of description of how such values can be ascertained clinically is described in the National Institute of Dental and Craniofacial Research (NIDCR) "periodontal destruction" examination.[1] This examination includes an assessment of periodontal attachment loss[58] as the distance (in millimeters) from the cemento-enamel junction to the bottom of the gingival sulcus. This distance was measured at the facial and mesiofacial sites of teeth in a randomly selected maxillary and mandibular quadrant using the indirect measurement method developed by Ramfjord.[58]

Translating Periodontal Conditions into Traditional Epidemiologic Measures of Disease Occurrence

The application of the traditional epidemiologic methods of risk, prevalence, and rate was challenging because medical epidemiologists typically deal with patients that do or do not have a particular condition of interest. Dental epidemiologists deal with as many of 188 sites per patient that may or may not bleed. In addition, these periodontal sites within patients are correlated, meaning that if a patient has one site that bleeds, it is more likely that this same patient also has other sites that will bleed. It is now well established that gingival bleeding is suppressed in smokers.[8] Therefore bleeding on probing in periodontal sites in a smoking patient tends to be more alike than bleeding on probing in periodontal sites among nonsmokers. In others words, sites within patients cannot be assumed to be statistically independent as a result of patient-related factors such as smoking or diet. The statistical methodology to deal with such correlated observations is complex, and for most of the twentieth century, it was challenging to calculate the confidence intervals for site-specific risks or prevalence.[33] As a result, clinicians could not obtain reliable information on whether a periodontal site infected with a particular microbiologic species was at an increased risk for periodontal attachment loss. These challenges greatly hampered progress in building causal models of periodontal disease that were based on the higher-level evidence of controlled clinical studies rather than the lower-level evidence of reasoned theory, case-series, and animal studies.

The most common approach to deal with this challenge of correlated observations was to summarize site-specific periodontal data at patient level. These summaries could be calculated in a variety of ways. Commonly, site-specific information was averaged at a patient level. However, a variety of other approaches has also been utilized. For example, the information on the presence of bleeding in up to 188 periodontal sites in a patient could be summarized as the presence of at least 1 bleeding site, or the presence of at least 5 bleeding sites, regardless of whether patients had 1 tooth or 32 teeth.

Some devised a more sophisticated approach to provide patient-level summary data for periodontal outcome data. For example, the extent and severity index (ESI) of periodontal disease was developed to provide separate estimates of the extent and severity of periodontal disease.[11] A threshold of disease must be established to calculate the extent score for an individual. In their initial study of the ESI, Carlos et al[11] considered a site to be diseased when attachment loss exceeded 1 mm. For an individual, the extent score is the percentage of sites examined that have attachment loss greater than 1 mm. The severity score for an individual is the average loss of attachment per site among the diseased sites. The ESI is expressed as a bivariate statistic: ESI = (extent, severity). For example, an individual's ESI of (20, 5.0) would be interpreted as 20% of sites examined had disease and of the diseased sites, the average loss of attachment was 5 mm. The ESI for a population would be the average extent and severity scores for the individuals examined.

The advent of modern statistical techniques to deal with the problem of correlated data made it possible to avoid summarizing site-specific information at a patient level and losing the richness in the clinical data that clinicians need.[15,16,27,32,33] These methods allow important clinical questions to be answered, such as determining whether the 3 mm loss of attachment at a site is related to site-specific factors (such as the amount of plaque at that site, the location of the site in the oral cavity, or the microflora present at that site), to host factors (such as glucose control, serum cotinine levels, or intake of fermentable carbohydrates), or to an interaction between a site-specific factor and a host factor.

True and Surrogate Measures of the Periodontal Condition

An important consideration in both epidemiology and clinical practice is the distinction between true and surrogate endpoints of periodontal disease. *True endpoints* are tangible outcomes that directly measure how a patient feels, functions, or survives.[18] A painful periodontal abscess is an example of a tangible event that can be precisely identified or realized by the patient. True endpoints include the oral health–related quality-of-life measurements[38,48,65] or self-reported problems such as a positive answer to the question: "When you brush or floss your teeth, do you notice bleeding that is both regular and involves spitting blood-stained saliva?" Synonyms for true endpoints include clinically relevant endpoints, clinically meaningful endpoints, terminal endpoints, or ultimate endpoints.

Surrogate endpoints are intangible to the patient.[68] Changes in bleeding on probing or pocket depth are examples of changes that

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are intangible to the patient. Examples of surrogate endpoints in periodontal research include anatomic measures (e.g., probing depth), measures of inflammation (e.g., gastrointestinal), microbiologic measures, and immunologic measures.[15] Surrogate endpoints are often objective because they can be measured by the clinician (rather than relying on self-report by patients) or by laboratory methods. Synonyms for surrogate endpoints include intermediate endpoints, biologic markers, or biomarkers.

Surrogate endpoints can be misleading when the goal is to provide reliable information on clinical decisions related to diagnosis, etiology, treatment, or prognosis. An overview of situations in clinical research in which surrogate endpoints have led to misleading conclusions is provided in Table 82-1 on the Expert Consult website. One periodontal example is systemic antibiotics that may have a beneficial impact on attachment gain[20] but a potential increased risk for tooth loss.[14]

Challenges in Obtaining Epidemiologic Measures of Periodontal Conditions and Diseases

Among the challenges that face the periodontal epidemiologist are the continuous changes in the type of surrogate data collected, the paucity of information on whether surrogate information provides reliable information on outcomes of tangible patient benefit (i.e., outcomes that the patient cares about), the lack of diagnostic codes for the reasons for tooth loss, and the difficulty in estimating the impact of tooth loss on surrogate outcomes.

The diversity of measures to assess periodontal condition or disease is truly staggering. One survey of periodontal clinical trials conducted over a mere 4-year period indicated that a total of 153 distinct surrogate endpoints were defined and that over 80% of the 153 surrogate endpoints were used in fewer than 5 of the 82 trials.[23] Another survey similarly identified the diversity in methodologies and definitions as a challenging issue in systematically reviewing evidence.[62] This continuous creation of "new and improved" surrogate outcomes in periodontal research is likely an important driver of false-positive conclusions.[26]

In addition, there is substantial secular variability in the type of periodontal measures that are favored, with new surrogate measures appearing on a regular basis. Russell developed the Periodontal Index,[60] which scored the supporting tissues for each tooth in the mouth according to a progressive scale that gives little weight to gingival inflammation and relatively great weight to advanced periodontal disease. Although the Periodontal Index was used in the first NHANES, thereby gaining national prominence in the US, it was not used again in any of the subsequent national surveys.

Similarly, in clinical periodontics, clinical attachment levels were once considered the gold standard for judging a treatment's efficacy but are now relegated as a mere safety measure in some drug-approval processes.[29] As a result of this continuous change in the type of surrogate periodontal measures that are in fashion, it was not possible until recently to provide reliable information on such simple questions as to whether the prevalence of destructive periodontal disease was increasing, decreasing, or remaining constant.

A second challenge in interpreting the clinical relevance is the paucity of information on whether the collected data are informative on any measure that matters to patients such as tooth loss or quality-of-life issues related to oral health. This situation creates challenges in interpreting evidence. It is similar to tracking prostate cancer by measuring inflammation or swelling of the prostate gland by digital palpation, without information on how this information relates to prostate cancer mortality, clinical symptoms of prostate disease, or anything that matters to the patient. This challenge is further compounded by the absence of diagnostic codes for tooth loss, which has largely prevented obtaining reliable information on how many teeth really are lost as the result of periodontal disease opposed to dental caries.

The attempt to track a disease by only collecting a surrogate outcome measure such as probing depth from those teeth that are present leads to a type of bias is typically referred to as *survival bias*. Most periodontal clinical trials in the twentieth century evaluated the effect of periodontal therapies on those teeth that survived the treatment. As in cancer research, in which studying the effect of chemotherapy on the survivors creates results that cannot be interpreted, studying the effect of periodontal therapies on survivor teeth creates unreliable data. Some methods, such as imputations, can provide an understanding of the extent to which such biases may alter the conclusions of epidemiologic studies.

In summary, the fundamental tool of periodontal epidemiology is a measure of the occurrence of periodontal conditions. These measures include epidemiologic statistics, such as prevalence, risk, and rate, and focus on either patient-specific or site-specific markers such as oral health–related quality of life, tooth loss, anatomic measures, or measures of gingival inflammation. This wealth of possibilities in defining periodontal conditions combined with statistical challenges in handling correlated data has made it difficult to answer even such a simple question as to whether a hidden periodontal disease epidemic occurred in the twentieth century.[28]

EPIDEMIOLOGIC STUDY DESIGNS

The essence of epidemiology and clinical epidemiology is to relate measures of disease occurrence to suspected causes or interventions. Can the recent dramatic drop in destructive periodontal disease prevalence in the US be attributed to a change in smoking prevalence? Can the presence of particular microbiologic species around a tooth be related to the risk of future tooth loss? Can the rate of tooth loss in a elderly sample be related to the use of an antimicrobial rinse? In an evidence-based approach, these questions can be most reliably answered by three epidemiologic study designs. As mentioned earlier in this chapter and briefly introduced in Chapter 2, these study designs (in order of decreasing reliability) are the randomized controlled trial, the cohort study, and the case-control study. For a comprehensive review of study designs, the reader can consult statistical or epidemiologic textbooks.

Randomized Controlled Trials

Randomized controlled trials in periodontics typically assign patients or some teeth within a patient randomly to a treatment and subsequent outcomes are assessed. Table 5-1 provides two examples of randomized controlled trials.

The randomized controlled trial is the only study design that can provide a probabilistic basis for making causal inference between an intervention and an outcome. Reliable inference on the

TABLE 5-1 Examples of Periodontal Randomized Controlled Trials

Periodontal Treatment	Outcome	Sample Size
Scaling and root planing in pregnant women[50]	Infant low birth weight	823
Biphasic calcium phosphate ceramic[51]	Clinical attachment level	137

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TABLE 5-2 Examples of Periodontal Cohort Studies

Periodontal Exposure	Outcome	Sample Size
Periodontal disease and tooth loss[34]	Coronary heart disease	51,529
Gingivitis[10]	Tooth loss	>500

TABLE 5-3 Examples of Periodontal Case-Control Studies

Case-Control Criteria	Investigated Risk Factors	Sample Size
Destructive periodontal disease[3]	Smoking	
Acute myocardial infarction[4]	Dental health	

causality of associations is only possible if there is a specific pretrial hypothesis that specifies the endpoint, the treatments to be compared, the patient population, and the degree of required precision and if the trial conduct adheres strictly to design and analysis principles of definitive trials. Factors important in obtaining reliable answers include a secure randomization process, masking of patients and clinicians, the presence of an independent data and safety monitoring board, and a strict adherence to the pretrial hypothesis, including an intent-to-treat analysis. Such trials are rare in any field including periodontal research. As a result, most of the trials published in the literature are in the category of exploratory trials typically reporting no pretrial hypothesis and concluding that the intervention was successful when compared to the control.[23] Such conclusions are almost always false-positive conclusions.[26] Only definitive trials can provide reliable answers on treatment efficacy. Unfortunately, these are the trials that are least likely to be published.

Cohort Studies

Cohort studies are sometimes referred to as *exposure-based study designs.* Subjects are classified with respect to an exposure such as cigarette smoking or diabetes and followed longitudinally for the assessment of periodontal outcomes. Table 5-2 provides two examples of cohort studies.

Cohorts can be defined by a geographic area, records, exposure status, or a combination of different criteria. In one study on causal factors of edentulism, the population of interest was defined as the inhabitants of the town of Tecumseh, Michigan. Persons within this community were examined in 1959 as part of a community-wide health study. Twenty-eight years later a subset of these patients was reexamined to study the risk factors for edentulism.[1] Several natural disease history studies of destructive periodontal disease have been conducted based on geographic location. Examples include the Norwegian Longitudinal Study,[3] the Veterans Administration Longitudinal Study,[37] and the Sri Lanka study.[4] A cohort can be defined by records (schools, health insurance plans, unions, industries, or professional organizations). Many cohort studies on periodontal disease outcomes are performed in patients belonging to a particular dental insurance company[14] or to a professional group.[34] Finally, cohorts can be defined based on a specific exposure. For example, different levels of fluoride concentration in the water supply has been used for the definition of cohorts.

Case-Control Studies

Case-control studies are typically referred to as *outcome-based study designs.* Persons with a condition or outcome of interest (cases) are compared to persons without a condition of interest (controls) with respect to the past history of the suspected causal factors. Many people intuitively think along the lines of a case-control study when evaluating disease etiology. For example, if one suffers from food poisoning after a party, he is likely to compare his past food intake history with the past food intake history of those individuals at the party that did not have food poisoning. Similarly, if one is diagnosed with a serious illness, a common reaction is: "Why me?" followed by a comparison of past history of exposures with other individuals that did not develop the serious illness. The primary goal in a case-control study is to find out what *past* exposures or factors are different between diseased patients (cases) and nondiseased patients? Table 5-3 provides two examples of case-control studies.

The case-control study is the most challenging type of study to conduct. Trying to minimize the role of bias in case-control studies requires careful planning, conduct, and analysis, and even when everything is done perfectly, one can come up with the wrong conclusions in case-control studies. A recent review of the quality of periodontal case-control studies suggested that they are frequently inadequately conducted and reported.[46]

Two important elements of the case-control study design are the definitions of case and control. A *case* is a person in the population or study group identified as having a particular disease, health disorder, or condition.[12] The case definition should be rigorous to minimize bias and misclassification and can be based on symptoms, signs, or results of diagnostic tests. For example, the case definition for a myocardial infarction in a case-control study on the relationship between dental health and acute myocardial infarction was as follows[4]:

1. Symptoms start within 36 hours before the admission.
2. No prior myocardial infarction.
3. Residing in Helsinki or immediate neighborhood.
4. Age <60 years for men and <65 years for woman.
5. Blood samples available at admission and at 4 weeks.

Three different types of cases can be distinguished based on the relationship between the date of disease onset and the data of study initiation. *Incident cases* are those whose date of diagnosis is after the date of study initiation. Using this approach, cases are interviewed at the closest possible time to the etiologic exposure. Factors affecting survival of cases may not have yet started to act. As a result, case-control studies with incident cases are least likely to confuse causes of disease with consequences of a disease. *Prevalent cases* are those cases whose date of diagnosis precedes the date of study initiation. The selection of prevalent cases has only one advantage: cases are readily available. Prevalent cases may represent the survivors of all cases. Under such circumstances, factors that affect survival may appear as etiologic factors. The latter is of particular concern in dental case-control studies in which diagnosis of case status is based on surviving teeth. *Decedent cases* are those cases who died before the date of study initiation. Decedent cases possess all the disadvantages of prevalent cases. In addition, it may be that proxies need to be interviewed to investigate past exposure histories.

In a case-control study, the *controls* should be at risk for developing the investigated disease and come from the same population that generated the cases. For example, if the investigated disease is root caries, the controls should be at risk for developing root caries (i.e., have exposed root surfaces) and originate from the same population that generated the cases that have root caries.

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ETIOLOGY

Human chronic diseases, such as cancer, diabetes, and destructive periodontal disease, have complex etiologies. The terms, *necessary cause, component cause,* and *sufficient cause,* help define the challenges in determining the etiology of a disease and in verbalizing the complexity of chronic disease etiology.[59]

The set of causes that initiate a chronic disease is referred to as a *sufficient cause.* Each sufficient cause consists of multiple component causes. Consider the hypothetical example where four sufficient causes exist for noniatrogenic destructive periodontal disease (Figure 5-2). The first sufficient cause in this example includes the following component causes: smoking, delayed neutrophil apoptosis, an interleukin-1 (IL-1) gene defect, dental plaque, a tooth, and an unspecified gene defect. These different elements of a sufficient cause are referred to as *component causes.* All component causes of a sufficient cause need to be present in order for the disease process to be initiated. Multiple sufficient causes may be responsible for a given disease. For example, two sufficient causes exist for destructive periodontal diseases that do not include smoking.

A component cause, which is an element of all the sufficient causes for a given disease, is referred to as a *necessary cause.* For example, fermentable carbohydrates are a necessary cause for dental caries. However, there are few examples of necessary causes—smoking is not a necessary cause of lung cancer or destructive periodontal disease, hepatitis B infection is not a necessary cause of liver cancer, or *Streptococcus viridans* is not a necessary cause of bacterial endocarditis. The search for necessary causes of disease is important, since its elimination would eradicate a disease.

The proportion of disease caused by different component causes does not add up to 100%. The component cause "smoking" is responsible for 80% of the destructive periodontal disease cases, plaque for 100%, and diabetes for 10%.

The complex causal web leading to the initiation and progression of chronic disease makes the reliable identification of causal components difficult. Over the last 50 years, epidemiology has succeeded in reliably identifying the highly prevalent causes for human chronic diseases. Now that those component causes responsible for a large proportion of cases for particular diseases (e.g., smoking for lung cancer) have been identified, the search for new causes is becoming significantly more challenging. For example, the hope existed that the genome project would lead to quick advances, but these hopes have not yet been fulfilled. Chronic diseases are typically caused not by one gene but by a set of many different genes, each one responsible for only a small proportion of the cases and acting in a variety of synergistic mechanisms on disease initiation.

Suspected Modifiable Etiologic Factors for Periodontal Disease

Tobacco Smoking. Tobacco smoking is recognized by several organizations as one of the primary drivers in periodontal disease epidemiology.[7] Many criteria for causality have been satisfied,[19] and smoking cessation has shown to slow the progression of periodontal disease.[36,37,55] The strong impact of tobacco smoking on periodontal disease has the potential to induce spurious causal associations in other suspected risk factors for periodontal disease. For example, smoking is a risk factor for both type II diabetes[72] and periodontal disease, making associations between type II diabetes and periodontal disease susceptible to biases. To obtain reliable inference on causal factors other than smoking, studies on periodontal disease epidemiology may need to be restricted to those who have never smoked.

Carbohydrate Metabolism. Several studies have shown relationships between periodontal disease and a variety of conditions that center on carbohydrate metabolism, including intake of dietary carbohydrates, exercise, obesity, prediabetes and diabetes. A systematic review of randomized controlled trials on carbohydrates suggested that an increased intake of fermentable carbohydrates will cause an increase in gingivitis.[24] Two systematic reviews suggested diabetes as a risk factor for destructive periodontal disease.[61,64]

Dental Plaque. Several systematic reviews have provided evidence that chemotherapeutic and mechanical plaque control will reduce gingival inflammation. Essential oils[6] and cetylpyridinium chloride-containing mouth rinses[21] may reduce gingival inflammation. Interdental brushes may reduce dental plaque, bleeding, and probing pocket depth.[66] Power-driven toothbrushes may be more effective than manual toothbrushes in removing plaque and reducing inflammation.[63] Self-performed dental flossing may not be effective in reducing plaque and gingival inflammation.[6] Although these systematic reviews provide evidence on the role of dental plaque in gingival inflammation, such studies do not necessarily suggest that dental plaque is the primary cause of gingival inflammation. Similar to the fluoride needed to prevent decay caused by fermentable carbohydrates, a toothbrush may be necessary to prevent gingivitis as the result of an unhealthy diet.

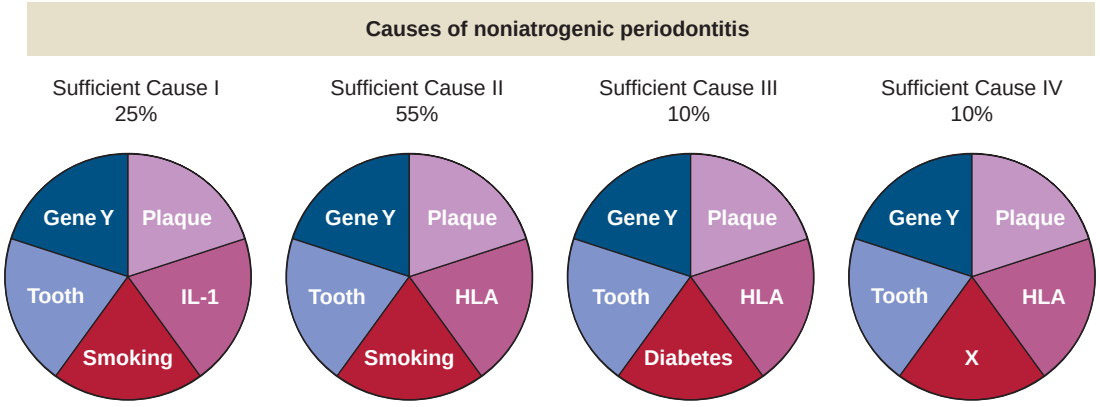

Figure 5-2 Causes of noniatrogenic periodontitis. *(Data from Rothman KJ:* Am J Epidemiol *141(2):90-95, 1995.)*

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There is no reliable randomized controlled trial evidence that oral hygiene has a beneficial impact on the prevention of periodontal destruction.[25]

The Cause of Periodontal Disease for the Patient Sitting in Your Chair

In clinical epidemiology, in a court of a law, and in modern-day clinical practice, uncertainty regarding the "cause" is a key consideration when discussing causality. The term *attributable risk percentage* is used to express the probability that a disease is caused by a suspected etiologic agent. In a smoker with lung cancer, there may be a 20% probability that the lung cancer was caused by a factor other than smoking (e.g., radon). In an obese person with diabetes, there may be a 10% chance that smoking played no role in the onset of diabetes. For a worker with leukemia in the nuclear industry, there may be an 80% chance that the leukemia was not caused by the low-level protracted radiation exposures. One can almost never determine with certainty what caused a particular condition or disease to appear in a patient. All one can do is to assign probabilities to the likelihood that a particular causal factor was responsible for the disease diagnosed in a patient. Destructive periodontal disease and periodontal inflammation are no exception to this general rule of uncertainty in determining disease etiology. As a result, diagnostic names, such as plaque-induced gingival disease or non-plaque–induced inflammatory gingival lesions,[41] can be considered misnomers, since such names imply diagnostic certainty that lead to circular reasoning.[4] In the absence of high-level evidence on the causes of these diseases, a declaration of certainty on causality reflects the strong belief in the low-level evidence that suggests that the cause of periodontal disease is infection. The principle of diagnostic uncertainty is important not only when considering disease etiology but also when it comes to diagnosing periodontal conditions.

DIAGNOSIS

Periodontal Conditions Versus Periodontal Diseases

Disease is defined as an attribute or a characteristic of a person, and diagnosis is the clinician's belief that the person has the attribute.[67] The World Health Organization (WHO) defined disease as those adverse health consequences that include physical or psychological impairment, activity restrictions, and/or role limitations.[67] Certain periodontal conditions have been associated with such adverse consequences and thus certain periodontal conditions qualify as diseases under the WHO definition. In one study, about 1 in 5 patients presenting to a periodontal specialist reported that teeth, gums, or dentures had an impact fairly often or very often on either eating; relaxing; avoiding going out; or feeling self-conscious, pain, or discomfort. In this same study, 4 out of 10 patients rated their oral health as fair or poor.[13] Other studies have shown that gingival conditions, such as necrotizing ulcerative gingivitis or attachment loss in high school students, are similarly related to oral health–related quality of life.[45]

An important question in periodontal diagnosis is determining which periodontal conditions can be diagnosed as "diseased." Can a patient with a couple of sites with 1 or 2 mm of attachment loss be classified as diseased? How about a patient with such subtle gingival inflammation that the majority of clinicians would not notice the inflammation and that even highly trained clinical examiners agree poorly on the presence of gingivitis? Disagreements on such questions is one of the reasons why the prevalence of gingivitis and destructive periodontal disease can range widely, depending on which reference levels are considered to be the cut-off for normal versus diseased.

Diagnostic Tests Available to Assess Periodontal Conditions

Diagnostic tests for periodontal disease can include anatomic measures of tissue destruction such as probing pocket depth and clinical attachment loss; measures of gingival inflammation such as redness, suppuration, bleeding, bleeding on probing, elevated gingival temperature, and a panoply of gingival crevicular fluid markers; radiographic measures of bone destruction and tooth mobility; and microbiologic measures. These test results combined with factors, such as age, past dental history, and systemic conditions, can be translated into a distinct set of periodontal diagnoses.

Translating Periodontal Diagnostic Test Results into Periodontal Disease Diagnosis

Three different methods can be distinguished to translate clinical conditions into diseases: (1) normative or arbitrary values, (2) risk-based reference values, or (3) treatment-based reference values.[30]

Normative or Arbitrary Values to Diagnose Periodontal Disease. Diseases can be defined based on normative or arbitrary reference values. If the normal periodontium is assumed to have no pocket depths deeper than 3 mm, then one could define destructive periodontal disease as a patient with any pocket deeper than or equal to 4 mm, or a patient with 3 pockets ≥5 mm could be classified as having destructive periodontal disease.

Alternatively, normative values could be based on parametric or nonparametric percentage cut-off values. For instance, the 97.5th percentile of the age-specific number of pockets deeper than 5 mm could be used to define destructive periodontal disease. Based on the NHANES III data, a 28-year-old individual with 2 pockets deeper than 5 mm could be diagnosed as having destructive periodontal disease, whereas 5 periodontal pockets deeper than 5 mm would be required in a 58-year-old individual.[30]

Diagnoses based on normative or arbitrary cut-offs result in normative or arbitrary disease prevalence levels, regardless of the distribution of underlying risk factors. Regardless of whether 5% or 95% of the population smoked 2 packs-a-day for 40 years, the prevalence of destructive periodontal disease would remain equal to the selected cut-off value. If all human chronic diseases were defined based on an arbitrary tenth percentile cut-off value, the prevalence of all chronic disease would be equal to 5% (e.g., 5% of the population would have blood pressure that is too high or 5% would have a blood glucose level that is too high).

Risk-Based Reference Values to Diagnose Periodontal Disease. The diagnosis of disease can be set at that point on the diagnostic marker in which a steep increased risk for adverse health outcomes is present. The cut-off is still somewhat arbitrary but is connected to clinical realities in terms of the risk of adverse health outcomes. There is a trade-off between the dangers of missed diagnoses when the cut-off is made too high (more specific) and the dangers of false-positive diagnoses when the cut-off is too low (more sensitive).

A risk-based diagnosis of destructive periodontal disease requires the conduct of longitudinal studies in which pocket depth at baseline is related to the risk of subsequent adverse outcomes such as tooth loss. Figure 5-1 represents such a plot and suggests that a pocket depth of 6 mm could be a diagnostic marker for destructive periodontal disease because a distinctive increased risk

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for tooth loss is associated with pocket depth values 6 mm or deeper.

Risk-based diagnosis of chronic diseases, similar to the use of normative or arbitrary values, can do more harm than good. A diagnosis of obesity based on a body mass index (BMI) of 28 may do more harm than good if weight loss treatments increase mortality risk.[35] A diagnosis of high blood pressure[57] or diabetes[49] may cause more harm than good if the prescribed treatment further increases the mortality risk. Similarly, a diagnosis of destructive periodontal disease based on the presence of periodontal pockets 6 mm or deeper may cause more harm than good if the suggested periodontal treatments increase periodontal morbidity.

Therapeutic Reference Values to Diagnose Periodontal Disease. A more attractive definition of disease is the therapeutic, or treatment-based, diagnosis. Under this definition, a person is defined as diseased only if the diagnosis of disease leads to tangible benefits. Most commonly, it is best to avoid diagnosing disease unless it can be shown that the diagnosis and the subsequent treatment actually provide tangible patient outcomes. Based on such an approach to diagnoses, periodontal disease should only be diagnosed if it leads to less morbidity.

Periodontal Disease Diagnoses

The Medical Subject Heading (MESH) term headings for periodontal disease, the classification systems for periodontal diseases developed by professional organizations, and a sampling of English-language periodontal textbooks indicates that periodontal disease diagnoses come and go at a fast rate. On PubMed, seven different entry terms are currently listed under the MESH heading of *Periodontitis,* reflecting some of the distinct periodontal diagnoses that have been used in the literature since 1965. A recent conference consensus[2] concluded that five of these seven terms listed in MESH are obsolete. The American Academy of Periodontology (AAP) reported 10 different classification systems in 20 years.[4] Periodontal textbooks have similarly reported different sets of periodontal diagnoses every decade.

Periodontal dystrophies provide one example on the apparent arbitrariness by which periodontal diagnoses come and go. Periodontal dystrophies were commonly reported from the eighteenth century until the 1960s. This diagnosis was rendered obsolete after two World Conferences in the 1970s concluded that some "as yet unclassified microorganism was found deep in the periodontal pocket."[56] Consequently, subsequent periodontal textbooks no longer referred to the diagnosis of *periodontosis.* Recently, an argument was made that this diagnosis should be resurrected.[53]

This example illustrates how profoundly the belief that "periodontal disease is an infectious disease" has influenced all aspects of clinical periodontics, *including* the system for classifying periodontal conditions. Periodontal diagnoses are currently based on the premise that periodontal diseases "follow an infection/host paradigm in which is it held that noxious materials from dental plaque bacteria induce an inflammatory response in the adjacent periodontal tissue … Central to this paradigm is the notion that the destruction of periodontal tissues is accompanied by an inflammatory response."[3] This means that even if the inflammatory response is not seen clinically, as in periodontosis, and if the microorganism cannot be identified, as is the case for periodontosis, it can still be theorized that the infection paradigm must be true and that inflammation that cannot be clinically observed and unidentifiable microbes must be at play.

Such beliefs on "periodontal-disease-as-an-infection" for creating a diagnostic classification of periodontal diseases are questionable on two points. First, high-level evidence from epidemiologic studies is required to determine that the belief on periodontal disease as an infection is valid. The emerging evidence suggests that smoking and diabetes may cause destructive periodontal disease independent of microbial colonizations, bringing into question this premise. Second, for chronic diseases with multiple causes, it is not possible to determine the cause of the disease and therefore it is of little clinical value to name the disease after a suspected cause. For example, periodontal disease in a diabetic patient cannot be referred to diabetic periodontitis. The clinician can only say that there is certain probability that the periodontal disease in a diabetic patient can be attributed to the diabetic condition.

From a clinical perspective, the ever-changing diagnostic classification systems that result from consensus conferences are largely irrelevant, since no reliable evidence exists that clinical use of such diagnostic systems improve patient outcomes. From a clinical perspective, the classification system developed by the American Dental Association (ADA) and the AAP has more clinical relevance because it provides information to the patient with respect to severity and prognosis and is free of assumptions when it comes to potential etiologic factors. The following four periodontal diagnoses are commonly recognized in the permanent dentition:

Case type I: Gingivitis
Case type II: Early periodontitis
Case type III: Moderate periodontitis
Case type IV: Advanced periodontitis

Such simple diagnostic systems of periodontal disease have many advantages in patient care because they provide useful information on both the severity of the disease and the prognosis.

UNITED STATES PREVALENCE DATA OF PERIODONTAL DISEASE

The US is one of the few countries where detailed information on changes in periodontal disease prevalence has been documented with national surveys (Dye, 2005) (see Tables 5-4 and 5-5). As a result, it may be worthwhile to take a closer look at the secular disease trends in this country as it may provide useful insights what may be happening worldwide.

But, before delving into the results of this report, the issue of defining periodontal disease needs to briefly revisited here (Section

TABLE 5-4 Periodontal Status of the United States Population among Adults

Adults (20–64 years)	1988–1994	1999–2004
Number of teeth present	24	25
Edentulism	6%	4%
Periodontal disease (one site with ≥3-mm attachment loss and ≥4-mm pocket depth	15%	9%
Periodontal disease among poor	28%	14%
Dental visits	66%	6%
Mean pocket depth	1.47 mm	1.02 mm
Mean loss of attachment	1.07 mm	0.72 mm
≥2 mm recession in at least one site	32%	21%
≥4 mm pocket depth in at least one site	23%	10%
≥4 mm attachment loss in at least one site	25%	17%

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TABLE 5-5 Periodontal Status of the United States Population among Seniors

Seniors (65 years and older)	1988–1994	1999–2004
Number of teeth present	18	19
Edentulism	34%	27%
Periodontal disease (one site with ≥3-mm attachment loss and ≥4-mm pocket depth	19.5%	10.5%
Periodontal disease among poor	26.3	16.6
Dental visits	54%	55%
Mean pocket depth	1.47 mm	1.07 mm
Mean loss of attachment	2.04 mm	1.55 mm
≥2-mm recession in at least one site	73%	48%
≥4-mm pocket depth in at least one site	22%	12%
≥4-mm attachment loss in at least one site	59%	50%

X). The use of expert-definitions of periodontal disease, as opposed to evidence-based definitions, causes the same issues in challenges in interpreting national survey data, as it does in interpreting research on treatment or etiology. Consider the following example: in the 1994–2000 survey, it was reported that approximately 1 in 10 seniors 75 years and older have periodontal disease (11.3%). If 1 in 10 seniors have periodontal disease, it appears plainly impossible that 2 in 10 seniors could have moderate-to-severe periodontitis (20.75%). Yet these are the statistics that are reported. Although several explanations are possible for such apparently contradictory findings, the arbitrary definitions of periodontal disease offer one plausible explanation (see Section X). The myriad of non-evidence based decisions for defining periodontal disease allows for a myriad of prevalence estimates that are not always logically consistent with each other. These expert decisions include which periodontal outcome measures to select, which cut-off values to choose for each of the selected outcome measures, at which periodontal locations these particular conditions need to be present, and whether these conditions need to co-occur at the same site. This multiplicity of choices is problematic for much of the periodontal research, including national surveys and can make prevalence estimates swing widely.

Regardless of these challenges in interpreting national survey data, the reported changes in periodontal disease parameters between the NHANES 1998–2004 survey and the 1994–2004 survey—and other sources of data—consistently suggest substantial improvements in periodontal disease prevalence (at least in terms of pocket depth and attachment levels) (Dye, 2005). Among adults, edentulism dropped by 2%, average number of teeth present in the oral cavity increased by one tooth per mouth, prevalence of moderate-to-severe periodontitis dropped by 4.3%, and prevalence of dental visits dropped by 6%. Among poor adults, prevalence of periodontitis dropped by 14%. Similar large changes occurred among the seniors. Edentulism dropped by 7%, average number of teeth present in the oral cavity increased by one tooth, prevalence of moderate-to-severe periodontitis dropped by 9%, and prevalence of dental visits remained unchanged. Among the poor seniors, prevalence of periodontitis dropped by almost 10%. If it would turn out that periodontal pockets are a valid surrogate for periodontal disease, these data imply that a fraction of the observed reduction in edentulism and tooth loss in the US population may be due to a reduction in periodontal disease severity.

Although relating population-level statistics to suspected causal factors can be misleading, hypotheses regarding the causes of these rapid changes can be analyzed. Large declines in periodontal disease prevalence appear difficult to explain by improved periodontal care or more effective periodontal procedures. The dramatic declines in periodontal disease prevalence are most pronounced among those with the least access to dental care: the poor. Moreover, these large declines occurred when studies reported prior large decreases in periodontal treatment utilization. A more plausible explanation for the decrease in periodontal disease prevalence is cigarette smoking. The large declines may at least in part be attributed to the continued decline in smoking. While these surveys were conducted, the prevalence of current smokers in the United States continued to decline (http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5644a2.htm). Worldwide changes in smoking habits and periodontitis prevalence will provide further opportunities to either refute or confirm cigarette smoking as a primary driver of the worldwide periodontitis epidemic. Other suspected etiological factors of periodontal disease appear opposite of expectations. Two suspected causal factors of periodontal disease, diabetes and obesity, increased during this decade suggesting that the prevalence of periodontal pockets and periodontal attachment loss would similarly increase. Yet the opposite trend was documented.

Causal hypotheses based on aggregate data have led to misleading conclusions on topics such as sugar consumption and dental caries, therefore one should be cautious in generating hypotheses regarding the primary drivers of the periodontal disease epidemic based on successive national survey data. More detailed patient-level epidemiological analyses will be required to explore answers to these questions as to why the periodontal disease prevalence as measured in terms of periodontal pocket depths and attachment levels is decreasing so rapidly. However, what can be concluded is that prevalence of periodontal pockets and periodontal treatment needs were opposite of predicted: both the need and the actual utilization for periodontal services appears to be decreasing as rapidly as the disease itself is decreasing.

A final note of caution should be considered when interpreting these national prevalence data. It would be wrong to imply that the pandemic of dental diseases has been conquered. Substantial improvements have occurred in unequivocal parameters of dental suffering such as edentulism. However, dental diseases remain more prevalent than most systemic chronic diseases and remain one of the most costly chronic conditions to treat. The cost-effective primary prevention of dental diseases appears to remain an elusive goal for most civilized societies.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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PART 3

Periodontal Pathology

Fermin A. Carranza

Thorough knowledge of the microscopic tissue changes in disease is essential to comprehend the biologic nature of the periodontal responses to injury and healing. This knowledge also provides an indispensable basis for the understanding and interpretation of the clinical and radiographic findings encountered in dental patients.

Part 3 provides information of the gingival diseases (Section I) and periodontal diseases (Section II). This latter section also includes chapters with detailed descriptions of the different diseases that can affect the periodontium.

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CHAPTER 6

Defense Mechanisms of the Gingiva

Jaime Bulkacz and Fermin A. Carranza

CHAPTER OUTLINE

SULCULAR FLUID
- Methods of Collection
- Permeability of Junctional and Sulcular Epithelia
- Amount
- Composition
- Cellular and Humoral Activity in Gingival Crevicular Fluid
- Clinical Significance
- Drugs in Gingival Crevicular Fluid

LEUKOCYTES IN THE DENTOGINGIVAL AREA

SALIVA
- Antibacterial Factors
- Salivary Antibodies
- Enzymes
- Salivary Buffers and Coagulation Factors
- Leukocytes
- Role in Periodontal Pathology

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some text and figures may be out of numeric order in this printed chapter.

The gingival tissue is constantly subjected to mechanical and bacterial aggressions. The saliva, the epithelial surface, and the initial stages of the inflammatory response provide resistance to these actions. Chapter 2 reviews the role of the epithelium, through its degree of keratinization and turnover rate. This chapter describes the permeability of the junctional and sulcular epithelia and the role of sulcular fluid, leukocytes, and saliva.

SULCULAR FLUID

The presence of sulcular fluid, or *gingival crevicular fluid* (GCF), has been known since the nineteenth century, but its composition and possible role in oral defense mechanisms were elucidated by the pioneering work of Waerhaug[122] and Brill and Krasse[14] in the 1950s. The latter investigators introduced filter paper into the gingival sulci of dogs previously injected intramuscularly with fluorescein; within 3 minutes the fluorescent material was recovered on the paper strips. This indicated the passage of fluid from the bloodstream through the tissues and exiting via the gingival sulcus.

In subsequent studies, Brill[11,12] confirmed the presence of GCF in humans and considered it a "transudate." However, others[73,123] demonstrated that GCF is an inflammatory exudate, not a continuous transudate. In strictly normal gingiva, little or no fluid can be collected.

More recently, interest in the development of tests for the detection or prediction of periodontal disease has resulted in numerous research papers on the components, origin, and function of GCF.

Methods of Collection

The most difficult hurdle to overcome when collecting GCF is the scarcity of material that can be obtained from the sulcus. Many collection methods have been tried.* These methods include the use of absorbing paper strips, twisted threads placed around and into the sulcus, micropipettes, and intracrevicular washings.

For more information on methods of collection and to view Figure 6-1, please visit the companion Expert CONSULT website at www.expertconsult.com.

Permeability of Junctional and Sulcular Epithelia

The initial studies by Brill and Krasse[14] with fluorescein were later confirmed with substances such as India ink[95] and saccharated iron oxide.[21] Substances shown to penetrate the sulcular epithelium include albumin,[94] endotoxin,[93,98] thymidine,[49] histamine,[28] phenytoin,[114] and horseradish peroxidase.[79] These findings indicate

*References 10, 13, 59, 63, 75, 76, 101.

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permeability to substances with a molecular weight of up to 1000 kD.

Squier and Johnson[113] reviewed the mechanisms of penetration through an intact epithelium. Intercellular movement of molecules and ions along intercellular spaces appears to be a possible mechanism. Substances taking this route do not traverse the cell membranes.

Amount

The amount of GCF collected on a paper strip can be evaluated in a variety of ways. The wetted area can be made more visible by staining with ninhydrin; it is then measured planimetrically on an enlarged photograph or with a magnifying glass or a microscope.

An electronic method has been devised for measuring the fluid collected on a "blotter" (Periopaper), employing an electronic transducer (Periotron, Harco Electronics, Winnipeg, Manitoba, Canada) (Figure 6-2). The wetness of the paper strip affects the flow of an electronic current and gives a digital readout. A comparison of the ninhydrin-staining method and the electronic method performed in vitro revealed no significant differences between the two techniques.[116]

The amount of GCF collected is extremely small. Measurements performed by Cimasoni[21] showed that a strip of paper 1.5 mm wide and inserted 1 mm within the gingival sulcus of a slightly inflamed gingiva absorbs about 0.1 mg of GCF in 3 minutes. Challacombe[19] used an isotope dilution method to measure the amount of GCF present in a particular space at any given time. His calculations in human volunteers with a mean gingival index of less than 1 showed that the mean GCF volume in proximal spaces from molar teeth ranged from 0.43 to 1.56 µl.

Composition

The components of GCF can be characterized according to individual proteins,[73,85,103] specific antibodies, antigens,[35,92] and enzymes of several specificities.[15] The GCF also contains cellular elements.[28,31,125]

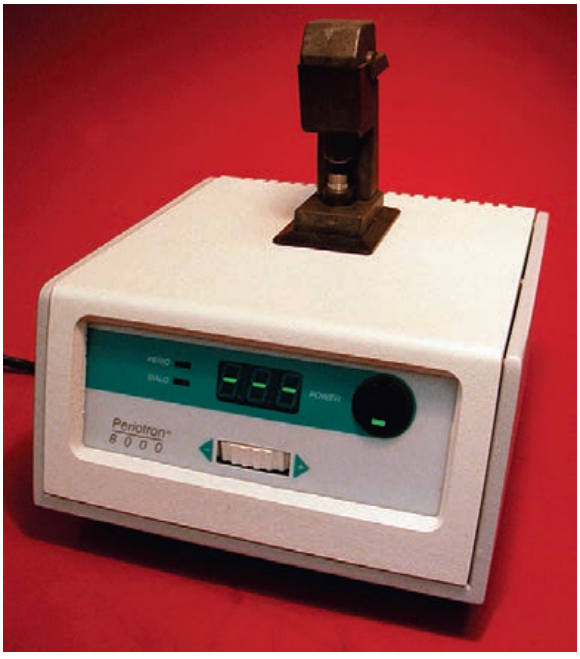

Figure 6-2 Electronic device for measuring the amount of fluid collected on filter paper.

Many research efforts have attempted to use GCF components to detect or diagnose active disease or to predict patients at risk for periodontal disease.[2] So far, more than 40 compounds found in GCF have been analyzed,[89] but their origin is not known with certainty. These compounds can be host derived or produced by the bacteria in the gingival crevice, but their source can be difficult to elucidate; examples include β-glucuronidase, a lysosomal enzyme, and lactic acid dehydrogenase, a cytoplasmic enzyme. The source for collagenases may be fibroblasts or polymorphonuclear leukocytes (PMNs, neutrophils),[87] or collagenases may be secreted by bacteria.[35] Phospholipases are lysosomal and cytoplasmic enzymes but are also produced by microorganisms.[15] The majority of GCF elements detected thus far have been enzymes, but there are nonenzymatic substances as well.

Cellular Elements. Cellular elements found in GCF include bacteria, desquamated epithelial cells, and leukocytes (PMNs, lymphocytes, monocytes/macrophages), which migrate through the sulcular epithelium.[28,31]

Electrolytes. Potassium, sodium, and calcium have been studied in GCF. Most studies have shown a positive correlation of calcium and sodium concentrations and the sodium/potassium ratio with inflammation.[56-58] (For further information, see references 12 and 13 and Box 6-2 online).

Organic Compounds. Both carbohydrates and proteins have been investigated. Glucose hexosamine and hexuronic acid are two of the compounds found in GCF.[47] Blood glucose levels do not correlate with GCF glucose levels; glucose concentration in GCF is three to four times greater than that in serum.[47] This is interpreted not only as a result of metabolic activity of adjacent tissues but also as a function of the local microbial flora.

The total protein content of GCF is much less than that of serum.[13,14] No significant correlations have been found between the concentration of proteins in GCF and the severity of gingivitis, pocket depth, or extent of bone loss.[7]

Metabolic and bacterial products identified in GCF include lactic acid,[48] urea,[42] hydroxyproline,[91] endotoxins,[109] cytotoxic substances, hydrogen sulfide,[112] and antibacterial factors.[27]

For more information on organic compounds, please visit the companion Expert CONSULT website at www.expertconsult.com. The online version of this chapter includes Box 6-1: Enzymes and Other Compounds Reported in the gingival Crevicular Fluid and Box 6-2: Compounds and Enzymes (Products) of possible Bacterial Origin Detected in gingival Crevicular Fluid, as well as the complete list of references for both boxes.

Cellular and Humoral Activity in Gingival Crevicular Fluid

Monitoring periodontal disease is a complicated task because very few noninvasive procedures can follow the initiation and progress of the disease. Analysis of GCF constituents in health and disease may be extremely useful because of GCF's simplicity and because GCF can be obtained with noninvasive methods.

Analysis of GCF has identified cell and humoral responses in both healthy individuals and those with periodontal disease.[64] The cellular immune response includes the appearance of cytokines in

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GCF (see Box 6-1 online), but there is no clear evidence of a relationship between cytokines and disease. However, interleukin-1 alpha (IL-1α) and IL-1β are known to increase the binding of PMNs and monocytes/macrophages to endothelial cells, stimulate the production of prostaglandin E_2 (PGE_2) and release of lysosomal enzymes, and stimulate bone resorption.[68] Preliminary evidence also indicates the presence of interferon-α in GCF,[64] which may have a protective role in periodontal disease because of its ability to inhibit the bone resorption activity of IL-1β.[44]

Because the amount of fluid recoverable from gingival crevices is small, only the use of very sensitive immunoassays permits the analysis of the specificity of antibodies.[27] A study comparing antibodies in different crevices with serum antibodies directed at specific microorganisms did not provide any conclusive evidence about the significance of the antibody presence in GCF in periodontal disease.[64]

Even though the role of antibodies in the gingival defense mechanisms is difficult to ascertain, the consensus is that in a patient with periodontal disease, (1) a reduction in antibody response is detrimental, and (2) an antibody response plays a protective role.[65]

Clinical Significance

As mentioned previously, GCF is an inflammatory exudate.[73] Its presence in clinically normal sulci can be explained because gingiva that appears clinically normal invariably exhibits inflammation when examined microscopically.

The amount of GCF is greater when inflammation is present[34,106] and is sometimes proportional to the severity of inflammation.[88] GCF production is not increased by trauma from occlusion[78] but is increased by mastication of coarse foods, toothbrushing and gingival massage, ovulation,[71] hormonal contraceptives,[70] and smoking.[80] Other factors that influence the amount of GCF are circadian periodicity and periodontal therapy.

Circadian Periodicity. There is a gradual increase in GCF amount from 6 AM to 10 PM and a decrease afterward.[9]

Sex Hormones. Female sex hormones increase GCF flow, probably because they enhance vascular permeability.[68] Pregnancy, ovulation,[67] and hormonal contraceptives[69] all increase gingival fluid production.

Mechanical Stimulation. Chewing[12] and vigorous gingival brushing stimulate the flow of GCF. Even the minor stimuli represented by intrasulcular placement of paper strips increases the production of fluid.

Smoking. Smoking produces an immediate transient but marked increase in GCF flow.[80]

Periodontal Therapy. There is an increase in GCF production during the healing period after periodontal surgery.[3]

Drugs in Gingival Crevicular Fluid

Drugs that are excreted through the GCF may be used advantageously in periodontal therapy. Bader and Goldhaber[6] demonstrated in dogs that tetracyclines are excreted through the GCF; this finding triggered extensive research that showed a concentration of tetracyclines in GCF compared with serum.[43] Metronidazole is another antibiotic that has been detected in human GCF[32] (see Chapter 47).

LEUKOCYTES IN THE DENTOGINGIVAL AREA

Leukocytes have been found in clinically healthy gingival sulci in humans and experimental animals. The leukocytes found are predominantly PMNs. They appear in small numbers extravascularly in the connective tissue adjacent to the bottom of the sulcus; from there, they travel across the epithelium[18,45] to the gingival sulcus, where they are expelled (Figures 6-3 and 6-4).

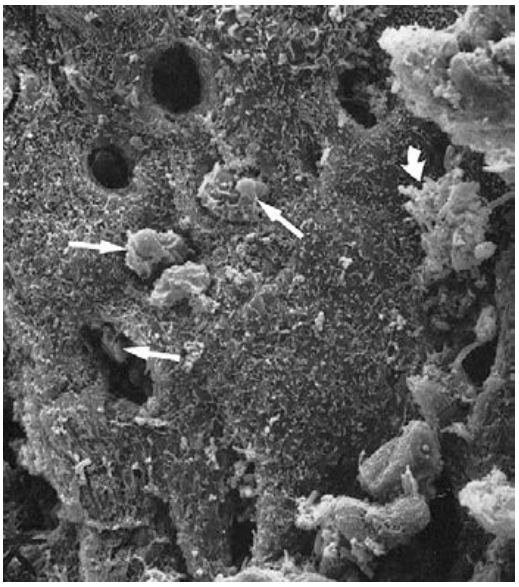

Figure 6-3 Scanning electron microscope view of periodontal pocket wall. Several leukocytes are emerging *(straight arrows)*, some partially covered by bacteria *(curved arrow)*. Empty holes correspond to tunnels through which leukocytes have emerged.

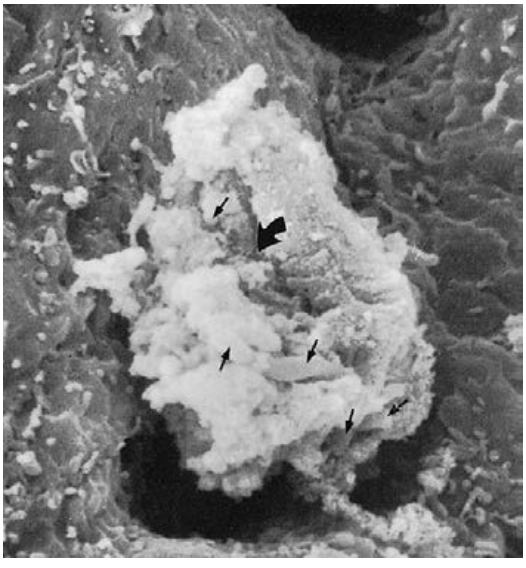

Figure 6-4 Scanning electron microscope view at higher magnification than Figure 6-3. A leukocyte emerging from the pocket wall is covered with bacteria *(small arrows)*. The large curved arrow points to a phagosomal vacuole through which bacteria are being engulfed.

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SCIENCE TRANSFER

Clinicians need to be aware of defense mechanisms of the gingiva because dental treatment can impair gingival defenses. The disruption of the epithelial lining of the gingival sulcus can occur with routine scaling and restorative procedures that involve the subgingival region, resulting in increased risk of inflammation as bacteria and their products have direct contact with the underlying connective tissue. The junctional and oral sulcular epithelium have the capacity to heal and reform the epithelial barrier in 7 to 10 days, and during this time, plaque control must be optimized to limit the risk of initiating periodontal breakdown. The use of gentle minimally traumatic techniques is also important.

Smoking may lessen the defense of the gingiva by impairing polynuclear leukocyte function in the tissues and the gingival sulcus. Further, smokers frequently have less gingival inflammation and bleeding on probing than nonsmokers with the same amount of periodontal attachment loss. Thus patients using tobacco should be carefully monitored for changes in pocket depth and given aggressive periodontal therapy.

Leukocytes are present in sulci even when histologic sections of adjacent tissue are free of inflammatory infiltrate. Differential counts of leukocytes from clinically healthy human gingival sulci have shown 91.2% to 91.5% PMNs and 8.5% to 8.8% mononuclear cells.[111,125]

Mononuclear cells were identified as 58% B lymphocytes, 24% T lymphocytes, and 18% mononuclear phagocytes. The ratio of T lymphocytes to B lymphocytes was found to be reversed from the normal ratio of about 3:1 found in peripheral blood to about 1:3 in GCF.[125]

Leukocytes are attracted by different plaque bacteria[54,124] but can also be found in the dentogingival region of germ-free adult animals.[74,100] Leukocytes were reported in the gingival sulcus in nonmechanically irritated (resting) healthy gingiva, indicating that their migration may be independent of an increase in vascular permeability.[5] The majority of these cells are viable and have phagocytic and killing capacity.[62,90,96] Therefore leukocytes constitute a major protective mechanism against the extension of plaque into the gingival sulcus.

Leukocytes are also found in saliva (see following discussion). The main port of entry of leukocytes into the oral cavity is the gingival sulcus.[104]

SALIVA

Salivary secretions are protective in nature because they maintain the oral tissues in a physiologic state (Table 6-1). Saliva exerts a major influence on plaque by mechanically cleansing the exposed oral surfaces, by buffering acids produced by bacteria, and by controlling bacterial activity.

Antibacterial Factors

Saliva contains numerous inorganic and organic factors that influence bacteria and their products in the oral environment. Inorganic factors include ions and gases, bicarbonate, sodium, potassium, phosphates, calcium, fluorides, ammonium, and carbon dioxide. Organic factors include lysozyme, lactoferrin, myeloperoxidase, lactoperoxidase, and agglutinins such as glycoproteins, mucins, β2-macroglobulins, fibronectins,[117] and antibodies.

Lysozyme is a hydrolytic enzyme that cleaves the linkage between structural components of the glycopeptide muramic acid–containing region of the cell wall of certain bacteria in vitro. Lysozyme works on both gram-negative and gram-positive organisms[50]; its targets include *Veillonella* species and *Actinobacillus actinomycetemcomitans*. It probably repels certain transient bacterial invaders of the mouth.[53]

TABLE 6-1 Role of Saliva In Oral Health

Function	Salivary Components	Probable Mechanism
Lubrication	Glycoproteins, mucoids	Coating similar to gastric mucin
Physical protection	Glycoproteins, mucoids	Coating similar to gastric mucin
Cleansing	Physical flow	Clearance of debris and bacteria
Buffering	Bicarbonate and phosphate	Antacids
Tooth integrity maintenance	Minerals Glycoprotein pellicle	Maturation, remineralization Mechanical protection
Antibacterial action	Immunoglobulin A	Control of bacterial colonization
	Lysozyme	Breaks bacterial cell walls
	Lactoperoxidase	Oxidation of susceptible bacteria

The *lactoperoxidase-thiocyanate system* in saliva has been shown to be bactericidal to some strains of *Lactobacillus* and *Streptococcus*[82,99] by preventing the accumulation of lysine and glutamic acid, both of which are essential for bacterial growth. Another antibacterial finding is lactoferrin, which is effective against *Actinobacillus* species.[55]

Myeloperoxidase, an enzyme similar to salivary peroxidase, is released by leukocytes and is bactericidal for *Actinobacillus*[81] but has the added effect of inhibiting the attachment of *Actinomyces* strains to hydroxyapatite.[16]

Salivary Antibodies

As with GCF, saliva contains antibodies that are reactive with indigenous oral bacterial species. Although immunoglobulins G (IgG) and M (IgM) are present, the preponderant immunoglobulin found in saliva is *immunoglobulin A* (IgA). However, IgG is more prevalent in GCF.[115] Major and minor salivary glands contribute all the secretory IgA (sIgA) and lesser amounts of IgG and IgM.

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GCF contributes most of the IgG, complement, and PMNs that in conjunction with IgG or IgM inactivate or opsonize bacteria.

Salivary antibodies appear to be synthesized locally because they react with strains of bacteria indigenous to the mouth but not with organisms characteristic of the intestinal tract.[36,38] Many bacteria found in saliva have been shown to be coated with IgA, and the bacterial deposits on teeth contain both IgA and IgG in quantities greater than 1% of their dry weight.[37] It has been shown that IgA antibodies present in parotid saliva can inhibit the attachment of oral *Streptococcus* species to epithelial cells.[33,122] Gibbons et al[36-38] suggested that antibodies in secretions may impair the ability of bacteria to attach to mucosal or dental surfaces.

Enzymes

The *enzymes* normally found in the saliva are derived from the salivary glands, bacteria, leukocytes, oral tissues, and ingested substances; the major enzyme is parotid amylase. Certain salivary enzymes have been reported in increased concentrations in periodontal disease: hyaluronidase and lipase,[17] β-glucuronidase and chondroitin sulfatase,[41] aspartate aminotransferase and alkaline phosphatase,[119] amino acid decarboxylases,[41] catalase, peroxidase, and collagenase.[60]

Proteolytic enzymes in the saliva are generated by both the host and oral bacteria. These enzymes have been recognized as contributors to the initiation and progression of periodontal disease.[49,77] To combat these enzymes, saliva contains antiproteases that inhibit cysteine proteases such as cathepsins[51] and antileukoproteases that inhibit elastase.[87] Another antiprotease, identified as a tissue inhibitor of matrix metalloproteinase (TIMP), has been shown to inhibit the activity of collagen-degrading enzymes.[26]

High-molecular-weight mucinous glycoproteins in saliva bind specifically to many plaque-forming bacteria. The glycoprotein-bacteria interactions facilitate bacterial accumulation on the exposed tooth surface.[33,36-38,124] The specificity of these interactions has been demonstrated. The interbacterial matrix of human plaque appears to contain polymers similar to salivary glycoproteins that may aid in maintaining the integrity of plaque. In addition, these glycoproteins selectively adsorb to the hydroxyapatite to make up part of the acquired pellicle. Other salivary glycoproteins inhibit the sorption of some bacteria to the tooth surface and to epithelial cells of the oral mucosa. This activity appears to be associated with the glycoproteins that possess blood group reactivity.[1,33,36,38,122] Another effect of mucin is the deletion of bacterial cells from the oral cavity by aggregation with mucin-rich films.

Glycoproteins and a glycolipid present on mammalian cell surfaces appear to serve as receptors for the attachment of some viruses and bacteria. Thus the close similarity between glycoproteins of salivary secretions and components of the epithelial cell surface suggests that the secretions can competitively inhibit antigen sorption and therefore may limit pathologic alterations.

Salivary Buffers and Coagulation Factors

The maintenance of physiologic hydrogen ion concentration (pH) at the mucosal epithelial cell surface and the tooth surface is an important function of salivary buffers. Their primary effect has been studied in relationship to dental caries. In saliva the most important salivary buffer is the bicarbonate–carbonic acid system.[76]

Saliva also contains coagulation factors (factors VIII, IX, and X; plasma thromboplastin antecedent [PTA]; Hageman factor) that hasten blood coagulation and protect wounds from bacterial invasion.[66] An active fibrinolytic enzyme may also be present.

Leukocytes

In addition to desquamated epithelial cells, the saliva contains all forms of leukocytes, of which the principal cells are PMNs. The number of PMNs varies from person to person at different times of the day and is increased in gingivitis. PMNs reach the oral cavity by migrating through the lining of the gingival sulcus. Living PMNs in saliva are sometimes referred to as *orogranulocytes,* and their rate of migration into the oral cavity is termed the *orogranulocytic migratory rate.* Some investigators believe the rate of migration correlates with the severity of gingival inflammation and is therefore a reliable index for assessing gingivitis.[111]

Role in Periodontal Pathology

Saliva exerts a major influence on plaque initiation, maturation, and metabolism. Salivary flow and composition also influence calculus formation, periodontal disease, and caries. The removal of the salivary glands in experimental animals significantly increases the incidence of dental caries[39] and periodontal disease[46] and delays wound healing.[107]

In humans, an increase in inflammatory gingival diseases, dental caries, and rapid tooth destruction associated with cervical or cemental caries is partially a consequence of decreased salivary gland secretion (xerostomia). Xerostomia may result from sialolithiasis, sarcoidosis, Sjögren's syndrome, Mikulicz's disease, irradiation, surgical removal of the salivary glands, and other factors (see Chapters 37 and 39).

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 7

Gingival Inflammation

Joseph P. Fiorellini, David M. Kim, N. Guzin Uzel

CHAPTER OUTLINE

STAGE I GINGIVITIS: THE INITIAL LESION
STAGE II GINGIVITIS: THE EARLY LESION
STAGE III GINGIVITIS: THE ESTABLISHED LESION
STAGE IV GINGIVITIS: THE ADVANCED LESION

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Pathologic changes in gingivitis are associated with the presence of oral microorganisms attached to the tooth and perhaps in or near the gingival sulcus.

For more information, please visit the companion Expert CONSULT website at www.expertconsult.com.

Morphologic and functional changes in the gingiva during plaque accumulation have been thoroughly investigated, especially in beagle dogs and humans.[39] A useful framework for organization and consideration of these data has been devised based on histopathologic, radiographic, and ultrastructural features and biochemical measurements.[40,42] The sequence of events cumulating in clinically apparent gingivitis is categorized into *initial*, *early*, and *established stages*, with periodontitis designated as the *advanced stage*[41] (Table 7-1). One stage evolves into the next, with no clear-cut dividing lines.

Despite extensive research, we still cannot distinguish definitively between normal gingival tissue and the initial stage of gingivitis.[39] Most biopsies of clinically normal human gingiva contain inflammatory cells consisting predominantly of T cells, with very few B cells or plasma cells.[39,53,54] These cells do not create tissue damage, but they appear to be important in the day-to-day host response to bacteria and other substances to which the gingiva is exposed.[39] Therefore, under normal conditions, a constant stream of neutrophils is migrating from the vessels of the gingival plexus through the junctional epithelium, to the gingival margin, and into the gingival sulcus and oral cavity.[46]

STAGE I GINGIVITIS: THE INITIAL LESION

The first manifestations of gingival inflammation are vascular changes consisting of dilated capillaries and increased blood flow. These initial inflammatory changes occur in response to microbial activation of resident leukocytes and the subsequent stimulation of endothelial cells. Clinically, this initial response of the gingiva to bacterial plaque (*subclinical gingivitis*[28]) is not apparent.

Microscopically, some classic features of acute inflammation can be seen in the connective tissue beneath the junctional epithelium. Changes in blood vessel morphologic features (e.g., widening of small capillaries or venules) and adherence of neutrophils to vessel walls (margination) occur within 1 week and sometimes as early as 2 days after plaque has been allowed to accumulate[19,44] (Figure 7-1). Leukocytes, mainly polymorphonuclear neutrophils (PMNs), leave the capillaries by migrating through the walls (diapedesis, emigration)[27,54,55] (Figure 7-2). They can be seen in increased quantities in the connective tissue, the junctional epithelium, and the gingival sulcus* (Figures 7-3 and 7-4). Exudation of fluid from the gingival sulcus[19] and extravascular proteins are present.[21,22]

* References 2, 3, 26, 37, 44, 49, 50.

However, these findings are not accompanied by manifestations of tissue damage perceptible at the light microscopic or ultrastructural level; they do not form an infiltrate, and their presence is not considered to indicate pathologic change.[39]

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TABLE 7-1 Stages of Gingivitis

Stage	Time (Days)	Blood Vessels	Junctional and Sulcular Epithelia	Predominant Immune Cells	Collagen	Clinical Findings
I. Initial lesion	2–4	Vascular dilation Vasculitis	Infiltration by PMNs	PMNs	Perivascular loss	Gingival fluid flow
II. Early lesion	4–7	Vascular proliferation	Same as stage I Rete pegs Atrophic areas	Lymphocytes	Increased loss around infiltrate	Erythema Bleeding on probing
III. Established lesion	14–21	Same as stage II, plus blood stasis	Same as stage II but more advanced	Plasma cells	Continued loss	Changes in color, size, texture, etc.

PMNs, Polymorphonuclear leukocytes (neutrophils).

SCIENCE TRANSFER

The initial lesion of gingivitis is focused on acute inflammation with vasodilation, edema, and accumulation of polymorphonuclear leukocytes. There is also ulceration of the epithelium lining the gingival sulcus so that bleeding on probing is present. In some patients, this is seen as early as 2 days after plaque accumulates and can be successfully treated by plaque and calculus removal. The plaque accumulation is then controlled by oral hygiene so that the gingiva can return to health within 7 to 10 days.

When gingivitis is established, the initial acute response is accompanied by chronic inflammation with emphasis on lymphocytic and plasma cell accumulation, capillary proliferation, and collagen destruction. This too can be reversed with initial therapy but will require a longer period for gingival tissues to heal and restore lost collagen fibers.

In some patients, gingivitis proceeds to periodontitis, which is much more difficult to treat. Therefore clinicians must be vigilant to detect the early stages of gingivitis and carry out effective treatment coupled with ongoing monitoring and therapy as part of a recall protocol.

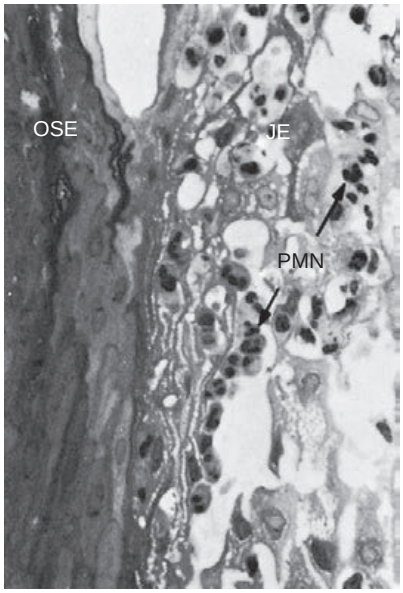

Figure 7-1 Human biopsy sample, experimental gingivitis. After 4 days of plaque accumulation, the blood vessels immediately adjacent to the junctional epithelium are distended and contain polymorphonuclear leukocytes (*PMNs,* neutrophils). Neutrophils have also migrated between the cells of the junctional epithelium *(JE). OSE,* Oral sulcular epithelium. (Magnification ×500.) *(From Payne WA, Page RC, Ogilvie AL, et al:* J Periodontal Res *10:51, 1975.)*

Subtle changes can also be detected in the junctional epithelium and perivascular connective tissue at this early stage. For example, the perivascular connective tissue matrix becomes altered, and there is exudation and deposition of fibrin in the affected area.[39] Also, lymphocytes soon begin to accumulate (see Figure 7-2, *D*). The increase in the migration of leukocytes and their accumulation within the gingival sulcus may be correlated with an increase in the flow of gingival fluid into the sulcus.[4]

The character and intensity of the host response determine whether this *initial lesion* resolves rapidly, with the restoration of the tissue to a normal state, or evolves into a chronic inflammatory lesion. If the latter occurs, an infiltrate of macrophages and lymphoid cells appears within a few days.

STAGE II GINGIVITIS: THE EARLY LESION

The *early lesion* evolves from the *initial lesion* within about 1 week after the beginning of plaque accumulation.[38,44] Clinically, the early lesion may appear as early gingivitis, and it overlaps with and evolves from the initial lesion with no clear-cut dividing line. As time goes on, clinical signs of erythema may appear, mainly because of the proliferation of capillaries and increased formation of capillary loops between rete pegs or ridges (Figure 7-5). Bleeding on probing may also be evident.[1] Gingival fluid flow and the numbers of transmigrating leukocytes reach their maximum between 6 and 12 days after the onset of clinical gingivitis.[28]

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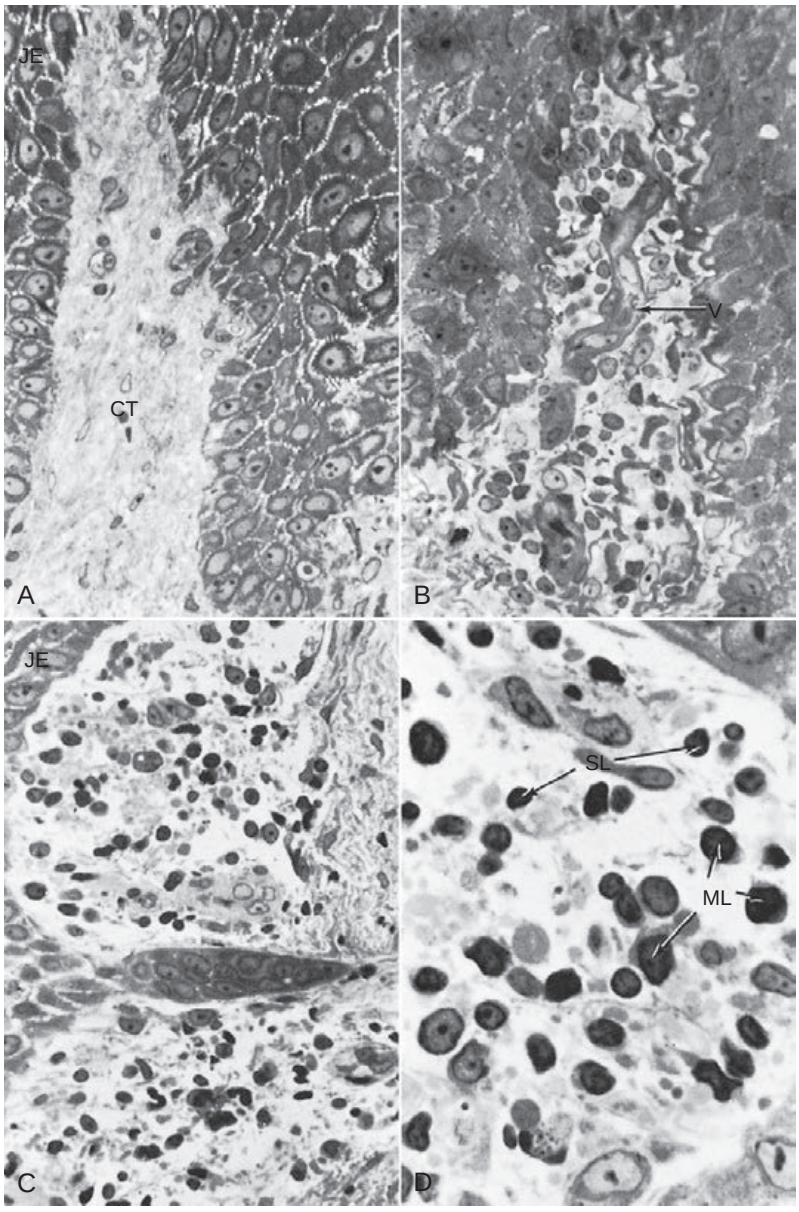

Figure 7-2 Human biopsy, experimental gingivitis. **A,** Control biopsy specimen from a patient with good oral hygiene and no detectable plaque accumulation. The junctional epithelium is at the left. The connective tissue *(CT)* shows few cells other than fibroblasts, blood vessels, and a dense background of collagen fibers. (×500.) **B,** Biopsy specimen taken after 8 days of plaque accumulation. The connective tissue is infiltrated with inflammatory cells, which displace the collagen fibers. A distended blood vessel *(V)* is seen in the center. (×500.) **C,** After 8 days of plaque accumulation, the connective tissue next to the junctional epithelium at the base of the sulcus shows a mononuclear cell infiltrate and evidence of collagen degeneration (clear spaces around cellular infiltrate). (×500.). **D,** The inflammatory cell infiltrate at higher magnification. After 8 days of plaque accumulation, numerous small *(SL)* and medium-sized *(ML)* lymphocytes are seen within the connective tissue. Most of the collagen fibers around these cells have disappeared, presumably as a result of enzymatic digestion. (×1250.) *(From Payne WA, Page RC, Ogilvie AL, et al:* J Periodontal Res *10:51, 1975.)*

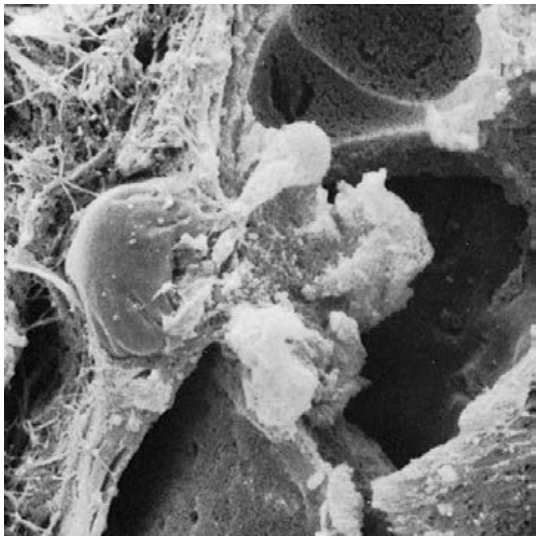

Figure 7-3 Scanning electron micrograph showing a leukocyte traversing the vessel wall to enter into the gingival connective tissue.

Microscopic examination of the gingiva reveals a leukocyte infiltration in the connective tissue beneath the junctional epithelium, consisting mainly of lymphocytes (75%, with the majority T cells)[44,51] but also composed of some migrating neutrophils, as well as macrophages, plasma cells, and mast cells. All the changes seen in the initial lesion continue to intensify with the early lesion.[16,30,32,38,51] The junctional epithelium becomes densely infiltrated with neutrophils, as does the gingival sulcus, and the junctional epithelium may begin to show development of rete pegs or ridges.

The amount of collagen destruction increases[12,30,51]; 70% of the collagen is destroyed around the cellular infiltrate. The main fiber groups affected appear to be the circular and dentogingival fiber assemblies. Alterations in blood vessel morphologic features and vascular bed patterns have also been described.[19,20]

PMNs that have left the blood vessels in response to chemotactic stimuli from plaque components travel to the epithelium,

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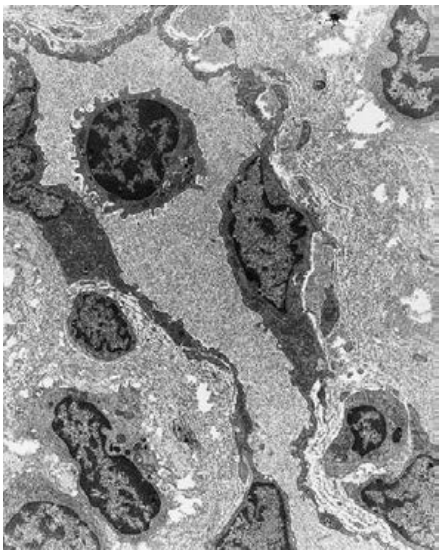

Figure 7-4 Early human gingivitis lesion. Area of lamina propria subjacent to the crevicular epithelium showing a capillary with several extravascular lymphocytes and one lymphocyte within the lumen. The specimen also exhibits considerable loss of perivascular collagen density. (×2500.) *(Courtesy Dr. Charles Cobb, Kansas City, MO.)*

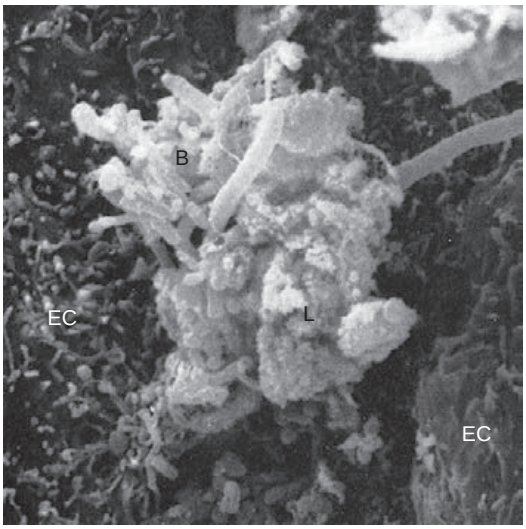

Figure 7-6 Scanning electron micrograph of leukocyte emerging to pocket wall and covered with bacteria and extracellular lysosomes. *EC*, Epithelial cells; *B*, bacteria; *L*, lysosomes.

Figure 7-5 Marginal gingivitis and irregular gingival contour.

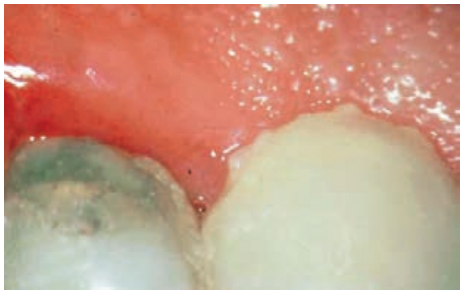

Figure 7-7 Marginal supragingival plaque and gingivitis.

cross the basement lamina, and are found in the epithelium, emerging in the pocket area (see Figure 7-3). PMNs are attracted to bacteria and engulf them in the process of phagocytosis (Figure 7-6). PMNs release their lysosomes in association with the ingestion of bacteria.[24] Fibroblasts show cytotoxic alterations,[43] with a decreased capacity for collagen.

Meanwhile, on the opposite side of molecular events, collagen degradation is related to matrix metalloproteins (MMPs). Different MMPs are responsible for extracellular matrix remodeling within 7 days of inflammation, which is directly related to MMP-2 and MMP-9 production and activation.[58]

STAGE III GINGIVITIS: THE ESTABLISHED LESION

Over time, the *established lesion* evolves, characterized by a predominance of plasma cells and B lymphocytes and probably in conjunction with the creation of a small gingival pocket lined with a pocket epithelium.[50] The B cells found in the established lesion are predominantly of the immunoglobulin G1 (IgG1) and G3 (IgG3) subclasses.[39]

In chronic gingivitis, which occurs 2 to 3 weeks after the beginning of plaque accumulation, the blood vessels become engorged and congested, venous return is impaired, and the blood flow becomes sluggish (Figure 7-7). The result is localized gingival *anoxemia*, which superimposes a somewhat bluish hue on the reddened gingiva.[18] Extravasation of erythrocytes into the connective tissue and breakdown of hemoglobin into its component pigments can also deepen the color of the chronically inflamed gingiva. The established lesion can be described as moderately to severely inflamed gingiva.

In histologic sections, an intense, chronic inflammatory reaction is observed. Several detailed cytologic studies have been performed on chronically inflamed gingiva.* A key feature that differentiates the established lesion is the increased number of plasma cells, which become the preponderant inflammatory cell type. Plasma cells invade the connective tissue not only immediately below the junctional epithelium but also deep into the connective tissue, around blood vessels, and between bundles of collagen fibers.[6] The junctional epithelium reveals widened intercellular spaces filled with granular cellular debris, including lysosomes derived from disrupted neutrophils, lymphocytes, and monocytes (Figure 7-8). The lysosomes contain acid hydrolases that can destroy tissue components. The junctional epithelium develops rete pegs or ridges that protrude into the connective tissue, and the basal lamina is destroyed in some areas. In the connective tissue, collagen fibers are destroyed around the infiltrate of intact and disrupted plasma cells, neutrophils, lymphocytes, monocytes, and mast cells (Figure 7-9).

* References 13, 15, 16, 43, 50, 53, 57.

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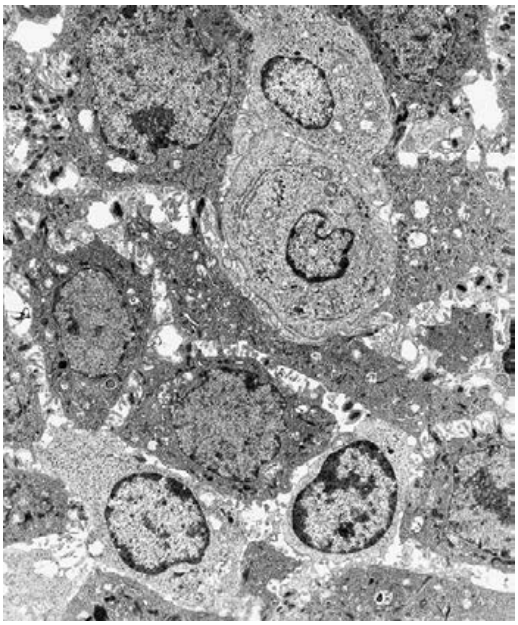

Figure 7-8 Established gingivitis in a human subject. Area of crevicular epithelium exhibiting enlarged intercellular spaces with numerous microvilli and desmosomal junctions. Several lymphocytes, both small and large, are seen migrating through the epithelial layer. (×3000.)

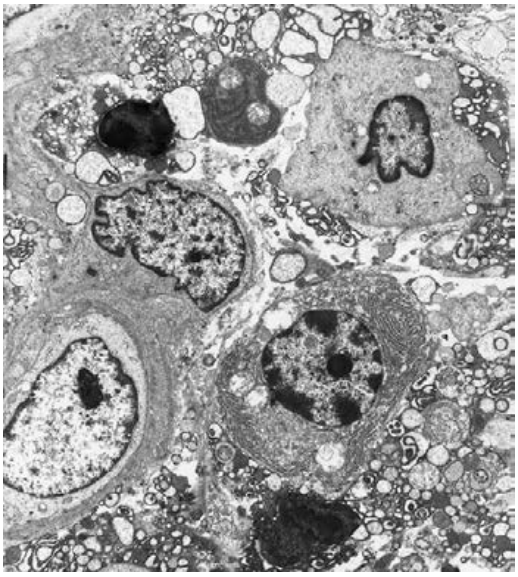

Figure 7-9 Advanced gingivitis in a human subject. Specimen from the lamina propria exhibiting plasma cell degeneration, with abundant cellular debris visible. (×3000.) *(Courtesy Dr. Charles Cobb, Kansas City, MO.)*

The predominance of plasma cells is thought to be a primary characteristic of the established lesion. However, several studies of human experimental gingivitis have failed to demonstrate plasma cell predominance in the affected connective tissues,[7,8,53] including one study of 6 months' duration.[1] Increases in the proportions of plasma cells were evident with long-standing gingivitis, but the time for the development of the classic "established lesion" may exceed 6 months.

An inverse relationship appears to exist between the number of intact collagen bundles and the number of inflammatory cells.[55] Collagenolytic activity is increased in inflamed gingival tissue[17] by the enzyme collagenase. Collagenase is normally present in gingival tissues[5] and is produced by some oral bacteria and by PMNs.

Enzyme histochemistry studies have shown that chronically inflamed gingivae have elevated levels of acid and alkaline phosphatase,[60] β-glucuronidase, β-glucosidase, β-galactosidase, esterases,[31] aminopeptidase,[36,45] and cytochrome oxidase.[9] Neutral mucopolysaccharide levels are decreased,[57] presumably as a result of degradation of the ground substance.

Established lesions of two types appear to exist; some remain stable and do not progress for months or years,[35,36,56] and others seem to become more active and to convert to progressively destructive lesions. Also, the established lesions appear to be reversible in that the sequence of events occurring in the tissues as a result of successful periodontal therapy seems to be essentially the reverse of the sequence of events observed as gingivitis develops. As the flora reverts from that characteristically associated with destructive lesions to that associated with periodontal health, the percentage of plasma cells decreases greatly, and the lymphocyte population increases proportionately.[29,33]

STAGE IV GINGIVITIS: THE ADVANCED LESION

Extension of the lesion into alveolar bone characterizes a fourth stage known as the *advanced lesion*[43] or *phase of periodontal breakdown.*[28] This is described in detail in Chapters 13 and 14.

Microscopically, there is fibrosis of the gingiva and widespread manifestations of inflammatory and immunopathologic tissue damage.[39] At the advanced stage, plasma cell presence dominates connective tissue and neutrophils continue dominating the junctional epithelium.

Patients with experimental gingivitis showed significantly more plaque accumulation, higher IL-1β, and lower IL-8 concentrations at 28 days.[11]

Gingivitis will progress to periodontitis only in individuals who are susceptible. Patients who had sites with consistent bleeding (gingival index [GI] = 2) had 70% more attachment loss than at sites that were not inflamed consistently (GI = 0). Teeth with noninflamed sites consistently had a 50-year survival rate of 99.5%, whereas teeth with consistently inflamed gingiva had a 63.4% survival rate over 50 years. Based on this longitudinal study on the natural history of periodontitis in a well-maintained male population, persistent gingivitis represents a risk factor for periodontal attachment loss and for tooth loss.[23]

However, whether periodontitis can occur without a precursor of gingivitis is not known at this time.

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CHAPTER 8

Clinical Features of Gingivitis

Joseph P. Fiorellini, David M. Kim and N. Guzin Uzel

CHAPTER OUTLINE

CHANGES IN GINGIVAL CONTOUR
COURSE AND DURATION
DESCRIPTION
CLINICAL FINDINGS
Gingival Bleeding on Probing
Gingival Bleeding Caused by Local Factors
Gingival Bleeding Associated with Systemic Changes
Color Changes in the Gingiva
Color Changes Associated with Systemic Factors
Changes in Consistency of the Gingiva
Changes in Surface Texture of the Gingiva
Changes in Position of the Gingiva
Changes in Gingival Contour

CHANGES IN GINGIVAL CONTOUR

Experimental gingivitis studies provided the first empiric evidence that accumulation of microbial biofilm on clean tooth surfaces results in the development of an inflammatory process around gingival tissue.[41,69] Research has also shown that the local inflammation will persist as long as the microbial biofilm is present adjacent to the gingival tissues and that the inflammation may resolve subsequent to a meticulous removal of the biofilm.[69]

The prevalence of gingivitis is evident worldwide. For example, epidemiologic studies indicate that more than 82% of adolescents in the United States (US) have overt gingivitis and signs of gingival bleeding. A similar or higher prevalence of gingivitis is reported for children and adolescents in other parts of the world.[7] A significant percentage of adults also show signs of gingivitis; more than half the US adult population is estimated to exhibit gingival bleeding, and other populations show even higher levels of gingival inflammation.[3,5,6,8,66] Whereas plaque remains the primary etiological factor causing gingivitis, other factors may affect the development of periodontal diseases. Recent findings suggest that distinct biologic phenotypes may play a role in the development of gingivitis.[59]

In general, clinical features of gingivitis may be characterized by the presence of any of the following clinical signs: redness and sponginess of the gingival tissue, bleeding on provocation, changes in contour, and presence of calculus or plaque with no radiographic evidence of crestal bone loss.[10] Histologic examination of inflamed gingival tissue reveals ulcerated epithelium. The presence of inflammatory mediators negatively affects epithelial function as a protective barrier. Repair of this ulcerated epithelium depends on the proliferative or regenerative activity of the epithelial cells. Removal of the etiologic agents triggering the gingival breakdown is essential.

COURSE AND DURATION

Gingivitis can occur with sudden onset and short duration and can be painful. A less severe phase of this condition can also occur.

Recurrent gingivitis reappears after having been eliminated by treatment or disappeared spontaneously.

Chronic gingivitis is slow in onset and of long duration. It is painless, unless complicated by acute or subacute exacerbations, and is the type most often encountered (Figure 8-1). Chronic gingivitis is a fluctuating disease in which inflammation persists or resolves and normal areas become inflamed.[30,36]

DESCRIPTION

Localized gingivitis is confined to the gingiva of a single tooth or group of teeth, whereas *generalized gingivitis* involves the entire mouth.

Marginal gingivitis involves the gingival margin and may include a portion of the contiguous attached gingiva. *Papillary gingivitis* involves the interdental papillae and often extends into the adjacent portion of the gingival margin. Papillae are involved more frequently than the gingival margin, and the earliest signs of gingivitis often occur in the papillae. *Diffuse gingivitis* affects the gingival margin, the attached gingiva, and the interdental papillae. Gingival disease in individual cases is described by combining the preceding terms as follows:

- *Localized marginal gingivitis* is confined to one or more areas of the marginal gingiva (Figure 8-2).
- *Localized diffuse gingivitis* extends from the margin to the mucobuccal fold in a limited area (Figure 8-3).
- *Localized papillary gingivitis* is confined to one or more interdental spaces in a limited area (Figure 8-4).

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Figure 8-1 Chronic gingivitis. The marginal and interdental gingivae are smooth, edematous, and discolored. Isolated areas of acute response are seen.

Figure 8-2 Localized, diffuse, intensely red area facial of tooth #7 and dark-pink marginal changes in remaining anterior teeth.

Figure 8-3 Generalized marginal gingivitis in the upper jaw with areas of diffuse gingivitis.

Figure 8-4 Generalized papillary gingivitis.

- *Generalized marginal gingivitis* involves the gingival margins in relation to all the teeth. The interdental papillae are usually affected (Figure 8-5).
- *Generalized diffuse gingivitis* involves the entire gingiva. The alveolar mucosa and attached gingiva are affected, so the mucogingival junction is sometimes obliterated (Figure 8-6). Systemic conditions can be involved in the cause of generalized diffuse gingivitis and should be evaluated if suspected as an etiologic cofactor.

Figure 8-5 Generalized marginal and papillary gingivitis.

Figure 8-6 Generalized diffuse gingivitis involves the marginal, papillary, and attached gingivae.

CLINICAL FINDINGS

A *systematic* approach is required in evaluation of the clinical features of gingivitis. The clinician should focus on subtle tissue alterations because these may be of diagnostic significance. A systematic clinical approach requires an orderly examination of the gingiva for color, contour, consistency, position, and ease and severity of bleeding and pain. This section discusses these clinical characteristics and the microscopic changes responsible for each.

Gingival Bleeding on Probing

The two earliest signs of gingival inflammation preceding established gingivitis are (1) increased gingival crevicular fluid production rate and (2) bleeding from the gingival sulcus on gentle probing (Figure 8-7). Chapter 6 discusses gingival crevicular fluid in detail.

Gingival bleeding varies in severity, duration, and ease of provocation. Bleeding on probing is easily detected clinically and therefore is of value for the early diagnosis and prevention of more advanced gingivitis. It has been shown that bleeding on probing appears earlier than a change in color or other visual signs of inflammation[36,37,46]; in addition, the use of bleeding rather than color changes to diagnose early gingival inflammation is advantageous in that bleeding is a more objective sign that requires less subjective estimation by the examiner. For example, it is estimated that 53.2 million (50.3%) of US adults 30 years and older exhibit gingival bleeding.[6] Probing pocket depth measurements by themselves are of limited value for the assessment of the extent and severity of gingivitis. For example, gingival recession may result in reduction of the probing depth and thus cause an inaccurate assessment of the periodontal status.[4] Therefore bleeding on

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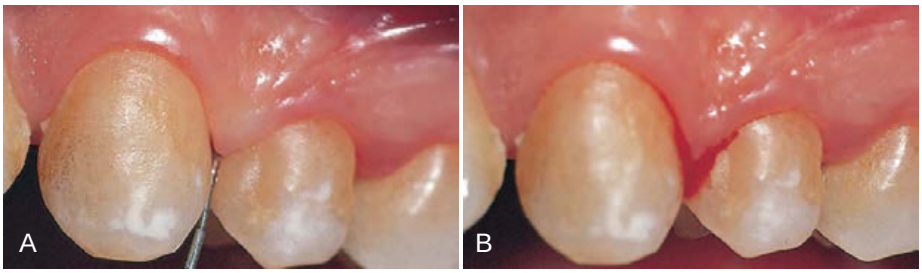

Figure 8-7 Bleeding on probing. **A,** Mild edematous gingivitis; a probe has been introduced to the bottom of the gingival sulcus. **B,** Bleeding appears after a few seconds.

probing is widely used by clinicians and epidemiologists to measure disease prevalence and progression, to measure outcomes of treatment, and to motivate patients with their home care.[24] Several gingival indices based on bleeding have been developed,[2,16,53] as described in Chapter 5. (For further considerations on probing, see Chapter 30.)

In general, gingival bleeding on probing indicates an inflammatory lesion both in the epithelium and in the connective tissue that exhibits specific histologic differences compared with healthy gingiva.[25] Even though gingival bleeding on probing may not be a good diagnostic indicator for clinical attachment loss, its absence is an excellent negative predictor of future attachment loss.[34] Therefore the absence of gingival bleeding on probing is desirable and implies a low risk of future clinical attachment loss. Longitudinal findings revealed that sites with consistent bleeding (gingival index [GI] = 2) had 70% more attachment loss than sites that were noninflamed over a 26-year period in 565 males. Thus persistent gingivitis can be considered as a risk factor for periodontal attachment loss that may lead to tooth loss.[35]

Interestingly, numerous studies show that current cigarette smoking suppresses the gingival inflammatory response, and smoking was found to exert a strong, chronic, dose-dependent suppressive effect on gingival bleeding on probing in the third National Health and Nutrition Examination Survey (NHANES III).[19] In addition, recent research reveals an increase in gingival bleeding on probing in patients who quit smoking.[54] Thus people who are committed to a smoking cessation program should be informed about the possibility of an increase in gingival bleeding associated with smoking cessation.

Gingival Bleeding Caused by Local Factors

Contributing factors to plaque retention that may lead to gingivitis include anatomic and developmental tooth variations, caries, frenum pull, iatrogenic factors, malpositioned teeth, mouth breathing, overhangs, partial dentures, lack of attached gingiva, and recession. In addition, orthodontic treatment and fixed retainers were associated with increased plaque retention and increased bleeding on probing.[38,78]

Chronic and Recurrent Bleeding. The most common cause of abnormal gingival bleeding on probing is chronic inflammation.[50] The bleeding is chronic or recurrent and is provoked by mechanical trauma (e.g., from toothbrushing, toothpicks, or food impaction) or by biting into solid foods such as apples.

Histologic evaluations on animal specimens revealed that in early stages of gingivitis, expression of cytokines responsible for connective tissue breakdown, matrix metalloproteinases (MMPs), is ubiquitous. Different MMPs play a role in this breakdown at different stages (e.g., a decrease of MMP-14 activity at 7 days of inflammation and an immediate increase in MMP-2, especially with fibroblastic stimulation). In addition, MMP-9 expression peaked 5 days after gingivitis occurrence, which was also regulated by macrophages and neutrophils. Thus extracellular matrix remodeling was regulated with MMP-2 and -9 production and activation by host inflammatory response.[42]

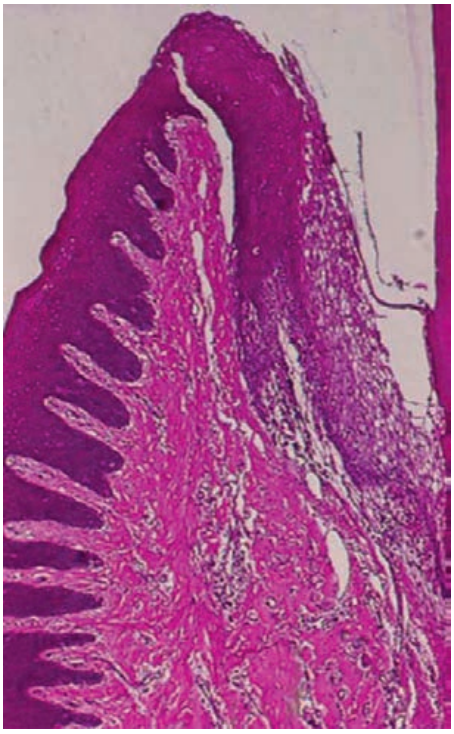

Figure 8-8 Microscopic view of interdental space in a human autopsy specimen. Note inflammatory infiltrate and thinned epithelium in area adjacent to the tooth, as well as collagenous tissue in outer half of the section.

In gingival inflammation, histopathologic alterations that result in abnormal gingival bleeding include dilation and engorgement of the capillaries and thinning or ulceration of the sulcular epithelium (Figure 8-8). Because the capillaries are engorged and closer to the surface, and the thinned, degenerated epithelium is less protective, stimuli that are normally innocuous cause rupture of the capillaries and gingival bleeding. Sites that bleed on probing have a greater area of inflamed connective tissue (i.e., cell-rich, collagen-poor tissue) than sites that do not bleed. In most cases the cellular infiltrate of sites that bleed on probing is predominantly lymphocytic (a characteristic of stage II, or early, gingivitis).[9,17,25]

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SCIENCE TRANSFER

Gingivitis is diagnosed by the presence of redness, swelling, and increased edema of the gingival tissues. There may be increased pocket depth without attachment loss caused by gingival enlargement, and bleeding on probing is a hallmark of gingivitis and periodontitis. Early diagnosis, simple nonsurgical therapy, and improvements in the patient's oral hygiene are all that is required to control gingivitis in most patients. Careful, detailed monitoring of the gingival status around each tooth, with recordings of pocket depths, bleeding on probing, and position of the gingival margin, is essential so that patients with gingivitis do not progress to periodontal bone loss.

Gingival recession must also be documented to see if it is progressive and to determine if mucogingival surgery is required to enhance the amount of attached keratinized gingiva and to treat unaesthetic problems.

The severity of bleeding and the ease of its provocation depend on the intensity of the inflammation. After the vessels are damaged and ruptured, interrelated mechanisms induce hemostasis.[73] The vessel walls contract, blood flow diminishes, blood platelets adhere to the edges of the tissue, and a fibrous clot is formed, which contracts and results in approximation of the edges of the injured area. Bleeding recurs when the area is irritated.

In cases of moderate or advanced periodontitis, the presence of bleeding on probing is considered a sign of active tissue destruction.

Acute episodes of gingival bleeding are caused by injury and can occur spontaneously in gingival disease. Laceration of the gingiva by toothbrush bristles during aggressive toothbrushing or by sharp pieces of hard food can cause gingival bleeding even in the absence of gingival disease. Gingival burns from hot foods or chemicals increase the ease of gingival bleeding.

Spontaneous bleeding or bleeding on slight provocation can occur in acute necrotizing ulcerative gingivitis. In this condition, engorged blood vessels in the inflamed connective tissue are exposed by ulceration of the necrotic surface epithelium.

Gingival Bleeding Associated with Systemic Changes

In some systemic disorders, gingival hemorrhage occurs spontaneously or after irritation and is excessive and difficult to control. These hemorrhagic diseases represent a wide variety of conditions that vary in etiologic factors and clinical manifestations. Such conditions have the common feature of a hemostatic mechanism failure and result in abnormal bleeding in the skin, internal organs, and other tissues, including the oral mucosa.[71]

Hemorrhagic disorders in which abnormal gingival bleeding is encountered include vascular abnormalities (vitamin C deficiency or allergy [e.g., Schönlein-Henoch purpura]), platelet disorders[27] (thrombocytopenic purpura), hypoprothrombinemia (vitamin K deficiency), other coagulation defects (hemophilia, leukemia, Christmas disease), deficient platelet thromboplastic factor (PF3) resulting from uremia,[47] multiple myeloma[12] and postrubella purpura.[30] The effects of hormonal replacement therapy, oral contraceptives, pregnancy, and the menstrual cycle are also reported to affect gingival bleeding.[43,65,79,80] In addition, in women, long-term depression-related stress exposure may increase concentrations of interleukin-6 (IL-6) in gingival crevicular fluid and may worsen periodontal conditions with elevated gingival inflammation and increased pocket depths.[32]

In addition, changes in androgenic hormones have long been established as significant modifying factors in gingivitis, especially among adolescents. Several reports have shown notable effects of fluctuating estrogen/progesterone levels on the periodontium, starting as early as puberty.[1,55] Among pathologic endocrine changes, diabetes is an endocrine condition with a well-characterized effect on gingivitis.[78] In diabetes, marked inflammation affects both the epithelial and connective tissues, which leads to degeneration of the dermal papilla, increase in the number of inflammatory cells, destruction of reticulin fibers, and accumulation of dense collagen fibers that causes fibrosis.[70]

Several medications have also been found to have adverse effects on the gingiva. For example, anticonvulsants, antihypertensive calcium channel blockers, and immunosuppressant drugs are known to cause gingival enlargement (see Chapter 9), which secondarily can cause gingival bleeding. The American Heart Association has recommended over-the-counter aspirin as a therapeutic agent for cardiovascular disease, and aspirin is often prescribed for rheumatoid arthritis, osteoarthritis, rheumatic fever, and other inflammatory joint diseases.[28] Thus it is important to consider aspirin's effect on bleeding during a routine dental examination to avoid false-positive readings, which could result in an inaccurate patient diagnosis.[67] (Chapter 27 discusses periodontal involvement in hematologic disorders.)

Color Changes in the Gingiva

The color of the gingiva is determined by several factors, including the number and size of blood vessels, epithelial thickness, quantity of keratinization, and pigments within the epithelium.

Color Changes in Gingivitis. *Change in color is an important clinical sign of gingival disease.* The normal gingival color is "coral pink" and is produced by the tissue's vascularity and modified by the overlying epithelial layers. For this reason, the gingiva becomes red when vascularization increases or the degree of epithelial keratinization is reduced or disappears. The color becomes pale when vascularization is reduced (in association with fibrosis of the corium) or epithelial keratinization increases. Thus chronic inflammation intensifies the red or bluish red color because of vascular proliferation and reduction of keratinization. Additionally, venous stasis will contribute a bluish hue. The gingival color changes with increasing chronicity of the inflammatory process. The changes start in the interdental papillae and gingival margin and spread to the attached gingiva (see Figure 8-1).

Proper diagnosis and treatment require an understanding of the tissue changes that alter the color of the gingiva at the clinical level.

Color changes in acute gingival inflammation differ in both nature and distribution from those in chronic gingivitis. The color changes may be marginal, diffuse, or patchlike, depending on the underlying acute condition. In acute necrotizing ulcerative

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gingivitis, the involvement is marginal; in herpetic gingivostomatitis, it is diffuse; and in acute reactions to chemical irritation, it is patchlike or diffuse.

Color changes vary with the intensity of the inflammation. Initially, there is an increase in erythema. If the condition does not worsen, this is the only color change until the gingiva reverts to normal. In severe acute inflammation, the red color gradually becomes a dull, whitish gray. The gray discoloration produced by tissue necrosis is demarcated from the adjacent gingiva by a thin, sharply defined erythematous zone. Chapter 10 provides detailed descriptions of the clinical and pathologic features of the various forms of acute gingivitis.

Metallic Pigmentation. Heavy metals (bismuth, arsenic, mercury, lead, and silver) absorbed systemically from therapeutic use or occupational or household environments may discolor the gingiva and other areas of the oral mucosa.[45] These changes are rare but still should be ruled out in suspected cases.

Typically, metals produce a black or bluish line in the gingiva that follows the contour of the margin (Figure 8-9). The pigmentation may also appear as isolated black blotches involving the interdental marginal and attached gingiva. This differs from the tattooing produced by the accidental embedding of amalgam or other metal fragments[15] (Figure 8-10).

Gingival pigmentation from systemically absorbed metals results from perivascular precipitation of metallic sulfides in the subepithelial connective tissue. Gingival pigmentation is not a result of systemic toxicity. It occurs only in areas of inflammation in which the increased permeability of irritated blood vessels permits seepage of the metal into the surrounding tissue. In addition to inflamed gingiva, mucosal areas irritated by biting or abnormal chewing habits (e.g., inner surface of lips, cheek at level of occlusal line, lateral border of tongue) are common sites of pigmentation. Pigmentation can be eliminated by treating the inflammatory changes without necessarily discontinuing the metal-containing medication.

Figure 8-9 Bismuth gingivitis. Note linear black discoloration of the gingiva in a patient receiving bismuth therapy.

Figure 8-10 Discoloration of the gingiva caused by embedded metal particles (amalgam).

Color Changes Associated with Systemic Factors

Many systemic diseases may cause color changes in the oral mucosa, including the gingiva.[22] In general, these abnormal pigmentations are nonspecific and should stimulate further diagnostic efforts or referral to the appropriate specialist.[68]

Endogenous oral pigmentations can be caused by melanin, bilirubin, or iron.[45] *Melanin* oral pigmentations can be normal physiologic pigmentations and are often found in highly pigmented ethnic groups (see Chapter 2). Diseases that increase melanin pigmentation include the following:

- *Addison's disease* is caused by adrenal dysfunction and produces isolated patches of discoloration varying from bluish black to brown.
- *Peutz-Jeghers syndrome* produces intestinal polyposis and melanin pigmentation in the oral mucosa and lips.
- *Albright's syndrome (polyostotic fibrous dysplasia)* and *von Recklinghausen's disease (neurofibromatosis)* produce areas of oral melanin pigmentation.

Skin and mucous membranes can also be stained by *bile pigments.* Jaundice is best detected by examination of the sclera, but the oral mucosa may also acquire a yellowish color. The deposition of *iron* in hemochromatosis may produce a blue-gray pigmentation of the oral mucosa. Several endocrine and metabolic disturbances, including diabetes and pregnancy, may result in color changes. Blood dyscrasias, such as anemia, polycythemia, and leukemia, may also induce color changes.

Exogenous factors capable of producing color changes in the gingiva include atmospheric irritants, such as coal and metal dust, and coloring agents in food or lozenges. Tobacco causes hyperkeratosis of the gingiva and also may induce a significant increase in melanin pigmentation of the oral mucosa.[56] Localized bluish black areas of pigment are often caused by amalgam implanted in the mucosa (see Figure 8-10).

In recent years the need for esthetics in dentistry has increased, with a growing demand for a pleasing smile. This has made many individuals more aware of their gingival pigmentation that may be apparent during smiling and speech.[20,21] Traditionally, gingival depigmentation has been carried out using nonsurgical and surgical procedures such as chemical, cryosurgical, and electrosurgical techniques. However, those techniques were met with skepticism because of varying degrees of success. More recently, lasers have been used to ablate cells producing the melanin pigment; a nonspecific laser beam destroys the epithelial cells, including those at the basal layer. In addition, selective ablation using a laser beam with a wavelength that is specifically absorbed in melanin effectively destroys the pigmented cells without damaging the nonpigmented cells. In both cases, radiation energy is transformed into ablation energy, resulting in cellular rupture and vaporization with minimal heating of the surrounding tissue.[76]

Changes in Consistency of the Gingiva

Both chronic and acute inflammations produce changes in the normal firm and resilient consistency of the gingiva. As previously noted, in chronic gingivitis, both destructive (edematous) and reparative (fibrotic) changes coexist, and the consistency of the gingiva is determined by their relative predominance (Figures 8-11 and 8-12). Table 8-1 summarizes the clinical alterations in the consistency of the gingiva and the microscopic changes that produce them.

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TABLE 8-1 Clinical and Histopathologic Changes in Gingival Consistency

Clinical Changes	Underlying Microscopic Features
Chronic Gingivitis	
1. Soggy puffiness that pits on pressure. 2. Marked softness and friability, with ready fragmentation on exploration with probe and pinpoint surface areas of redness and desquamation. 3. Firm, leathery consistency.	1. Infiltration by fluid and cells of inflammatory exudate. 2. Degeneration of connective tissue and epithelium associated with injurious substances that provoke the inflammation and inflammatory exudate; change in connective tissue–epithelium relationship, with inflamed, engorged connective tissue expanding to within a few epithelial cells of surface, thinning of epithelium and degeneration associated with edema and leukocyte invasion, separated by areas in which rete pegs are elongated to connective tissue. 3. Fibrosis and epithelial proliferation associated with long-standing chronic inflammation.
Acute Forms of Gingivitis	
1. Diffuse puffiness and softening. 2. Sloughing with grayish, flakelike particles of debris adhering to eroded surface. 3. Vesicle formation.	1. Diffuse edema of acute inflammatory origin, fatty infiltration in xanthomatosis. 2. Necrosis with formation of pseudomembrane composed of bacteria, polymorphonuclear leukocytes, and degenerated epithelial cells in fibrinous meshwork. 3. Intercellular and intracellular edema with degeneration of nucleus and cytoplasm and rupture of cell wall.

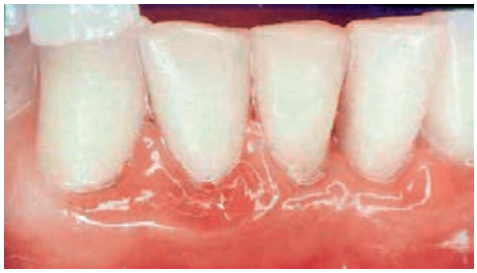

Figure 8-11 Chronic gingivitis. Swelling, loss of stippling, and discoloration occur when inflammatory exudate and edema are the predominant microscopic changes. The gingiva is soft and friable and bleeds easily.

Figure 8-12 Chronic gingivitis. Firm gingiva is produced when fibrosis predominates in the inflammatory process.

Calcified Masses in the Gingiva. Calcified microscopic masses may be found in the gingiva.[14,52] They can occur alone or in groups and vary in size, location, shape, and structure. Such masses may be calcified material removed from the tooth and traumatically displaced into the gingiva during scaling,[52] root remnants, cementum fragments, or cementicles. Chronic inflammation and fibrosis, an occasional foreign body, and giant cell activity occur in relation to these masses.

They are sometimes enclosed in an osteoidlike matrix. Crystalline foreign bodies have also been described in the gingiva, but their origin has not been determined.[60]

Toothbrushing. Toothbrushing has various effects on the consistency of the gingiva such as promoting keratinization of the oral epithelium, enhancing capillary gingival circulation, and thickening alveolar bone.[44,49,77] In animal studies, mechanical stimulation by toothbrushing was found to increase the proliferative activity of the junctional basal cells in dog gingiva by 2.5 times compared with using a scaler.[31] These findings may indicate that toothbrushing causes an increased turnover rate and desquamation of the junctional epithelial surfaces. This process may repair small breaks in the junctional epithelium and prevent direct access to the underlying tissue by periodontal pathogens.[81]

Changes in Surface Texture of the Gingiva

The surface of normal gingiva usually exhibits numerous small depressions and elevations, giving the tissue an orange-peel appearance referred as *stippling*.[13] Stippling is restricted to the attached gingiva and is predominantly localized to the subpapillary area, but it extends to a variable degree into the interdental papilla.[61] Although the biologic significance of gingival stippling is not known, some investigators conclude that loss of stippling is an early sign of gingivitis.[33,61] However, clinicians must take into consideration that its pattern and extent vary in different mouth areas, among patients, and with age.

In chronic inflammation the gingival surface is either smooth and shiny or firm and nodular, depending on whether the dominant changes are exudative or fibrotic. Smooth surface texture is also produced by epithelial atrophy in atrophic gingivitis, and peeling of the surface occurs in chronic desquamative gingivitis. Hyperkeratosis results in a leathery texture, and drug-induced gingival overgrowth produces a nodular surface.

Changes in Position of the Gingiva

Traumatic Lesions. One of the unique features of the most recent gingival disease classification is the recognition of

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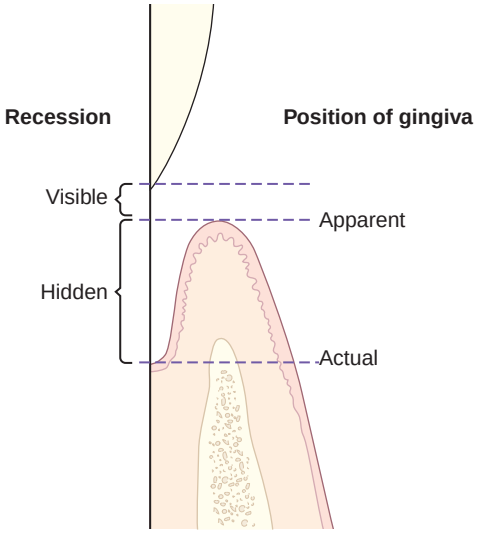

Figure 8-13 Diagram of apparent and actual positions of the gingiva and visible and hidden recession.

non–plaque-induced traumatic gingival lesions as distinct gingival conditions.[11] Traumatic lesions, whether chemical, physical, or thermal, are among the most common lesions in the mouth. Sources of chemical injuries include aspirin, hydrogen peroxide, silver nitrate, phenol, and endodontic materials. Physical injuries can include lip, oral, and tongue piercing, which can result in gingival recession. Thermal injuries can result from hot drinks and foods. In acute cases, the appearance of slough (necrotizing epithelium), erosion, or ulceration and the accompanying erythema are common features. In chronic cases, permanent gingival defects are usually present in the form of gingival recession. Typically, the localized nature of the lesions and the lack of symptoms readily eliminate them from the differential diagnosis of systemic conditions that may be present with erosive or ulcerative oral lesions.[64]

Gingival Recession. Gingival recession is a common finding. The prevalence, extent, and severity of gingival recession increase with age and are more prevalent in males.[6]

Positions of the Gingiva. By clinical definition, recession is exposure of the root surface by an apical shift in the position of the gingiva. To understand recession, it helps to distinguish between the actual and apparent positions of the gingiva. The *actual position* is the level of the coronal end of the epithelial attachment on the tooth, whereas the *apparent position* is the level of the crest of the gingival margin (Figure 8-13). *The severity of recession is determined by the actual position of the gingiva, not its apparent position.* For example, in periodontal disease, the inflamed pocket wall covers part of the denuded root; thus some of the recession is hidden and some may be visible. The total amount of recession is the sum of the two.

Recession refers to the location of the gingiva not its condition. Receded gingiva can be inflamed but may be normal except for its position (Figure 8-14). Recession may be localized to one tooth or a group of teeth, or it may be generalized throughout the mouth.

Gingival recession increases with age; the incidence varies from 8% in children to 100% after age 50 years.[83] This has led some investigators to assume that recession may be a physiologic process related to aging. However, no convincing evidence has been presented for a physiologic shift of the gingival attachment.[39] The gradual apical shift is most likely the result of the cumulative effect of minor pathologic involvement and repeated minor direct trauma to the gingiva. In some populations without access to dental care, however, recession may be the result of increasing periodontal disease.[29,40]

Figure 8-14 Different degrees of recession. Recession is slight in teeth #26 and #29 and marked in #27 and #28. The change in gingival contour and the recession, as seen in tooth #28, are referred to as Stillman's clefts.

The following etiologic factors have been implicated in gingival recession: faulty toothbrushing technique (gingival abrasion), tooth malposition, friction from soft tissues (gingival ablation),[72] gingival inflammation, abnormal frenum attachment, and iatrogenic dentistry. Trauma from occlusion has been suggested in the past, but its mechanism of action has never been demonstrated. For example, a deep overbite has been associated with gingival inflammation and recession. Excessive incisal overlap may result in a traumatic injury to the gingiva. Orthodontic movement in a labial direction in monkeys has been shown to result in loss of marginal bone and connective tissue attachment, as well as in gingival recession.[74]

Standard oral hygiene procedures, whether toothbrushing or flossing, may lead to a frequent transient and minimal gingival injury.[18,62] Although toothbrushing is important for gingival health, faulty toothbrushing technique or brushing with hard bristles may cause significant injury. This type of injury may present as lacerations, abrasions, keratosis, and recession, with the facial marginal gingiva most affected.[59] Thus, in these cases, recession tends to be more frequent and severe in patients with clinically healthy gingiva, little bacterial plaque, and good oral hygiene.[23,57,58]

Susceptibility to recession is also influenced by the position of teeth in the arch,[82] the root-bone angle, and the mesiodistal curvature of the tooth surface.[51] On rotated, tilted, or facially displaced teeth, the bony plate is thinned or reduced in height. Pressure from mastication or moderate toothbrushing damages the unsupported gingiva and produces recession. The effect of the angle of the root in the bone on recession is often observed in the maxillary molar area. If the lingual inclination of the palatal root is prominent or the buccal roots flare outward, the bone in the cervical area is thinned or shortened, and recession results from repeated trauma of the thin, marginal gingiva.

The health of the gingival tissue also depends on properly designed and placed restorative materials. Pressure from a poorly designed partial denture, such as an ill-fitting denture clasp, can cause gingival trauma and recession.[84] Overhanging dental restorations have long been viewed as a contributing factor to gingivitis because of plaque retention. In addition, there is general agreement that placing restorative margins within the biologic width frequently leads to gingival inflammation, clinical attachment loss, and eventually, bone loss. Clinically, the violation of biologic width

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typically manifests as gingival inflammation, deepened periodontal pockets, and gingival recession.

A relationship may exist between smoking and gingival recession (see Chapter 26). The multifactorial mechanisms may include reduced gingival blood flow and altered immune response but are not as yet conclusive.[26,63]

Clinical Significance. Several aspects of gingival recession make it clinically significant. Exposed root surfaces are susceptible to caries. Abrasion or erosion of the cementum exposed by recession leaves an underlying dentinal surface that can be sensitive. Hyperemia of the pulp and associated symptoms may also result from excessive exposure of the root surface.[48] Interproximal recession creates oral hygiene problems and resulting plaque accumulation.

Changes in Gingival Contour

Changes in gingival contour are primarily associated with gingival enlargement (see Chapter 9), but such changes may also occur in other conditions. Of historical interest are the descriptions of indentations of the gingival margin referred to as *Stillman's clefts* (see Figure 8-14)[75] and the *McCall festoons*. The term "Stillman's clefts" has been used to describe a specific type of gingival recession consisting of a narrow, triangular-shaped gingival recession. As the recession progresses apically, the cleft becomes broader, exposing the cementum of the root surface. When the lesion reaches the mucogingival junction, the apical border of oral mucosa is usually inflamed because of the difficulty in maintaining adequate plaque control at this site.

The term "McCall festoons" has been used to describe a rolled, thickened band of gingiva usually seen adjacent to the cuspids when recession approaches the mucogingival junction. Initially, Stillman's clefts and McCall festoons were attributed to traumatic occlusion, and the recommended treatment was occlusal adjustment. However, this association was never proved, and these indentations merely represent peculiar inflammatory changes of the marginal gingiva.[14]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 9

Gingival Enlargement

Fermin A. Carranza and Eva L. Hogan

CHAPTER OUTLINE

INFLAMMATORY ENLARGEMENT
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Acute Inflammatory Enlargement
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General Information
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Immunosuppressants
Calcium Channel Blockers
IDIOPATHIC GINGIVAL ENLARGEMENT
ENLARGEMENTS ASSOCIATED WITH SYSTEMIC DISEASES
Conditioned Enlargements
Systemic Diseases That Cause Gingival Enlargement
NEOPLASTIC ENLARGEMENT (GINGIVAL TUMORS)
Benign Tumors of the Gingiva
Malignant Tumors of the Gingiva
FALSE ENLARGEMENT
Underlying Osseous Lesions
Underlying Dental Tissues

Increase in size of the gingiva is a common feature of gingival disease. Accepted current terminology for this condition is *gingival enlargement* or *gingival overgrowth*. These are strictly clinical descriptive terms and avoid the erroneous pathologic connotations of terms used in the past, such as "hypertrophic gingivitis" or "gingival hyperplasia."

The many types of gingival enlargement can be classified according to etiologic factors and pathologic changes as follows:

I. Inflammatory enlargement
   A. Chronic
   B. Acute
II. Drug-induced enlargement
   A. General information
   B. Anticonvulsants
   C. Immunosuppressants
   D. Calcium channel blockers
III. Enlargements associated with systemic diseases or conditions
   A. Conditioned enlargement
      1. Pregnancy
      2. Puberty
      3. Vitamin C deficiency
      4. Plasma cell gingivitis
      5. Nonspecific conditioned enlargement (pyogenic granuloma)
   B. Systemic diseases causing gingival enlargement
      1. Leukemia
      2. Granulomatous diseases (e.g., Wegener's granulomatosis, sarcoidosis)
IV. Neoplastic enlargement (gingival tumors)
   A. Benign tumors
   B. Malignant tumors
V. False enlargement

Using the criteria of location and distribution, gingival enlargement is designated as follows:

- *Localized:* Limited to the gingiva adjacent to a single tooth or group of teeth.
- *Generalized:* Involving the gingiva throughout the mouth.
- *Marginal:* Confined to the marginal gingiva.
- *Papillary:* Confined to the interdental papilla.
- *Diffuse:* Involving the marginal and attached gingivae and papillae.
- *Discrete:* An isolated sessile or pedunculated, tumorlike enlargement.

The degree of gingival enlargement can be scored as follows[16]:

- *Grade 0:* No signs of gingival enlargement.
- *Grade I:* Enlargement confined to interdental papilla.
- *Grade II:* Enlargement involves papilla and marginal gingiva.
- *Grade III:* Enlargement covers three quarters or more of the crown.

INFLAMMATORY ENLARGEMENT

Gingival enlargement may result from chronic or acute inflammatory changes; chronic changes are much more common. In addition, inflammatory enlargements usually are a secondary complication to any of the other types of enlargement, creating a

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combined gingival enlargement. In these cases, it is important to understand the double etiology and treat them adequately.

Chronic Inflammatory Enlargement

Clinical Features. Chronic inflammatory gingival enlargement originates as a slight ballooning of the interdental papilla and marginal gingiva. In the early stages, it produces a life preserver–shaped bulge around the involved teeth. This bulge can increase in size until it covers part of the crowns. The enlargement may be localized or generalized and progresses slowly and painlessly, unless it is complicated by acute infection or trauma (Figures 9-1 and 9-2).

Occasionally, chronic inflammatory gingival enlargement occurs as a discrete sessile or pedunculated mass resembling a tumor. It may be interproximal or on the marginal or attached gingiva. The lesions are slow-growing masses and usually painless. They may undergo spontaneous reduction in size, followed by exacerbation and continued enlargement. Painful ulceration sometimes occurs in the fold between the mass and the adjacent gingiva.

Histopathology Chronic inflammatory gingival enlargements show the exudative and proliferative features of chronic inflammation (Figure 9-3). Lesions that are clinically deep red or bluish red are soft and friable with a smooth, shiny surface, and they bleed easily. They also have a preponderance of inflammatory cells and fluid, with vascular engorgement, new capillary formation, and associated degenerative changes. Lesions that are relatively firm, resilient, and pink have a greater fibrotic component with an abundance of fibroblasts and collagen fibers.

Etiology. Chronic inflammatory gingival enlargement is caused by prolonged exposure to dental plaque. Factors that favor plaque accumulation and retention[48] include poor oral hygiene, irritation by anatomic abnormalities, and improper restorative and orthodontic appliances.

Gingival Changes Associated with Mouth Breathing. Gingivitis and gingival enlargement are often seen in mouth breathers. The gingiva appears red and edematous, with a diffuse surface shininess of the exposed area. The maxillary anterior region is the common site of such involvement. In many cases the altered gingiva is clearly demarcated from the adjacent unexposed normal gingiva (Figure 9-4). The exact manner in which mouth breathing affects gingival changes has not been demonstrated. Its harmful effect is generally attributed to irritation from surface dehydration. However, comparable changes could not be produced by air drying the gingiva of experimental animals.[67]

Acute Inflammatory Enlargement

Gingival Abscess. A gingival abscess is a localized, painful, rapidly expanding lesion that usually has a sudden onset (Figure 9-5). It is generally limited to the marginal gingiva or interdental papilla. In its early stages, it appears as a red swelling with a smooth, shiny surface. Within 24 to 48 hours, the lesion usually becomes fluctuant and pointed with a surface orifice from which a purulent exudate may be expressed. The adjacent teeth are often sensitive to percussion. If permitted to progress, the lesion generally ruptures spontaneously.

Figure 9-1 Chronic inflammatory gingival enlargement localized to the anterior region.

Figure 9-2 Chronic inflammatory gingival enlargement.

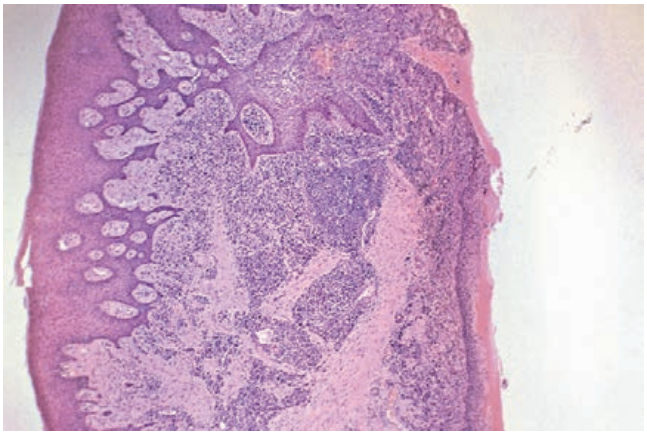

Figure 9-3 Survey section of chronic inflammatory gingival enlargement showing the inflamed connective tissue core and strands of proliferating epithelium.

Figure 9-4 Gingival enlargement in a mouth breather. Note the lesion sharply circumscribed to anterior marginal and papillary areas.

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Histopathology The gingival abscess consists of a purulent focus in the connective tissue, surrounded by a diffuse infiltration of polymorphonuclear leukocytes (PMNs), edematous tissue, and vascular engorgement. The surface epithelium has varying degrees of intracellular and extracellular edema, invasion by leukocytes, and sometimes ulceration.

Etiology. Acute inflammatory gingival enlargement results from bacteria carried deep into the tissues when a foreign substance (e.g., toothbrush bristle, piece of apple core, or lobster shell fragment) is forcefully embedded into the gingiva. The lesion is confined to the gingiva and should not be confused with periodontal or lateral abscesses.

Periodontal (Lateral) Abscess. Periodontal abscesses involve the supporting periodontal tissues and generally produce enlargement of the gingiva. For a detailed description of periodontal abscesses, see Chapter 13.

Figure 9-5 Gingival abscess on facial gingival surface, in space between cuspid and lateral incisor, unrelated to the gingival sulcus area.

DRUG-INDUCED GINGIVAL ENLARGEMENT

Gingival enlargement is a well-known consequence of the administration of some anticonvulsants, immunosuppressants, and calcium channel blockers and may create speech, mastication, tooth eruption, and aesthetic problems.

Clinical and microscopic features of the enlargements caused by the different drugs are similar.[19,87] These are presented first, followed by a description of the particular features of each drug.

General Information

Clinical Features. The growth starts as a painless, beadlike enlargement of the interdental papilla and extends to the facial and lingual gingival margins (Figure 9-6). As the condition progresses, the marginal and papillary enlargements unite and may develop into a massive tissue fold covering a considerable portion of the crowns; they may interfere with occlusion (Figure 9-7).

When uncomplicated by inflammation, the lesion is mulberry shaped, firm, pale pink, and resilient, with a minutely lobulated surface and no tendency to bleed. The enlargement characteristically appears to project from beneath the gingival margin, from which it is separated by a linear groove. However, the presence of the enlargement makes plaque control difficult, often resulting in a secondary inflammatory process that complicates the gingival overgrowth caused by the drug.

The resultant enlargement then becomes a combination of the increase in size caused by the drug and the complicating inflammation caused by bacteria. Secondary inflammatory changes not only add to the size of the lesion caused by the drug but also produce a red or bluish red discoloration, obliterate the lobulated surface demarcations, and increase bleeding tendency (Figure 9-8).

The enlargement is usually generalized throughout the mouth but is more severe in the maxillary and mandibular anterior regions. It occurs in areas in which teeth are present, not in edentulous spaces, and the enlargement disappears in areas from which teeth are extracted. Hyperplasia of the mucosa in edentulous mouths has been reported but is rare.[26,27]

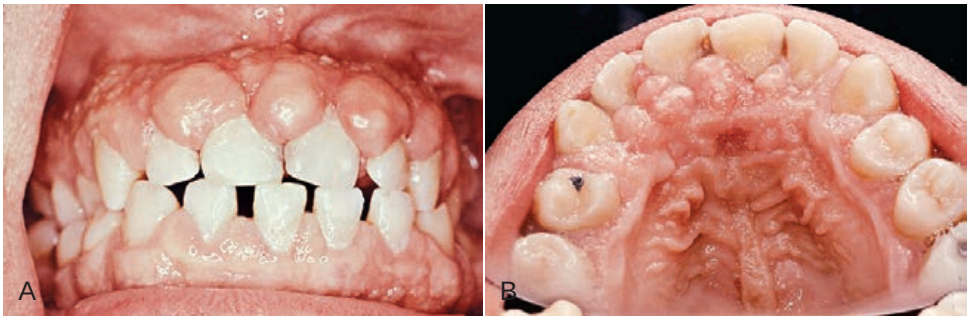

Figure 9-6 Gingival enlargement associated with phenytoin therapy. **A,** Facial view; note the prominent papillary lesions and the firm, nodular surface. **B,** Occlusal view of upper jaw.

SCIENCE TRANSFER

Gingival enlargement is seen in many different forms and has many different etiologies. Often there is a history of medication that has induced gingival hyperplasia where the response to plaque bacteria has an accentuation of tissue proliferation, e.g., hydantoin, calcium channel blockers, and immunosuppressants. Other causes such as pregnancy, puberty, mouth breathing, leukemia, sarcoidosis, and Wegener's granulomatosis are also to be considered. A localized gingival enlargement that has no obvious etiological basis should be biopsied so that malignant neoplastic disease is identified and treated and also to provide the basis for treatment of other less dangerous entities such as fibromas, papillomas, peripheral giant cell granulomas, and gingival cysts.

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Drug-induced enlargement may occur in mouths with little or no plaque and may be absent in mouths with abundant deposits. Some investigators, however, believe that inflammation is a prerequisite for development of the enlargement, which therefore could be prevented by plaque removal and fastidious oral hygiene.[22,37,79,102] Oral hygiene by means of toothbrushing or use of a chlorhexidine toothpaste[91] reduces the inflammation but does not lessen or prevent the overgrowth. Hassell et al[42,44] have hypothesized that in noninflamed gingiva, fibroblasts are less active or even quiescent and do not respond to circulating phenytoin, whereas fibroblasts within inflamed tissue are in an active state as a result of the inflammatory mediators and the endogenous growth factors present.

A genetic predisposition is a suspected factor[45,84] in determining whether a person treated with phenytoin will develop gingival enlargement or not.

The enlargement is chronic and slowly increases in size. When surgically removed, it recurs. Spontaneous disappearance occurs within a few months after discontinuation of the drug. See Chapter 58 for treatment of gingival enlargements, including the substitution of drugs that do not induce gingival overgrowth.

Figure 9-7 Phenytoin gingival enlargement in a 5-year-old child covering most of the clinical crowns of teeth.

Figure 9-8 Combined gingival enlargement resulting from the inflammatory involvement of a phenytoin-induced overgrowth.

Histopathology Drug-induced gingival enlargement consists of a pronounced hyperplasia of the connective tissue and epithelium (Figure 9-9). There is acanthosis of the epithelium, and elongated rete pegs extend deep into the connective tissue, which exhibits densely arranged collagen bundles with an increase in the number of fibroblasts and new blood vessels.[90] An abundance of amorphous ground substance has also been reported.[68] Structural changes in the outer epithelial cell surface have been reported in cyclosporine-induced enlargements.[3]

The enlargement begins as a hyperplasia of the connective tissue core of the marginal gingiva and increases by its proliferation and expansion beyond the crest of the gingival margin. An inflammatory infiltrate may be found at the bottom of the sulcus, or pocket. Cyclosporine enlargements usually have a more highly vascularized connective tissue with foci of chronic inflammatory cells, particularly plasma cells.[75]

The "mature" phenytoin enlargement has a fibroblast/collagen ratio equal to that of normal gingiva from normal individuals, suggesting that at some point in the development of the lesion, fibroblastic proliferation must have been abnormally high.[43] Recurring phenytoin enlargements appear as granulation tissue composed of numerous young capillaries and fibroblasts and irregularly arranged collagen fibrils with occasional lymphocytes.

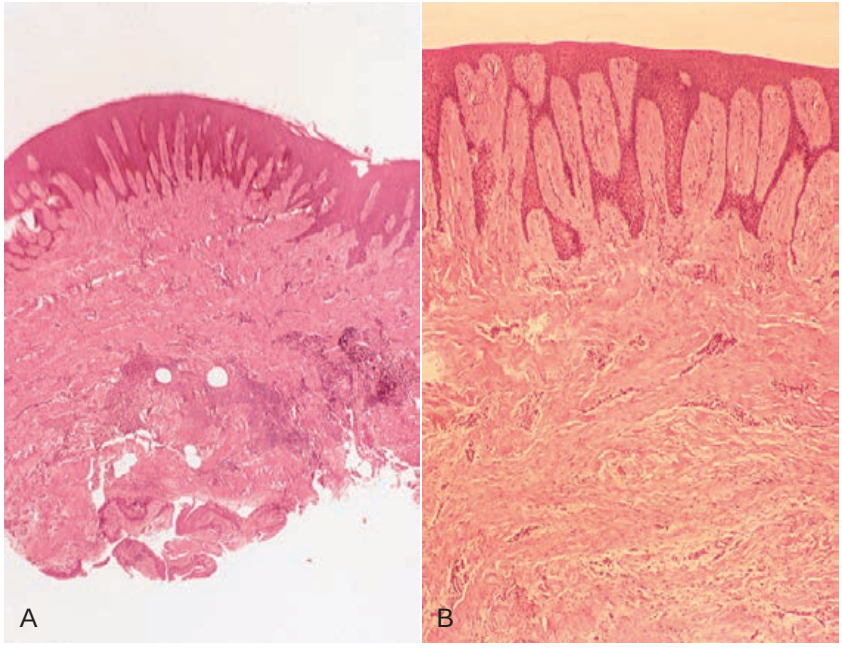

Figure 9-9 Microscopic view of gingival enlargement associated with phenytoin therapy. **A,** Hyperplasia and acanthosis of the epithelium and densely collagenous connective tissue, with evidence of inflammation in the area adjacent to the gingival sulcus (pocket). **B,** High-power view showing extension of deep rete pegs into the connective tissue.

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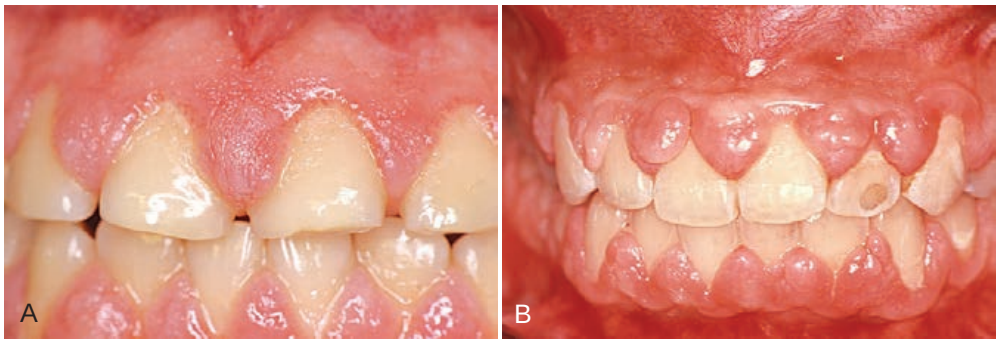

Figure 9-10 Cyclosporine-associated gingival enlargement. **A,** Mild involvement located particularly on papillae between teeth #9 and 10 and teeth #10 and 11. **B,** Advanced generalized enlargement.

Anticonvulsants

The first drug-induced gingival enlargements reported were those produced by phenytoin (Dilantin). Dilantin is a hydantoin, introduced by Merritt and Putnam[69] in 1938 for the treatment of all forms of epilepsy, except petit mal. Shortly thereafter, its relationship with gingival enlargement was reported.[35,57]

Other hydantoins known to induce gingival enlargement are ethotoin (Peganone) and mephenytoin (Mesantoin). Other anticonvulsants that have the same side effect are the succinimides (ethosuximide [Zarontin], methsuximide [Celontin]), and valproic acid [Depakene]).[38]

Gingival enlargement occurs in about 50% of patients receiving the drug,[110] although different authors have reported incidences from 3% to 84.5%.[2,35,80] It occurs more often in younger patients.[5] Its occurrence and severity are not necessarily related to the dosage after a threshold level has been exceeded.[97] Phenytoin appears in the saliva. There is no consensus, however, on whether the severity of the overgrowth is related to the levels of phenytoin in plasma or saliva.[2,5,6,117] Some reports indicate a relation between the drug dosage and the degree of gingival overgrowth.[54,58]

Tissue culture experiments indicate that phenytoin stimulates proliferation of fibroblast-like cells[98] and epithelium.[99] Two analogs of phenytoin (1-allyl-5-phenylhydantoinate and 5-methyl 5-phenylhydantoinate) have a similar effect on fibroblast-like cells.[99] Fibroblasts from a phenytoin-induced gingival overgrowth show increased synthesis of sulfated glycosaminoglycans in vitro.[53] Phenytoin may induce a decrease in collagen degradation as a result of the production of an inactive fibroblastic collagenase.[42]

Experimental attempts to induce gingival enlargement with phenytoin administration in laboratory animals have been successful only in the cat,[50] the ferret, and the *Macaca speciosa* monkey.[104] In experimental animals, phenytoin causes gingival enlargement that is independent of local inflammation.

In cats, one of the metabolic products of phenytoin is 5-(parahydroxyphenyl)-5-phenylhydantoin; administration of this metabolite to cats also induces gingival enlargement in some cases. This led Hassell et al[43] to hypothesize that gingival enlargement may result from the genetically determined ability or inability of the host to deal effectively with prolonged administration of phenytoin.

Systemic administration of phenytoin accelerates the healing of gingival wounds in nonepileptic humans[101] and increases the tensile strength of healing abdominal wounds in rats.[25,100] The administration of phenytoin may precipitate a megaloblastic anemia[66] and folic acid deficiency.[105]

In conclusion, the pathogenesis of gingival enlargement induced by phenytoin is not known, but some evidence links it to a direct effect on specific, genetically predetermined subpopulations of fibroblasts, inactivation of collagenase, and plaque-induced inflammation.

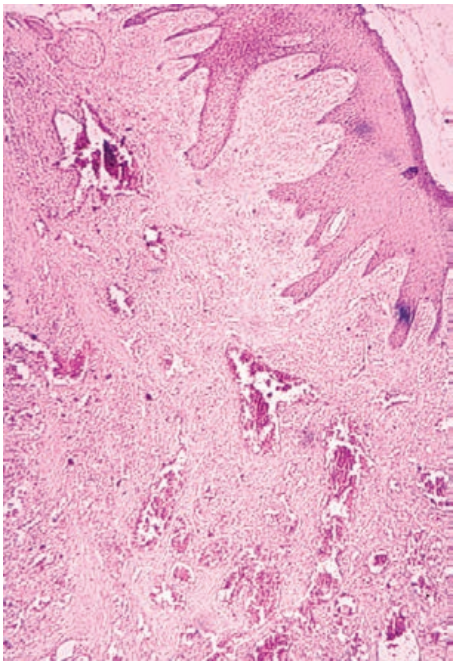

Figure 9-11 Microscopic view of cyclosporine-associated gingival enlargement. Note the epithelial hyperplasia and fibrous stroma with abundant vascularization.

Immunosuppressants

Cyclosporine is a potent immunosuppressive agent used to prevent organ transplant rejection and to treat several diseases of autoimmune origin.[20] Its exact mechanism of action is not well known, but it appears to selectively and reversibly inhibit helper T cells, which play a role in cellular and humoral immune responses. Cyclosporin A (Sandimmune, Neoral) is administered intravenously or by mouth, and dosages greater than 500 mg/day have been reported to induce gingival overgrowth.[24]

Cyclosporine-induced gingival enlargement is more vascularized than phenytoin enlargement (Figures 9-10 and 9-11)[86,92,119]. Its occurrence varies according to different studies from 25% to 70%,[89] it affects children more frequently, and its magnitude appears to be related more to the plasma concentration than to the patient's periodontal status.[96] Gingival enlargement is greater in

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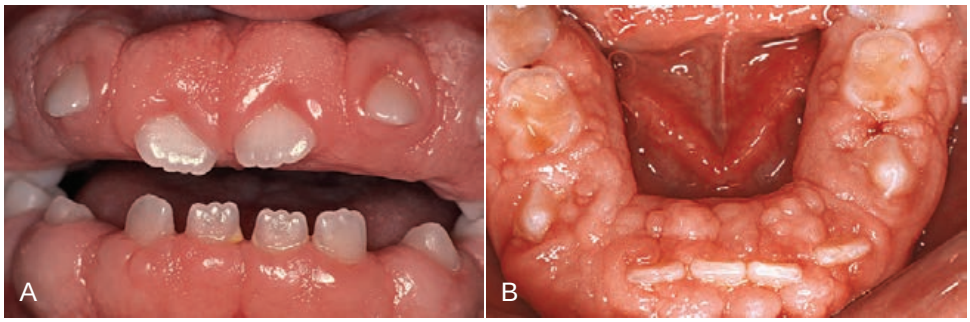

Figure 9-12 Idiopathic gingival enlargement in 14-year-old white male patient. **A,** Facial view; gingiva is firm, with nodular, pebbled surface and partially covers the crowns of the teeth. **B,** Occlusal view of lower jaw.

patients who are medicated with both cyclosporine and calcium channel blockers.[102,112,113]

The microscopic finding of many plasma cells plus the presence of an abundant amorphous extracellular substance has suggested that the enlargement is a hypersensitivity response to the cyclosporine.[68]

In experimental animals (rats), oral administration of cyclosporine was reported also to induce abundant formation of new cementum.[4]

In addition to gingival enlargement, cyclosporine induces other major side effects such as nephrotoxicity, hypertension, and hypertrichosis. Another immunosuppressive drug, *tacrolimus,* has been used effectively and is also nephrotoxic, but it results in much less severe hypertension, hypertrichosis, and gingival overgrowth.[7,71,103]

Calcium Channel Blockers

Calcium channel blockers are drugs developed for the treatment of cardiovascular conditions such as hypertension, angina pectoris, coronary artery spasms, and cardiac arrhythmias. They inhibit calcium ion influx across the cell membrane of heart and smooth muscle cells, blocking intracellular mobilization of calcium. This induces direct dilation of the coronary arteries and arterioles, improving oxygen supply to the heart muscle; it also reduces hypertension by dilating the peripheral vasculature.

These drugs are the *dihydropyridine derivatives* (amlodipine [Lotrel, Norvasc], felodipine [Plendil], nicardipine [Cardene], nifedipine [Adalat, Procardia]), the *benzothiazine derivatives* (diltiazem [Cardizem, Dilacor XR, Tiazac]), and the *phenylalkylamine derivatives* (verapamil [Calan, Isoptin, Verelan, Covera HS]).[38]

Some of these drugs can induce gingival enlargement. Nifedipine, one of the most often used,[39,63,65,77] induces gingival enlargement in 20% of patients.[8] Diltiazem, felodipine, nitrendipine, and verapamil also induce gingival enlargement.[14,46] The dihydropyridine derivative, isradipine, can replace nifedipine in some cases and does not induce gingival overgrowth.[116]

Nifedipine is also used with cyclosporine in kidney transplant recipients, and the combined use of both drugs induces larger overgrowths.[13] Nifedipine gingival enlargement has been induced experimentally in rats, where it appears to be dose dependent[32]; in humans, however, this dose dependency is not clear. One report indicates that nifedipine increases the risk of periodontal destruction in patients with diabetes mellitus type 2.[64]

IDIOPATHIC GINGIVAL ENLARGEMENT

Idiopathic gingival enlargement is a rare condition of undetermined cause. It has been designated by such terms as gingivomatosis, elephantiasis, idiopathic fibromatosis, hereditary gingival hyperplasia, and congenital familial fibromatosis.

Clinical Features. The enlargement affects the attached gingiva, as well as the gingival margin and interdental papillae, in contrast to phenytoin-induced overgrowth, which is often limited to the gingival margin and interdental papillae (see Figure 9-6). The facial and lingual surfaces of the mandible and maxilla are generally affected, but the involvement may be limited to either jaw. The enlarged gingiva is pink, firm, and almost leathery in consistency and has a characteristic minutely pebbled surface (Figure 9-12). In severe cases the teeth are almost completely covered, and the enlargement projects into the oral vestibule. The jaws appear distorted because of the bulbous enlargement of the gingiva. Secondary inflammatory changes are common at the gingival margin.

Histopathology Idiopathic gingival enlargement shows a bulbous increase in the amount of connective tissue that is relatively avascular and consists of densely arranged collagen bundles and numerous fibroblasts. The surface epithelium is thickened and acanthotic with elongated rete pegs.

Etiology. The cause is unknown, and thus the condition is designated as "idiopathic." Some cases have a hereditary basis,[28,120,121] but the genetic mechanisms involved are not well understood. A study of several families found the mode of inheritance to be autosomal recessive in some cases and autosomal dominant in others.[52,84] In some families the gingival enlargement may be linked to impairment of physical development.[56] The enlargement usually begins with the eruption of the primary or secondary dentition and may regress after extraction, suggesting that the teeth (or the plaque attached to them) may be initiating factors. The presence of bacterial plaque is a complicating factor.

Gingival enlargement has been described in *tuberous sclerosis,* which is an inherited condition characterized by a triad of epilepsy, mental deficiency, and cutaneous angiofibromas.[107,111]

ENLARGEMENTS ASSOCIATED WITH SYSTEMIC DISEASES

Many systemic diseases can develop oral manifestations that may include gingival enlargement. These diseases and conditions can affect the periodontium by two different mechanisms, as follows:

1. *Magnification of an existing inflammation initiated by dental plaque.* This group of diseases, discussed in the Conditioned Enlargements section, includes some hormonal conditions (e.g., pregnancy and puberty), nutritional diseases such as vitamin C deficiency, and some cases in which the systemic influence is not identified (nonspecific conditioned enlargement).

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2. *Manifestation of the systemic disease independently of the inflammatory status of the gingiva.* This group is described in the section on Systemic Diseases Causing Gingival Enlargement and Neoplastic Enlargement (Gingival Tumors).

Conditioned Enlargements

Conditioned enlargements occur when the systemic condition of the patient exaggerates or distorts the usual gingival response to dental plaque. The specific manner in which the clinical picture of conditioned gingival enlargement differs from that of chronic gingivitis depends on the nature of the modifying systemic influence. *Bacterial plaque is necessary for the initiation of this type of enlargement.* However, plaque is not the sole determinant of the nature of the clinical features.

The three types of conditioned gingival enlargement are *hormonal* (pregnancy, puberty), *nutritional* (associated with vitamin C deficiency), and *allergic*. Nonspecific conditioned enlargement is also seen.

Enlargement in Pregnancy. Pregnancy gingival enlargement may be marginal and generalized or may occur as single or multiple tumor-like masses (see Chapters 27 and 38).

During pregnancy, there is an increase in levels of both progesterone and estrogen, which by the end of the third trimester reach levels 10 and 30 times the levels during the menstrual cycle, respectively.[1] These hormonal changes induce changes in vascular permeability, leading to gingival edema and an increased inflammatory response to dental plaque. The subgingival microbiota may also undergo changes, including an increase in *Prevotella intermedia*.[61,83]

Marginal Enlargement. Marginal gingival enlargement during pregnancy results from the aggravation of previous inflammation, and its incidence has been reported as 10%[18] and 70%.[122]

The clinical picture varies considerably. The enlargement is usually generalized and tends to be more prominent interproximally than on the facial and lingual surfaces. The enlarged gingiva is bright red or magenta, soft, and friable and has a smooth, shiny surface. Bleeding occurs spontaneously or on slight provocation.

Tumorlike Gingival Enlargement. The so-called pregnancy tumor is not a neoplasm; it is an inflammatory response to bacterial plaque and is modified by the patient's condition. It usually appears after the third month of pregnancy but may occur earlier. The reported incidence is 1.8% to 5%.[67]

The lesion appears as a discrete, mushroomlike, flattened spherical mass that protrudes from the gingival margin or more often from the interproximal space and is attached by a sessile or pedunculated base (Figure 9-13). It tends to expand laterally, and pressure from the tongue and the cheek perpetuates its flattened appearance. Generally dusky red or magenta, it has a smooth, glistening surface that often exhibits numerous deep-red, pinpoint markings. It is a superficial lesion and usually does not invade the underlying bone. The consistency varies; the mass is usually semifirm, but it may have various degrees of softness and friability. It is usually painless unless its size and shape foster accumulation of debris under its margin or interfere with occlusion, in which case, painful ulceration may occur.

Although the microscopic findings are characteristic of gingival enlargement in pregnancy, they are not pathognomonic because they cannot be used to differentiate pregnant and nonpregnant patients.[67]

Figure 9-13 Localized gingival enlargement in a 27-year-old pregnant patient.

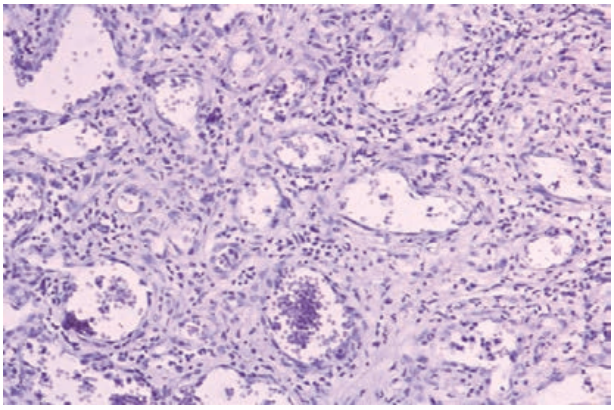

Figure 9-14 Microscopic view of gingival enlargement in pregnant patient showing abundance of blood vessels and interspersed inflammatory cells.

Histopathology Gingival enlargement in pregnancy is called *angiogranuloma.* Both marginal and tumorlike enlargements consist of a central mass of connective tissue, with numerous diffusely arranged, newly formed, and engorged capillaries lined by cuboid endothelial cells (Figure 9-14), as well as a moderately fibrous stroma with varying degrees of edema and chronic inflammatory infiltrate. The stratified squamous epithelium is thickened, with prominent rete pegs and some degree of intracellular and extracellular edema, prominent intercellular bridges, and leukocytic infiltration.

Most gingival disease during pregnancy can be prevented by the removal of plaque and calculus, as well as the institution of fastidious oral hygiene at the outset. In pregnancy, treatment of the gingiva that is limited to the removal of tissue without complete elimination of local irritants is followed by recurrence of gingival enlargement. Although spontaneous reduction in the size of gingival enlargement typically follows the termination of pregnancy, complete elimination of the residual inflammatory lesion requires the removal of all plaque deposits and factors that favor its accumulation. For additional information on gingival disease in pregnancy, see Chapter 58.

Enlargement in Puberty. Enlargement of the gingiva is sometimes seen during puberty (see Chapter 27). It occurs in both male and female adolescents and appears in areas of plaque accumulation.

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The size of the gingival enlargement greatly exceeds that usually seen in association with comparable local factors. It is marginal and interdental and is characterized by prominent bulbous interproximal papillae (Figure 9-15). Often, only the facial gingivae are enlarged, and the lingual surfaces are relatively unaltered; the mechanical action of the tongue and the excursion of food prevent a heavy accumulation of local irritants on the lingual surface.

Gingival enlargement during puberty has all the clinical features generally associated with chronic inflammatory gingival disease. It is the *degree* of enlargement and its tendency to recur in the presence of relatively scant plaque deposits that distinguish pubertal gingival enlargement from uncomplicated chronic inflammatory gingival enlargement. After puberty the enlargement undergoes spontaneous reduction but does not disappear completely until plaque and calculus are removed.

A longitudinal study of 127 children 11 to 17 years of age showed a high initial prevalence of gingival enlargement that tended to decline with age.[108] When the mean number of inflamed gingival sites per child was determined and correlated with the time at which the maximum number of inflamed sites was observed and with the oral hygiene index at that time, a pubertal peak in gingival inflammation unrelated to oral hygiene factors clearly occurred. A longitudinal study of subgingival microbiota of children between ages 11 and 14 and their association with clinical parameters has implicated *Capnocytophaga* species in the initiation of pubertal gingivitis.[72] Other studies have reported that hormonal changes coincide with an increase in the proportion of *Prevotella intermedia* and *Prevotella nigrescens*.[74,118]

Histopathology The microscopic appearance of gingival enlargement in puberty is chronic inflammation with prominent edema and associated degenerative changes.

Figure 9-15 Conditioned gingival enlargement in puberty in a 13-year-old male.

Enlargement in Vitamin C Deficiency. Enlargement of the gingiva is generally included in classic descriptions of scurvy. Acute vitamin C deficiency itself does not cause gingival inflammation, but it does cause hemorrhage, collagen degeneration, and edema of the gingival connective tissue. These changes modify the response of the gingiva to plaque to the extent that the normal defensive delimiting reaction is inhibited, and the extent of the inflammation is exaggerated,[33,34] resulting in the massive gingival enlargement seen in scurvy (Figure 9-16) (see Chapter 27).

Gingival enlargement in vitamin C deficiency is marginal; the gingiva is bluish red, soft, and friable and has a smooth, shiny surface. Hemorrhage, occurring either spontaneously or on slight provocation, and surface necrosis with pseudomembrane formation are common features.

Histopathology In vitamin C deficiency the gingiva has a chronic inflammatory cellular infiltration with a superficial acute response. There are scattered areas of hemorrhage, with engorged capillaries. Marked diffuse edema, collagen degeneration, and scarcity of collagen fibrils or fibroblasts are striking findings.

Plasma Cell Gingivitis. Plasma cell gingivitis consists of a mild marginal gingival enlargement that extends to the attached gingiva. The gingiva appears red, friable, and sometimes granular and bleeds easily; usually it does not induce a loss of attachment (Figure 9-17). This lesion is located in the oral aspect of the attached gingiva and therefore differs from plaque-induced gingivitis.

Figure 9-16 Gingival enlargement in patient with vitamin C deficiency. Note the prominent hemorrhagic areas. *(Courtesy Dr. Gerald Shklar, Boston.)*

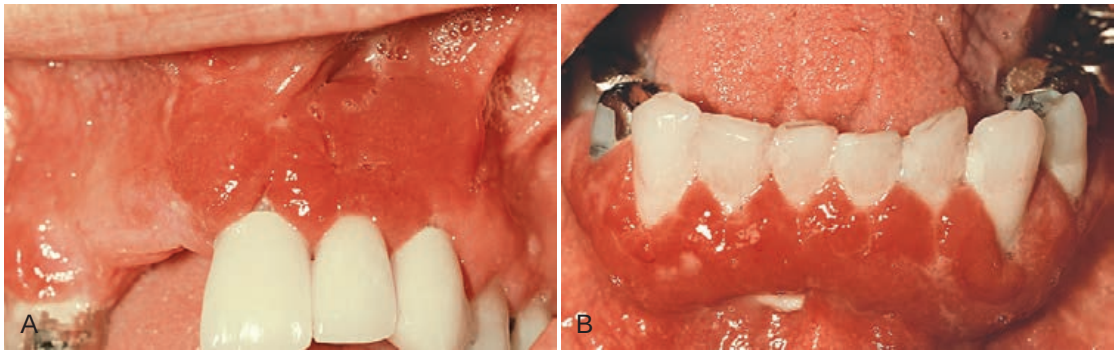

Figure 9-17 Plasma cell gingivitis. **A,** Diffuse lesions on facial surface of the anterior maxilla. **B,** Mandibular lesions. *(Courtesy Dr. Kim D. Zussman, Thousand Oaks, CA.)*

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Figure 9-18 Pyogenic granuloma. *(Courtesy Dr. Silvia Oreamuno, San José, Costa Rica.)*

Figure 9-19 Leukemic gingival enlargement (acute myelocytic leukemia). *(Courtesy Dr. Spencer Wolfe, Dublin, Ireland.)*

Histopathology In plasma cell gingivitis the oral epithelium shows spongiosis and infiltration with inflammatory cells; ultrastructurally, there are signs of damage in the lower spinous layers and the basal layers. The underlying connective tissue contains a dense infiltrate of plasma cells that also extends to the oral epithelium, inducing a dissecting type of injury.[76]

An associated cheilitis and glossitis have been reported.[55,94] Plasma cell gingivitis is thought to be allergic in origin, possibly related to components of chewing gum, dentifrices, or various diet components. Cessation of exposure to the allergen brings resolution of the lesion.

In rare instances, marked inflammatory gingival enlargements with a predominance of plasma cells can appear; these are associated with rapidly progressive periodontitis.[78]

A solitary plasma cell tumor or *plasmocytoma* has been described in the nasopharynx and rarely in the oral mucosa.[12,92] It is a slow-growing pedunculated tumor with a pink and smooth surface and is composed of normal plasma cells. It is usually benign, but in rare cases, it can be an oral manifestation of multiple myeloma, a malignant tumor of the bone marrow.

Nonspecific Conditioned Enlargement (Pyogenic Granuloma). *Pyogenic granuloma* is a tumorlike gingival enlargement that is considered an exaggerated conditioned response to minor trauma (Figure 9-18). The exact nature of the systemic conditioning factor has not been identified.[11] Pyogenic granuloma is similar in clinical and microscopic appearance to the conditioned gingival enlargement seen in pregnancy. Differential diagnosis is based on the patient's history.

Treatment consists of removal of the lesions plus the elimination of irritating local factors. The recurrence rate is about 15%.

Systemic Diseases That Cause Gingival Enlargement

Several systemic diseases may result in gingival enlargement by different mechanisms. These are uncommon cases and are only briefly discussed.

Leukemia. Leukemic gingival enlargement may be diffuse or marginal and localized or generalized (see Chapter 27). It may appear as a diffuse enlargement of the gingival mucosa, an oversized extension of the marginal gingiva (Figure 9-19), or a discrete tumorlike interproximal mass.

Clinical Features. In leukemic enlargement the gingiva is generally bluish red and has a shiny surface. The consistency is moderately firm, but there is a tendency toward friability and hemorrhage, occurring either spontaneously or on slight irritation. Acute painful necrotizing ulcerative inflammatory involvement may occur in the crevice formed at the junction of the enlarged gingiva and the contiguous tooth surfaces.

Patients with leukemia may also have a simple chronic inflammation without the involvement of leukemic cells and may present with the same clinical and microscopic features seen in patients without the systemic disease. Most cases, however, reveal features of both simple chronic inflammation and leukemic infiltrate.

True leukemic enlargement often occurs in acute leukemia but may also be seen in subacute leukemia. It seldom occurs in chronic leukemia.

Histopathology Gingival enlargements in leukemic patients show various degrees of chronic inflammation. Mature leukocytes and areas of connective tissue are infiltrated with a dense mass of immature and proliferating leukocytes, the specific nature of which varies with the type of leukemia. Engorged capillaries, edematous and degenerated connective tissue, and epithelium with various degrees of leukocytic infiltration and edema are found. Isolated surface areas of acute necrotizing inflammation with a pseudomembranous meshwork of fibrin, necrotic epithelial cells, PMNs, and bacteria are often seen.

Granulomatous Diseases

Wegener's Granulomatosis. Wegener's granulomatosis is a rare disease characterized by acute granulomatous necrotizing lesions of the respiratory tract, including nasal and oral defects. Renal lesions develop, and acute necrotizing vasculitis affects the blood vessels. The initial manifestations of Wegener's granulomatosis may involve the orofacial region and include oral mucosal ulceration, gingival enlargement,[47] abnormal tooth mobility, exfoliation of teeth, and delayed healing response.[17]

The granulomatous papillary enlargement is reddish purple and bleeds easily on stimulation (see Figure 12-30).

Histopathology Chronic inflammation occurs, with scattered giant cells and foci of acute inflammation and microabscesses covered by a thin, acanthotic epithelium. Vascular changes have not been described with gingival enlargement in Wegener's granulomatosis, probably because of the small size of the gingival blood vessels.[51]

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The cause of Wegener's granulomatosis is unknown, but the condition is considered an immunologically mediated tissue injury.[23] At one time the usual outcome was death from kidney failure within a few months, but more recently the use of immunosuppressive drugs has produced prolonged remissions in more than 90% of patients.[60]

Sarcoidosis. Sarcoidosis is a granulomatous disease of unknown etiology. It starts in individuals in their twenties or thirties, predominantly affects blacks, and can involve almost any organ, including the gingiva, in which a red, smooth, painless enlargement may appear.

Histopathology Sarcoid granulomas consist of discrete, noncaseating whorls of epithelioid cells and multinucleated, foreign body–type giant cells with peripheral mononuclear cells.[88]

NEOPLASTIC ENLARGEMENT (GINGIVAL TUMORS)

This section provides only a brief description of some of the more common neoplastic and pseudoneoplastic lesions of the gingiva. The reader is referred to texts on oral pathology for more comprehensive coverage.[88,92]

Benign Tumors of the Gingiva

Epulis is a generic term used clinically to designate all discrete tumors and tumorlike masses of the gingiva. It serves to locate the tumor but not to describe it. Most lesions referred to as "epulis" are inflammatory rather than neoplastic.

Neoplasms account for a comparatively small proportion of gingival enlargements and make up a small percentage of the total number of oral neoplasms. In a survey of 257 oral tumors, approximately 8% occurred on the gingiva.[70] In another study of 868 growths of the gingiva and palate, of which 57% were neoplastic and the remainder inflammatory, the following incidence of tumors was noted: carcinoma, 11.0%; fibroma, 9.3%; giant cell tumor, 8.4%; papilloma, 7.3%; leukoplakia, 4.9%; mixed tumor (salivary gland type), 2.5%; angioma, 1.5%; osteofibroma, 1.3%; sarcoma, 0.5%; melanoma, 0.5%; myxoma, 0.45%; fibropapilloma, 0.4%; adenoma, 0.4%; and lipoma, 0.3%.[9]

Fibroma. Fibromas of the gingiva arise from the gingival connective tissue or from the periodontal ligament. They are slow-growing, spherical tumors that tend to be firm and nodular but may be soft and vascular. Fibromas are usually pedunculated. Hard fibromas of the gingiva are rare; most of the lesions diagnosed clinically as "fibromas" are inflammatory enlargements.[93]

Histopathology Fibromas are composed of bundles of well-formed collagen fibers with a scattering of fibrocytes and a variable vascularity.

The so-called giant cell fibroma contains multinucleated fibroblasts. In another variant, mineralized tissue (bone, cementum-like material, and dystrophic calcifications) may be found; this type of fibroma is called *peripheral ossifying fibroma.*

Papilloma. Papillomas are benign proliferations of surface epithelium that are in many but not all cases associated with the human papillomavirus (HPV). Gingival papillomas appear as solitary wartlike or cauliflower-like protuberances (Figure 9-20). They may be small and discrete or broad, hard elevations with minutely irregular surfaces.

Histopathology The papilloma lesion consists of fingerlike projections of stratified squamous epithelium, often hyperkeratotic, with a central core of fibrovascular connective tissue.

Peripheral Giant Cell Granuloma. Giant cell lesions of the gingiva arise interdentally or from the gingival margin, occur most frequently on the labial surface, and may be sessile or pedunculated. They vary in appearance from smooth, regularly outlined masses to irregularly shaped, multilobulated protuberances with surface indentations (Figure 9-21). Ulceration of the margin is occasionally seen. The lesions are painless, vary in size, and may cover several teeth. They may be firm or spongy, and the color varies from pink to deep red or purplish blue. There are no pathognomonic clinical features whereby these lesions can be differentiated from other forms of gingival enlargement. Microscopic examination is required for definitive diagnosis.

The prefix "peripheral" is needed to differentiate them from comparable lesions that originate within the jawbone (central giant cell granulomas).

In some cases the giant cell granuloma of the gingiva is locally invasive and causes destruction of the underlying bone (Figure 9-22). Complete removal leads to uneventful recovery.

Figure 9-20 Papilloma of the gingiva in 26-year-old male.

Figure 9-21 Gingival giant cell granuloma.

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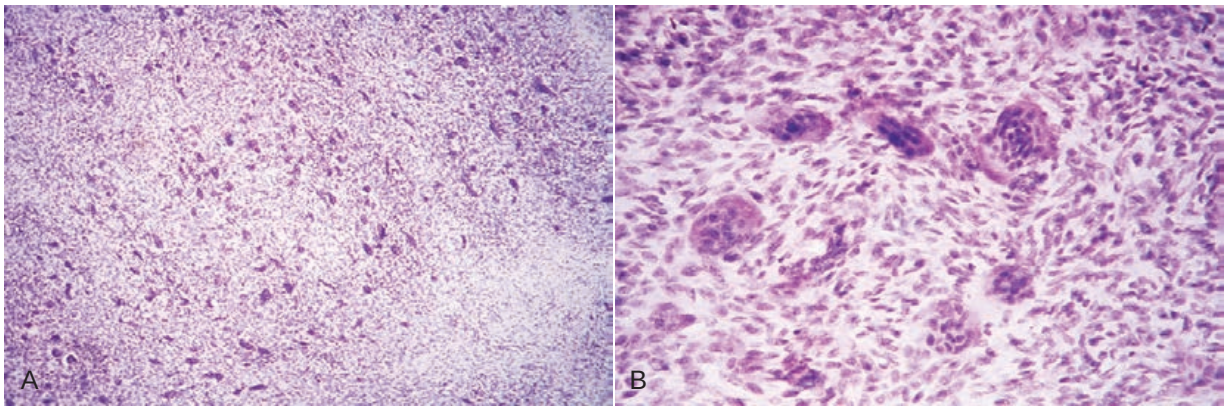

Figure 9-22 **A,** Microscopic survey of peripheral giant cell granuloma. **B,** High-power study of the lesion demonstrating the giant cells and intervening stroma that make up the major portion of the mass.

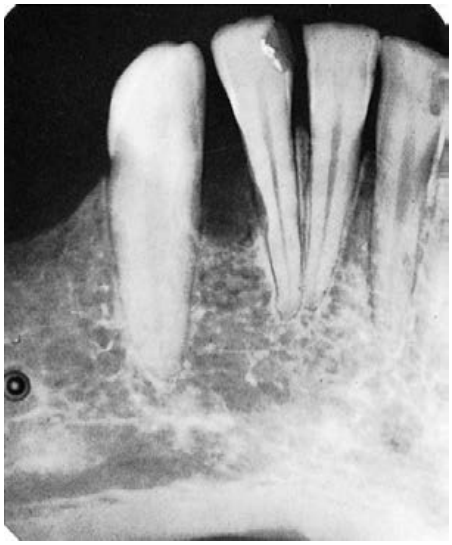

Figure 9-23 Bone destruction in the interproximal space between the canine and lateral incisor caused by the extension of a peripheral giant cell reparative granuloma of the gingiva. *(Courtesy Dr. Sam Toll.)*

Histopathology The giant cell granuloma has numerous foci of multinuclear giant cells and hemosiderin particles in a connective tissue stroma (see Figure 9-22). Areas of chronic inflammation are scattered throughout the lesion, with acute involvement occurring at the surface. The overlying epithelium is usually hyperplastic, with ulceration at the base. Bone formation occasionally occurs within the lesion (Figure 9-23).

Central Giant Cell Granuloma. Giant cell lesions arise within the jaws and produce central cavitation. They occasionally create a deformity of the jaw that makes the gingiva appear enlarged.

Mixed tumors, salivary gland type of tumors, and plasmacytomas of the gingiva have also been described but are not often seen.

Leukoplakia. *Leukoplakia* is a strictly clinical term defined by the World Health Organization as a white patch or plaque that does not rub off and cannot be diagnosed as any other disease. The cause of leukoplakia remains obscure, although it is associated with

Figure 9-24 Leukoplakia of the gingiva.

the use of tobacco (smoke or smokeless). Other probable factors are *Candida albicans,* HPV-16 and HPV-18, and trauma. Leukoplakia of the gingiva varies in appearance from a grayish white, flattened, scaly lesion (Figure 9-24) to a thick, irregularly shaped, keratinous plaque.

Most leukoplakias (80%) are benign; the remaining 20% are malignant or premalignant, and only 3% of these are invasive carcinomas.[31] Biopsy of all leukoplakias is necessary, selecting the most suspicious area, to arrive at a correct diagnosis and institute proper therapy.[31,92]

Histopathology Leukoplakia exhibits hyperkeratosis and acanthosis. Premalignant and malignant cases have a variable degree of atypical epithelial changes that may be mild, moderate, or severe, depending on the extent of involvement of the epithelial layers. When dysplastic changes involve all layers, it is diagnosed as a *carcinoma in situ,* and this may become invasive carcinoma when the basement membrane is breached.[31] Inflammatory involvement of the underlying connective tissue is a common associated finding.

Gingival Cyst. Gingival cysts of microscopic proportions are common, but they seldom reach a clinically significant size.[73] When they do, they appear as localized enlargements that may involve the marginal and attached gingiva. The cysts occur in the mandibular canine and premolar areas, most often on the lingual surface. They are painless, but with expansion, they may cause erosion of the surface of the alveolar bone. The gingival cyst should

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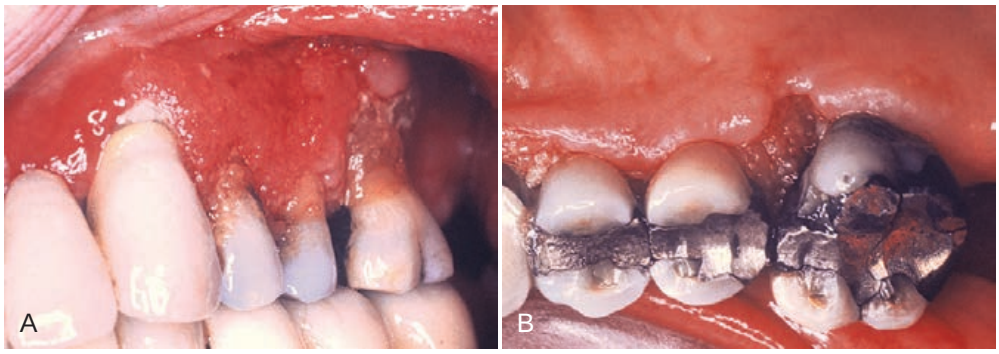

Figure 9-25 Squamous cell carcinoma of the gingiva. **A,** Facial view. Note the extensive verrucous involvement. **B,** Palatal view. Note the mulberry-like tissue emerging between the second premolar and the first molar.

be differentiated from the lateral periodontal cyst (see Chapter 13), which arises within the alveolar bone, adjacent to the root, and is developmental in origin. Gingival cysts develop from odontogenic epithelium or from surface or sulcular epithelium traumatically implanted in the area. Removal is followed by uneventful recovery.

Histopathology A gingival cyst cavity is lined by a thin, flattened epithelium with or without localized areas of thickening. Less frequently, the following types of epithelium can be found: unkeratinized stratified squamous epithelium, keratinized stratified squamous epithelium, and parakeratinized epithelium with palisading basal cells.[16]

Other Benign Masses. Other benign tumors have also been described as rare or infrequent findings in the gingiva. They include nevus,[10] myoblastoma,[36] hemangioma,[109] neurilemoma,[31] neurofibroma,[82] mucus-secreting cysts (mucoceles),[114] and ameloblastoma.[106]

Malignant Tumors of the Gingiva

Carcinoma. Oral cancer accounts for less than 3% of all malignant tumors in the body but is the sixth most common cancer in males and the twelfth in females.[75] The gingiva is not a frequent site of oral malignancy (6% of oral cancers[62]).

Squamous cell carcinoma is the most common malignant tumor of the gingiva. It may be *exophytic,* presenting as an irregular outgrowth, or *ulcerative,* appearing as flat, erosive lesions. It is often symptom free, going unnoticed until complicated by inflammatory changes that may mask the neoplasm but cause pain; sometimes it becomes evident after tooth extraction. These masses are locally invasive, involving the underlying bone and periodontal ligament of adjoining teeth and the adjacent mucosa (Figure 9-25). Metastasis is usually confined to the region above the clavicle; however, more extensive involvement may include the lung, liver, or bone.

Malignant Melanoma. Malignant melanoma is a rare oral tumor that tends to occur in the hard palate and maxillary gingiva of older persons.[75,85] It is usually darkly pigmented and is often preceded by localized pigmentation.[21] It may be flat or nodular and is characterized by rapid growth and early metastasis. It arises from melanoblasts in the gingiva, cheek, or palate. Infiltration into the underlying bone and metastasis to cervical and axillary lymph nodes are common.

Sarcoma. Fibrosarcoma, lymphosarcoma, and reticulum cell sarcoma of the gingiva are rare; only isolated cases have been described in the literature.[40,110] Kaposi's sarcoma often occurs in the oral cavity of patients with acquired immunodeficiency syndrome (AIDS), particularly in the palate and the gingiva (see Chapter 19).

Metastasis. Tumor metastasis to the gingiva occurs infrequently. Such metastasis has been reported with various tumors, including adenocarcinoma of the colon,[49] lung carcinoma, melanoma,[30] renal cell carcinoma,[15] hypernephroma,[81] chondrosarcoma,[110] and testicular tumor.[29]

The low incidence of oral malignancy should not mislead the clinician. Ulcerations that do not respond to therapy in the usual manner, as well as all gingival tumors and tumorlike lesions, must be biopsied and submitted for microscopic diagnosis (see Chapter 35).

FALSE ENLARGEMENT

False enlargements are not true enlargements of the gingival tissues but may appear as such as a result of increases in size of the underlying osseous or dental tissues. The gingiva usually presents with no abnormal clinical features except the massive increase in size of the area.

Underlying Osseous Lesions

Enlargement of the bone subjacent to the gingival area occurs most often in *tori* and *exostoses,* but it can also occur in Paget's disease, fibrous dysplasia, cherubism, central giant cell granuloma, ameloblastoma, osteoma, and osteosarcoma. Figure 9-26 shows fibrous dysplasia (florid type) in a 38-year-old black female that induced an osseous enlargement in the mandibular molar area appearing as a gingival enlargement. The gingival tissue can appear normal or may have unrelated inflammatory changes.

Underlying Dental Tissues

During the various stages of eruption, particularly of the primary dentition, the labial gingiva may show a bulbous marginal distortion caused by superimposition of the bulk of the gingiva on the normal prominence of the enamel in the gingival half of the crown. This enlargement has been termed *developmental enlargement* and often persists until the junctional epithelium has migrated from the enamel to the cementoenamel junction.

In a strict sense, developmental gingival enlargements are physiologic and usually present no problems. However, when

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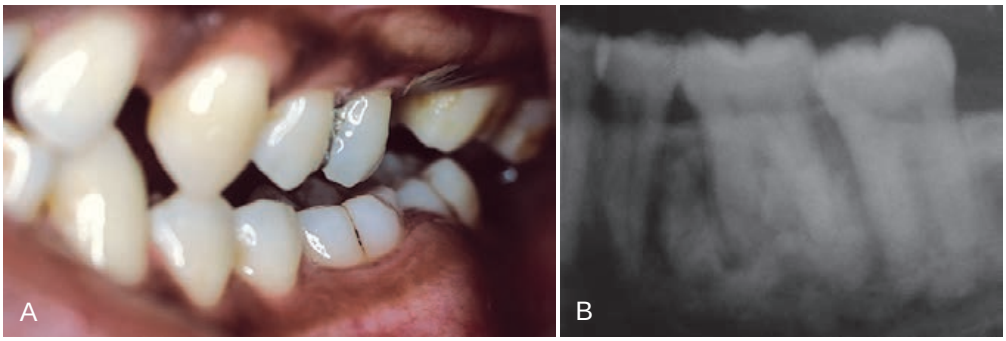

Figure 9-26 **A,** Apparent gingival enlargement associated with bone augmentation in patient with fibrous dysplasia. **B,** Radiograph of case shown in **A,** depicting a ground-glass, mottled pattern.

Figure 9-27 Developmental gingival enlargement. The normal bulbous contour of the gingiva around the incompletely erupted anterior teeth is accentuated by chronic inflammation.

such enlargement is complicated by marginal inflammation, the composite picture gives the impression of extensive gingival enlargement (Figure 9-27). Treatment to alleviate the marginal inflammation, rather than resection of the enlargement, is sufficient in these patients.

NOTE: Chapter 85 in the online version of this book presents numerous examples of gingival enlargements.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 10

Acute Gingival Infections

Perry R. Klokkevold and Fermin A. Carranza

CHAPTER OUTLINE

NECROTIZING ULCERATIVE GINGIVITIS
- Clinical Features
- Relation of Bacteria to the Necrotizing Ulcerative Gingivitis Lesion
- Diagnosis
- Etiology
- Epidemiology and Prevalence
- Communicability

PRIMARY HERPETIC GINGIVOSTOMATITIS
- Clinical Features
- Diagnosis
- Differential Diagnosis
- Communicability

PERICORONITIS
- Clinical Features
- Complications

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Some text and figures may be out of numeric order in this printed chapter.

NECROTIZING ULCERATIVE GINGIVITIS

Necrotizing ulcerative gingivitis (NUG) is a microbial disease of the gingiva in the context of an impaired host response. It is characterized by the death and sloughing of gingival tissue and presents with characteristic signs and symptoms.

Clinical Features

NUG is usually identified as an *acute* disease. However, the term "acute" in this case is a clinical descriptor and should not be used as a diagnosis because there is no chronic form of the disease. The acronym *ANUG,* although frequently used, is a misnomer.[59] NUG often undergoes a diminution in severity without treatment, leading to a *subacute* stage with milder clinical symptoms. Thus patients may have a history of repeated remissions and exacerbations, and the condition can also recur in previously treated patients. Involvement may be limited to a single tooth or group of teeth (Figure 10-1, *A* and *B* [see Figure 10-1, *C*]) or may be widespread throughout the mouth (see Figure 10-1, *D*).

Figures 10-1 C, D can be found on the Expert CONSULT companion website at www.expertconsult.com.

NUG can cause tissue destruction involving the periodontal attachment apparatus,[41] especially in patients with long-standing disease or severe immunosuppression. When bone loss occurs, the condition is called *necrotizing ulcerative periodontitis* (NUP) (see Chapter 17).

History. NUG is characterized by sudden onset, sometimes after an episode of debilitating disease or acute respiratory tract infection. A change in living habits, protracted work without adequate rest, poor nutrition, tobacco use, and psychologic stress are frequent features of the patient's history.

Oral Signs. Characteristic lesions are *punched-out, craterlike depressions at the crest of the interdental papillae*, subsequently extending to the marginal gingiva and rarely to the attached gingiva and oral mucosa. The surface of the gingival craters is covered by a *gray, pseudomembranous slough,* demarcated from the remainder of the gingival mucosa by a pronounced *linear erythema* (see Figure 10-1, *A*). In some cases the lesions are denuded of the surface pseudomembrane, exposing the gingival margin, which is red, shiny, and hemorrhagic. The characteristic lesions may progressively destroy the gingiva and underlying periodontal tissues (see Figure 10-1, *B*).

Spontaneous gingival hemorrhage or pronounced bleeding after the slightest stimulation are additional characteristic clinical signs (see Figure 10-1, *B,* and Figure 10-1, *C*). Other signs often found are fetid odor and increased salivation.

NUG can occur in otherwise disease-free mouths or can be superimposed on chronic gingivitis or periodontal pockets. However, NUG or NUP does not usually lead to periodontal pocket formation because the necrotic changes involve the junctional epithelium; a viable junctional epithelium is needed for pocket deepening (see Chapter 13). It is rare in edentulous mouths, but isolated spherical lesions occasionally occur in the soft palate.[58]

Oral Symptoms. The lesions are *extremely sensitive to touch,* and the patient often complains of a constant radiating, gnawing

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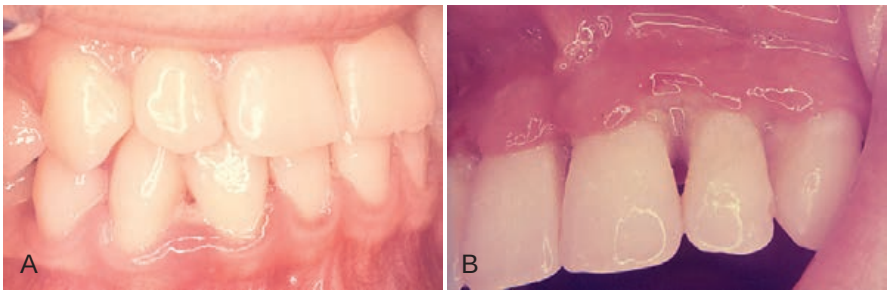

Figure 10-1 Necrotizing ulcerative gingivitis. **A,** Typical punched-out papilla between mandibular canine and lateral incisor, covered by grayish white pseudomembrane. **B,** More advanced case showing destruction of papillae resulting in irregular marginal contour.

pain that is intensified by eating spicy or hot foods and chewing. There is a "metallic" foul taste, and the patient is conscious of an excessive amount of "pasty" saliva.

Extraoral and Systemic Signs and Symptoms. Patients are usually ambulatory and have a minimum of systemic symptoms. Local lymphadenopathy and a slight elevation in temperature are common features of the mild and moderate stages of the disease. In severe cases, there may be high fever, increased pulse rate, leukocytosis, loss of appetite, and general lassitude. Systemic reactions are more severe in children. Insomnia, constipation, gastrointestinal disorders, headache, and mental depression sometimes accompany the condition.

In very rare cases, severe sequelae, such as gangrenous stomatitis and noma, have been described.[1,2,18,33]

Clinical Course. The clinical course can vary. If untreated, NUG may lead to a progressive destruction of the periodontium and gingival recession, accompanied by an increase in the severity of systemic complications.[31,51]

Histopathology Microscopically, the NUG lesion is a nonspecific acute necrotizing inflammation of the gingival margin, involving both the stratified squamous epithelium and the underlying connective tissue. The surface epithelium is destroyed and replaced by a meshwork of fibrin, necrotic epithelial cells, polymorphonuclear leukocytes (PMNs, neutrophils), and various types of microorganisms (see Figure 10-2). This is the zone that appears clinically as the surface pseudomembrane. At the immediate border of the necrotic pseudomembrane, the epithelium is edematous, and the individual cells exhibit varying degrees of hydropic degeneration. In addition, there is an infiltration of PMNs in the intercellular spaces.

The underlying connective tissue is markedly hyperemic, with numerous engorged capillaries and a dense infiltration of PMNs. This acutely inflamed zone appears clinically as the linear erythema beneath the surface pseudomembrane. Numerous plasma cells may appear in the periphery of the infiltrate; this is interpreted as an area of established chronic gingivitis on which the acute lesion became superimposed.[30]

The epithelium and connective tissue alterations decrease as the distance from the necrotic gingival margin increases, blending gradually with the uninvolved gingiva.

It is noteworthy that the microscopic appearance of NUG is nonspecific. Comparable changes result from trauma, chemical irritation, or the application of caustic medications.

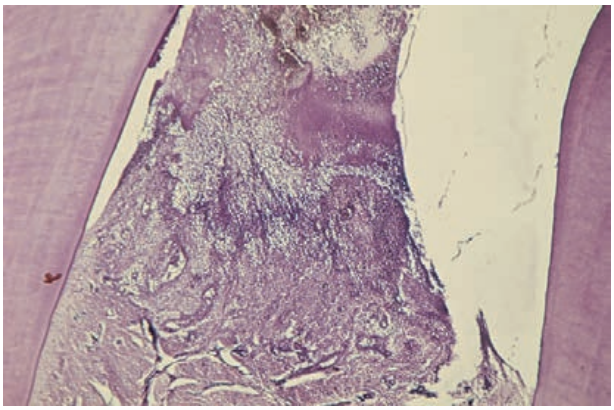

Figure 10-2 Survey section of interdental papilla in necrotizing ulcerative gingivitis. Top portion of the section shows the necrotic tissue that forms the gray marginal pseudomembrane. In lower portion, note the ulceration and accumulation of leukocytes and fibrin.

Relation of Bacteria to the Necrotizing Ulcerative Gingivitis Lesion

Light and electron microscopy have been used to study the relationship of bacteria to the characteristic lesion of NUG. Light microscopy shows that the exudate on the surface of the necrotic lesion contains microorganisms that morphologically resemble cocci, fusiform bacilli, and spirochetes.[76] The layer between the necrotic and living tissue contains enormous numbers of fusiform bacilli and spirochetes, in addition to leukocytes and fibrin. Spirochetes and other bacteria[4,12,16,38] invade the underlying living tissue.

Spirochetes have been found as deep as 300 µm from the surface. The majority of spirochetes in the deeper zones are morphologically different from cultivated strains of *Treponema microdentium*. They occur in nonnecrotic tissue before other types of bacteria and may be present in high concentrations intercellularly in the epithelium adjacent to the ulcerated lesion and in the connective tissue.[37]

Smears from the lesions (Figure 10-3) show scattered bacteria, predominantly spirochetes and fusiform bacilli, desquamated epithelial cells, and occasional polymorphonuclear leukocytes (PMNs). Spirochetes and fusiform bacteria are usually seen with other oral spirochetes, vibrios, and filaments.

Diagnosis

Diagnosis is based on clinical findings of gingival pain, ulceration, and bleeding. A bacterial smear is not necessary or definitive because the bacterial picture is not appreciably different from that in marginal gingivitis, periodontal pockets, pericoronitis, or primary

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SCIENCE TRANSFER

The two most common acute gingival infections are necrotizing gingivitis and herpetic gingivostomatitis. These diseases occur most often in healthy patients, but patients with depressed immunologic responses such as acquired immunodeficiency syndrome (AIDS), leukemia, and cyclic neutropenia have an increased risk for these gingival entities. Necrotizing gingivitis is usually treatable with local root planing, curettage, and plaque control; however, in patients with immunologic deficiencies or patients with evidence of spread beyond the gingival tissues then systemic antibiotics are indicated. Tissue of immunologically incompetent patients can destruct rapidly and should be seen and treated on a daily basis until the necrotizing lesions are controlled.

Primary herpetic gingivostomatitis is accompanied by fever and malaise and as long as there are vesicles, the patient is infectious. Secondary infections of the virus can also cause gingival vesicles and is sometimes triggered by tissue trauma occurring with periodontal treatment such as periodontal surgery and root planing.

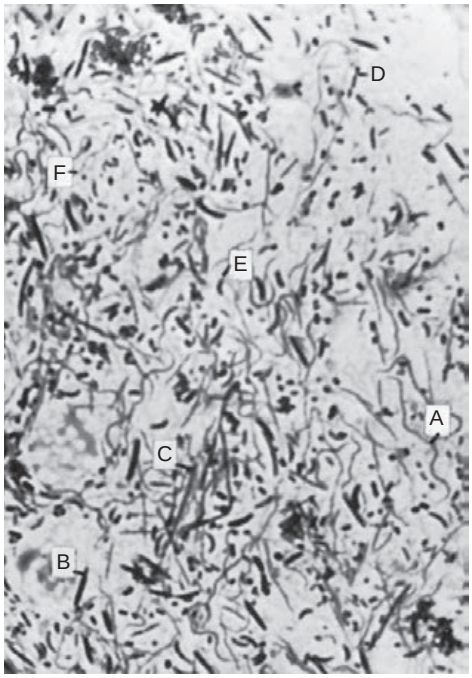

Figure 10-3 Bacterial smear from lesion in necrotizing ulcerative gingivitis. *A,* Spirochete; *B, Bacillus fusiformis; C,* filamentous organism (*Actinomyces* or *Leptotrichia*); *D, Streptococcus; E, Vibrio; F, Treponema microdentium.*

herpetic gingivostomatitis.[57] Bacterial studies are useful, however, in the differential diagnosis of NUG and specific infections of the oral cavity such as diphtheria, thrush, actinomycosis, and streptococcal stomatitis.

Microscopic examination of a biopsy specimen is not sufficiently specific to be diagnostic. It can be used to differentiate NUG from specific infections, such as tuberculosis, or from neoplastic disease, but it does not differentiate between NUG and other necrotizing conditions of nonspecific origin such as those produced by trauma or caustic medications.

Differential Diagnosis. NUG should be differentiated from other conditions that resemble it in some respects, such as herpetic gingivostomatitis (see Table 10-1); chronic periodontitis; desquamative gingivitis (see Table 10-2); streptococcal gingivostomatitis; aphthous stomatitis; gonococcal gingivostomatitis; diphtheritic and syphilitic lesions (see Table 10-3); tuberculous gingival lesions; candidiasis, agranulocytosis, and dermatoses (pemphigus, erythema multiforme, and lichen planus); and stomatitis venenata. Treatment options for these diseases vary dramatically, and improper treatment may exacerbate the condition. In the case of primary herpetic gingivostomatitis, early diagnosis may result in treatment with antiviral drugs that would be ineffective for NUG, whereas treatment of a case of herpes with the debridement required for NUG could exacerbate herpes. (See Chapter 12 for a description of most of these conditions.)

Tables 10-1 to 10-3 can be found on the Expert CONSULT companion website at www.expertconsult.com.

Streptococcal gingivostomatitis is a rare condition characterized by a diffuse erythema of the gingiva and other areas of the oral mucosa.[44] In some cases, it is confined as a marginal erythema with marginal hemorrhage. Necrosis of the gingival margin is not a feature of this disease, and there is no notable fetid odor. Bacterial smears show a preponderance of streptococcal forms, which were identified as *Streptococcus viridans*, but other studies report it to be group A β-hemolytic streptococcus.[39]

Agranulocytosis is characterized by a marked decrease in the number of circulating PMNs, lesions of the throat and other mucous membranes, and ulceration and necrosis of the gingiva, which may resemble that of NUG, and occurs most commonly after chemotherapy in cancer patients or in patients with leukemia. The oral condition in agranulocytosis is primarily necrotizing but lacks the severe inflammatory reaction seen in NUG. Blood studies serve to differentiate between NUG and the gingival necrosis in agranulocytosis.

Vincent's angina is a fusospirochetal infection of the oropharynx and throat, as distinguished from NUG, which affects the marginal gingiva. Patients with Vincent's angina have a painful membranous ulceration of the throat, with edema and hyperemic patches breaking down to form ulcers covered with pseudomembranous material. The process may extend to the larynx and middle ear.

For more information on NUG, please visit flu companion Expert CONSULT companion website at www.expertconsult.com.

Etiology

Role of Bacteria. Plaut[53] in 1894 and Vincent[78] in 1896 postulated that NUG is caused by specific bacteria: fusiform bacillus and a spirochetal organism.

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Opinions still differ regarding whether bacteria are the primary causative factors in NUG. Several observations support this concept, including that spirochetal organisms and fusiform bacilli are always found in the disease, with other organisms also involved. Rosebury et al[57] described a fusospirochetal complex consisting of *T. microdentium*, intermediate spirochetes, vibrios, fusiform bacilli, and filamentous organisms, in addition to several *Borrelia* species.

Loesche et al[40] described a predominant constant flora and a variable flora associated with NUG. The constant flora is composed of *Prevotella intermedia*, in addition to *Fusobacterium*, *Treponema*, and *Selenomonas* species. The variable flora consists of a heterogeneous array of bacterial types.

Treatment with metronidazole results in a significant reduction of *Treponema* species, *Prevotella intermedia*, and *Fusobacterium*, with resolution of the clinical symptoms.[15,40] The antibacterial spectrum of this drug provides evidence for the anaerobic members of the flora as etiologic agents.

These bacteriologic findings have been supported by immunologic data[8] that reported increased immunoglobulin (IgG and IgM) antibody titers for medium-sized spirochetes and *P. intermedia* in NUG patients compared with titers in those with chronic gingivitis and healthy controls.

Role of the Host Response. Regardless of whether specific bacteria are implicated in the etiology of NUG, the presence of these organisms appears to be insufficient to cause the disease.

In the first place, the fusiform-spirochete flora are frequently found in patients who do not have NUG. Also, exudates from NUG lesions produce fusospirochetal abscesses rather than typical NUG, when inoculated subcutaneously in experimental animals.[56] Local intracutaneous injection of a hyaluronidase- and chondroitinase-containing cell-free filtrate of oral microaerophilic diphtheroid bacilli aggravated spirochetal lesions that were produced by oral treponemes.[30] Only in one animal experiment has the transmission of lesions comparable with those seen in humans been reported.[1]

The role of an impaired host response in NUG has long been recognized. Even in the early descriptions of the disease, NUG has been associated with physical and emotional stress[10,59] and decreased resistance to infection. NUG has not been produced experimentally in humans or animals by just inoculation of bacterial exudates from the lesions. In the animal model, local or systemic immunosuppression with glucocorticoids (e.g., ketoconazole) results in more characteristic lesions of NUG in infected animals. Furthermore, NUG is not found in well-nourished individuals with a fully functional immune system. All the predisposing factors for NUG are associated with immunosuppression. Cogen et al[9] described a depression in host defense mechanisms, particularly in PMN chemotaxis and phagocytosis, in NUG patients. (For further details on the host-bacteria interactions in NUG, see Chapters 22, 23, and 25.)

It is essential for the clinician to determine the predisposing factors leading to immunodeficiency in NUG in order to address the continued susceptibility of the patient and to determine whether an underlying systemic disease is present. Immunodeficiency may be related to varying levels of nutritional deficiency, fatigue caused by chronic sleep deprivation, other health habits (e.g., alcohol or drug abuse), psychosocial factors, or systemic disease. Importantly, NUG may be the presenting symptom for patients with immunosuppression related to human immunodeficiency virus (HIV) infection.

Local Predisposing Factors. Preexisting gingivitis, injury to the gingiva, and smoking are important predisposing factors. Although NUG may appear in an otherwise disease-free mouth, it most often occurs superimposed on preexisting chronic gingival disease and periodontal pockets. Deep periodontal pockets and pericoronal flaps are particularly vulnerable areas because they offer a favorable environment for the proliferation of anaerobic fusiform bacilli and spirochetes. Areas of the gingiva traumatized by opposing teeth in malocclusion, such as the palatal surface behind the maxillary incisors and the labial gingival surface of the mandibular incisors, may predispose to NUG.

The relationship between NUG and *smoking* has often been mentioned in the literature. Pindborg[51] reported that 98% of his patients with NUG were smokers and that the frequency of this disease increases with an increasing exposure to tobacco smoke. The effect of smoking on periodontal disease in general has been the subject of numerous studies in the past two decades, and smoking has been established as a high risk factor for disease (see Chapter 26).

Systemic Predisposing Factors. NUG is not found in a well-nourished individual with a fully functional immune system. Therefore it is important for the clinician to determine the predisposing factors leading to immunodeficiency. Again, immunodeficiency may be related to varying levels of nutritional deficiency; fatigue caused by chronic sleep deficiency; other health habits (e.g., alcohol or drug abuse), and systemic disease (e.g., diabetes, debilitating infection).

Nutritional Deficiency. Necrotizing gingivitis has been produced by giving animals nutritionally deficient diets.[6,34,47,74,77] Several researchers found an increase in the fusospirochetal flora in the mouths of the experimental animals, but the bacteria were regarded as opportunists, proliferating only when the tissues were altered by the deficiency. A poor diet has been cited as a predisposing factor in NUG and its sequelae in developing African countries, although the effects appear primarily to diminish the effectiveness of the immune response.[19,20,35]

In a controlled study of malnourished Nigerian children with and without NUG, those with NUG had a 38% increase in cortisol levels and higher levels of proinflammatory cytokines (interleukin [IL]-6, IL-8, IL-10, IL-18, and IL1 β) as compared to controls whose values were close to normal.[20]

Nutritional deficiencies (e.g., vitamin C, vitamin B_2) accentuate the severity of the pathologic changes induced when the fusospirochetal bacterial complex is injected into animals.[72]

Debilitating Disease. Debilitating systemic disease may predispose patients to the development of NUG. Such systemic disturbances include chronic diseases (e.g., syphilis, cancer), severe gastrointestinal disorders (e.g., ulcerative colitis), blood dyscrasias (e.g., leukemia, anemia), and acquired immunodeficiency syndrome (AIDS). Nutritional deficiency resulting from debilitating disease may be an additional predisposing factor. Experimentally-induced leukopenia in animals may produce ulcerative gangrenous stomatitis.[47,75,76] Ulceronecrotic lesions appear in the gingival margins of hamsters exposed to total-body irradiation[42]; these lesions can be prevented with systemic antibiotics.[43]

Psychosomatic Factors. Psychologic factors appear to be important in the etiology of NUG. The disease often occurs in association with stressful situations (e.g., induction into the armed forces, school examinations).[25] Psychologic disturbances,[26] as well as increased adrenocortical secretion,[64] are common in patients with the disease.

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Significant correlation between disease incidence and two personality traits, dominance and abasement, suggests the presence of an NUG-prone personality.[23]

The mechanisms whereby psychologic factors create or predispose to gingival damage have not been established, but alterations in digital and gingival capillary responses suggesting increased autonomic nervous activity have been demonstrated in patients with NUG.[24]

Cohen-Cole et al[10] suggested that a psychiatric disturbance (e.g., trait anxiety, depression, or psychopathic deviance) and the impact of negative life events (stress) may lead to activation of the hypothalamic-pituitary-adrenal axis. This results in elevation of serum and urine cortisol levels, which is associated with a depression of lymphocyte and PMN function that may predispose to NUG.

It can be concluded that opportunistic bacteria are the primary etiologic agents of NUG in patients who demonstrate immunosuppression. Stress, smoking, and preexisting gingivitis are common predisposing factors.

Epidemiology and Prevalence

The prevalence of NUG appears to have been rather low in the United States and Europe before 1914. During World Wars I and II, numerous "epidemics" broke out among the Allied troops, but German soldiers did not seem to have been similarly affected. Epidemic-like outbreaks have also occurred among civilian populations. A study at a dental clinic in Prague, Czech Republic, reported the incidence of NUG as 0.08% in patients age 15 to 19 years, 0.05% in those age 20 to 24, and 0.02% in those age 25 to 29.[67]

NUG occurs at all ages, with the highest incidence reported between ages 20 and 30 years[14,36,72] and ages 15 and 20 years.[67] It is not common in children in the United States, Canada, and Europe, but it has been reported in children from low socioeconomic groups in underdeveloped countries.[33] In India, 54%[46] and 58%[52] of the patients in two studies were under age 10 years.

In a random school population in Nigeria, NUG occurred in 11.3% of children between ages 2 and 6 years[65]; in a Nigerian hospital population, it was present in 23% of children under age 10 years.[18] It has been reported in several members of the same family in low socioeconomic groups. NUG is more common in children with Down syndrome than in other children with mental deficiencies.[3]

Opinions differ as to whether NUG is more common during the winter,[36,49] summer, or fall[65] and whether there is a peak seasonal incidence.[13]

Communicability

NUG often occurs in groups in an epidemic pattern. At one time, NUG was considered contagious and had to be reported to the community health department, but it was later concluded not to be communicable.[55,61] A distinction must be made between "communicability" and "transmissibility" when referring to the characteristics of disease. The term *transmissible* denotes a capacity for the maintenance of an infectious agent in successive passages through a susceptible animal host.[55] The term *communicable* signifies a capacity for the maintenance of infection by natural modes of spread such as direct contact through drinking water, food, and eating utensils; via the airborne route; or by means of arthropod vectors. A disease that is communicable is described as *contagious.* It has been demonstrated that disease associated with the fusospirochetal bacterial complex is transmissible; however, *it has not been shown to be communicable or contagious.*

Attempts have been made to spread NUG from human to human, without success.[62] King[35] traumatized an area in his gingiva and introduced debris from a patient with a severe case of NUG. There was no response until he happened to fall ill shortly thereafter; subsequent to his illness, he observed the characteristic lesion in the experimental area. It may be inferred with reservation from this experiment that systemic debility is a prerequisite for the contagion of NUG.

It is a common impression that because NUG often occurs in groups using the same kitchen facilities, the disease is spread by bacteria on eating utensils. Growth of fusospirochetal organisms requires carefully controlled conditions and an anaerobic environment; they do not ordinarily survive on eating utensils.[11,29]

The occurrence of NUG in epidemic-like outbreaks does not necessarily mean that it is contagious. The affected groups may be afflicted by the disease because of common predisposing factors rather than because of its spread from person to person. In all likelihood, both a predisposed immunocompromised host and the presence of appropriate bacteria are necessary for the production of this disease.

PRIMARY HERPETIC GINGIVOSTOMATITIS

Primary herpetic gingivostomatitis is an infection of the oral cavity caused by the herpes simplex virus type 1 (HSV-1).[14,44,45,60] It occurs most often in infants and children younger than 6 years of age,[5,60,63] but it is also seen in adolescents and adults. It occurs with equal frequency in male and female patients. In most persons, however, the primary infection is asymptomatic.

As part of the primary infection, the virus ascends through sensory and autonomic nerves, where it persists as latent HSV in neuronal ganglia that innervate the site. In approximately one-third of the world's population, secondary manifestations result from various stimuli such as sunlight, trauma, fever, and stress. These secondary manifestations include herpes labialis (Figure 10-4), herpetic stomatitis, herpes genitalis, ocular herpes, and herpetic encephalitis. Secondary herpetic stomatitis can occur on the palate, gingiva (see Figure 10-5), or on the mucosa as a result of dental treatment that traumatizes or stimulates the latent virus in the ganglia innervating the area and may present as pain away from the site of treatment 2 to 4 days later. Careful inspection for characteristic vesicles may be diagnostic (see Figure 10-4).

Figures 10-5 to 10-8 can be found on the Expert CONSULT companion website at www.expertconsult.com.

Clinical Features

Oral Signs. Primary herpetic gingivostomatitis appears as a diffuse, erythematous, shiny involvement of the gingiva and the adjacent oral mucosa, with varying degrees of edema and gingival bleeding. In its initial stage, it is characterized by the presence of discrete, spherical gray vesicles, which may occur on the gingiva, labial and buccal mucosae, soft palate, pharynx, sublingual mucosa, and tongue (see Figure 10-6). After approximately 24 hours, the vesicles rupture and form painful, small ulcers with a red, elevated, halolike margin and a depressed, yellowish or grayish white central portion. These occur either in widely separated areas or in clusters where confluence occurs (see Figure 10-7).

Occasionally, primary herpetic gingivitis may occur without overt vesiculation. The clinical picture consists of diffuse, erythematous, shiny discoloration and edematous enlargement of the gingivae with a tendency toward bleeding.

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Figure 10-4 Recurrent herpetic vesicles in the lip. **A,** Early stage. **B,** Late stage showing brownish crusted lesions. *(From Sapp JP, Eversole, LR, Wysocki GP:* Contemporary oral and maxillofacial pathology, *ed 2, St Louis, 2002, Mosby.)*

The course of the disease is limited to 7 to 10 days. The diffuse gingival erythema and edema that appear early in the disease persist for several days after the ulcerative lesions have healed. Scarring does not occur in the areas of healed ulcerations.

Oral Symptoms. The disease is accompanied by generalized "soreness" of the oral cavity, which interferes with eating, drinking, and oral hygiene. The ruptured vesicles are the focal sites of pain and are particularly sensitive to touch, thermal changes, foods such as condiments and fruit juices, and the action of coarse foods. In infants, the disease is marked by irritability and refusal to take food.

Extraoral and Systemic Signs and Symptoms. Cervical adenitis, fever as high as 101° F to 105° F (38° C to 40.6° C), and generalized malaise are common.

History. Primary herpetic gingivostomatitis is the result of an acute infection by HSV and has an acute onset.

Histopathology The virus targets the epithelial cells, which show "ballooning degeneration" that consists of acantholysis, nuclear clearing, and nuclear enlargement. These cells are called *Tzanck cells.* Infected cells fuse, forming multinucleated cells, and intercellular edema leads to formation of an intraepithelial vesicles that rupture and develop a secondary inflammatory response with a fibropurulent exudate[48] (see Figure 10-8). Discrete ulcerations resulting from rupture of the vesicles have a central portion of acute inflammation, with varying degrees of purulent exudate, surrounded by engorged blood vessels.

Diagnosis

It is critical to arrive at a diagnosis as early as possible in primary herpetic infections. Treatment with antiviral medications can dramatically alter the course of the disease, reducing symptoms and potentially reducing recurrences. The diagnosis is usually established from the patient's history and the clinical findings. Material may be obtained from the lesions and submitted to the laboratory for confirmatory tests, including virus culture and immunologic tests using monoclonal antibodies or deoxyribonucleic acid (DNA) hybridization techniques.[5,54] This should not delay treatment if strong clinical evidence exists for primary gingivostomatitis.

Differential Diagnosis

Primary herpetic gingivostomatitis should be differentiated from several conditions. NUG can be differentiated in different ways (see Table 10-1).

Erythema multiforme can be differentiated because its vesicles are generally more extensive than those in primary herpetic gingivostomatitis and on rupture demonstrate a tendency toward pseudomembrane formation. In addition, the tongue is usually very involved in erythema multiforme, with infection of the ruptured vesicles resulting in varying degrees of ulceration. Oral involvement in erythema multiforme may be accompanied by skin lesions. The duration of erythema multiforme may be comparable with that of primary herpetic gingivostomatitis, but prolonged involvement may occur for weeks.

Stevens-Johnson syndrome is a comparatively rare form of erythema multiforme, characterized by vesicular hemorrhagic lesions in the oral cavity, hemorrhagic ocular lesions, and bullous skin lesions.

Bullous lichen planus is a very rare and painful condition. It is characterized by large blisters on the tongue and cheek that rupture and undergo ulceration; it runs a prolonged, indefinite course. Patches of linear, gray, lacelike lesions of lichen planus are often interspersed among the bullous eruptions. Lichen planus involvement of the skin may coexist with the oral lesions and facilitate differential diagnosis.

Desquamative gingivitis is characterized by diffuse involvement of the gingiva, with varying degrees of "peeling" of the epithelial surface and exposure of the underlying tissue. It is a chronic condition (see Chapter 12).

Lesions of *recurrent aphthous stomatitis* (RAS)[21] range from occasional small (0.5 to 1 cm in diameter), well-defined, round or ovoid, shallow ulcers with a yellowish gray central area surrounded by an erythematous halo, which heal in 7 to 10 days without scarring, to larger (1 to 3 cm in diameter) oval or irregular ulcers, which persist for weeks and heal with scarring (Figure 10-9). The cause is unknown, although immunopathologic mechanisms appear to play a role. RAS is a different clinical entity from primary herpetic gingivostomatitis. The ulcerations may look the same in the two conditions, but diffuse erythematous involvement of the gingiva and acute toxic systemic symptoms do not occur in RAS. A history of previous episodes of painful mucosal ulcerations suggests RAS rather than primary HSV.

Communicability

Primary herpetic gingivostomatitis is contagious.[7,39] Most adults have developed immunity to HSV as the result of infection during

Figure 10-9 Aphthous lesion in the lip. The depressed gray center is surrounded by an elevated red border. *(From Sapp JP, Eversole, LR, Wysocki GP:* Contemporary oral and maxillofacial pathology, *ed 2, St Louis, 2002, Mosby.)*

childhood, which in most cases is subclinical. For this reason, acute herpetic gingivostomatitis usually occurs in infants and children. Recurrent herpetic gingivostomatitis has been reported,[28] although it is not often clinically significant unless immunity is destroyed by debilitating systemic disease. Studies demonstrating HSV in periodontal pockets suggest more recurrence of viral replication than previously recognized.[68] Secondary herpetic infection of the skin, such as herpes labialis, does recur.[66]

PERICORONITIS

The term *pericoronitis* refers to inflammation of the gingiva in relation to the crown of an incompletely erupted tooth (Figure 10-10). It occurs most often in the mandibular third molar area. Pericoronitis may be acute, subacute, or chronic.

Clinical Features

The partially erupted or impacted mandibular third molar is the most common site of pericoronitis. The space between the crown of the tooth and the overlying gingival flap (operculum) is an ideal area for the accumulation of food debris and bacterial growth. Even in patients with no clinical signs or symptoms, the gingival flap is often chronically inflamed and infected and has varying degrees of ulceration along its inner surface. Acute inflammatory involvement is a constant possibility and may be exacerbated by trauma, occlusion, or a foreign body trapped underneath the tissue flap (e.g., popcorn husk, nut fragment).

Acute pericoronitis is identified by varying degrees of inflammatory involvement of the pericoronal flap and adjacent structures, as well as by systemic complications. The inflammatory fluid and cellular exudate increase the bulk of the flap, which then may interfere with complete closure of the jaws and can be traumatized

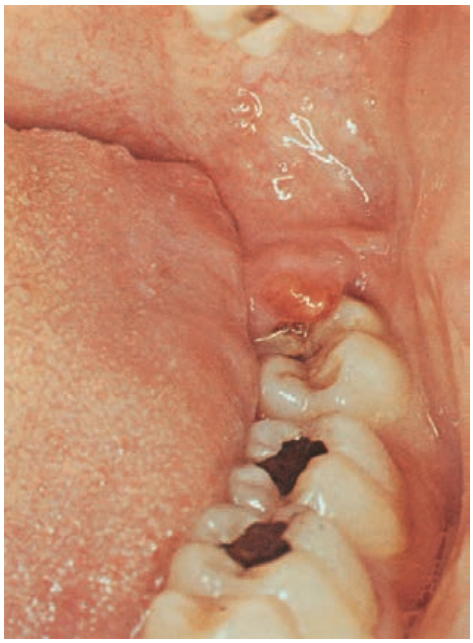

Figure 10-10 Pericoronitis. Inflamed coronal flap covering disto-occlusal surface of impacted mandibular third molar. Note swelling and redness. *(From Glickman I, Smulow J:* Periodontal disease: clinical, radiographic and histopathologic features, *Philadelphia, 1974, Saunders.)*

by contact with the opposing jaw, aggravating the inflammatory involvement.

The resultant clinical picture is a red, swollen, suppurating lesion that is exquisitely tender, with radiating pains to the ear, throat, and floor of the mouth. The patient is extremely uncomfortable because of a foul taste and an inability to close the jaws, in addition to the pain. Swelling of the cheek in the region of the angle of the jaw and lymphadenitis are common findings. Trismus may also be a presenting complaint. The patient may also have systemic complications such as fever, leukocytosis, and malaise.

Complications

Involvement may be localized in the form of a pericoronal abscess. It may spread posteriorly into the oropharyngeal area and medially to the base of the tongue, making it difficult for the patient to swallow. Depending on the severity and extent of the infection, there is involvement of the submaxillary, posterior cervical, deep cervical, and retropharyngeal lymph nodes.[32,50] Peritonsillar abscess formation, cellulitis, and Ludwig's angina are infrequent but potential sequelae of acute pericoronitis.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 11

Gingival Diseases in Childhood*

Clarice S. Law, Donald F. Duperon, James J. Crall, and Fermin A. Carranza

CHAPTER OUTLINE

PERIODONTIUM OF THE PRIMARY DENTITION
PERIODONTAL CHANGES ASSOCIATED WITH NORMAL DEVELOPMENT
Tooth Eruption
Ankylosed Teeth
Primary Tooth Exfoliation
GINGIVAL DISEASES OF CHILDHOOD
Plaque-Induced Gingival Disease
Non–Plaque-Induced Gingival Lesions
PERIODONTAL DISEASES OF CHILDHOOD
Aggressive Periodontitis
Chronic Periodontitis
GINGIVAL MANIFESTATION OF SYSTEMIC DISEASE IN CHILDREN
Endocrine Disorders and Hormonal Changes
Hematologic Disorders and Immune Deficiencies
ORAL MUCOSA IN CHILDHOOD DISEASES
THERAPEUTIC CONSIDERATIONS IN THE PEDIATRIC PATIENT
SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Periodontal disease in adults is partly precipitated by gingival inflammation in the formative years of childhood and early adolescence. The nondestructive gingival inflammation of childhood without appropriate intervention may progress to the more significant periodontal diseases seen in the adult population.

After first reviewing anatomic and physiologic changes in the periodontium and dentition, this chapter presents the gingival changes associated with childhood and adolescence. Periodontal diseases in these early periods of life are presented in the chapters dealing with the respective diseases (see Chapters 16, 17, 18, and 27).

PERIODONTIUM OF THE PRIMARY DENTITION

The normal gingiva of the primary dentition is somewhat different from that found in adults. The tissues are pale pink but to a lesser degree than the attached gingiva of adults because the thinness of the keratinized layer causes the underlying vessels in children to be more visible.[36] Stippling appears at about 3 years of age and has been reported in 56% of children between ages 3 and 10 years, with little difference between maxillary and mandibular arches or males and females throughout childhood[10] (Figure 11-1).

The *interdental gingiva* is broad buccolingually and narrow mesiodistally, consistent with the morphology of the primary dentition. Its structure and composition are similar to adult gingiva.

Gingival sulcular depth is shallower in the primary dentition than in the permanent dentition. Probing depths range from 1 to 2 mm, with an increase in depth from anterior to posterior.[8,20,49]

The *attached gingiva* varies in width anteroposteriorly, with a range of 3 to 6 mm. On the buccal surfaces, the width decreases from anterior to posterior, with some data indicating a narrowing over the canines (Figure 11-2). The lingual attached gingiva shows an inverse relationship, with an increase in width from anterior to posterior.[20] The gingival width normally increases with age as children transition from primary to permanent dentition.[5,8,14,49] Interestingly, the junctional epithelium is thicker in the primary dentition than in the permanent dentition,[9] which is a phenomenon thought to reduce the permeability of the epithelium to bacterial toxins.

Radiographically, the lamina dura is prominent in the primary dentition, with a wider periodontal space than in the permanent dentition. The marrow spaces of the bone are larger, and the crests of the interdental bony septa are flat, with bony crests within 1 to 2 mm of the cementoenamel junction[21] (Figure 11-3).

PERIODONTAL CHANGES ASSOCIATED WITH NORMAL DEVELOPMENT

Significant changes occur in the periodontium as the dentition changes from primary to permanent teeth. Most of the changes are associated with eruption and are physiologic in nature. These

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Figure 11-1 Normal gingiva of a 5-year-old child showing light stippling and flattened interproximal gingival in areas of physiologic spacing.

Figure 11-2 Normal gingiva demonstrating width of the attached gingiva, as illustrated by the pigmentation that occurs only in the attached gingival area. This 6-year-old African-American youngster has erupting lower central incisors. As the incisors erupt, the attached gingiva will widen as the alveolus grows.

Figure 11-3 Bite-wing radiograph of a 5-year-old child illustrating the flattened interseptal bone, which is normal in the primary dentition.

changes should be distinguished from gingival disease, which may occur simultaneously.

Tooth Eruption

Before eruption of a primary or permanent tooth, the gingiva reveals a bulge that is firm and pink or blanched because of the underlying tooth crown (Figure 11-4). Occasionally, an *eruption cyst*, which may be filled with blood and generally presents as a bluish or deep red enlargement of the gingiva over the erupting tooth, may be evident. The most common sites are the primary lower incisors and the permanent first molars. Many resolve without treatment but may be marsupialized if they are painful or interfere with occlusion.[13]

Figure 11-4 On the right side, the central incisor bulge is evident, a normal finding at this stage of eruption. On the left side, an eruption cyst is evident. These cysts may exhibit the blue color illustrated here or may be hemorrhagic in nature.

As a tooth erupts, the gingival margin and sulcus develop. At this point the margin is rounded, edematous, and reddened. During the period of active tooth eruption, it is normal for the marginal gingiva surrounding partially erupted teeth to appear prominent; this is most evident in the maxillary anterior region. The prominence is caused by the height of contour of the erupting tooth and mild inflammation from mastication. Poor oral hygiene can contribute to the development of significant gingivitis in unprotected gingival areas.

Ankylosed Teeth

External trauma, occlusal trauma, or developmental disturbances may result in ankylosis of primary teeth and occasionally of permanent teeth. These teeth appear to be intruded or sunken relative to adjacent teeth. Ankylosed primary teeth may interfere with alveolar bone growth around erupting permanent teeth and may prevent or delay their timely emergence into the oral cavity. Ankylosis of primary first molars often resolves spontaneously as the succedaneous premolar erupts; however, a significant number of ankylosed second primary molars will prevent the eruption of the underlying premolar and should be managed appropriately before the completion of root development of the premolar.

Primary Tooth Exfoliation

Like tooth eruption, the process of tooth exfoliation involves changes in the periodontium. The depth of the gingival sulcus increases as the junctional epithelium migrates down the resorbing root of an exfoliating tooth.[7,9] During this process, there may be changes in the permeability and integrity of the junctional epithelium, making the exfoliating tooth more susceptible to inflammation.[9]

Normal functional occlusal forces may traumatize the remaining supporting periodontal tissues of an exfoliating primary tooth or its succedaneous replacement.[22] During the process of exfoliation, teeth may change position, possibly leading to changes in occlusion. Malalignment caused by spacing and changes in skeletal relationships related to erupting teeth also may contribute to significant trauma of periodontal structures.

Microscopically, minor traumatic changes may demonstrate compression, ischemia, and hyalinization of the periodontal ligament.[26,42] With more severe injury, crushing and necrosis of the periodontal ligament may occur. In most patients, these injuries are spontaneously resolved as teeth exfoliate, erupt, and align through the normal growth and development processes.

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Figure 11-5 Gingivitis secondary to crowding and poor oral hygiene.

Figure 11-6 Crowding of the dentition can lead to gingivitis because of the difficulty of cleansing the area.

Relation of Periodontal Status to Malocclusion. Data indicate an association between abnormal tooth position and gingivitis.[17] Crowding in the mixed dentition can often make plaque and food removal more difficult, leading to an increased incidence of gingivitis (Figures 11-5 and 11-6). Severe changes may include gingival enlargement, discoloration, occasional ulceration, and the formation of deep pockets or pseudopockets. Generally, gingival health can be restored by orthodontic correction, but failure to align teeth does not necessarily have an effect on periodontal disease later in life.[17]

Mucogingival Problems. The prevalence of mucogingival problems and recession in children ranges from 1% to 19%, depending on the criteria used to assess the condition.[30] Evidence suggests that some mucogingival problems may start in the primary dentition as a consequence of developmental aberrations in eruption and deficiencies in the thickness of the periodontium.[36,37] Frenum pull may also be a factor in the development of mucogingival problems.[45,50] In the mixed dentition, recession is most often found on the facial aspect of mandibular permanent incisors secondary to rotations or labial positioning related to space problems (Figure 11-7). Although erupting permanent lower incisors often show minimal attached gingiva, gingival width often increases as the teeth erupt and stimulate bone development[14] (see Figure 11-2).

The maxillary canine region is also prone to localized gingival recession. Late-erupting canines in a crowded dentition may be displaced buccally, erupting into or near unattached gingiva or mucosa, and increasing the risk of insufficient gingival tissue width and recession. Recession also may be associated with an anterior open bite secondary to labial inclination of the teeth.[33] Orthodontic

Figure 11-7 Labial stripping secondary to a class II growth pattern and lower incisor crowding. The labially erupting lower incisor demonstrates an associated gingivitis from poor oral hygiene. Immediate orthodontic treatment should be undertaken followed by appropriate grafting, if necessary.

Figure 11-8 Mucosal injury secondary to excessive toothbrushing by the parent. Both the mucosa and the bone have receded more than two-thirds of the root length. When the practice was discontinued, some healing occurred.

treatment and realignment may be necessary to protect the integrity of the attached gingiva.

Mucogingival problems also can result from factitious habits or excessive toothbrushing either by a parent or child (Figure 11-8). Because the width of the attached gingiva increases with age, any of these problems may resolve spontaneously, suggesting a cautious approach to treatment with judicious monitoring instead of immediate surgical intervention.[14,46]

GINGIVAL DISEASES OF CHILDHOOD

Plaque-Induced Gingival Disease

Gingivitis is extremely common in children and adolescents, affecting up to 70% of children older than seven years of age.[15,40] Inflammation is generally limited to the marginal gingiva (Figure 11-9), with undetectable loss of bone or connective tissue attachment in most cases. Although gingivitis does not always progress to periodontitis, management of the gingival disease in children is important because periodontitis is always preceded by gingivitis.[40]

In children, as in adults, the primary cause of gingivitis is dental plaque, which is related to poor oral hygiene. The relationship between plaque and the gingival index, however, is weak and remains unclear.[7,34,35] Although gingivitis is highly prevalent in

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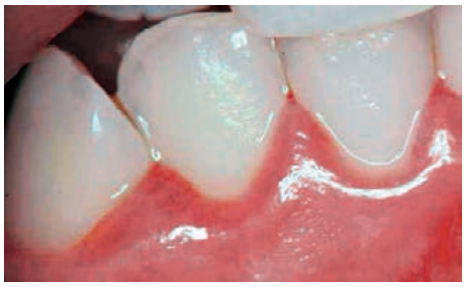

Figure 11-9 Chronic marginal gingivitis secondary to poor oral hygiene. Note the erythematous and edematous tissues of the gingival margin.

Figure 11-10 Eruption gingivitis complicated with severe marginal gingivitis secondary to the illustrated poor oral hygiene.

children, its severity generally is less intense than in adults.[9] Similar oral hygiene conditions generally produce less severe forms of disease in children compared to adults.[40]

As children age, their tendency to develop gingivitis increases.[9] The prevalence of disease is lowest during the preschool years and increases throughout childhood, peaking during puberty. Increases in gingivitis do not fully correlate with the amount of plaque, suggesting the influence of other factors.

Clinical Features. The most prevalent type of gingival disease in childhood is *chronic marginal gingivitis* (see Figure 11-9). The gingival tissues exhibit changes in color, size, consistency, and surface texture similar to chronic inflammation in the adult. Red, linear inflammation is accompanied by underlying chronic changes, including swelling, increased vascularization, and hyperplasia. Bleeding and increased pocket depth are not found as often in children as in adults but may be observed if severe gingival hypertrophy or hyperplasia occur.[7,40] Chronic gingivitis in children is characterized by loss of collagen in the area around the junctional epithelium and an infiltrate consisting mostly of lymphocytes, with small numbers of polymorphonuclear leukocytes, plasma cells, monocytes, and mast cells. Lesions generally have relatively few plasma cells and resemble the early nondestructive, nonprogressive lesions seen in adults.

Gingivitis in children also differs from adult gingivitis in that the response is dominated by T lymphocytes, with few B lymphocytes and plasma cells in the infiltrate. This difference could explain why gingivitis in children rarely progresses to periodontitis.[29,31,32,35,47]

Gingival histology in children also demonstrates other unique features that may contribute to a decreased tendency to progress to severe gingivitis. The junctional epithelium of the primary dentition tends to be thicker than in the permanent dentition,[9] which is thought to reduce the permeability of the gingival structures to bacterial toxins that initiate the inflammatory response.

Calculus. Calculus deposits are uncommon in infants and toddlers, but may increase with age. About 9% of 4- to 6-year-old children exhibit calculus deposits. By age 7 to 9 years, 18% of children present with calculus deposits, and by age 10 to 15 years, 33% to 43% have some calculus formation. Within the category of special needs patients, children with cystic fibrosis have a higher incidence of number of calculus deposits, which may be caused by increased calcium and phosphate concentrations in their saliva.[54] Children fed exclusively with gastric or nasogastric tubes show significant calculus buildup secondary to lack of function and increased oral pH.

Microbiology of Disease. Because the intensity of gingival disease increases as a child develops into adulthood, it is important to understand the microbiology of disease, which is discussed more fully in Chapter 23. Interestingly, the composition of the oral microflora also changes as the child matures.[9] Yang et al[55] analyzed samples of dental plaque in children and reported that 71% of 18- to 48-month-old children were infected with at least one periodontal pathogen. Sixty-eight percent were infected with *Porphyromonas gingivalis* and 20% exhibited *Bacteroides forsythus* (*Tannerella forsythia*).[55] A moderate correlation also has been found between *B. forsythus* in children and periodontal disease in their mothers. *B. forsythus* also has been associated with gingival bleeding in children.

In a similar study, 60% of children between 2 and 18 years of age had detectable levels of *P. gingivalis* in their plaque and 75% showed similar levels of *Actinobacillus actinomycetemcomitans*. The presence of *P. gingivalis* was most strongly associated with the progression of gingivitis and the onset of periodontitis in healthy children.[39]

Experimental gingivitis models in children have demonstrated increased subgingival levels of *Actinomyces*, *Capnocytophaga*, *Leptotrichia* and *Selenomonas*[15]—pathogens that generally are not seen in adult gingivitis—thereby raising interest in their potential role in the etiology of childhood gingivitis.

Eruption Gingivitis. Gingivitis associated with tooth eruption is so common that the term *eruption gingivitis* has come into common use. Tooth eruption per se does not cause gingivitis; however, inflammation associated with plaque accumulation around erupting teeth, perhaps secondary to discomfort caused by brushing these friable areas, may contribute to gingivitis.[9] The gingiva around erupting teeth may appear reddened because gingival margins have not yet keratinized fully and sulcus development is incomplete (Figure 11-10).

Exfoliating and severely carious primary teeth often contribute to gingivitis caused by plaque accumulation secondary to pain during brushing or food impaction in areas of tooth destruction. As a normal part of exfoliation, the junctional epithelium migrates under the resorbing tooth, increasing pocket depth and potentially creating a niche for pathogenic bacteria.[9] The discomfort of chewing on severely infected teeth often leads to unilateral chewing on the unaffected side.

Puberty Gingivitis. As mentioned previously, the incidence of marginal gingivitis increases as a child matures, peaking at 9 to 14 years of age, then decreasing slightly after puberty.[9] Gingival disease that behaves in such a manner is often referred to as *pubertal*

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Figure 11-11 Pubertal gingival inflammation and enlargement secondary to poor oral hygiene and hormonal influences. Little or no calculus was found when the child's teeth were scaled. This most often clears with improved oral hygiene and natural stabilization of estrogen and testosterone levels.

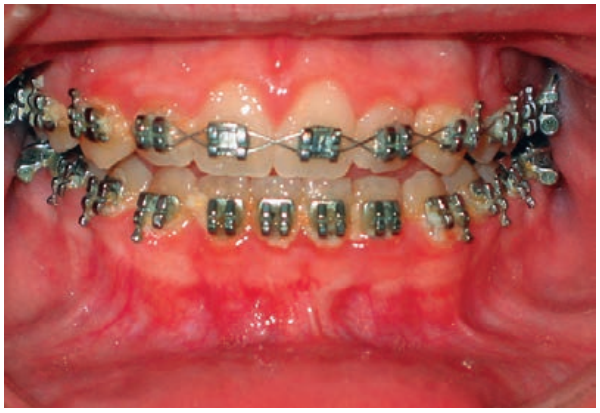

Figure 11-12 Chronic marginal gingivitis secondary to orthodontic therapy and inadequate oral hygiene. Improved hygiene coupled with chlorhexidine mouthwash may help to reduce the inflammation in this patient.

(or puberty) *gingivitis.* Chapters 9 and 27 continue the discussion of this condition.

The most frequent manifestation of puberty gingivitis is bleeding and inflammation in interproximal areas. Inflammatory gingival enlargement may also be noted in both males and females and generally subsides after puberty[38] (Figure 11-11).

The altered gingival response during this developmental stage is thought to be the result of hormonal changes that magnify the vascular and inflammatory response to dental plaque[9,40] and modify reactions of dental plaque microbes.[19]

Drug-Induced Gingival Enlargement. Gingival enlargement, discussed in Chapter 9, may result from the use of certain drugs. Cyclosporine, phenytoin, and calcium channel blockers—drugs used to treat conditions encountered in childhood—result in a higher prevalence of gingival enlargement. Although complicated by the plaque levels along the gingival margin, this form of gingival disease has features that are not typical of chronic marginal gingivitis.[40]

Figure 11-13 Patient with gingivitis secondary to compulsive mouth breathing secondary to allergic rhinitis. This is a typical gingival response to chronic desiccation in adolescent patients. This was originally diagnosed as "allergic gingivitis" by the patient's pediatrician because it subsided when he gave the patient antihistamines. Of course, the antihistamines simply allowed him to breathe through his nose so he could keep his mouth closed.

Gingival Changes Related to Orthodontic Appliances. Gingival enlargement can be related to the presence of fixed orthodontic appliances, which complicate plaque removal (Figure 11-12). Gingival changes can occur within 1 to 2 months of appliance placement, are generally transient, and only rarely produce long-term damage to periodontal tissues.[17] The fact that most orthodontic treatment is provided to individuals during puberty when they are subject to the inflammatory changes associated with puberty gingivitis may exacerbate the observed effect.

Mouth Breathing. Mouth breathing and lip incompetence, or an open mouth posture, are often associated with increased plaque and gingival inflammation.[17] The area of inflammation is often limited to the gingiva of the maxillary incisors. There is often a clear line of demarcation in which the gingiva is uncovered by the lip (Figure 11-13).

Non–Plaque-Induced Gingival Lesions

Intraoral soft tissue lesions may be encountered in the pediatric population as in the adult population. The six most common pediatric intraoral lesions are primary herpetic gingivostomatitis, recurrent herpes simplex, recurrent aphthous stomatitis, candidiasis, angular cheilitis, and geographic tongue.[40] Most of these lesions present without significant difference between the pediatric and adult population. Two have specific pediatric considerations.

Primary Herpetic Gingivostomatitis. Primary herpetic gingivostomatitis is an acute-onset viral infection that occurs early in childhood, with a heightened incidence from 1 to 3 years of age (see Chapter 10). In children with primary herpetic infections, 99% are symptom-free or have symptoms that are attributed to teething. The remaining 1% can develop significant gingival inflammation and ulceration of the lips and mucous membranes[24,40] (Figure 11-14).

Candidiasis. Candidiasis results from an overgrowth of *Candida albicans*, usually after a course of antibiotics or as a result of congenital or acquired immunodeficiencies. It is far less common in the child than the adult and is rarely associated with a healthy child.[12]

PERIODONTAL DISEASES OF CHILDHOOD

Although gingivitis is considered to be "nearly universal" in children over the age of 7 years,[15,40] frank periodontal disease with loss

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Children have a high incidence of gingivitis but are generally less susceptible to chronic periodontitis than adults. Aggressive periodontitis most commonly begins early in life, and there are other forms of gingival and periodontal diseases that have a predilection for first appearing in children (e.g., mouth breathing gingivitis, herpetic gingivostomatitis, and neutropenia-related changes.

Children should have a periodontal evaluation, including recording of pocket depths, gingival recession, and gingival hyperplasia soon after the deciduous teeth erupt, followed by annual re-evaluations. Oral hygiene habits should be imprinted early in life with instructions on techniques and directions on frequencies of plaque removal procedures. These will form a foundation for a lifetime of dedication to periodontal health.

Clinicians should be aware of the specific periodontal needs of children with particular abnormalities such as gingival hyperplasia associated with immune suppression protocols used with organ transplants, antiseizure medications, and increased severity of periodontal disease in diabetic children. Physically and mentally handicapped children deserve special care to ensure that appropriate preventive techniques are available; these may include the use of electric toothbrushes and antibacterial mouthrinses.

Figure 11-14 Acute herpetic gingivostomatitis in an 18-month-old child. Several active lesions still exist on the tongue. The gingiva demonstrates the typical red, swollen appearance associated with the herpes virus. The infection is mostly limited to the attached gingiva, tongue, palate, and lips. It is most important to control hydration with bland, nonacetic fluids. Hospitalization may be necessary for rehydration in severe cases.

Figure 11-15 Acute necrotizing ulcerative gingivitis in a preschool child.

of periodontal attachment and supporting bone is far less common in the pediatric population than in adults.[15] The incidence of disease begins increasing between age 12 to 17 years, but the prevalence of severe attachment loss involving multiple teeth remains low at 0.2% to 0.5%.[15] When comparing the different presentations of periodontal disease, chronic periodontitis has been shown to be more prevalent in adults and aggressive periodontitis is more common in children and adolescents.[15]

See Chapters 16, 17, and 18 for detailed descriptions of the different types of periodontal disease.

For more information on aggressive and chronic periodontitis, please visit the companion Expert CONSULT website at www.expertconsult.com.

GINGIVAL MANIFESTATION OF SYSTEMIC DISEASE IN CHILDREN

Systemic diseases resulting in periodontitis occur more frequently in children than adults.[2] Chapter 27 discusses some of the general systemic diseases and disorders that impact periodontal health. Many diseases, however, are expressed differently in children than in adults and therefore merit special mention.

Acute necrotizing gingivitis (Figure 11-15) is very rarely seen except in cases of primary or secondary immune suppression, Down syndrome, or severe malnutrition.[16,23,43] The breath is fetid, and the child complains of pain and discomfort when eating (see Chapter 10).

Endocrine Disorders and Hormonal Changes

Diabetes Mellitus. Type 1 or insulin-dependent diabetes mellitus occurs more frequently in children and young adults than type 2 or non–insulin-dependent diabetes mellitus. As in the diabetic adult, gingival inflammation and periodontitis are more prevalent in affected children than in unaffected individuals.[40,44] Clinical consequences include premature tooth loss and impaired immune response to the oral flora. The severity of periodontal disease is worse in children with poor metabolic control.

Although destructive changes are rather rare in healthy children, periodontal destruction can be observed in diabetic children, usually appearing around the time of puberty and becoming progressively worse as children mature into adulthood. Disease prevention and fastidious oral hygiene measures should be highly promoted.[27]

Hematologic Disorders and Immune Deficiencies

Leukemias. Leukemia is the most common type of cancer in children. Acute lymphocytic leukemia accounts for the majority of

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cases in children under the 7 years of age. Leukemia must be considered in the differential diagnosis for children who present with the hallmark features of acute gingival enlargement, ulceration, bleeding, and infection.[1]

Leukocyte (Neutrophil) Disorders. As mentioned in Chapter 27, neutrophil disorders impair defenses against infections, making afflicted individuals susceptible to severe periodontal destruction. Many neutrophil disorders are genetic, including some forms of neutropenia, Chediak-Higashi syndrome, leukocyte adhesion deficiency, and Papillon-Lefèvre syndrome. Therefore diagnosis of the systemic disorder generally will have occurred before any signs of periodontal destruction appear. Since periodontal changes are difficult to reverse in children with neutrophil disorders, disease management includes oral hygiene measures, mechanical debridement, antimicrobial therapy, and supportive care for resultant tissue destruction or tooth loss. Treatment success is unpredictable as a result of the impact of systemic disease.[15]

Congenital Anomalies. Down syndrome is another congenital condition that would be diagnosed before expression of periodontal disease. Afflicted individuals experience a high prevalence of severe aggressive periodontitis in early adulthood. The disease process is thought to be related to some kind of host susceptibility resulting in an exaggerated immune-inflammatory response rather than a reaction to a specific causative microbe.[3,4,16]

ORAL MUCOSA IN CHILDHOOD DISEASES

Some childhood diseases present alterations or lesions of the oral mucosa and underlying tissues such as rubeola (rubella, measles), varicella (chickenpox), diphtheria, and scarlatina (scarlet fever). For a discussion of these diseases, the reader is referred to texts on oral and pediatric pathology.

For more information on therapeutic considerations, please visit the companion Expert CONSULT website at www.expertconsult.com.

SUMMARY

- The periodontium of the primary dentition differs from that of the permanent dentition.
- Normal development can result in changes to the periodontium.
- Plaque-induced gingivitis is very common in children, although it may be less intense than in adults.
- With the exception of localized aggressive periodontitis, children rarely show signs of periodontitis.
- Some systemic disorders commonly associated with periodontal disease present initially during childhood.
- Recommendations regarding home plaque control routines should be individualized according to each patient's periodontal disease status and developmental stage.

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 12

Desquamative Gingivitis

Alfredo Aguirre, Jose Luis Tapia Vazquez, and Russell J. Nisengard

CHAPTER OUTLINE

CHRONIC DESQUAMATIVE GINGIVITIS
DIAGNOSIS OF DESQUAMATIVE GINGIVITIS: A SYSTEMATIC APPROACH
DISEASES CLINICALLY PRESENTING AS DESQUAMATIVE GINGIVITIS
Lichen Planus
Pemphigoid
Pemphigus Vulgaris
Chronic Ulcerative Stomatitis
Linear IgA Disease (Linear IgA Dermatosis)
Dermatitis Herpetiformis
Lupus Erythematosus
Erythema Multiforme
DRUG ERUPTIONS
MISCELLANEOUS CONDITIONS MIMICKING DESQUAMATIVE GINGIVITIS

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures may be out of numeric order in this printed chapter.

CHRONIC DESQUAMATIVE GINGIVITIS

Although first recognized and reported in 1894,[156] the term *chronic desquamative gingivitis* was coined in 1932 by Prinz[122] to describe a peculiar condition characterized by intense erythema, desquamation, and ulceration of the free and attached gingiva[50,100] (Figure 12-1). Patients may be asymptomatic; when symptomatic, however, their complaints range from a mild burning sensation to an intense pain. Approximately 50% of desquamative gingivitis cases are localized to the gingiva, although patients can have involvement of the gingiva plus other intraoral and even extraoral sites.[56,112] Initially, the cause of this condition was unclear, with a variety of possibilities suggested. Because most cases were diagnosed in women in the fourth to fifth decades of life (although desquamative gingivitis may occur as early as puberty or as late as the seventh or eighth decade), a hormonal derangement was suspected. In 1960, however, McCarthy et al[94] suggested that *desquamative gingivitis was not a specific disease entity, but a gingival response associated with a variety of conditions.* This concept has been further supported by numerous immunopathologic studies.[77,114,130,151]

Use of clinical and laboratory parameters have revealed that approximately 75% of desquamative gingivitis cases have a dermatologic genesis. Cicatricial pemphigoid and lichen planus account for more than 95% of the dermatologic cases.[110] However, many other mucocutaneous autoimmune conditions, such as bullous pemphigoid, pemphigus vulgaris, linear immunoglobulin A (IgA) disease, dermatitis herpetiformis, lupus erythematosus, and chronic ulcerative stomatitis, can clinically manifest as desquamative gingivitis.[138]

Other conditions that must be considered in the differential diagnosis of desquamative gingivitis include chronic bacterial, fungal, and viral infections, as well as reactions to medications, mouthwashes, and chewing gum. Although less common, Crohn's disease, sarcoidosis, some leukemias, and even factitious lesions have also been reported to present clinically as desquamative gingivitis.[138,160]

Therefore it is of paramount importance to ascertain the identity of the disease responsible for desquamative gingivitis to establish the appropriate therapeutic approach and management. To achieve this goal, the clinical examination must be coupled with a thorough history and routine histologic and immunofluorescence studies.[160] Despite this diagnostic approach, however, the cause of desquamative gingivitis cannot be elucidated in up to one-third of the cases.[125]

DIAGNOSIS OF DESQUAMATIVE GINGIVITIS: A SYSTEMATIC APPROACH

Desquamative gingivitis is only a clinical term, not a diagnosis per se. Once it is rendered, a series of laboratory procedures should be used to arrive at a final diagnosis. Thus the success of any given therapeutic approach depends on the establishment of an accurate final diagnosis. The following discussion represents a systematic

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approach to elucidate the disease triggering desquamative gingivitis (Figure 12-2).

Clinical History. A thorough clinical history is mandatory to begin the assessment of desquamative gingivitis.[113] Data regarding the symptomatology associated with this condition, as well as its historical aspects (i.e., when did the lesion start, has it worsened, or is there a habit that exacerbates the condition), provide the foundation for a thorough examination. Information regarding previous therapy to alleviate the condition should also be documented.

Clinical Examination. Recognition of the pattern of distribution of the lesions (i.e., focal or multifocal, with or without confinement to the gingival tissues) provides leading information to begin the formulation of a differential diagnosis. In addition, a simple clinical maneuver. such as Nikolsky's sign, offers insight into the plausibility of the presence of a vesiculobullous disorder.[97]

Figure 12-1 Chronic desquamative gingivitis. Irregular, conspicuous erythema involves the free and attached gingival tissues.

Biopsy. Given the extent and number of lesions that may be present in an individual, an incisional biopsy is the best alternative to begin the microscopic and immunologic evaluation. An important consideration is selection of the biopsy site. A perilesional incisional biopsy should avoid areas of ulceration because necrosis and epithelial denudation severely hamper the diagnostic process. Once the tissue is excised from the oral cavity, the specimen can be bisected and then submitted for microscopic examination. Buffered formalin (10%) should be used to fix the tissue for conventional hematoxylin and eosin (H&E) evaluation. Michell's buffer (ammonium sulfate buffer, pH 7.0) is used as transport solution for immunofluorescence assessment. In general, an incisional biopsy of uninvolved (normal) mucosa will show the same immunofluorescent findings as the biopsy of the perilesional tissue. However, there are notable exceptions, such as lichen planus and chronic cutaneous lupus erythematosus, in which only the lesional tissue will exhibit the corresponding immunologic markers (Table 12-1).

Microscopic Examination. Sections of approximately 5 µm of formalin-fixed, paraffin-embedded tissue stained with conventional H&E are obtained for light microscopy examination.

Immunofluorescence. For *direct* immunofluorescence, unfixed frozen sections are incubated with a variety of

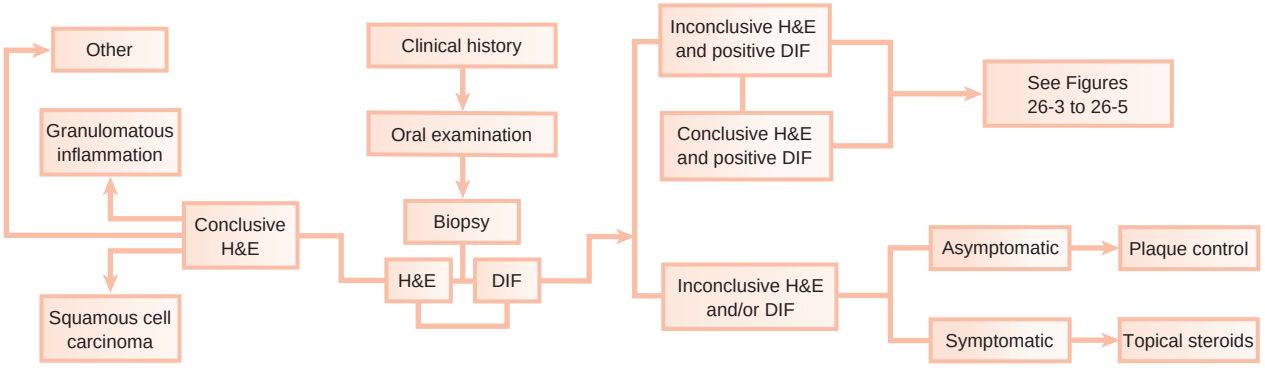

Figure 12-2 Diagnostic approach for desquamative gingivitis. *H&E,* Hematoxylin and eosin; *DIF,* direct immunofluorescence.

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In many instances, the initial diagnosis of desquamative lesions of the gingiva is the responsibility of the dental clinician. Biopsy of lesions accompanied by appropriate histologic and immunofluorescence tests are needed to confirm the clinical diagnosis. In cases with dermatologic or systemic components, patients should be managed in conjunction with appropriate physician referral. Many of the disease entities are made worse by normal mechanical oral hygiene techniques and so chlorhexidine rinses are generally used for plaque control to reduce any secondary gingival inflammation caused by the biofilms and to reduce patient discomfort.

Allergic manifestations can themselves result in gingival desquamation and also may exacerbate the destruction from other causes. Patients need to be informed to stop exposure to the most common allergens found in the diet, such as cinnamon used as a flavoring agent and pyrophosphates found in tartar control toothpaste.

Clinicians should be aware that in some cases, squamous cell carcinomas can initially present as "desquamative gingivitis," which is yet another reason for the use of biopsy on all desquamative lesions.

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TABLE 12-1 Histopathologic, Direct, and Indirect Immunofluorescence Findings in Select Conditions that may Present Clinically as Desquamative Gingivitis

		DIRECT IMMUNOFLUORESCENCE		INDIRECT IMMUNOFLUORESCENCE
Disease	**Histopathology**	**Biopsy Perilesional Mucosa**	**Biopsy Uninvolved Mucosa**	**Serum**
Pemphigus	Intraepithelial clefting above basal cell layer; basal cells have characteristic "tombstone" appearance; acantholysis present.	Intercellular deposits in epithelium; IgG in all cases, C3 in most cases.	Same as for perilesional mucosa.	Intercellular (IgG) antibodies in ≥90% of cases.
Cicatricial pemphigoid	Subepithelial clefting with epithelial separation from underlying lamina propria, leaving intact basal layer.	Linear deposits of C3, with or without IgG at basement membrane zone in almost all cases.	Same as for perilesional mucosa.	Basement membrane zone (IgG) antibodies in 10% of cases.
Bullous pemphigoid	Subepithelial clefting with epithelial separation from underlying lamina propria, leaving intact basal layer.	Linear deposits of C3, with or without IgG at basement membrane zone in almost all cases.	Same as for perilesional mucosa.	Basement membrane zone in 40%-70% of cases.
Epidermolysis bullosa acquisita	Similar to bullous and cicatricial pemphigoid.	Linear deposits of IgG and C3 at basement membrane zone in almost cases.	Same as for perilesional mucosa.	Basement membrane zone (IgG) antibodies in 25% of cases.
Lichen planus	Hyperkeratosis, hydropic degeneration of basal layer, sawtooth rete pegs; lamina propria exhibits dense, bandlike infiltrate primarily of T lymphocytes; colloid bodies present.	Fibrillar deposits of fibrin at dermal-epidermal junction.	Negative.	Negative.
Chronic ulcerative stomatitis	Similar to erosive lichen planus; hyperkeratosis, acanthosis, basal cell layer liquefaction, subepithelial clefting, lymphohistiocytic chronic infiltrate in bandlike configuration.	IgG deposits in nuclei of basal layer of epithelial cells.	Same as for perilesional mucosa.	ANA specific for basal cells of stratified squamous epithelium.
Linear IgA disease	Similar to erosive lichen planus.	Linear deposits of IgA at basement membrane zone.	Same as for perilesional mucosa.	IgA basement membrane zone (IgA) antibodies in 30% of cases.
Dermatitis herpetiformis	Collection of neutrophils, eosinophils, and fibrin in connective tissue papillae.	IgA deposits in dermal papillae in 85% of cases.	IgA deposits in dermal papillae in 100% of cases.	IgA endomysial antibodies in 70% of cases; gliadin antibodies in 30% of cases.
Systemic lupus erythematosus	Hyperkeratosis, basal cell degeneration, epithelial atrophy, perivascular inflammation.	IgG or IgM, with or without C3 deposits at dermal-epidermal junction.	Same as for perilesional mucosa.	ANA in >95% of cases; DNA and ENA antibodies in >50% of cases.
Chronic cutaneous erythematosus	Hyperkeratosis, basal cell degeneration, epithelial atrophy, perivascular inflammation.	IgG or IgM, with or without C3 deposits at dermal-epidermal junction.	Negative.	Usually negative.
Subacute lupus erythematosus	Less inflammatory cell infiltrate than systemic and chronic cutaneous forms but with similar microscopic features.	IgG or IgM, with or without C3 deposits at dermal-epidermal junction in 60% of cases; granular IgG deposits in basal cell cytoplasm in 30% of cases.	Same as for perilesional mucosa.	ANA in 60%-90%, Ro (SSA) in 80%, RF in 30%, and anti-RNP in 10% of cases

Modified from Rinaggio J, Neiders ME, Aguirre A, et al: *Compendium* 20:943, 1999.

ANA, Antinuclear antibodies; *DNA*, deoxyribonucleic acid; *ENA*, extractable nuclear antigens; *IgA*, immunoglobulin A; *RF*, rheumatoid factor; *RNP*, ribonucleoprotein.

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fluorescein-labeled, antihuman serum (anti-IgG, anti-IgA, anti-IgM, antifibrin, and anti-C3). With *indirect* immunofluorescence, unfixed frozen sections of oral or esophageal mucosa from an animal such as a monkey are first incubated with the patient's serum to allow attachment of any serum antibodies to the mucosal tissue. The tissue is then incubated with fluorescein-labeled antihuman serum. Immunofluorescence tests are positive if a fluorescent signal is observed in the epithelium, its associated basement membrane, or the underlying connective tissue (see Table 12-1).

Management. Once the diagnosis is established, the dentist must choose the optimum management for the patient. This is accomplished according to three factors: (1) practitioner's experience, (2) systemic impact of the disease, and (3) systemic complications of the medications. A detailed consideration of these three factors dictates three different scenarios.

In the *first scenario,* the dental practitioner takes direct and exclusive responsibility for the treatment of the patient, as with erosive lichen planus that is responsive to topical steroids (Figure 12-3).

In the *second scenario,* the dentist collaborates with another health care provider to evaluate and/or treat a patient concurrently. The classic example is seen with cicatricial pemphigoid, in which dentists and ophthalmologists work together (Figure 12-4). Although the dentist addresses the oral lesions, the ophthalmologist monitors the integrity of the ocular conjunctiva.

In the *third scenario,* the patient is immediately referred to a dermatologist for further evaluation and treatment. This occurs with conditions in which the systemic impact of the disease transcends the boundaries of the oral cavity and results in significant morbidity and even mortality. Pemphigus vulgaris is a clear example of a condition that, once diagnosed by the dentist, requires immediate referral to a dermatologist (Figure 12-5). In addition, the complications of chronically administered systemic medications that are indicated for the management of diseases, such as pemphigus vulgaris or nonresponsive mucous membrane pemphigoid (e.g.,

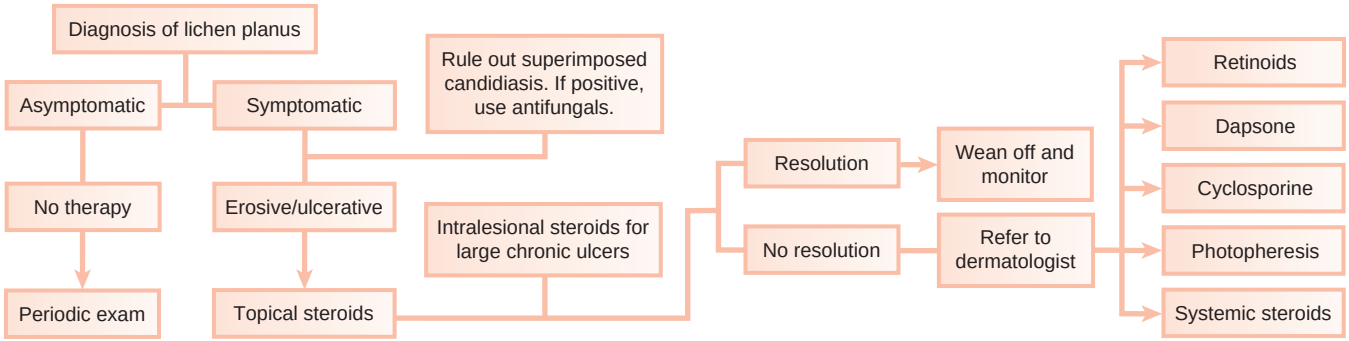

Figure 12-3 Treatment of lichen planus.

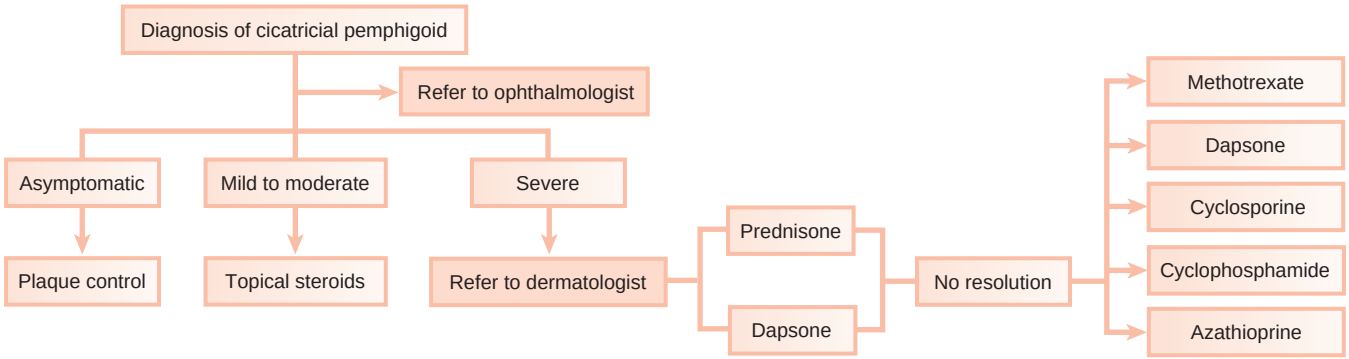

Figure 12-4 Treatment of cicatricial pemphigoid.

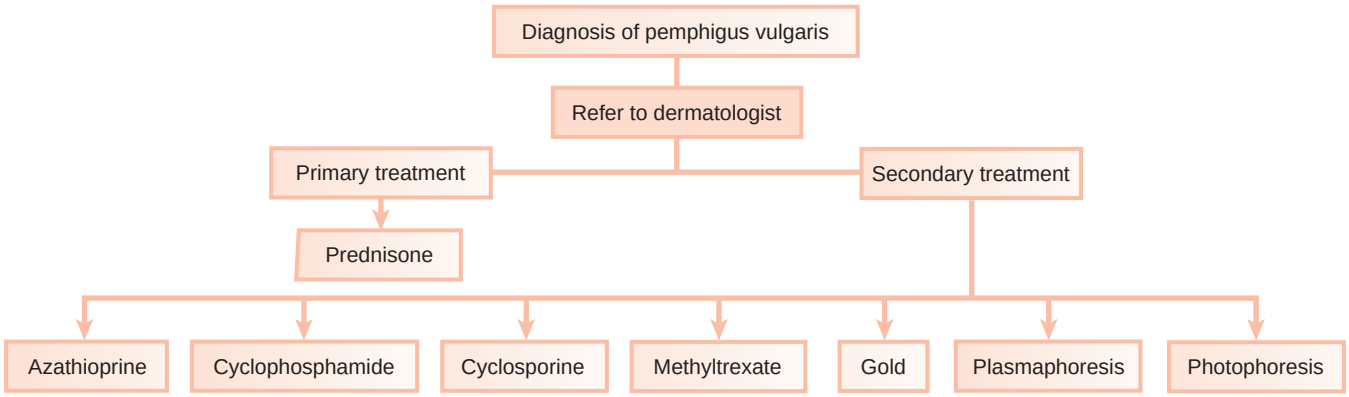

Figure 12-5 Treatment of pemphigus vulgaris.

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diabetes mellitus, osteoporosis, or methemoglobinemia), warrant the referral to a dermatologist or a specialist in internal medicine.

When oral treatment is provided, periodic evaluation is needed to monitor the response of the patient to the selected therapy. Initially, the patient should be evaluated at 2 to 4 weeks after beginning treatment to ensure that the condition is under control. This observation should continue until the patient is free of discomfort. Appointments every 3 to 6 months would then be appropriate. Doses of medication(s) are usually adjusted during this interval.

Table 12-2 summarizes suggested contemporary therapeutic approaches used to treat select conditions that clinically present as desquamative gingivitis. Dentists clearly play an important role in the diagnosis and management of desquamative gingivitis. The importance of being able to recognize and properly diagnose this condition is accentuated by the fact that a serious and life-threatening disease, such as squamous cell carcinoma, may mimic desquamative gingivitis.[127]

DISEASES CLINICALLY PRESENTING AS DESQUAMATIVE GINGIVITIS

Lichen Planus

Lichen planus is an inflammatory mucocutaneous disorder that may involve mucosal surfaces (e.g., oral cavity, genital tract, and other mucosae) and the skin (including the scalp and the nails).[131] The current evidence suggests that lichen planus is an immunologically-mediated mucocutaneous disorder in which host T lymphocytes play a central role.[11,64,92] Although the oral cavity may present lichen planus lesions with a distinct clinical configuration and distribution, the clinical presentation sometimes may simulate other mucocutaneous disorders. Therefore a clinical diagnosis of oral lichen planus should be accompanied by a broad differential diagnosis. Numerous epidemiologic studies have shown that oral lichen planus presents in 0.1% to 4% of the population.[139,142] The majority of patients with oral lichen planus are middle-aged and older females, with a 2:1 ratio of females to males. Children are rarely affected. In a dental setting, cutaneous lichen planus is observed in about one-third of the patients diagnosed with oral lichen planus.[88] In contrast, two-thirds of patients seen in dermatologic clinics exhibit oral lichen planus.[136]

Oral Lesions. Although there are several clinical forms of oral lichen planus (reticular, patch, atrophic, erosive, and bullous), the most common are the reticular and erosive subtypes. The typical *reticular* lesions are asymptomatic and bilateral and consist of interlacing white lines on the posterior region of the buccal mucosa. The lateral border and dorsum of the tongue, hard palate, alveolar ridge, and gingiva may also be affected. In addition, the reticular lesions may have an erythematous background, which is a feature associated with coexisting candidiasis. Oral lichen planus lesions follow a chronic course and have alternating, unpredictable periods of quiescence and flare-ups.

The *erosive* subtype of lichen planus is often associated with pain and clinically manifests as atrophic, erythematous, and often ulcerated areas. Fine, white radiating striations are observed bordering the atrophic and ulcerated zones. These areas may be sensitive to heat, acid, and spicy foods (Figure 12-6).

Gingival Lesions. Seven percent to 10% of patients with oral lichen planus have lesions restricted to the gingival tissue[99,136] that may occur as one or more types of four distinctive patterns, as follows:

TABLE 12-2 Accepted Contemporary Therapeutic Approaches Used To Treat Select Conditions That Clinically Present As Desquamative Gingivitis

Disease	Therapy
Erosive lichen planus	*Mild cases:* Delivery of therapeutic agent can be enhanced with use of vacuum-formed custom trays. **Rx:** Lidex (0.05% fluocinonide) gel **Disp:** One tube 1.5 g **Sig:** Apply to affected area pc and hs. Monitoring patient's oral cavity is warranted because candidiasis may develop after a few weeks of topical steroid use; concomitant use of antifungal may be necessary. **Rx:** Nystatin oral pastilles (100,000 IU) **Disp:** 60 pastilles **Sig:** Dissolve in mouth bid, then expectorate for 30 consecutive days. *Recalcitrant cases:* **Rx:** Protopic (0.1% tacrolimus) ointment **Disp:** One tube 15 g **Sig:** Apply to affected area bid. *Severe or refractory cases:* Refer to dermatologist for management with systemic corticosteroids.
Cicatricial pemphigoid	*Mild cases:* **Rx:** Lidex (0.05% fluocinonide) gel **Disp:** One tube 1.5 g **Sig:** Apply to affected area pc and hs. **Rx:** Temovate (0.05% clobetasol propionate) **Disp:** One tube 1.5 g **Sig:** Apply to affected area qid. *Severe or refractory cases:* Refer to a dermatologist for management with prednisone (20-30 mg/day); concomitant use of azathioprine may be needed; dapsone, sulfonamide, and tetracycline are other alternatives.
Pemphigus	Refer to dermatologist for management with prednisone (20-30 mg/day); concomitant use of azathioprine may be needed.
Chronic ulcerative stomatitis	*Mild cases:* **Rx:** Lidex (0.05% fluocinonide) gel **Disp:** One tube 1.5 g **Sig:** Apply to affected area qid. **Rx:** Temovate (0.05% clobetasol propionate) **Disp:** One tube 1.5 g **Sig:** Apply to affected area qid.

g, Grams; *pc,* after meals; *hs,* at bedtime; *IU,* international units; *bid,* twice daily; *qid,* four times daily.

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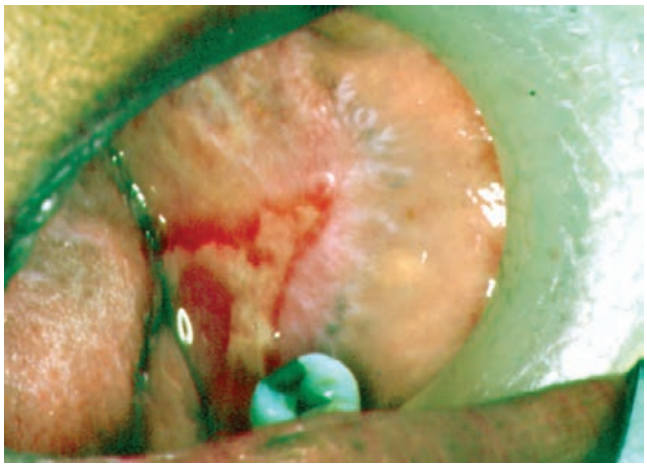

Figure 12-6 Erosive lichen planus. Large ulcerative lesion on the left buccal mucosa exhibits bordering erythema. The typical white striations of lichen planus are evident in the periphery of the ulcer.

Figure 12-7 Erosive lichen planus presenting as desquamative gingivitis. The gingival tissues are erythematous, ulcerated, and painful. *(Courtesy Dr. Luis Gaitan, Oral Pathology Laboratory, Faculty of Odontology, National Autonomous University of Mexico (UNAM), Mexico City.)*

1. *Keratotic lesions.* These raised white lesions may present as groups of individual papules, linear or reticular lesions, or plaquelike configurations.
2. *Erosive or ulcerative lesions.* These extensive erythematous areas with a patchy distribution may present as focal or diffuse hemorrhagic areas. These lesions are exacerbated by slight trauma (e.g., toothbrushing) (Figure 12-7).
3. *Vesicular or bullous lesions.* These raised, fluid-filled lesions are uncommon and short lived on the gingiva, quickly rupturing and leaving an ulceration.
4. *Atrophic lesions.* Atrophy of the gingival tissues with ensuing epithelial thinning results in erythema confined to the gingiva.

Histopathology Microscopically, the three main features that characterize oral lichen planus are (1) hyperkeratosis or parakeratosis, (2) hydropic degeneration of the basal layer, and (3) a dense, bandlike infiltrate primarily of T lymphocytes in the lamina propria (Figure 12-8). Classically, the epithelial rete ridges have a "sawtooth" configuration. Hydropic degeneration of the basal layer of the epithelium may be sufficiently extensive that the epithelium becomes thin and atrophic or detaches from the underlying connective tissue and produces either a subepithelial vesicle or an ulcer. Colloid bodies (Civatte bodies) are often seen at the epithelium–connective tissue interface. A microscopic diagnosis of oral lichen planus is straightforward in the keratotic lesions, and biopsy specimens should be obtained from these areas if possible. However, these classic histologic features may be obscured in the areas of ulceration, making a conclusive diagnosis of oral lichen planus difficult based solely on conventional microscopy. Electron microscopy studies indicate that separation of the basal lamina from the basal cell layer is an early manifestation of lichen planus.[61]

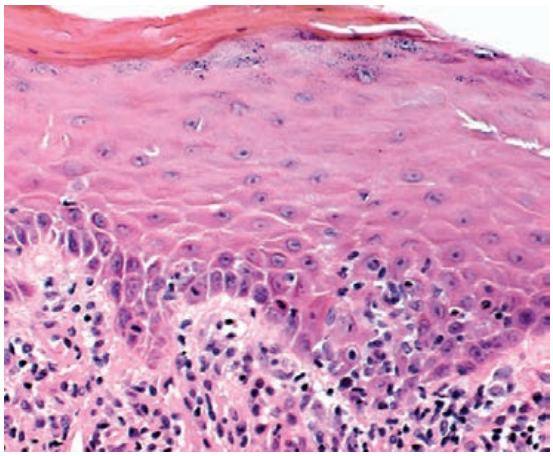

Figure 12-8 Microscopic appearance of lichen planus. Biopsy from gingival lesion shows hyperkeratosis and mild hypergranulosis. Focal basal cell degeneration, lymphocytic exocytosis, and thickening of the basement membrane are apparent. The rete pegs exhibit a slight serrated configuration. The papillary lamina propria shows a bandlike infiltrate of lymphohistiocytic, chronic inflammatory cells. (Hematoxylin and eosin [H&E] stain; original magnification ×100.)

It is noteworthy that oral lesions of lichen planus may change in pattern, and in certain unusual cases, a second or even a third biopsy may be necessary to arrive at a definitive diagnosis. More importantly, controversy exists about the malignant potential of oral lichen planus. In recent studies, it has been estimated that oral cancer emerges in up to 2% of patients with oral lichen planus.[41,55,62,63,150] In contrast, other researchers reject or question the connection between oral lichen planus and oral cancer.[42,66,87,123] Despite this controversy, biopsy and close follow-up are warranted in these patients.

Immunopathology. Direct immunofluorescence of both lesional and perilesional oral lichen planus biopsy specimens reveals linear fibrillar ("shaggy") deposits of fibrin in the basement membrane zone (Figure 12-9), along with scattered, immunoglobulin-staining cytoid bodies in the upper areas of the lamina propria (Figure 12-10). Serum tests using indirect immunofluorescence are negative in lichen planus (see Table 12-1).

Differential Diagnosis. The classic clinical presentation of oral lichen planus can be simulated by other conditions, mainly by lichenoid mucositis. If oral lichen planus is confined to the gingival tissues (erosive oral lichen planus), the identification of fine, white radiating striations bordering the erosive areas support a clinical diagnosis of oral lichen planus. If the white striations are absent, the differential diagnosis should primarily include mucous membrane pemphigoid and pemphigus vulgaris. Other, less common

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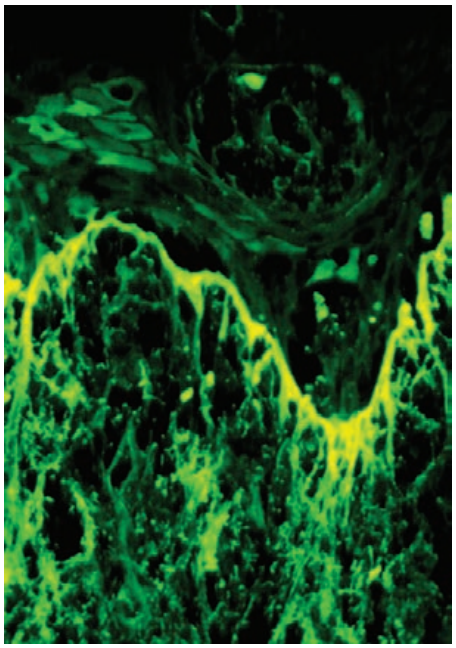

Figure 12-9 Direct immunofluorescence of lichen planus. Fibrin deposits along the basement membrane of the epithelium exhibit a "shaggy" configuration.

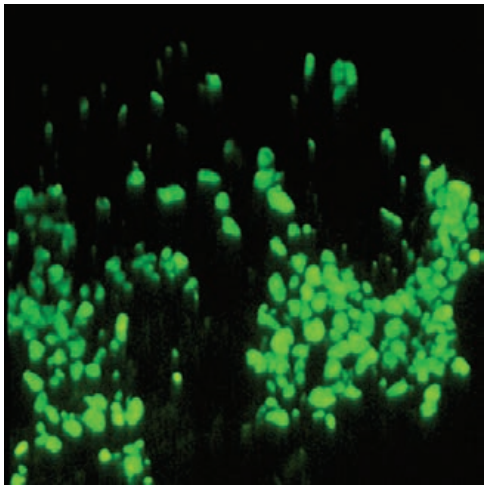

Figure 12-10 Direct immunofluorescence of lichen planus. Clusters of cytoid bodies exhibit immunoglobulin M (IgM) deposits in the lamina propria.

possibilities include linear IgA disease (LAD) and chronic ulcerative stomatitis.

Treatment. The keratotic lesions of oral lichen planus are asymptomatic and do not require treatment once the microscopic diagnosis is established. However, follow-up of the patient every 6 to 12 months is warranted to monitor suspicious clinical changes and the emergence of an erosive component.

In contrast, the erosive, bullous, or ulcerative lesions of oral lichen planus are treated with high-potency topical steroids, such as 0.05% fluocinonide gel (Lidex, three times daily). Lidex can also be mixed 1:1 with carboxymethyl cellulose (Orabase) paste or other adhesive ointment. A gingival tray can also be used to deliver Lidex or 0.05% clobetasol propionate with 100,000 IU/ml of nystatin in Orabase. Three 5-minute applications of this mixture daily appear to be effective in controlling erosive lichen planus.[57] Intralesional injections of triamcinolone acetonide (10 to 20 mg) or short-term regimens of 40 mg of prednisone daily for 5 days, followed by 10 to 20 mg daily for an additional 2 weeks, have also been used in more severe cases.[112] Because of the potential side effects, administration of systemic steroids should be prescribed and monitored by a dermatologist. Other treatment modalities (e.g., retinoids, hydroxychloroquine, cyclosporine, azathioprine, cyclophosphamide, or free gingival grafts) have also been used.[112,119] A promising therapeutic agent, tacrolimus (0.1% Protopic ointment, twice daily), is an immunosuppressant that is effective in controlling lesions of erosive lichen planus.[72,91,104,144]

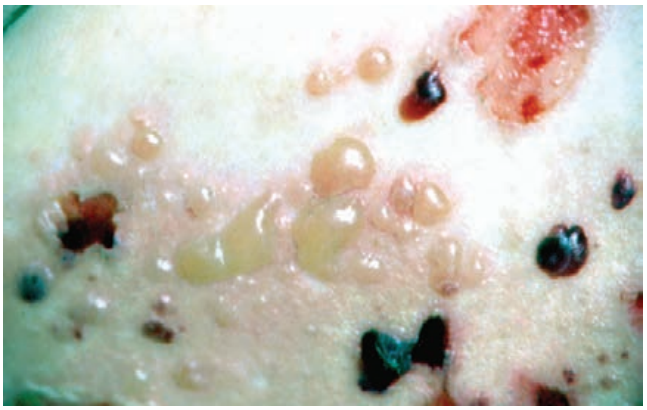

Figure 12-11 Bullous pemphigoid. Coalescing cutaneous bullae are seen, some of them are hemorrhagic. Rupture of bullae leads to formation of serpiginous ulcers.

Because candidiasis is often associated with symptomatic oral lichen planus and topical steroid therapy promotes fungal growth, treatment should also include a topical antifungal agent.[16,52,67]

Pemphigoid

The term *pemphigoid* applies to a number of cutaneous, immune-mediated, subepithelial bullous diseases characterized by a separation of the basement membrane zone, including bullous pemphigoid, mucous membrane pemphigoid, and pemphigoid (herpes) gestationis.[118,140] Among these conditions, bullous pemphigoid and mucous membrane pemphigoid, also known as *benign mucous membrane pemphigoid* and *cicatricial pemphigoid,* have received considerable attention. Current molecular findings on these two diseases clearly indicate that they are separate entities.[140] However, considerable histologic and immunopathologic overlap exists between the two diseases, so their differentiation may be impossible based on these two criteria.[118] In many cases, the clinical findings are probably the best cognitive element to discriminate between them. Accordingly, the term *bullous pemphigoid* is preferred when the disease is nonscarring and mainly affects the skin. The term *cicatricial pemphigoid* is favored when scarring occurs and the disease is mainly confined to mucous membranes (although scarring may be absent in some subtypes of mucous membrane pemphigoid).[157] Until more research allows better understanding of this family of diseases, bullous pemphigoid and mucous membrane pemphigoid are discussed separately.

Bullous Pemphigoid. Bullous pemphigoid is a chronic, autoimmune, subepidermal bullous disease with tense cutaneous bullae that rupture and become flaccid (Figure 12-11). Oral involvement occurs in about a third of the patients.[148]

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Although the skin lesions of bullous pemphigoid clinically resemble those of pemphigus, the microscopic picture is quite distinct.

Histopathology There is no evidence of acantholysis, and the developing vesicles are subepithelial rather than intraepithelial. The epithelium separates from the underlying connective tissue at the basement membrane zone. Electron microscopic studies show an actual horizontal splitting or replication of the basal lamina. The separating epithelium remains relatively intact, and the basal layer is present and appears to be regular. The two major antigenic determinants for bullous pemphigoid are the 230 kD protein plaque known as BP1 and the 180 kD collagen-like transmembrane protein BP2.[105,121,129]

Immunofluorescence. Immunologically, bullous pemphigoid is characterized by immunoglobulin G (IgG) and complement 3 (C3) immune deposits along epithelial basement membranes and circulating IgG antibodies to the epithelial basement membrane.[71,109] Direct immunofluorescence is positive in 90% to 100% of these patients, whereas indirect immunofluorescence is positive in 40% to 70% of affected patients[110] (see Table 12-1).

Oral Lesions. Oral lesions have been reported to occur secondarily in up to 40% of cases. There is an erosive or desquamative gingivitis presentation and occasional vesicular or bullous lesions.[148]

Treatment. Because its etiologic factors are unknown, treatment of bullous pemphigoid is designed to control its signs and symptoms.[71,109] The primary treatment is a moderate dose of systemic prednisone. Steroid-sparing strategies (prednisone plus other immunomodulator drugs) are used when high doses of steroids are needed or the steroid alone fails to control the disease.[38] For localized lesions of bullous pemphigoid, high-potency topical steroids or tetracycline, with or without nicotinamide, can be effective.[112]

Mucous Membrane Pemphigoid (Cicatricial Pemphigoid).

Mucous membrane pemphigoid, also known as *cicatricial pemphigoid,* is a chronic, vesiculobullous autoimmune disorder of unknown cause that predominantly affects women in the fifth decade of life. Although rare, it has been reported in young children.[22,106,140]

Cicatricial pemphigoid involves the oral cavity, conjunctiva, and the mucosa of the nose, vagina, rectum, esophagus, and urethra. In about 20% of patients, however, the skin may also be involved. Recent investigations suggest that cicatricial pemphigoid encompasses a group of heterogeneous conditions with distinct clinical and molecular features.[33,101,133] An elaborate cascade of events is involved in the pathogenesis of cicatricial pemphigoid. Initially, antigen-antibody complexing occurs at the basement membrane zone, followed by complement activation and subsequent leukocyte recruitment. Next, proteolytic enzymes are released and dissolve or cleave the basement membrane zone, usually at the level of the lamina lucida.[45] The two major antigenic determinants for cicatricial pemphigoid are bullous pemphigoid 1 and 2 (BP1 and BP2). Most cases of cicatricial pemphigoid are the result of an immune response directed against BP2 and less often against BP1, epiligrin (laminin-5, a lamina lucida protein in basement membrane of stratified epithelium), and β-4 integrins.[8,20,33]

Current research strongly suggests that there are at least five subtypes of cicatricial pemphigoid: oral pemphigoid, antiepiligrin pemphigoid, anti-BP antigen mucosal pemphigoid, ocular pemphigoid, and multiple-antigens pemphigoid.[140] Recent studies have shown that the sera of ocular pemphigoid patients recognize β4 integrin subunit while patients with oral pemphigoid recognize the βα6 unit.[124]

Ocular Lesions. In cases presenting first to the dentist (mainly desquamative gingivitis), the eyes are affected in approximately 25% of patients.[110] In contrast, in cases presenting first to the dermatologist, 66% of patients present with conjunctival lesions, whereas in ophthalmic studies, 100% of the patients have ocular involvement.[51,81,102,103] The initial lesion is characterized by unilateral conjunctivitis that becomes bilateral within 2 years. Subsequently, there may be adhesions of eyelid to eyeball *(symblepharon)* (Figure 12-12). Adhesions at the edges of the eyelids *(ankyloblepharon)* may lead to a narrowing of the palpebral fissure. Small vesicular lesions may develop on the conjunctiva, which may eventually produce scarring, corneal damage, and blindness.

Oral Lesions. The most characteristic feature of oral involvement is the presence of desquamative gingivitis, typically with areas of erythema, desquamation, ulceration, and vesiculation, of the attached gingiva[53,146] (Figure 12-13). Vesiculobullous lesions may occur elsewhere in the mouth.[53] The bullae tend to have a relatively thick roof and rupture in 2 to 3 days, leaving irregularly-shaped areas of ulceration. Healing of the lesions may take up to 3 weeks or longer.

Figure 12-12 Mucous membrane pemphigoid. Characteristic ocular lesion (symblepharon) in a patient presenting with cicatricial pemphigoid. *(Courtesy Dr. Carl Allen, The Ohio State University, Columbus, OH.)*

Figure 12-13 Gingival mucous membrane pemphigoid. Lesions are confined to the gingival tissues, producing a typical desquamative gingivitis appearance. *(Courtesy Dr. Stuart L. Fischman, State University of New York at Buffalo.)*

Histopathology The microscopic appearance of the oral lesions, although not completely diagnostic for mucous membrane pemphigoid, is sufficiently distinctive that a tentative diagnosis can be considered. A striking subepithelial vesiculation, with the epithelium separated from the underlying lamina propria, leaves an intact basal layer (Figure 12-14). The separation of the epithelium and the connective tissue occurs at the basement membrane zone. Electron microscopic studies demonstrate a split in the basal lamina.[154] A mixed inflammatory infiltrate (lymphocytes, plasma cells, neutrophils, and scarce eosinophils) is observed in the underlying fibrous connective tissue.

Immunofluorescence. Positive findings along the basement membrane area have been reported using both direct and indirect immunofluorescence.[34,70,76] In biopsy tests by direct immunofluorescence, the main immunoreactants are IgG and C3, which are confined to the basement membrane (Figure 12-15). Some studies indicate that positive indirect immunofluorescence is rare in these patients (<25%).[96] The lack of indirect immunofluorescence findings may be caused by an earlier diagnosis of mucous membrane pemphigoid, resulting in the identification of patients with less extensive disease.[2,79] In any event, circulating autoantibodies do not appear to play a role in the pathogenesis of the disease.

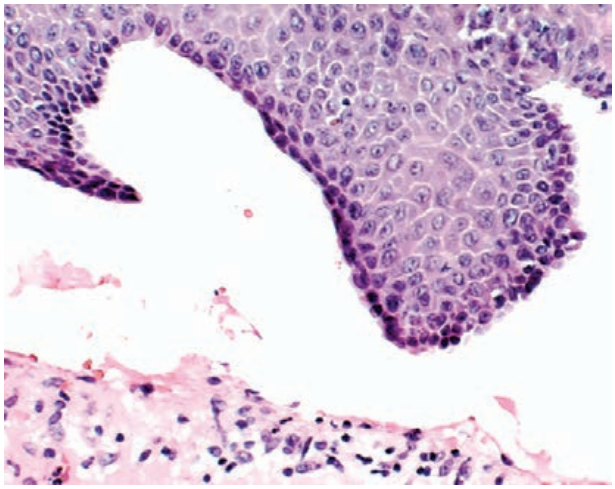

Figure 12-14 Microscopic features of oral mucous membrane pemphigoid. Separation of the epithelium from the subjacent connective tissue (subepithelial clefting). An intact basal cell layer remains attached to the epithelium. (H&E stain; ×100.)

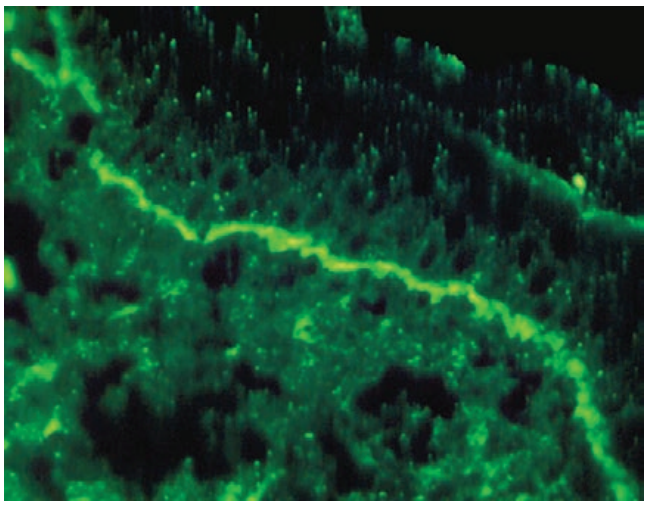

Figure 12-15 Direct immunofluorescence of mucous membrane pemphigoid. C3 deposits confined along the basement membrane.

Differential Diagnosis. Several disease entities present with similar clinical and histologic (subepithelial bulla) features.[40] These include bullous pemphigoid, bullous lichen planus, dermatitis herpetiformis, LAD, erythema multiforme, herpes gestationis, and epidermolysis bullosa acquisita.

Pemphigus may be confined to the oral cavity in its early stage, and the vesicular and ulcerative lesions may resemble those of mucous membrane pemphigoid. An erosive or desquamative gingivitis may also be seen in pemphigus as a rare manifestation. Biopsy studies can quickly rule out pemphigus by revealing the absence or presence of acantholytic changes. In erythema multiforme, there are obvious vesiculobullous lesions, but the onset is usually acute rather than chronic, labial involvement is severe, and the gingivae are usually not affected. Desquamative gingivitis is an unusual finding in erythema multiforme, although occasional vesicular lesions may develop. A biopsy study of an oral lesion reveals an unusual degeneration of the upper stratum spinosum, characteristically seen in oral erythema multiforme lesions.

Cicatricial pemphigoid must be differentiated from epidermolysis bullosa acquisita, which can present with similar histopathology and immunopathology. When the biopsy is treated with salt to separate the dermis from the epidermis, basement membrane immunodeposits occur on the epidermal side in pemphigoid and the dermal side in epidermolysis bullosa acquisita.[44]

Treatment. Topical steroids are the mainstay of treatment for mucous membrane pemphigoid, particularly when localized lesions are present. Fluocinonide (0.05%) and clobetasol propionate (0.05%) in an adhesive vehicle can be used three times a day for up to 6 months. When the oral lesions of mucous membrane pemphigoid are confined to the gingival tissues, topical corticosteroids are effectively delivered with vacuum-formed custom trays or veneers.[140] Optimal oral hygiene is essential because local irritants on the tooth surface result in an exaggerated gingival inflammatory response. Gingival irritation from any dental prosthesis should also be minimized.

If the disease is not severe and symptoms are mild, systemic corticosteroids may be omitted. If ocular involvement exists, systemic corticosteroids are indicated.

When lesions do not respond to steroids, systemic dapsone (4-4′-diaminodiphenylsulfone) has proved to be effective.[28,49,101,108] Because of the systemic side effects to dapsone, including hemolysis and methemoglobinemia, particularly in patients with glucose-6-phosphate dehydrogenase deficiency, referral to a dermatologist is often indicated.[115] Systemic steroids can also be combined with azathioprine or cyclophosphamide.[7,137] Some authors also advocate sulfonamides and tetracycline; surgery, although not a treatment for mucous membrane pemphigoid, is used in some patients to prevent blindness, as well as esophageal and upper airways stenosis.[140] Connective tissue grafting to alleviate root surface sensitivity and improve esthetics has been used with success to manage gingival recession in a patient with cicatricial pemphigoid.[84]

Pemphigus Vulgaris

The pemphigus diseases are a group of autoimmune bullous disorders that produce cutaneous and mucous membranes blisters

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Figure 12-16 Cutaneous lesion of patient with pemphigus vulgaris. A large bulla is present in the flexor surface of the wrist.

Figure 12-17 Pemphigus vulgaris of the oral cavity. Multiple and coalescent areas of ulceration are covered by pseudomembranes of necrotic epithelium. This patient presented with large ulcers in the labial mucosa, tongue, and soft palate.

(Figures 12-16 and 12-17). Pemphigus vulgaris is the most common of the pemphigus diseases, which also include pemphigus foliaceous, pemphigus vegetans, and pemphigus erythematosus.[128] Pemphigus vulgaris is a potentially lethal, chronic condition (10% mortality rate) with a worldwide incidence of 0.1 to 0.5 cases per year per 100,000 individuals.[12,128,135] A predilection in women, usually after the fourth decade of life, has been observed.[112] However, pemphigus vulgaris has also been reported in unusually young children and even in newborns.[26,128,143,158]

The epidermal and mucous membrane blisters occur when the cell-to-cell adhesion structures are damaged by the action of circulating and in vivo binding of autoantibodies to the pemphigus vulgaris antigens, which are cell surface glycoproteins present in keratinocytes. These pemphigus vulgaris glycoproteins are members of the *desmoglein* (DSG) subfamily of the cadherin superfamily of cell-cell adhesion molecules, which are present in desmosomes.[78] Whereas high levels of desmoglein 3 (Dsg3) autoantibodies correlate with severity of oral disease in patients with pemphigus vulgaris, elevated levels of desmoglein 1 (Dsg1) autoantibodies are associated with severity of cutaneous disease.[60] Recent evidence suggests that DSG3, the gene coding for pemphigus vulgaris, is located in chromosomes.[18]

Most cases of pemphigus vulgaris are idiopathic. However, medications, such as penicillamine and captopril, can produce drug-induced pemphigus, which is usually reversible on withdrawal of the causative drug. Paraneoplastic pemphigus is antigenically distinct from pemphigus vulgaris and is associated with underlying malignancies.[107]

Figure 12-18 Pemphigus vulgaris of the gingiva. Clinical appearance of patient with oral lesions confined to the gingivae that were clinically diagnosed as "consistent with desquamative gingivitis." *(Courtesy Dr. Beatriz Aldape, Faculty of Odontology, National Autonomous University of Mexico (UNAM), Mexico City.)*

In approximately 60% of patients with pemphigus vulgaris, the oral lesions are the first sign of the disease and may herald the dermatologic involvement by a year or more.[98,149]

Oral Lesions. Oral lesions can range from small vesicles to large bullae. When the bullae rupture, they leave extensive areas of ulceration (Figure 12-18). Virtually any region of the oral cavity can be involved, but multiple lesions often develop at sites of irritation or trauma. The soft palate is more often involved (80%), followed by the buccal mucosa (46%), ventral aspect or dorsum of tongue (20%), and lower labial mucosa (10%). Oral lesions of pemphigus vulgaris are confined less often to the gingival tissues.[75] In these patients, a clinical diagnosis of erosive gingivitis or desquamative gingivitis is seen as the sole manifestation of oral pemphigus.

Histopathology Lesions of pemphigus demonstrate a characteristic intraepithelial separation, which occurs above the basal cell layer. The intraepithelial vesiculation begins as a microscopic alteration (Figure 12-19) and gradually results in a grossly visible, fluid-filled bulla. Occasionally the entire superficial layers of epithelium are lost, leaving only the basal cells attached to the underlying lamina propria, conferring a characteristic "tombstone" appearance to the epithelial cells. *Acantholysis,* a separation of the epithelial cells of the lower stratum spinosum, takes place and is characterized by the presence of round rather than polyhedral epithelial cells. The intercellular bridges are lost, and the nuclei are large and hyperchromatic.[32,80,161] The underlying connective tissue usually presents as a mild-to-moderate, chronic inflammatory cell infiltrate. As the vesicle or bulla ruptures, the ulcerated lesion becomes infiltrated with polymorphonuclear leukocytes (PMNs), and the surface may show suppuration.

Immunofluorescence. The presence of autoantibodies can be demonstrated in the oral mucosa of patients with oral pemphigus by the use of immunofluorescence techniques. For direct immunofluorescence, perilesional unfixed frozen sections are incubated with fluorescein-labeled human anti-IgG. In indirect immunofluorescence, unfixed frozen sections of oral or esophageal mucosa from an animal such as a monkey is first incubated with the patient's serum to allow attachment of any serum antibodies to

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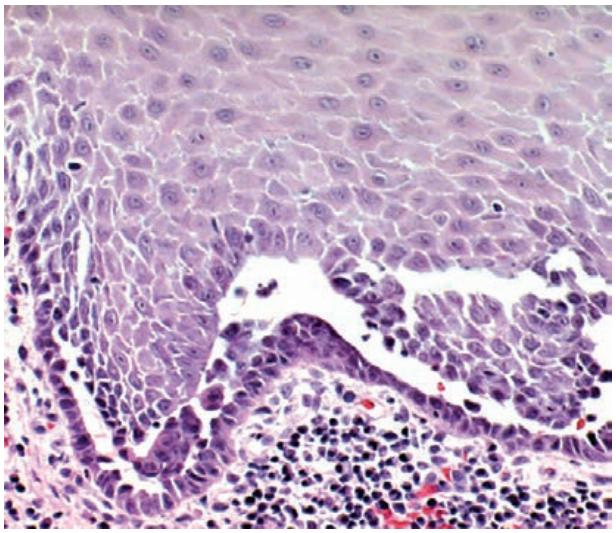

Figure 12-19 Microscopic features of pemphigus vulgaris. Typical intraepithelial clefting with "tombstone" appearance of basal cells, which remain attached to subjacent basement membrane and fibrous connective tissue. Acantholysis of epithelial cells with formation of "Tzanck" cells is seen in the intraepithelial cleft. (H&E stain; ×100.)

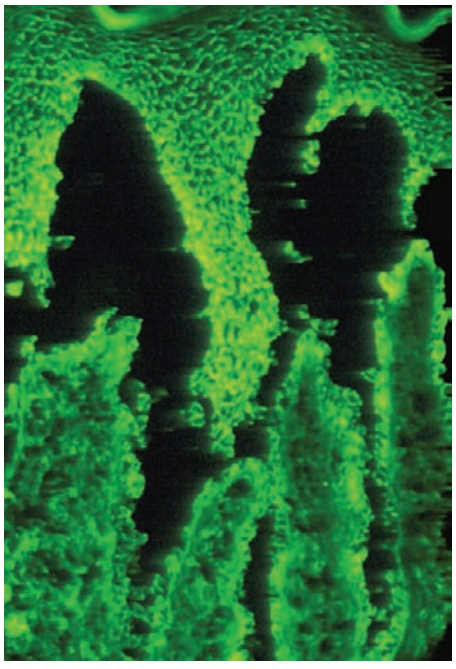

Figure 12-20 Direct immunofluorescence of oral pemphigus. Positive intercellular signal for immunoglobulin G (IgG) deposits is seen in keratinocytes of the stratified squamous epithelium.

the mucosal tissue. The tissue is then incubated with fluorescein-labeled, antihuman IgG serum. The test is positive if immunofluorescence is observed in the intercellular spaces of the stratified squamous epithelium of the mucosa (Figure 12-20).

The indirect technique is less sensitive than the direct technique and may be negative in early stages of the disease, particularly in localized forms (see Table 12-1). In most cases, however, the indirect immunofluorescence titers are helpful to monitor disease activity and have prognostic value.

Differential Diagnosis. The oral lesions of pemphigus vulgaris may be similar to those seen in erythema multiforme. In erythema multiforme, however, recurrent active episodes of comparatively short duration are followed by long intervals free of skin or oral lesions. Erythema multiforme affects the lips with considerable severity. Microscopic examination with conventional H&E and direct immunofluorescence can discriminate oral lesions of pemphigus from those of erythema multiforme. Pemphigus vulgaris will show characteristic intraepithelial clefting at the basal-spinous cell layers and interface with acantholysis, whereas erythema multiforme shows microvesiculation of the superficial epithelial layers and numerous necrotic keratinocytes. Pemphigus vulgaris shows an intercellular and intraepithelial signal with direct immunofluorescence. In contrast, erythema multiforme exhibits negative immunofluorescence.

Pemphigoid may clinically resemble pemphigus. Microscopic examination and direct immunofluorescence studies are needed to establish a correct diagnosis. Bullous pemphigoid and mucous membrane pemphigoid exhibit detachment of the epithelium from the underlying connective tissue ("lifting off"), instead of the acantholytic lesion characteristic of pemphigus.

Bullous lichen planus must also be considered in the differential diagnosis. The primary lesion of pemphigus may be of a bullous character, followed by erosion with associated pain and discomfort. In lichen planus, however, the characteristic reticular lesions are invariably found associated with the bullae. Microscopic examination and direct immunofluorescence studies are necessary to differentiate this condition from pemphigus. Bullous lichen planus shows separation of the epithelium from the underlying fibrous connective tissue, "sawtooth" rete pegs, and a bandlike, chronic inflammatory infiltrate in the lamina propria. Direct immunofluorescence reveals linear fibrillar deposits of fibrin in the basement membrane of bullous lichen planus, whereas pemphigus vulgaris has intercellular immunoglobulin deposition within the epithelium.

If the oral lesions of pemphigus vulgaris are restricted to the gingival tissues, erosive lichen planus, pemphigoid, LAD, and chronic ulcerative stomatitis should be ruled out.

Treatment. The main therapy for pemphigus vulgaris is systemic corticosteroids with or without the addition of other immunosuppressive agents.[149] If the patient responds well to corticosteroids, the dosage can be gradually reduced, but a low maintenance dosage is usually necessary to prevent or minimize the recurrence of lesions. Many dermatologists monitor the dose of steroids by periodic evaluation of the titers of DsG3 and DsG1 antibodies. Increasing titers are often associated with an impending exacerbation and warrant an increase of the steroid dose. A decrease in titer justifies a reduction of the steroid dose.[23] In patients not responsive to corticosteroids or who gradually adapt to them, "steroid-sparing" therapies are used; these consist of combinations of steroids plus other medications, such as azathioprine, cyclophosphamide, cyclosporine, dapsone, gold, and methotrexate, as well as photoplasmapheresis and plasmapheresis.[112] Rituximab is an anti-CD20 monoclonal antibody aimed to remove desmoglein-reactive autoantibodies that is currently being evaluated as an adjunct to treat pemphigus vulgaris.[43,141]

The maintenance phase aims to control the disease with the lowest dose of medication. To minimize the risk of morbidity associated with the long-term use of steroids, alternate-day steroid therapy, steroid-sparing drugs, and topical steroids can be combined. Because topical steroids may promote the development of candidiasis, topical antifungal medication may also be needed.[90]

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Figure 12-21 Chronic ulcerative stomatitis. Erythema and ulceration of the gingiva consistent with a clinical diagnosis of desquamative gingivitis. Direct and indirect immunofluorescence studies demonstrate the presence of stratified epithelium–specific antinuclear antibodies (SES-ANA). *(Courtesy Dr. Douglas Damm, University of Kentucky, Lexington.)*

Minimizing oral irritation is important in patients with oral pemphigus vulgaris. Optimal oral hygiene is essential because there is usually widespread involvement of the marginal and attached gingivae in pemphigus vulgaris, as well as in other areas of the mouth, which can be exacerbated by plaque-associated gingivitis and periodontitis. Periodontal care is an important issue of the overall management of patients with pemphigus vulgaris. To prevent flare-ups, patients in the maintenance phase should receive prednisone before professional oral prophylaxis and periodontal surgery.[128] In addition, the fit and design of removable prosthetic appliances should receive special attention because even slight irritation from these prostheses can cause severe inflammation with vesiculation and ulceration.

Chronic Ulcerative Stomatitis

Chronic ulcerative stomatitis, first reported in 1990,[68] clinically presents with chronic oral ulcerations and has a predilection for women in their fourth decade of life. The erosions and ulcerations present predominantly in the oral cavity, with only a few cases exhibiting cutaneous lesions.[25,82,159]

Oral Lesions. Painful, solitary small blisters and erosions with surrounding erythema are present mainly on the gingiva and the lateral border of the tongue. Because of the magnitude and clinical features of the gingival lesions, a diagnosis of desquamative gingivitis is considered (Figure 12-21). The buccal mucosae and hard palate may also present similar lesions.[152]

Histopathology The microscopic features of chronic ulcerative stomatitis are similar to those observed in erosive lichen planus. Hyperkeratosis, acanthosis, and liquefaction of the basal cell layer with areas of subepithelial clefting are prominent features of the epithelium. The underlying lamina propria exhibits a lymphohistiocytic, chronic infiltrate in a bandlike configuration.

Immunofluorescence. Direct immunofluorescence of normal and perilesional tissues reveals typical stratified epithelium–specific antinuclear antibodies (SES-ANA). These are nuclear deposits of IgG with a speckled pattern, mainly in the basal cell layer of the normal epithelium (Figure 12-22). In addition, fibrin deposits are visualized at the epithelial tissue–connective tissue interface. Indirect immunofluorescence studies using an esophageal substrate also reveal the presence of SES-ANA.[153]

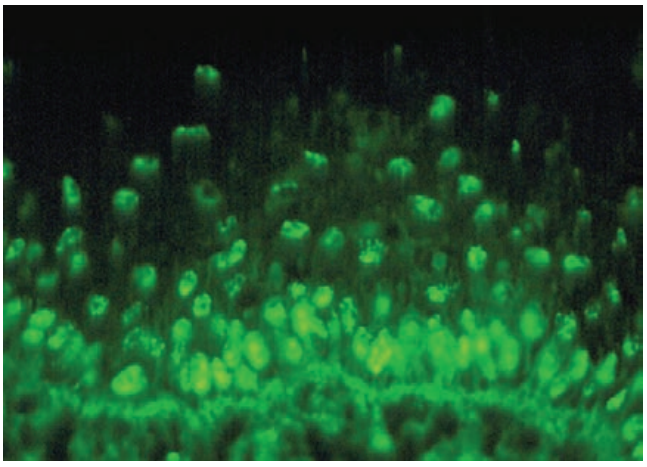

Figure 12-22 Direct immunofluorescence of chronic ulcerative stomatitis. Nuclear deposits of IgG are prominent in the basal cell layer and fade toward the superficial layers. *(Courtesy Dr. Douglas Damm, University of Kentucky, Lexington.)*

Diagnosis. Chronic ulcerative stomatitis is clinically similar to erosive lichen planus. In addition, pemphigus vulgaris, mucous membrane pemphigoid, LAD, bullous pemphigoid, and lupus erythematosus have to be included in the clinical differential diagnosis. Microscopic examination usually reduces the number of possibilities to chronic ulcerative stomatitis, LAD, and erosive lichen planus. Direct and indirect immunofluorescence studies are needed to arrive at the correct diagnosis.

Treatment. For mild cases, topical steroids (fluocinonide, clobetasol propionate) and topical tetracycline may produce clinical improvement; however, recurrences are common.[85] For severe cases, a high dose of systemic corticosteroids is needed to achieve remission. Unfortunately, reduction of the corticosteroid dose results in relapse of the lesions. Hydroxychloroquine sulfate at a dosage of 200 to 400 mg per day seems to be the treatment of choice to produce complete, long-lasting remission.[13,27,65,68] However, a long-term follow-up study demonstrated that combined therapy (small doses of corticosteroids and chloroquine) may be required because the initial good response to chloroquine ceases after several months or even years of treatment.[25]

Linear Immunoglobulin A Disease (Linear Immunoglobulin A Dermatosis)

Linear IgA disease (LAD), also known as *linear IgA dermatosis,* is an uncommon mucocutaneous disorder with a predilection for women. The etiopathogenic aspects of LAD are not fully understood, although drug-induced LAD triggered by angiotensin-converting enzyme (ACE) inhibitors has been reported.[48] LAD clinically presents as a pruritic vesiculobullous rash, usually during middle age and later, although younger individuals may be affected. Characteristic plaques or crops with an annular presentation surrounded by a peripheral rim of blisters affect the skin of the upper and lower trunk, shoulders, groin, and lower limbs. The face and perineum may also be affected. Mucosal involvement, including the oral mucosa, ranges from 50% to 100% of the cases published.[21,29,69]

LAD may mimic lichen planus both clinically and histologically. Immunofluorescence studies are needed to establish the correct diagnosis.

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Figure 12-23 Linear immunoglobulin A (IgA) disease. Intense erythema and ulceration of the gingiva consistent with desquamative gingivitis.

Oral Lesions. Oral manifestations of LAD consist of vesicles, painful ulcerations or erosions, and erosive gingivitis/cheilitis. The hard and soft palates are affected more often; tonsillar pillars, buccal mucosa, tongue, and gingiva follow in frequency. Rarely, oral lesions may be the only manifestation of LAD for several years before the presentation of cutaneous lesions.[19] In addition, oral lesions of LAD have been clinically reported as desquamative gingivitis[37,116,120,121] (Figure 12-23).

Histopathology The microscopic features of linear immunoglobulin A (IgA) disease (LAD) are similar to those observed in erosive lichen planus.

Immunofluorescence. Linear deposits of IgA are observed at the epithelial tissue–connective tissue interface. They differ from the granular pattern observed in dermatitis herpetiformis.

Differential Diagnosis. The differential diagnosis of LAD includes erosive lichen planus, chronic ulcerative stomatitis, pemphigus vulgaris, bullous pemphigoid, and lupus erythematosus. Microscopic examination and immunofluorescence studies are necessary to establish the correct diagnosis.

Treatment. The primary treatment for LAD is a combination of sulfones and dapsone. Small amounts of prednisone (10 to 30 mg/day) can be added if the initial response is inadequate.[24] Alternatively, tetracycline (2 g/day) combined with nicotinamide (1.5 g/day) have shown promising results.[117] Recently, mycophenolate (1 g twice daily [bid]) combined with prednisolone (30 mg daily) resulted in the resolution of refractory ulcerations associated with LAD.[83]

Dermatitis Herpetiformis

Dermatitis herpetiformis is a chronic condition that usually develops in young adults (ages 20 to 30 years) and has a slight predilection for men.[39] Currently, evidence indicates that dermatitis herpetiformis is a cutaneous manifestation of *celiac disease.* Approximately 25% of patients with celiac disease have dermatitis herpetiformis. The etiology of celiac disease is obscure; however, tissue transglutaminase seems to be the predominant autoantigen in the intestine, skin, and sometimes mucosae.[31] Gluten enteropathy can be severe in about two-thirds of patients and mild or subclinical in the remaining third. In severe cases, patients may complain of dysphagia, weakness, diarrhea, and weight loss.[93]

Clinically, dermatitis herpetiformis presents with bilateral and symmetric pruritic papules or vesicles primarily restricted to the extensor surfaces of the extremities. The sacrum, buttocks, and occasionally the face, as well as the oral cavity, may also be affected.[15,39] The name "herpetic" derives from the initial presentation of this disease, in which clusters of vesicles or papules arise on the skin. These vesicles or papules eventually resolve and are followed by hyperpigmentation of the skin, which ultimately wanes. The oral lesions of dermatitis herpetiformis range from painful ulcerations preceded by the collapse of ephemeral vesicles or bullae to erythematous lesions.[111]

Histopathology Microscopic examination of the initial lesions of dermatitis herpetiformis reveals focal aggregates of neutrophils and eosinophils among deposits of fibrin at the apices of the dermal pegs.[162]

Immunofluorescence. Direct immunofluorescence shows that IgA and C3 are present at the dermal papillary apices. These findings are present in perilesional and normal, uninvolved tissue. In contrast, biopsies taken from lesional sites may fail to exhibit IgA or C3, resulting in false-negative results.[162] Although no circulating autoantibodies to epithelial basement membrane are present in dermatitis herpetiformis, almost 80% of patients have antiendomysial and gliadin antibodies.[14]

Treatment. A gluten-free diet is essential in the treatment of celiac disease and dermatitis herpetiformis. Oral dapsone is usually needed in newly detected dermatitis herpetiformis to alleviate symptoms promptly.[19,31]

Lupus Erythematosus

Lupus erythematosus is an autoimmune disease with three different clinical presentations: systemic, chronic cutaneous, and subacute cutaneous.

Systemic Lupus Erythematosus. Systemic lupus erythematosus (SLE) is a severe disease with a predilection for women over men (10:1) that affects vital organs such as the kidneys and heart, as well as the skin and mucosa. The classic cutaneous lesions characterized by the presence of a rash on the malar area with a butterfly distribution are actually uncommon[35] (Figure 12-24). The oral lesions of SLE are usually ulcerative or similar to lichen planus. Oral ulcerations present in 36% of patients with SLE. In about 4% of patients, hyperkeratotic plaques reminiscent of lichen planus appear on the buccal mucosa and palate.[17] Direct immunofluorescence of the perilesional and normal tissue reveals immunoglobulins and C3 deposits at the dermal-epidermal interface. Antinuclear antibodies (ANA) are present in more than 95% of cases, whereas deoxyribonucleic acid (DNA) and extractable nuclear antigen (ENA) antibodies are present in more than 50% of patients (see Table 12-1).

Chronic Cutaneous Lupus Erythematosus. Chronic cutaneous lupus erythematosus (CCLE) usually has no systemic signs or symptoms, with lesions being limited to skin or mucosal surfaces. The skin lesions are referred to as *discoid lupus erythematosus* (DLE). DLE merely describes the chronic scarring, atrophy-producing lesion that may develop into hyperpigmentation or hypopigmentation of the healing area (Figure 12-25). In the oral cavity, about 9% of patients with CCLE present lichen planus–like plaques on the palate and buccal mucosa.[5,17] The gingiva may be affected and clinically present as desquamative gingivitis (Figure 12-26).

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Figure 12-24 Systemic lupus erythematosus producing erythema on bridge of the nose with a "butterfly" pattern. *(Courtesy of the Department of Dermatology, Hospital General Manuel Gea González, Mexico City.)*

Figure 12-25 Chronic cutaneous lupus erythematosus. Multiple facial lesions with irregular hyperpigmented borders, some of which exhibit central scarring with cutaneous atrophy. Other lesions consist of hyperpigmented cutaneous patches.

Figure 12-26 Lupus erythematosus of the oral cavity presenting as desquamative gingivitis. Intense erythema with ulceration is bordered by white radial lines. *(Courtesy Dr. Stuart L. Fischman, State University of New York at Buffalo.)*

Histopathology The histopathology of the oral lesions of chronic cutaneous lupus erythematosus consists of hyperkeratosis, alternated acanthosis and atrophy, and hydropic degeneration of the basal layer of the epithelium. The lamina propria exhibits a chronic inflammatory cell infiltrate similar to that observed in lichen planus. However, a more diffuse and deeper inflammatory infiltrate with a perivascular pattern is typically observed.[147]

Direct immunofluorescence of lesional tissue reveals immunoglobulins and C3 deposits at the dermal-epidermal junction of the lesional or perilesional tissue but not in normal tissue. This seems to differentiate SLE from DLE. Indirect immunofluorescence reveals the presence of ANAs in more than 95% of patients, whereas DNA- and ENA-circulating antibodies are present in more than 50%.

Subacute Cutaneous Lupus Erythematosus. Patients with subacute cutaneous lupus erythematosus (SCLE) present cutaneous lesions that are similar to DLE but lack the development of scarring and atrophy.[18] In addition, arthritis/arthralgia, low-grade fever, malaise, and myalgia may present in up to 50% of SCLE patients.[18,155] Direct immunofluorescence reveals immunoglobulins and C3 deposits at the dermal-epidermal junction in 60% of cases, and granular IgG deposits in the cytoplasm of basal cells in 30%. About 80% of patients with SCLE have Ro (SSA) antibodies to nuclear antigens, whereas 25% to 30% have La (SSB) antibodies to nuclear antigens. Rheumatoid factor (RF) is positive in about 30% of these patients, ANA is positive in 60% to 90%, and 10% of cases have antiribonucleoprotein (anti-RNP) antibodies to nuclear antigens (see Table 12-1).

Differential Diagnosis. Erosive lichen planus, erythema multiforme, and pemphigus vulgaris may sometimes simulate the lesions observed in lupus erythematosus. The diagnosis of DLE confined to the oral cavity is difficult to make, but microscopic studies may suggest the characteristic histopathology.[4] Biopsy studies (H&E and direct immunofluorescence) aid in differentiating between lupus erythematosus and other erosive diseases.

Treatment. The therapy for SLE depends on the severity and extent of the disease ranging from topical steroids to nonsteroidal antiinflammatory drug (NSAIDs) and for severe systemic organ involvement, moderate-to-high doses of prednisone. Immunosuppressive drugs, such as cytotoxic agents (cyclophosphamide and azathioprine), and plasmapheresis alone or with steroids are useful.[112] Recently, rituximab has been utilized and produced dramatic long-term remissions.[35] For CCLE, topical steroids are effective to manage the cutaneous and oral lesions. For patients resistant to topical therapy, systemic antimalarial drugs may be used with good results.[110]

Erythema Multiforme

Erythema multiforme (EM) is an acute bullous and macular inflammatory mucocutaneous disease that affects mainly young adults 20 to 40 years of age and rarely children (up to 20%).[134] The genesis of the mucocutaneous lesions is believed to reside in the development of immune complex vasculitis. This is followed by complement fixation, leading to leukocytoclastic destruction of vascular walls and small vessel occlusion. The culmination of these

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events produces ischemic necrosis of the epithelium and underlying connective tissue.[45] Target or "iris" lesions with central clearing are the "hallmark" of erythema multiforme. It may be a mild condition *(erythema multiforme minor)* or a severe, possibly life-threatening condition *(erythema multiforme major*, or *Stevens-Johnson syndrome).* An underdiagnosed type of erythema multiforme is the oral form in which most patients have chronic or recurrent oral lesions only.[6]

Erythema multiforme minor lasts approximately 4 weeks and exhibits a moderate cutaneous and mucosal involvement. Stevens-Johnson syndrome may last a month or longer and involves the skin, conjunctiva, oral mucosa, and genitalia, requiring more aggressive therapy. Some researchers consider toxic epidermal necrolysis as the most severe form of erythema multiforme; however, other investigators believe that these are unrelated entities.[10] The two most common etiologic factors for the development of erythema multiforme are (1) herpes simplex infection and (2) drug reactions. The most common causative drugs are sulfonamides, penicillins, quinolones, chlormezanone, barbiturates, oxicam NSAIDs, anticonvulsant drugs, protease inhibitors, and allopurinol.[46]

Oral lesions in erythema multiforme are common and present in more than 70% of patients with skin involvement.[47,89,95] In rare instances, however, erythema multiforme may be confined to the mouth.[6,86,138] The oral lesions consist of multiple, large, shallow painful ulcers with an erythematous border. They may affect the entire oral mucosa in approximately 20% of patients with erythema multiforme. The lesions are so painful that chewing and swallowing are impaired (Figure 12-27). The buccal mucosa and tongue are the most frequently affected sites, followed by the labial mucosa. Less often affected are the floor of the mouth, hard and soft palate, and the gingiva.[47] There are rare instances in which erythema multiforme may be confined exclusively to the gingival tissues, resulting in a clinical diagnosis of desquamative gingivitis.[9] Hemorrhagic crusting of the vermilion border of the lips may occur, which is helpful in arriving at a clinical diagnosis of erythema multiforme.

Histopathology Common microscopic findings of erythema multiforme include liquefaction degeneration of the upper epithelium and development of intraepithelial microvesicles but without the acantholysis that occurs in pemphigus.[145] In addition, acanthosis, pseudoepitheliomatous hyperplasia, and necrotic keratinocytes are observed in the epithelium. Degenerative changes also occur in the basement membrane. In some cases the junction between the epithelium and the lamina propria is indistinct because of a dense, inflammatory cell infiltrate. Edema of the lamina propria, vascular dilation, and congestion are also present. Deeper layers of the connective tissue stroma exhibit a perivascular, chronic inflammatory cell infiltrate. However, neutrophils and eosinophils may also be present.

Immunofluorescence. Immunofluorescence examination is negative in erythema multiforme. Its value resides in ruling out other vesiculobullous and ulcerative disorders.

Treatment. There is no specific treatment for erythema multiforme. For mild symptoms, systemic and local antihistamines together with topical anesthetics and debridement of lesions with an oxygenating agent are adequate. In patients with bullous or ulcerative lesions and severe symptoms, corticosteroids are considered the drug of choice, although their use is controversial and not completely accepted.[46]

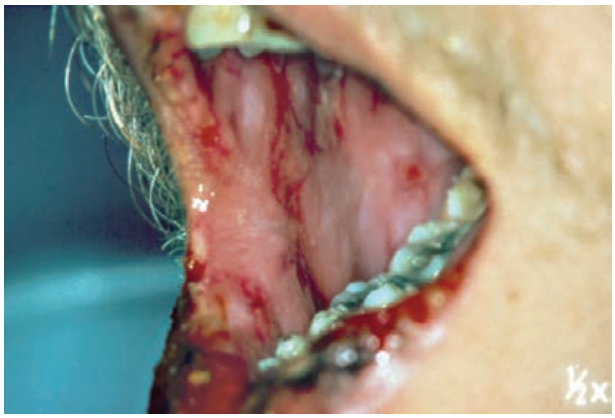

Figure 12-27 Erythema multiforme. Large, shallow, and painful ulcers involving the labial and buccal mucosae. Hemorrhagic crusting of the mandibular vermilion border of the lips is observed. *(Courtesy Dr. Stuart L. Fischman, State University of New York at Buffalo.)*

DRUG ERUPTIONS

An increase in the incidence of skin and oral manifestations of hypersensitivity to drugs has been noted since the advent of sulfonamides, barbiturates, and various antibiotics. The cutaneous and oral lesions are attributed to the drug acting as an allergen that sensitizes the tissues.

Eruptions in the oral cavity resulting from sensitivity to drugs that have been taken by mouth or parenterally are termed *stomatitis medicamentosa.* The local reaction from the use of a medicament in the oral cavity (e.g., stomatitis resulting from topical penicillin) is referred to as *stomatitis venenata* or *contact stomatitis.* In many cases, skin eruptions may accompany the oral lesions.

In general, drug eruptions in the oral cavity are multiform. Vesicular and bullous lesions occur most often, but pigmented or nonpigmented macular lesions are also frequently observed. Erosions, often followed by deep ulceration with purpuric lesions, may also occur. The lesions are seen in different areas of the oral cavity, with the gingiva often affected.[1,54]

The development of gingival lesions caused by contact allergy to mercurial compounds present in dental amalgam has been clearly documented.[73] Because of financial considerations, biopsy and patch testing may be indicated before the indiscriminate replacement of dental amalgam restorations. Similarly, desquamative gingivitis has been reported with the use of tartar control toothpaste. Pyrophosphates and flavoring agents have been identified as the main causative agents of this unusual condition.[36] Oral reactions to cinnamon compounds (cinnamon oil, cinnamic acid, or cinnamic aldehyde) used to mask the taste of pyrophosphates in tartar control toothpaste include an intense erythema of the attached gingival tissues characteristic of plasma cell gingivitis[3,74] (Figure 12-28). A thorough clinical history usually discloses the source of the gingival disturbance. Elimination of the offending agent (e.g., tartar control toothpaste) leads to resolution of the gingival lesions within a week. Challenge with the offending agent leads to recurrence of the oral lesions. If removal of the offending medication is not possible, topical corticosteroids and topical tacrolimus can be used to treat the lesions.[66]

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Figure 12-28 Plasma cell gingivitis. The gingiva presents a band of moderate-to-severe inflammation reminiscent of desquamative gingivitis.

MISCELLANEOUS CONDITIONS MIMICKING DESQUAMATIVE GINGIVITIS

For more information on conditions mimicking desquamative gingivitis and to view Figures 12-29 and 12-30, please visit the companion Expert CONSULT website at www.expertconsult.com.

Failure to evaluate properly and systematically a patient with a clinical condition that is consistent with desquamative gingivitis can lead to unpleasant outcomes. This is particularly true when therapy for a putative desquamative gingivitis is established before a biopsy of the lesional tissue is obtained. Every year we see in our laboratory at least two examples of clinically diagnosed desquamative gingivitis for which microscopic and immunofluorescence studies were not performed to rule out the genesis of the gingival lesions. In these cases the patients have been either "carefully" followed up or prescribed topical steroids for several months. The lack of response of the gingival tissues eventually impels the clinician to obtain a biopsy, which reveals that the gingival lesions are *squamous cell carcinomas.* Thus the clinician should be alert to the possibility of squamous cell carcinoma of the gingival tissues presenting initially as "desquamative gingivitis."

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 13

The Periodontal Pocket

Fermin A. Carranza and Paulo M. Camargo

CHAPTER OUTLINE

CLASSIFICATION
CLINICAL FEATURES
PATHOGENESIS
HISTOPATHOLOGY
- Bacterial Invasion
- Mechanisms of Tissue Destruction
- Microtopography of Gingival Wall
- Periodontal Pockets as Healing Lesions
- Pocket Contents
- Root Surface Walls

PERIODONTAL DISEASE ACTIVITY
SITE SPECIFICITY
PULP CHANGES ASSOCIATED WITH PERIODONTAL POCKETS
RELATIONSHIP OF ATTACHMENT LOSS AND BONE LOSS TO POCKET DEPTH
AREA BETWEEN BASE OF POCKET AND ALVEOLAR BONE
RELATIONSHIP OF POCKET TO BONE
PERIODONTAL ABSCESS
LATERAL PERIODONTAL CYST

The periodontal pocket, defined as a pathologically deepened gingival sulcus, is one of the most important clinical features of periodontal disease. All different types of periodontitis, as outlined in Chapter 4, share histopathologic features such as tissue changes in the periodontal pocket, mechanisms of tissue destruction, and healing mechanisms. They differ, however, in their etiology, natural history, progression, and response to therapy.[32]

CLASSIFICATION

Deepening of the gingival sulcus may occur by coronal movement of the gingival margin, apical displacement of the gingival attachment, or a combination of the two processes (Figure 13-1). Pockets can be classified as follows:

Gingival pocket is formed by gingival enlargement without destruction of the underlying periodontal tissues. The sulcus is deepened because of the increased bulk of the gingiva (Figure 13-2, *A*).

Periodontal pocket produces destruction of the supporting periodontal tissues, leading to loosening and exfoliation of the teeth. The remainder of this chapter refers to this type of pocket.

Two types of periodontal pockets exist, as follows:

Suprabony *(supracrestal* or *supraalveolar),* in which the bottom of the pocket is coronal to the underlying alveolar bone (Figure 13-2, *B*).

Intrabony *(infrabony, subcrestal, intraalveolar),* in which the bottom of the pocket is apical to the level of the adjacent alveolar bone. In this second type, the lateral pocket wall lies between the tooth surface and the alveolar bone (Figure 13-2, *C*).

Pockets can involve one, two, or more tooth surfaces and can be of different depths and types on different surfaces of the same tooth and on approximating surfaces of the same interdental space.[30,38] Pockets can also be spiral (i.e., originating on one tooth surface and twisting around the tooth to involve one or more additional surfaces) (Figure 13-3). These types of pockets are most common in furcation areas.

CLINICAL FEATURES

Clinical signs that suggest the presence of periodontal pockets include a bluish red, thickened marginal gingiva; a bluish red, vertical zone from the gingival margin to the alveolar mucosa; gingival bleeding and suppuration; tooth mobility; diastema formation; and symptoms such as localized pain or pain "deep in the bone." The only reliable method of locating periodontal pockets and determining their extent is careful probing of the gingival margin along each tooth surface (Figure 13-4 and Table 13-1). On the basis of depth alone, however, it is sometimes difficult to differentiate between a deep normal sulcus and a shallow periodontal pocket. In such borderline cases, pathologic changes in the gingiva distinguish the two conditions.

For a more detailed discussion of the clinical aspects of periodontal pockets, see Chapter 30.

PATHOGENESIS

The initial lesion in the development of periodontitis is the inflammation of the gingiva in response to a bacterial challenge. Changes involved

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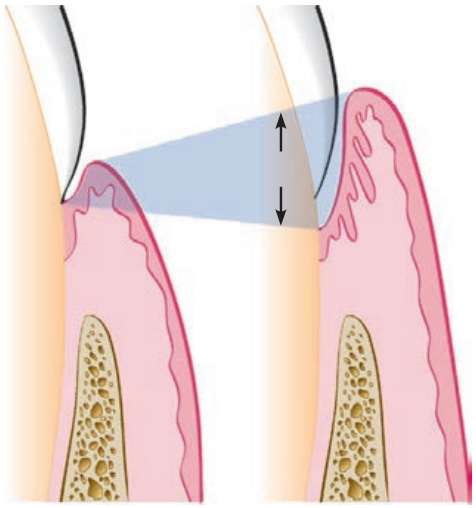

Figure 13-1 Illustration of pocket formation indicating expansion in two directions *(arrows)* from the normal gingival sulcus *(left)* to the periodontal pocket *(right)*.

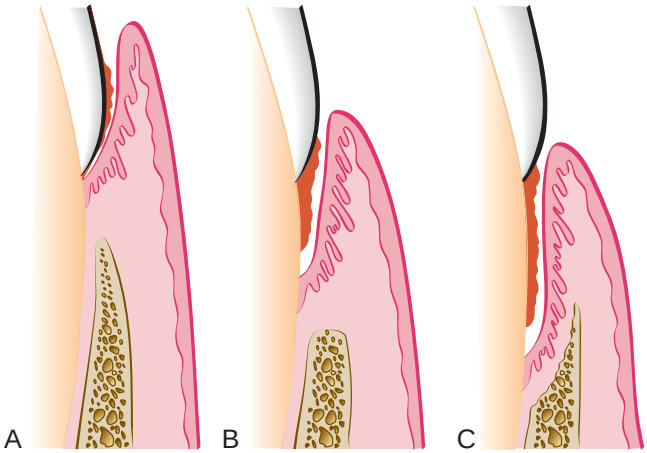

Figure 13-2 Different types of periodontal pockets. **A,** Gingival pocket. There is no destruction of the supporting periodontal tissues. **B,** Suprabony pocket. The base of the pocket is coronal to the level of the underlying bone. Bone loss is horizontal. **C,** Intrabony pocket. The base of the pocket is apical to the level of the adjacent bone. Bone loss is vertical.

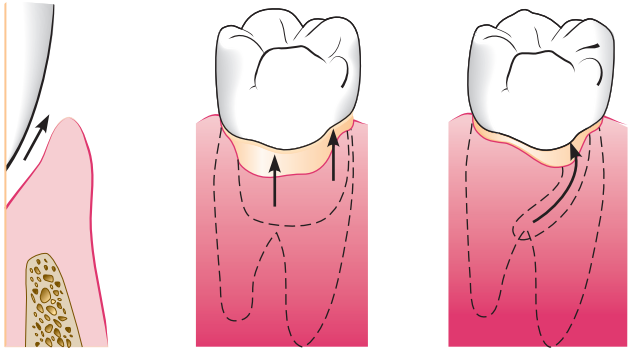

Figure 13-3 Classification of pockets according to involved tooth surfaces. **A,** Simple pocket. **B,** Compound pocket. **C,** Complex pocket.

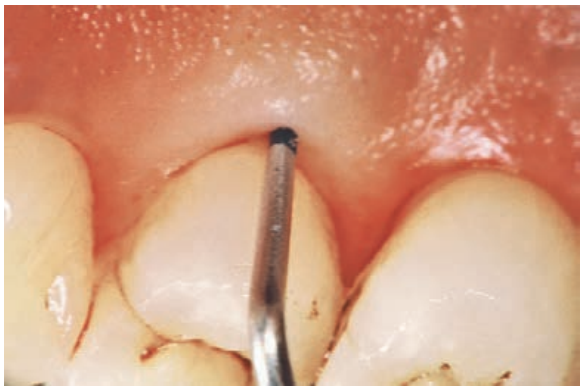

Figure 13-4 Probing of a deep periodontal pocket. Entire length of the periodontal probe has been inserted to the base of the pocket in palatal surface of first premolar.

TABLE 13-1 Correlation of Clinical and Histopathologic Features of the Periodontal Pocket

Clinical Features	Histopathologic Features
1. Gingival wall of pocket presents various degrees of bluish red discoloration; flaccidity; a smooth, shiny surface; and pitting on pressure.	1. The discoloration is caused by circulatory stagnation; the flaccidity, by destruction of gingival fibers and surrounding tissues; the smooth, shiny surface, by atrophy of epithelium and edema; and the pitting on pressure, by edema and degeneration.
2. Less frequently, gingival wall may be pink and firm.	2. In such cases, fibrotic changes predominate over exudation and degeneration, particularly in relation to outer surface of pocket wall. However, despite external appearance of health, inner wall of pocket invariably presents some degeneration and is often ulcerated (see Figure 13-15).
3. Bleeding is elicited by gently probing soft tissue wall of pocket.	3. Ease of bleeding results from increased vascularity, thinning and degeneration of epithelium, and proximity of engorged vessels to inner surface.
4. When explored with a probe, inner aspect of pocket is generally painful.	4. Pain on tactile stimulation is caused by ulceration of inner aspect of pocket wall.
5. In many cases, pus may be expressed by applying digital pressure.	5. Pus occurs in pockets with suppurative inflammation of inner wall.

in the transition from the normal gingival sulcus to the pathologic periodontal pocket are associated with different proportions of bacterial cells in dental plaque. Healthy gingiva is associated with few microorganisms, mostly coccoid cells and straight rods. Diseased gingiva is associated with increased numbers of spirochetes and motile rods.[40,41,43] However, the microbiota of diseased sites cannot be used as a predictor of future attachment or bone loss because their presence alone is not sufficient for disease to start or progress.[35]

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Figure 13-5 Interdental papilla with suprabony pockets on proximal tooth surfaces. Note the densely inflamed connective tissue, with the infiltrate extending between the collagen fibers, and the proliferating and ulcerated pocket epithelium.

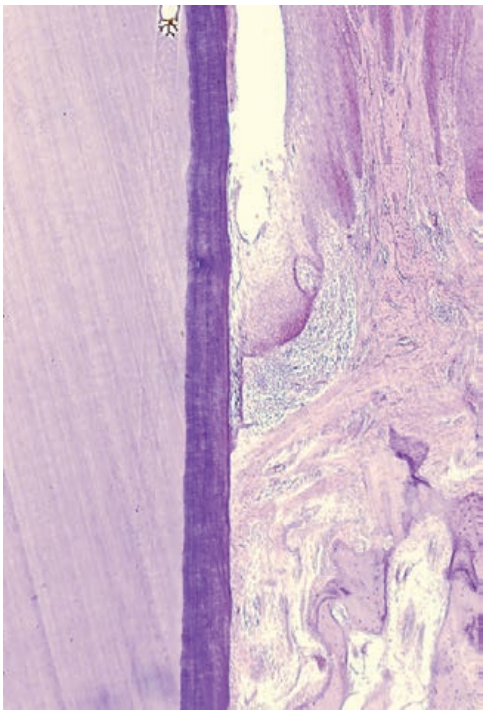

Figure 13-6 Low-power view of base of periodontal pocket and apical area. Note the dense inflammatory infiltrate on area of destroyed collagen fibers and the thin, fingerlike extension of epithelium covering the cementum denuded of fibers.

Pocket formation starts as an inflammatory change in the connective tissue wall of the gingival sulcus. The cellular and fluid inflammatory exudate causes degeneration of the surrounding connective tissue, including the gingival fibers. Just apical to the junctional epithelium, collagen fibers are destroyed[20,61] and the area is occupied by inflammatory cells and edema (Figure 13-5).

Early concepts assumed that after the initial bacterial attack, periodontal tissue destruction continued to be linked to bacterial action. More recently, it was established that the host's immunoinflammatory response to the initial and persistent bacterial attack unleashes mechanisms that lead to collagen and bone destruction. These mechanisms are related to various cytokines, some produced normally by cells in noninflamed tissue and others by cells involved in the inflammatory process such as polymorphonuclear leukocytes (PMNs), monocytes, and other cells, leading to collagen and bone destruction. This chapter describes the histologic aspects of gingival inflammation and tissue destruction. For further information on the molecular biology aspects of these mechanisms of tissue destruction the reader is referred to Chapter 25 and online.

The two mechanisms associated with collagen loss are (1) collagenases and other enzymes secreted by various cells in healthy and inflamed tissue, such as fibroblasts,[74] PMNs,[73] and macrophages,[53] become extracellular and destroy collagen (these enzymes that degrade collagen and other matrix macromolecules into small peptides are called *matrix metalloproteinases*[75]) and (2) fibroblasts phagocytize collagen fibers by extending cytoplasmic processes to the ligament-cementum interface and degrade the inserted collagen fibrils and the fibrils of the cementum matrix.[20,21]

As a consequence of the loss of collagen, the apical cells of the junctional epithelium proliferate along the root, extending fingerlike projections two or three cells in thickness (Figure 13-6).

As a result of inflammation, PMNs invade the coronal end of the junctional epithelium in increasing numbers (Figure 13-7). The PMNs are not joined to one another or to the epithelial cells by desmosomes. When the relative volume of PMNs reaches approximately 60% or more of the junctional epithelium, the tissue loses cohesiveness and detaches from the tooth surface. Thus the coronal portion of the junctional epithelium detaches from the root as the apical portion migrates, resulting in its apical shift, and the oral sulcular epithelium gradually occupies an increasing portion of the sulcus (then a pocket) lining.[62]

Extension of the junctional epithelium along the root requires the presence of healthy epithelial cells. Marked degeneration or necrosis of the junctional epithelium impairs rather than accelerates pocket formation. (This occurs in necrotizing ulcerative gingivitis resulting in an ulcer and not in pocket formation.)

Degenerative changes seen in the junctional epithelium at the base of periodontal pockets are usually less severe than those in the epithelium of the lateral pocket wall (see Figure 13-7). Because migration of the junctional epithelium requires healthy, viable cells, it is reasonable to assume that the degenerative changes seen in this area occur after the junctional epithelium reaches its position on the cementum.

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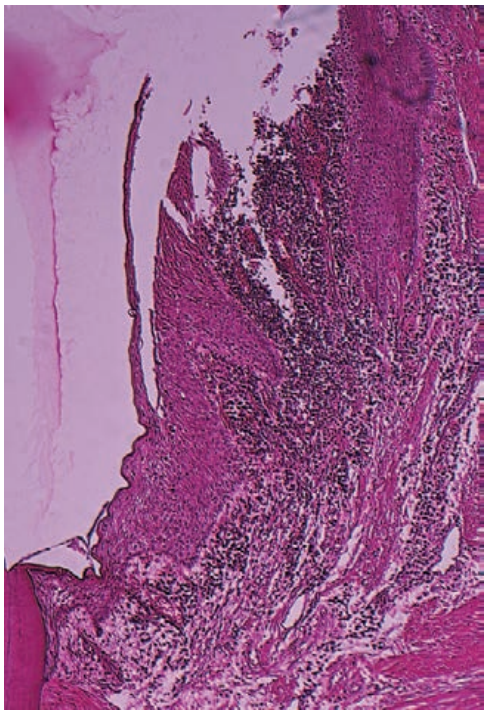

Figure 13-7 View of the ulcerated lateral pocket wall of a periodontal pocket. Note the extension of epithelial cells and the dense accumulation of leukocytes within the epithelium and in the connective tissue.

The degree of leukocyte infiltration of the junctional epithelium is independent of the volume of inflamed connective tissue, thus this process may occur in gingiva with only slight signs of clinical inflammation.[61]

With continued inflammation, the gingiva increases in bulk and the crest of the gingival margin extends coronally. The apical cells of the junctional epithelium continue to migrate along the root and its coronal cells continue to separate from it. The epithelium of the lateral wall of the pocket proliferates to form bulbous, cordlike extensions into the inflamed connective tissue. Leukocytes and edema from the inflamed connective tissue infiltrate the epithelium lining the pocket, resulting in various degrees of degeneration and necrosis.

The transformation of a gingival sulcus into a periodontal pocket creates an area where plaque removal becomes impossible, and a feedback mechanism is established. The rationale for pocket reduction is based on the need to eliminate areas of plaque accumulation.

HISTOPATHOLOGY

Changes occurring in the initial stages of gingival inflammation are presented in Chapter 7. Once the pocket is formed, several microscopic features are present, as discussed in this section.

Soft Tissue Wall

The connective tissue is edematous and densely infiltrated with plasma cells (approximately 80%), lymphocytes, and a scattering of PMNs. The blood vessels are increased in number, dilated, and engorged, particularly in the subepithelial connective tissue layer.[10]

The connective tissue exhibits varying degrees of degeneration. Single or multiple necrotic foci are occasionally present.[52] In addition to exudative and degenerative changes, the connective tissue shows proliferation of the endothelial cells, with newly formed capillaries, fibroblasts, and collagen fibers (see Figure 13-5).

The junctional epithelium at the base of the pocket is usually much shorter than that of a normal sulcus. Although marked variations are found as to length, width, and condition of the epithelial cells,[63] usually the coronoapical length of the junctional epithelium is reduced to only 50 to 100 µm.[14] The cells may be well formed and in good condition or may exhibit slight-to-marked degeneration (see Figures 13-6 and 13-9).

The most severe degenerative changes in the periodontal pocket occur along the lateral wall (see Figure 13-7). The epithelium of the lateral wall of the pocket presents striking proliferative and degenerative changes. Epithelial buds or interlacing cords of epithelial cells project from the lateral wall into the adjacent inflamed connective tissue and may extend farther apically than the junctional epithelium (Figures 13-8, *A,* and 13-9). These epithelial projections, as well as the remainder of the lateral epithelium, are densely infiltrated by leukocytes and edema from the inflamed connective tissue. The cells undergo vacuolar degeneration and rupture to form vesicles. Progressive degeneration and necrosis of the epithelium lead to ulceration of the lateral wall, exposure of the underlying inflamed connective tissue, and suppuration. In some cases, acute inflammation is superimposed on the underlying chronic changes.

A comparative study of gingival changes in aggressive and chronic periodontitis revealed more pronounced degenerative changes in the epithelium of aggressive cases with more open intercellular spaces, with microclefts and necrotic areas.[35]

The severity of the degenerative changes is not necessarily related to pocket depth. Ulceration of the lateral wall may occur in shallow pockets, and deep pockets are occasionally observed in which the lateral epithelium is relatively intact or shows only slight degeneration.

The epithelium at the gingival crest of a periodontal pocket is generally intact and thickened, with prominent rete pegs.

For a detailed electron microscopic study of the pocket epithelium in experimentally induced pockets in dogs, see Müller-Glauser and Schröder.[49]

Bacterial Invasion

Bacterial invasion of the apical and lateral areas of the pocket wall has been described in human chronic periodontitis. Filaments, rods, and coccoid organisms with predominant gram-negative cell walls have been found in intercellular spaces of the epithelium.[25,26] Hillmann et al[35] have reported the presence of *Porphyromonas gingivalis* and *Prevotella intermedia* in the gingiva of aggressive periodontitis cases. *Actinobacillus actinomycetemcomitans* has also been found in the tissues.[16,47,58]

Bacteria may invade the intercellular space under exfoliating epithelial cells, but they are also found between deeper epithelial cells and accumulating on the basement lamina. Some bacteria traverse the basement lamina and invade the subepithelial connective tissue[60] (Figures 13-10 and 13-11).

The presence of bacteria in the gingival tissues has been interpreted by different investigators as bacterial invasion or as "passive translocation" of plaque bacteria.[42] This important point has significant clinicopathologic implications and has not yet been clarified.[17,39,43]

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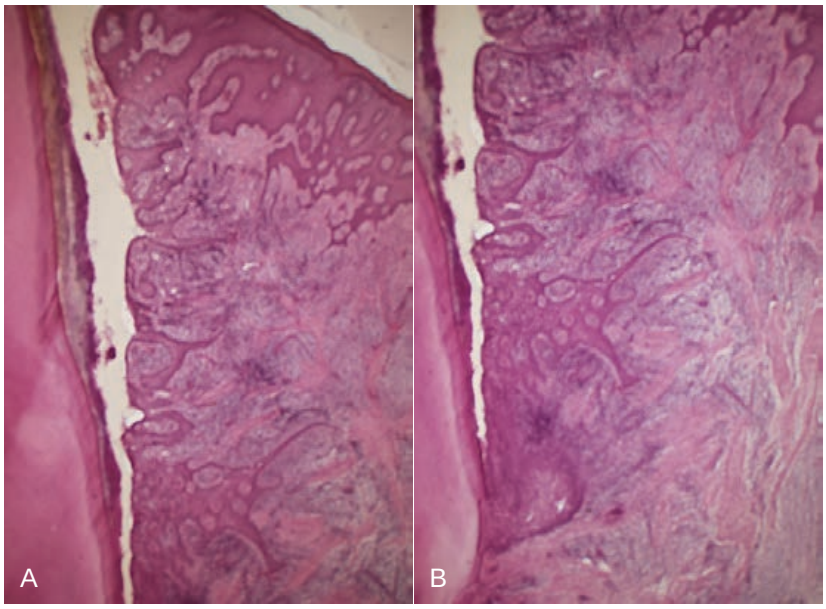

Figure 13-8 **A,** Lateral wall of periodontal pocket showing epithelial proliferative and atrophic changes and marked inflammatory infiltrate and destruction of collagen fibers. **B,** Slightly apical view of the same case showing the shortened junctional epithelium.

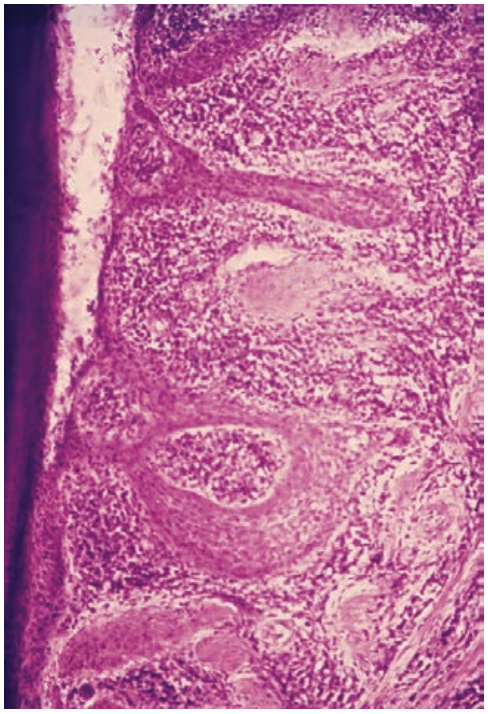

Figure 13-9 Base of periodontal pocket showing extensive proliferation of lateral epithelium next to atrophic areas, dense inflammatory infiltrate, remnants of destroyed collagen fibers, and the junctional epithelium, apparently in a less altered state than lateral pocket epithelium.

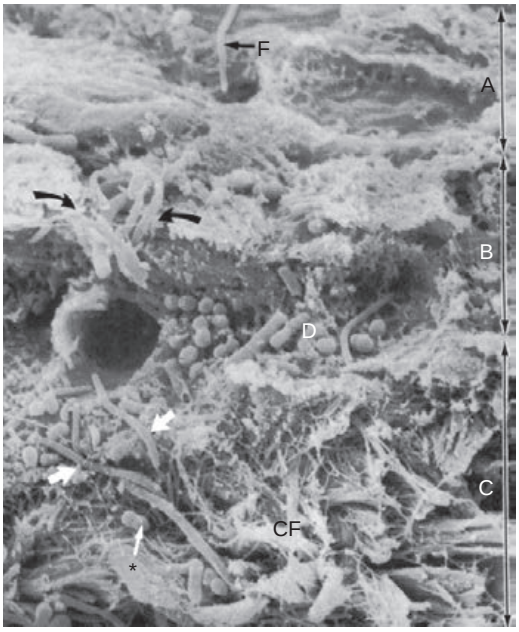

Figure 13-10 Scanning electron micrograph of section of pocket wall in advanced periodontitis in human specimen showing bacterial penetration into the epithelium and connective tissue. Scanning electron microscope view of surface of pocket wall *(A),* sectioned epithelium *(B),* and sectioned connective tissue *(C).* Curved arrows point to areas of bacterial penetration into the epithelium. Thick white arrows point to bacterial penetration into the connective tissue through a break in the continuity of the basal lamina. *CF,* Connective tissue fibers; *D,* accumulation of bacteria (rods, cocci, filaments) on basal lamina; *F,* filamentous organism on surface of epithelium. Asterisk points to coccobacillus in connective tissue.

Mechanisms of Tissue Destruction

The inflammatory response triggered by bacterial plaque unleashes a complex cascade of events aimed at destroying and removing bacteria, necrotic cells, and deleterious agents. However, this process is nonspecific and in an attempt to restore health, the host's cells, such as neutrophiles, macrophages, fibroblasts, epithelial cells and others, produce proteinases, cytokines, and prostaglandins that can damage or destroy the tissues.

Chapter 25 describes in detail these aspects of inflammation and the mechanisms of tissue destruction at the molecular level.

Microtopography of Gingival Wall

Scanning electron microscopy has permitted the description of several areas in the soft tissue (gingival) wall of the periodontal pocket in which different types of activity take place.[59] These areas are irregularly oval or elongated and adjacent to one another and measure about 50 to 200 µm. These findings suggest that the pocket wall is constantly changing as a result of the interaction

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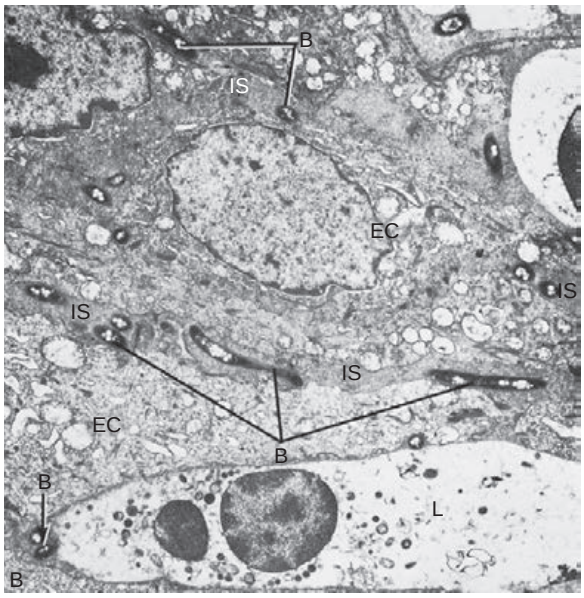

Figure 13-11 Transmission electron micrograph of the epithelium in the periodontal pocket wall showing bacteria in the intercellular spaces. *B,* Bacteria; *EC,* epithelial cell; *IS,* intercellular space; L, leukocyte about to engulf bacteria. (×8000.)

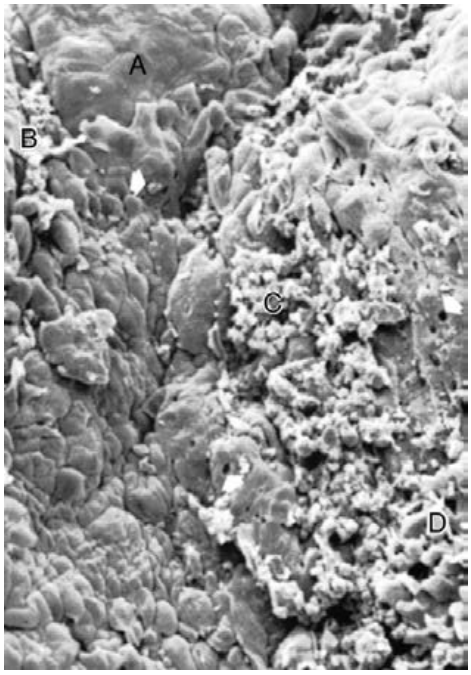

Figure 13-12 Scanning electron frontal micrograph of the periodontal pocket wall. Different areas can be seen in the pocket wall surface. *A,* Area of quiescence; *B,* bacterial accumulation; *C,* bacterial-leukocyte interaction; *D,* intense cellular desquamation. Arrows point to emerging leukocytes and holes left by leukocytes in the pocket wall. (×800.)

between the host and the bacteria. The following areas have been noted:

1. *Areas of relative quiescence,* showing a relatively flat surface with minor depressions and mounds and occasional shedding of cells (Figure 13-12, area *A*).
2. *Areas of bacterial accumulation,* which appear as depressions on the epithelial surface, with abundant debris and bacterial clumps penetrating into the enlarged intercellular spaces. These bacteria are mainly cocci, rods, and filaments, with a few spirochetes (Figure 13-12, area *B*).
3. *Areas of emergence of leukocytes,* in which leukocytes appear in the pocket wall through holes located in the intercellular spaces (Figure 13-13).
4. *Areas of leukocyte-bacteria interaction,* in which numerous leukocytes are present and covered with bacteria in an apparent process of phagocytosis. Bacterial plaque associated with the epithelium is seen either as an organized matrix covered by a fibrinlike material in contact with the surface of cells or as bacteria penetrating into the intercellular spaces (Figure 13-12, area *C*).
5. *Areas of intense epithelial desquamation,* which consist of semiattached and folded epithelial squames, sometimes partially covered with bacteria (Figure 13-12, area *D*).
6. *Areas of ulceration,* with exposed connective tissue (Figure 13-14).
7. *Areas of hemorrhage,* with numerous erythrocytes.

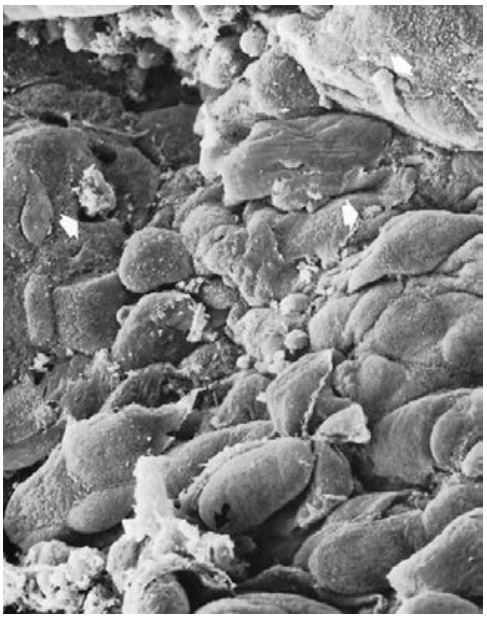

Figure 13-13 Scanning electron micrograph of the periodontal pocket wall, frontal view, in a patient with advanced periodontitis. Note the desquamating epithelial cells and leukocytes *(white arrows)* emerging onto the pocket space. Scattered bacteria can also be seen *(black arrow).* (×1500.)

The transition from one area to another could result from bacteria accumulating in previously quiescent areas and triggering the emergence of leukocytes and the leukocyte-bacteria interaction. This would lead to intense epithelial desquamation and finally to ulceration and hemorrhage.

Periodontal Pockets as Healing Lesions

Periodontal pockets are chronic inflammatory lesions and thus are constantly undergoing repair. Complete healing does not occur because of the persistence of the bacterial attack, which continues to stimulate an inflammatory response, causing degeneration of the new tissue elements formed in the continuous effort at repair.

The condition of the soft tissue wall of the periodontal pocket results from the interplay of the destructive and constructive tissue changes. Their balance determines clinical features such as color, consistency, and surface texture of the pocket wall. If the inflammatory fluid and cellular exudate predominate, the pocket wall is

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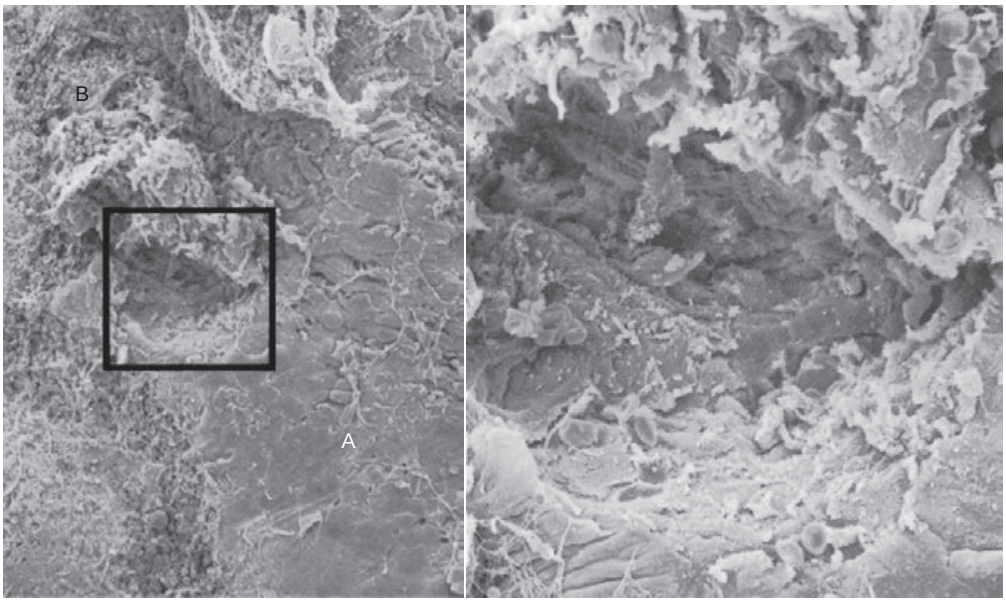

Figure 13-14 *Left,* Area of ulceration in the lateral wall of a deep periodontal pocket in a human specimen. *A,* Surface of pocket epithelium in a quiescent state; *B,* area of hemorrhage. (×800.) *Right,* Magnification of the square on left. Connective tissue fibers and cells can be seen in the bottom of the ulcer. (Scanning electron microscopy, ×3000.)

SCIENCE TRANSFER

Clinicians should use their knowledge of the histopathology of periodontal disease as a basis for clinical decision making. As inflammation spreads apically to involve the connective tissue apical to the junctional epithelium, gingivitis then becomes periodontitis. Periodontal probing with gentle pressure generally allows the probe to extend to the apical end of the junctional epithelium in patients with gingival health; however, when inflammatory changes result in less dense collagen fibers in early and advanced periodontitis, special care must be taken not to push the probe too far apically, which results in inaccurate pocket depth recordings and pain for the patient.

One of the earliest and common changes in gingivitis is ulceration of the gingival sulcular epithelium that results in bleeding on probing, which will persist as the disease progresses to periodontitis. This is a sign of continuing bacterial destruction of periodontal tissues. In more severe disease, purulence may be seen in the gingival fluid, which indicates more dramatic and aggressive tissue breakdown.

bluish red, soft, spongy, and friable, with a smooth, shiny surface; at the clinical level, this is generally referred to as an *edematous pocket wall.* If there is a relative predominance of newly formed connective tissue cells and fibers, the pocket wall is more firm and pink, clinically referred to as a *fibrotic pocket wall* (see Table 13-1 and Chapter 30).

Edematous and fibrotic pockets represent opposite extremes of the same pathologic process not different disease entities. They are subject to constant modification, depending on the relative predominance of exudative and constructive changes.

Fibrotic pocket walls may be misleading because they do not necessarily reflect what is taking place throughout the pocket wall. The most severe degenerative changes in periodontal tissues occur adjacent to the tooth surface and subgingival plaque. In some cases, inflammation and ulceration on the inside of the pocket are walled off by fibrous tissue on the outer aspect (Figure 13-15). Externally the pocket appears pink and fibrotic, despite the inflammatory changes occurring internally.

Pocket Contents

Periodontal pockets contain debris consisting principally of microorganisms and their products (enzymes, endotoxins, and other metabolic products), gingival fluid, food remnants, salivary mucin, desquamated epithelial cells, and leukocytes. Plaque-covered calculus usually projects from the tooth surface (Figure 13-16). Purulent exudate, if present, consists of living, degenerated, and necrotic leukocytes; living and dead bacteria; serum; and a scant amount of fibrin.[46] The contents of periodontal pockets filtered free of organisms and debris have been demonstrated to be toxic when injected subcutaneously into experimental animals.[31]

Pus is a common feature of periodontal disease, but it is only a secondary sign. The presence of pus or the ease with which it can be expressed from the pocket merely reflects the nature of the inflammatory changes in the pocket wall. It is not an indication of the depth of the pocket or the severity of the destruction of the supporting tissues. Extensive pus formation may occur in shallow pockets, whereas deep pockets may exhibit little or no pus.

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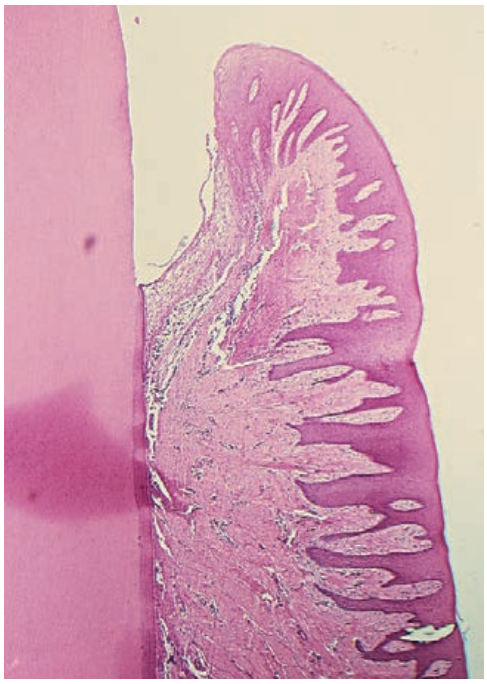

Figure 13-15 Periodontal pocket wall. The inner half is inflamed and ulcerated; the outer half is densely collagenous.

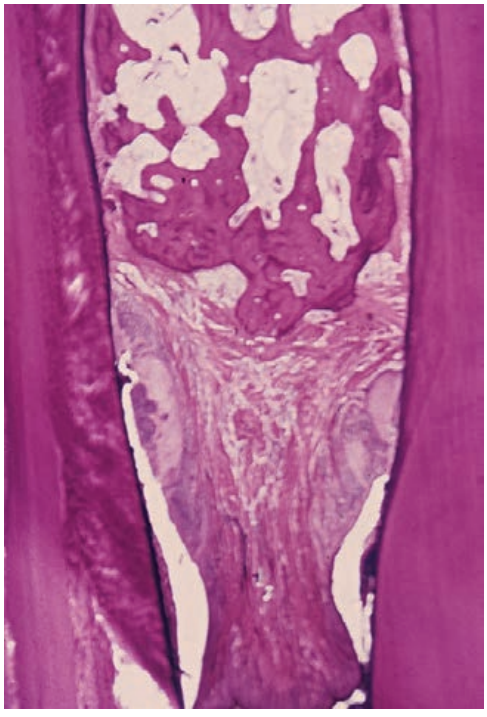

Figure 13-16 Interdental papilla with ulcerated suprabony periodontal pockets on its mesial and distal aspects. Calculus is present on the approximal tooth surfaces and within the gingiva.

Localized accumulation of pus constitutes an abscess, which is discussed later in this chapter.

Root Surface Walls

The root surface wall of periodontal pockets often undergoes changes that are significant because they may perpetuate the periodontal infection, cause pain, and complicate periodontal treatment.[11]

As the pocket deepens, collagen fibers embedded in the cementum are destroyed and cementum becomes exposed to the oral environment. Collagenous remnants of Sharpey's fibers in the cementum undergo degeneration, creating an environment favorable to the penetration of bacteria. Viable bacteria have been found in the roots of 87% of periodontally diseased noncarious teeth.[2] Bacterial penetration into the cementum can be found as deep as the cementodentinal junction[1,19] and may also enter the dentinal tubules.[29,31] Penetration and growth of bacteria leads to fragmentation and breakdown of the cementum surface and results in areas of necrotic cementum, separated from the tooth by masses of bacteria.

Pathologic granules[9] have been observed with light and electron microscopy[6,7] and may represent areas of collagen degeneration or areas in which collagen fibrils have not been fully mineralized initially.

In addition, bacterial products, such as endotoxins,[3,5] have also been detected in the cementum wall of periodontal pockets. When root fragments from teeth with periodontal disease are placed in tissue culture, they induce irreversible morphologic changes in the cells of the culture. Such changes are not produced by normal roots.[33] Diseased root fragments also prevent the in vitro attachment of human gingival fibroblasts, whereas normal root surfaces allow the cells to attach freely.[4] When placed in the oral mucosa of the patient, diseased root fragments induce an inflammatory response even if they are autoclaved.[44]

These changes manifest clinically as softening of the cementum surface, which is usually asymptomatic, but painful when a probe or explorer penetrates the area. They also constitute a possible reservoir for reinfection of the area after treatment. In the course of treatment, these necrotic areas are removed by root planing until a hard, smooth surface is reached. Cementum is very thin in the cervical areas, and scaling and root planing often remove it entirely, exposing the underlying dentin. Sensitivity to cold may result until secondary dentin is formed by the pulp tissue.

Decalcification and Remineralization of Cementum. Areas of *increased mineralization*[65] are probably a result of an exchange, on exposure to the oral cavity, of minerals and organic components at the cementum-saliva interface. The mineral content of exposed cementum increases,[64] and the minerals that are increased in diseased root surfaces include calcium,[67] magnesium,[50,67] phosphorus,[50] and fluoride.[50] Microhardness, however, remains unchanged.[55,81] The development of a highly mineralized superficial layer may increase the tooth resistance to decay.[4]

The hypermineralized zones are detectable by electron microscopy and are associated with increased perfection of the crystal structure and organic changes suggestive of a subsurface cuticle.[64,65] These zones have also been seen in microradiographic studies[66] as a layer 10 to 20 mm thick, with areas as thick as 50 mm. No decrease in mineralization was found in deeper areas, thereby indicating that increased mineralization does not come from adjacent areas. A loss of or reduction in the crossbanding of collagen near the cementum surface[27,28] and a subsurface condensation of organic material of exogenous origin[64] have also been reported.

Areas of demineralization are often related to **root caries**. Exposure to oral fluid and bacterial plaque results in proteolysis of the embedded remnants of Sharpey's fibers; the cementum may be

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softened and may undergo fragmentation and cavitation.[34] Unlike enamel caries, root surface caries tend to progress around rather than into the tooth.[48] Active root caries lesions appear as well-defined yellowish or light-brown areas, are frequently covered by plaque, and have a softened or leathery consistency on probing. Inactive lesions are well-defined darker lesions with a smooth surface and a harder consistency on probing.[24]

The dominant microorganism in root surface caries is *Actinomyces viscosus,*[72] although its specific role in the development of the lesion has not been established.[24] Other bacteria, such as *Actinomyces naeslundii, Streptococcus mutans, Streptococcus salivarius, Streptococcus sanguis,* and *Bacillus cereus,* have been found to produce root caries in animal models. Quirynen et al[54] reported that when plaque levels and pocket depths decrease after periodontal therapy (both nonsurgical and surgical), a shift in oral bacteria occurs, leading to a reduction in periodontal pathogens and an increase in *S. mutans* and the development of root caries.

A prevalence rate study of root caries in 20- to 64-year-old individuals revealed that 42% had one or more root caries lesions and that these lesions tended to increase with age.[37]

The tooth may not be painful, but exploration of the root surface reveals the presence of a defect, and penetration of the involved area with a probe causes pain. Caries of the root may lead to pulpitis, sensitivity to sweets and thermal changes, or severe pain. Pathologic exposure of the pulp occurs in severe cases. Root caries may be the cause of toothache in patients with periodontal disease and no evidence of coronal decay.

Caries of the cementum requires special attention when the pocket is treated. The necrotic cementum must be removed by scaling and root planing until firm tooth surface is reached, even if this entails extension into the dentin.

Areas of cellular resorption of cementum and dentin are common in roots unexposed by periodontal disease.[68] These areas are of no particular significance because they are symptom free, and as long as the root is covered by the periodontal ligament, they are likely to undergo repair. However, if the root is exposed by progressive pocket formation before repair occurs, these areas appear as isolated cavitations that penetrate into the dentin. These areas can be differentiated from caries of the cementum by their clear-cut outline and hard surface. They may be sources of considerable pain, requiring placement of a restoration.

Surface Morphology of Tooth Wall.[78] The following zones can be found in the bottom of a periodontal pocket (Figure 13-17):

1. *Cementum covered by calculus,* in which all the changes described in the preceding paragraphs can be found.
2. *Attached plaque,* which covers calculus and extends apically from it to a variable degree, probably 100 to 500 µm.
3. The zone of *unattached plaque* that surrounds attached plaque and extends apically to it.
4. The zone of *attachment of the junctional epithelium to the tooth.* The extension of this zone, which in normal sulci is more than 500 µm, is usually reduced in periodontal pockets to less than 100 µm.
5. A zone of *semidestroyed connective tissue fibers* may be apical to the junctional epithelium[60] (see section on Pathogenesis).

Zones 3, 4, and 5 make up the "plaque-free zone" seen in extracted teeth.[8,12,36,57,78] The total width of the plaque-free zone varies according to the type of tooth (wider in molars than in incisors) and the depth of the pocket (narrower in deeper pockets).[56] It is important to remember that the term *plaque-free zone* refers only to attached plaque because unattached plaque contains a variety of gram-positive and gram-negative morphotypes, including cocci, rods, filaments, fusiforms, and spirochetes. The most apical zone contains predominantly gram-negative rods and cocci.[76]

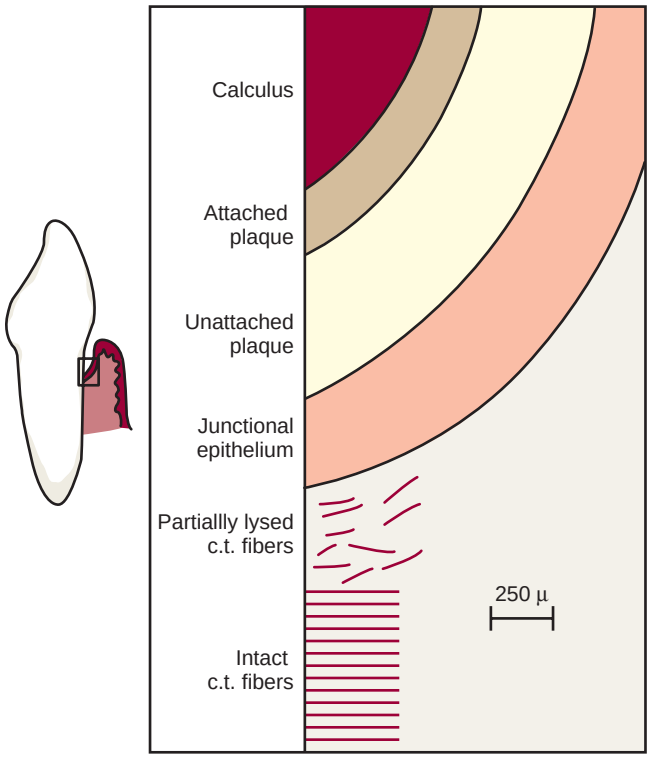

Figure 13-17 Diagram of the area at the bottom of a pocket; *c.t.,* connective tissue.

PERIODONTAL DISEASE ACTIVITY

For many years the loss of attachment produced by periodontal disease was thought to be a slow but continuously progressive phenomenon. More recently, as a result of studies on the specificity of plaque bacteria, the concept of periodontal disease activity has evolved.

According to this concept, periodontal pockets go through periods of exacerbation and quiescence, resulting from episodic bursts of activity followed by periods of remission. *Periods of quiescence* are characterized by a reduced inflammatory response and little or no loss of bone and connective tissue attachment. A buildup of unattached plaque, with its gram-negative, motile, and anaerobic bacteria (see Chapter 23), starts a *period of exacerbation* in which bone and connective tissue attachment are lost and the pocket deepens. This period may last for days, weeks, or months and is eventually followed by a period of remission or quiescence in which gram-positive bacteria proliferate and a more stable condition is established. Based on a study of radioiodine^{125}I absorptiometry, McHenry et al[45] confirmed that bone loss in untreated periodontal disease occurs in an episodic manner.

These periods of quiescence and exacerbation are also known as *periods of inactivity* and *periods of activity.* Clinically, active periods show bleeding, either spontaneously or with probing, and greater amounts of gingival exudate. Histologically, the pocket epithelium appears thin and ulcerated, and an infiltrate composed predominantly of plasma cells,[19] PMNs,[53] or both are seen. Bacterial samples

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from the pocket lumen analyzed with dark-field microscopy show high proportions of motile organisms and spirochetes.[43]

Methods to detect periods of activity or inactivity are currently being investigated (see online).

SITE SPECIFICITY

Periodontal destruction does not occur in all parts of the mouth at the same time but rather on a few teeth at a time or even only some aspects of some teeth at any given time. This is referred to as the *site specificity* of periodontal disease. Sites of periodontal destruction are often found next to sites with little or no destruction. Therefore the severity of periodontitis increases with the development of new disease sites and with the increased breakdown of existing sites.

PULP CHANGES ASSOCIATED WITH PERIODONTAL POCKETS

The spread of infection from periodontal pockets may cause pathologic changes in the pulp. Such changes may give rise to painful symptoms or may adversely affect the response of the pulp to restorative procedures. Involvement of the pulp in periodontal disease occurs through either the apical foramen or the lateral pulp canals after pocket infection reaches then. Atrophic and inflammatory pulpal changes occur in such cases (see Chapters 52 and 63).

RELATIONSHIP OF ATTACHMENT LOSS AND BONE LOSS TO POCKET DEPTH

The severity of the attachment loss in pocket formation is generally but not always correlated with the depth of the pocket. This is because the degree of attachment loss depends on the location of the base of the pocket on the root surface, whereas pocket depth is the distance between the base of the pocket and the crest of the gingival margin. Pockets of the same depth may be associated with different degrees of attachment loss (Figure 13-18), and pockets of different depths may be associated with the same amount of attachment loss (Figure 13-19).

Severity of bone loss is generally, but not always, correlated with pocket depth. Extensive attachment and bone loss may be associated with shallow pockets if the attachment loss is accompanied by recession of the gingival margin, and slight bone loss can occur with deep pockets.

AREA BETWEEN BASE OF POCKET AND ALVEOLAR BONE

Normally, the distance between the apical end of the junctional epithelium and the alveolar bone is relatively constant. The distance between the apical extent of calculus and the alveolar crest in human periodontal pockets is most constant, having a mean length of 1.97 mm (±33.16%).[71,77]

The distance from attached plaque to bone is never less than 0.5 mm and never more than 2.7 mm.[78-80] These findings suggest that the bone-resorbing activity induced by the bacteria is exerted within these distances. However, the finding of isolated bacteria or clumps of bacteria in the connective tissue[60] and on the bone surface[26] may modify these considerations.

RELATIONSHIP OF POCKET TO BONE

In infrabony pockets, the base of the pocket is apical to the crest of the alveolar bone, and the pocket wall lies between the tooth and the bone. The bone loss is in most cases vertical. In suprabony pockets on the other hand, the base is coronal to the crest of the alveolar bone and the pocket wall lies coronal to the bone. The type of bone loss is always horizontal.

This creates some microscopic differences that have some therapeutic importance. They are the relationship of the soft tissue wall of the pocket to the alveolar bone, the pattern of bone destruction, and the direction of the transseptal fibers of the periodontal ligament[15] (Figures 13-20 to 13-22).

In suprabony pockets, the alveolar crest gradually attains a more apical position in relation to the tooth but retains its general morphology and architecture. The interdental fibers that run over the bone from one tooth to the other maintain their usual horizontal direction. In infrabony pockets, the morphology of the alveolar crest changes completely, with the formation of an angular bony defect. The interdental fibers in this case run over the bone in an oblique direction between the two teeth of the interdental space.[15,80] This may affect the function of the area and also necessitate a

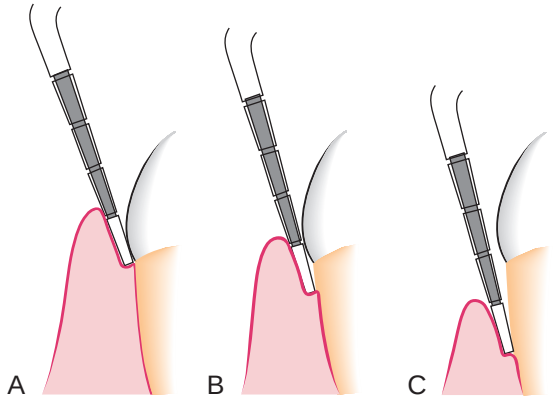

Figure 13-18 Same pocket depth with different amounts of recession. **A,** Gingival pocket with no recession. **B,** Periodontal pocket of similar depth as in **A** but with some degree of recession. **C,** Pocket depth same as in A and **B** but with still more recession.

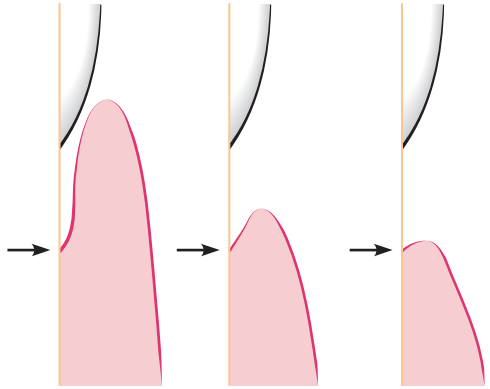

Figure 13-19 Different pocket depths with the same amount of attachment loss. Arrows point to bottom of the pocket. The distance between the arrow and the cementoenamel junctions remains the same despite different pocket depths.

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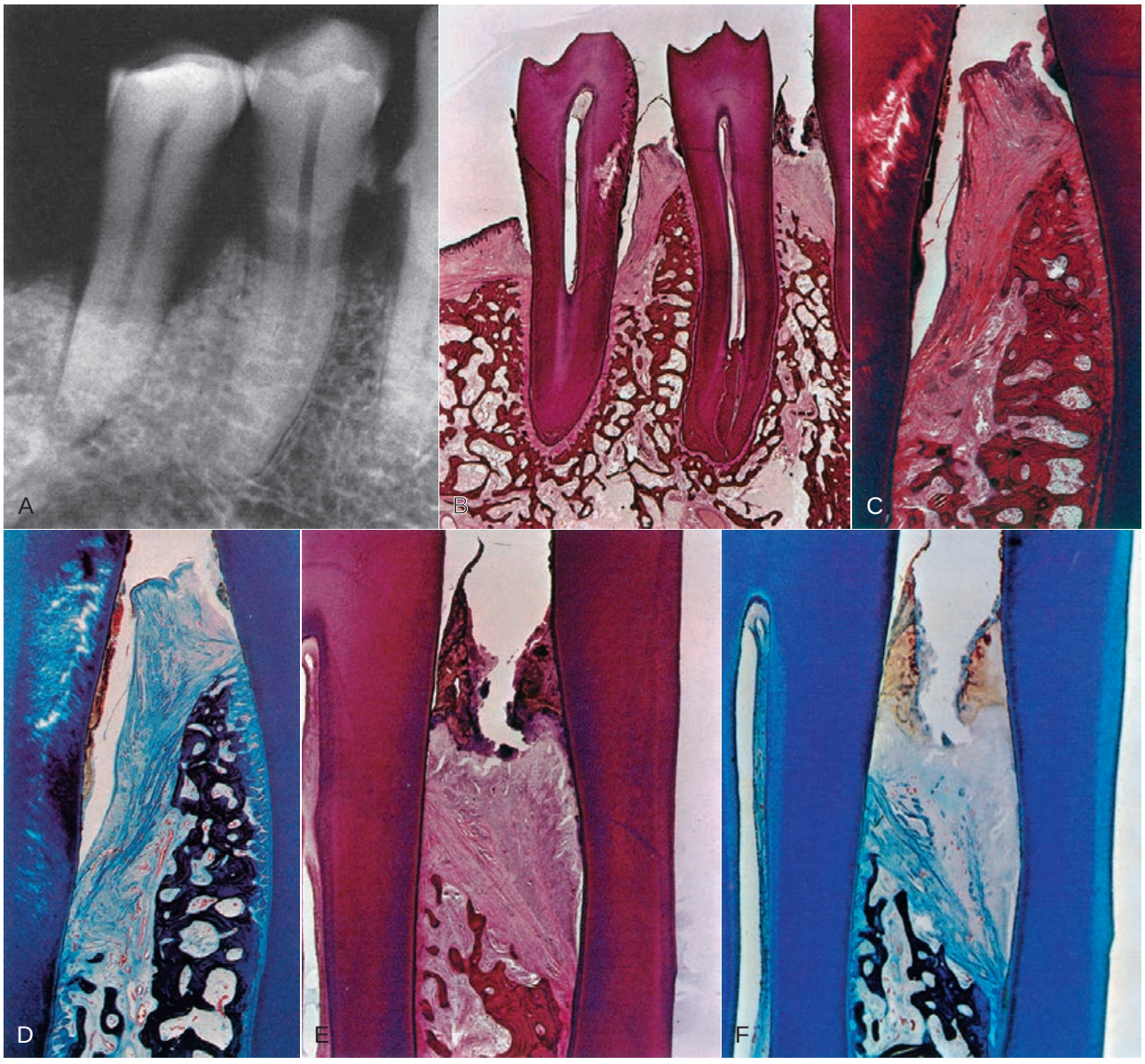

Figure 13-20 Radiographic and microscopic features of intrabony pockets. **A,** Radiograph of mandibular canine and premolar area showing angular bone loss mesial to second premolar. Type of bone loss between first premolar and canine is not radiographically apparent. **B,** Mesiodistal histologic section of teeth seen in **A** showing intrabony pocket mesial to second premolar, as well as suprabony pockets distal to second premolar and mesial and distal to first premolar. The mesial suprabony pocket of first premolar appears coronal to a vertical bone loss. **C,** Higher-power view of area between the premolars. Note the angular bone loss and transseptal fibers covering the bone. **D,** Higher-power view of area between the premolars stained with Mallory connective tissue stain, clearly showing the direction of transseptal fibers. **E,** Higher-power view of area between first premolar and canine. Note the abundant calculus, dense leukocytic infiltration of the gingiva, and angulation of transseptal fibers and bone. The pocket is still suprabony. **F,** Mallory stain of similar area as in **E.** Note destruction of the gingival fibers caused by inflammation and the angular fibers formed over angular bone loss and less affected by the inflammation. Transseptal fibers extend from distal surface of premolar over crest of alveolar bone into the intrabony pocket. Note the leukocytic infiltration of the transseptal fibers. *(From Glickman I, Smulow J:* Periodontal disease: clinical, radiographic and histopathologic features, *Philadelphia, 1974, Saunders.)*

modification in treatment techniques (see Chapters 60 and 62).[13,15] Table 13-2 summarizes the distinguishing features of suprabony and infrabony pockets. The classification of infrabony pockets is discussed in Chapter 14.

PERIODONTAL ABSCESS

A periodontal abscess is a localized purulent inflammation in the periodontal tissues (Figure 13-23). It is also known as a *lateral abscess* or *parietal abscess.* Abscesses localized in the gingiva, caused by injury to the outer surface of the gingiva, and not involving the supporting structures are called *gingival abscesses.* Gingival abscesses may occur in the presence or absence of a periodontal pocket (see Chapter 9).

Periodontal abscess formation may occur in the following ways:

1. Extension of infection from a periodontal pocket deeply into the supporting periodontal tissues and localization of the suppurative inflammatory process along the lateral aspect of the root.
2. Lateral extension of inflammation from the inner surface of a periodontal pocket into the connective tissue of the pocket

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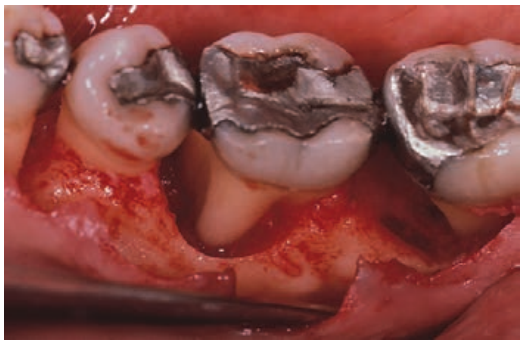

Figure 13-21 After raising a flap for the treatment of the infrabony pockets, the vertical bone loss around the mesial roots of the mandibular first and second molars can be seen.

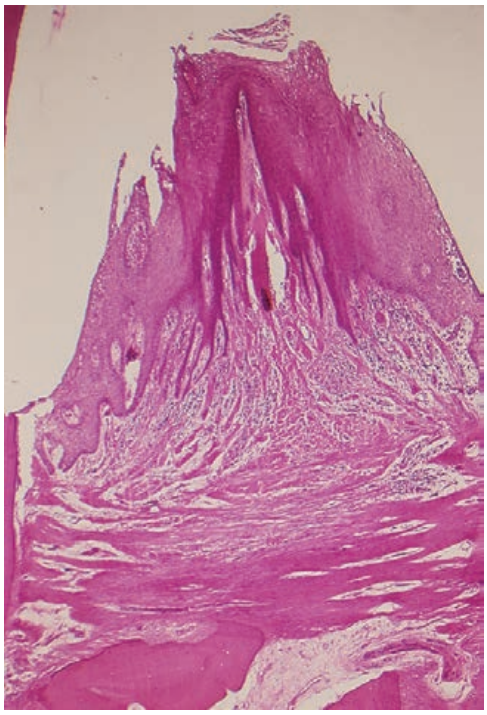

Figure 13-22 Two suprabony pockets in an interdental space. Note the normal horizontal arrangement of the transseptal fibers.

wall. Formation of the abscess results when drainage into the pocket space is impaired.

3. Formation in a pocket with a tortuous course around the root. A periodontal abscess may form in the cul-de-sac, the deep end of which is shut off from the surface.
4. Incomplete removal of calculus during treatment of a periodontal pocket. The gingival wall shrinks, occluding the pocket orifice, and a periodontal abscess occurs in the sealed-off portion of the pocket.
5. After trauma to the tooth or with perforation of the lateral wall of the root in endodontic therapy. In these situations, a periodontal abscess may occur in the absence of periodontal disease.

Periodontal abscesses are classified according to location as follows:

1. Abscess in the *supporting periodontal tissues* along the lateral aspect of the root. In this condition, a sinus generally occurs in the bone that extends laterally from the abscess to the external surface.
2. Abscess in the *soft tissue wall* of a deep periodontal pocket.

Figure 13-23 Periodontal abscess on an upper right central incisor.

TABLE 13-2 Distinguishing Features of Suprabony and Intrabony Periodontal Pockets

Suprabony Pocket	Intrabony Pocket
1. Base of pocket is coronal to level of alveolar bone.	1. Base of pocket is apical to crest of alveolar bone so that the bone is adjacent to soft tissue wall (see Figure 13-2).
2. Pattern of destruction of underlying bone is horizontal.	2. Pattern of bone destruction is vertical (angular) (see Figures 13-20 and 13-21).
3. Interproximally, transseptal fibers that are restored during progressive periodontal disease are arranged horizontally in the space between base of pocket and alveolar bone (see Figure 13-22).	3. Interproximally, transseptal fibers are oblique rather than horizontal. They extend from cementum beneath base of pocket along alveolar bone and over crest to cementum of adjacent tooth (see Figure 13-20).
4. On facial and lingual surfaces, periodontal ligament fibers beneath pocket follow their normal horizontal-oblique course between the tooth and bone.	4. On facial and lingual surfaces, periodontal ligament fibers follow angular pattern of adjacent bone. They extend from cementum beneath base of pocket along alveolar bone and over crest to join with outer periosteum.

Microscopically, an abscess is a localized accumulation of viable and nonviable PMNs within the periodontal pocket wall. The PMNs liberate enzymes that digest the cells and other tissue structures, forming the liquid product known as *pus,* which constitutes the center of the abscess. An acute inflammatory reaction surrounds the purulent area, and the overlying epithelium exhibits intracellular and extracellular edema and invasion of leukocytes (Figure 13-24).

The localized acute abscess becomes a chronic abscess when its purulent content drains through a fistula into the outer gingival surface or into the periodontal pocket and the infection causing the abscess is not resolved.

Bacterial invasion of tissues has been reported in abscesses; the invading organisms were identified as gram-negative cocci, diplococci, fusiforms, and spirochetes. Invasive fungi were also found and were interpreted as being "opportunistic invaders."[22] Microorganisms that colonize the periodontal abscess have been reported to be primarily gram-negative anaerobic rods.[51]

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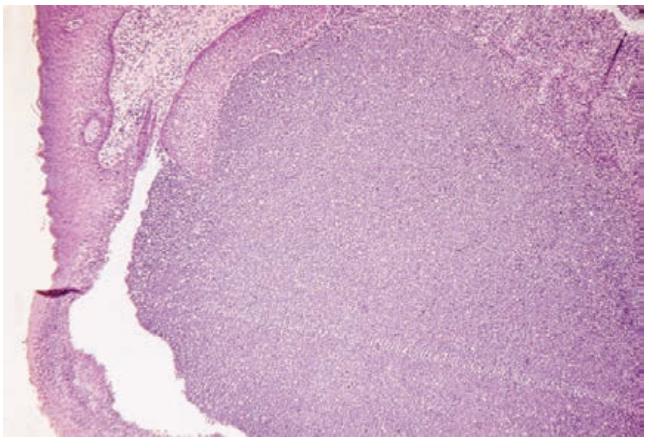

Figure 13-24 Microscopic view of a periodontal abscess showing dense accumulation of polymorphonuclear leukocytes (PMNs) covered by squamous epithelium.

LATERAL PERIODONTAL CYST

The periodontal cyst, also called *lateral periodontal cyst*, is an uncommon lesion that produces localized destruction of the periodontal tissues along a lateral root surface, most often in the mandibular canine-premolar area.[23,70] It is considered to be derived from rests of Malassez or other proliferating odontogenic rests.[69]

A periodontal cyst is usually asymptomatic, without grossly detectable changes, but it may present as a localized, tender swelling. Radiographically, an interproximal periodontal cyst appears on the side of the root as a radiolucent area bordered by a radiopaque line. Its radiographic appearance cannot be differentiated from that of a periodontal abscess.

Microscopically, the cystic lining may be (1) a loosely arranged, thin, nonkeratinized, epithelium, sometimes with thicker proliferating areas or (2) an odontogenic keratocyst.[23]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 14

Bone Loss and Patterns of Bone Destruction

Fermin A. Carranza, Paulo M. Camargo, and Henry H. Takei

CHAPTER OUTLINE

BONE DESTRUCTION CAUSED BY EXTENSION OF GINGIVAL INFLAMMATION
- Radius of Action
- Rate of Bone Loss
- Periods of Destruction
- Mechanisms of Bone Destruction
- Bone Formation in Periodontal Disease

BONE DESTRUCTION CAUSED BY TRAUMA FROM OCCLUSION

BONE DESTRUCTION CAUSED BY SYSTEMIC DISORDERS

FACTORS DETERMINING BONE MORPHOLOGY IN PERIODONTAL DISEASE
- Normal Variation in Alveolar Bone
- Exostoses
- Trauma from Occlusion
- Buttressing Bone Formation
- Food Impaction
- Aggressive Periodontitis

BONE DESTRUCTION PATTERNS IN PERIODONTAL DISEASE
- Horizontal Bone Loss
- Bone Deformities (Osseous Defects)
- Vertical or Angular Defects
- Osseous Craters
- Bulbous Bone Contours
- Reversed Architecture
- Ledges
- Furcation Involvement

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some text and figures may be out of numeric order in this printed chapter.

Although periodontitis is an infectious disease of the gingival tissue, changes that occur in bone are crucial because the destruction of bone is responsible for tooth loss.

The height and density of the alveolar bone are normally maintained by an equilibrium, regulated by local and systemic influences, between bone formation and bone resorption.[12,14] When resorption exceeds formation, both bone height and bone density may be reduced.

The level of bone is the consequence of past pathologic experiences, whereas changes in the soft tissue of the pocket wall reflect the present inflammatory condition. Therefore the degree of bone loss is not necessarily correlated with the depth of periodontal pockets, severity of ulceration of the pocket wall, or presence or absence of pus.

BONE DESTRUCTION CAUSED BY EXTENSION OF GINGIVAL INFLAMMATION

The most common cause of bone destruction in periodontal disease is the extension of inflammation from the marginal gingiva into the supporting periodontal tissues. The inflammatory invasion of the bone surface and the initial bone loss that follows mark the transition from gingivitis to periodontitis.

Periodontitis is always preceded by gingivitis, but not all gingivitis progress to periodontitis. Some cases of gingivitis apparently never become periodontitis, and other cases go through a brief gingivitis phase and rapidly develop into periodontitis. The factors that are responsible for the extension of inflammation to the supporting structures, thus initiating the conversion of gingivitis to periodontitis, are not clearly understood at this time.

The transition from gingivitis to periodontitis is associated with changes in the composition of bacterial plaque. In advanced stages of disease, the number of motile organisms and spirochetes increases, whereas the number of coccoid rods and straight rods decreases.[34] The cellular composition of the infiltrated connective tissue also changes with increasing severity of the lesion (see Chapter 7). Fibroblasts and lymphocytes predominate in stage I gingivitis, whereas the number of plasma cells and blast cells increases gradually as the disease progresses. Seymour et al[58,59] have postulated a stage of "contained" gingivitis in which T lymphocytes are preponderant; they believe that as the lesion becomes a B-lymphocyte lesion, it becomes progressively destructive.

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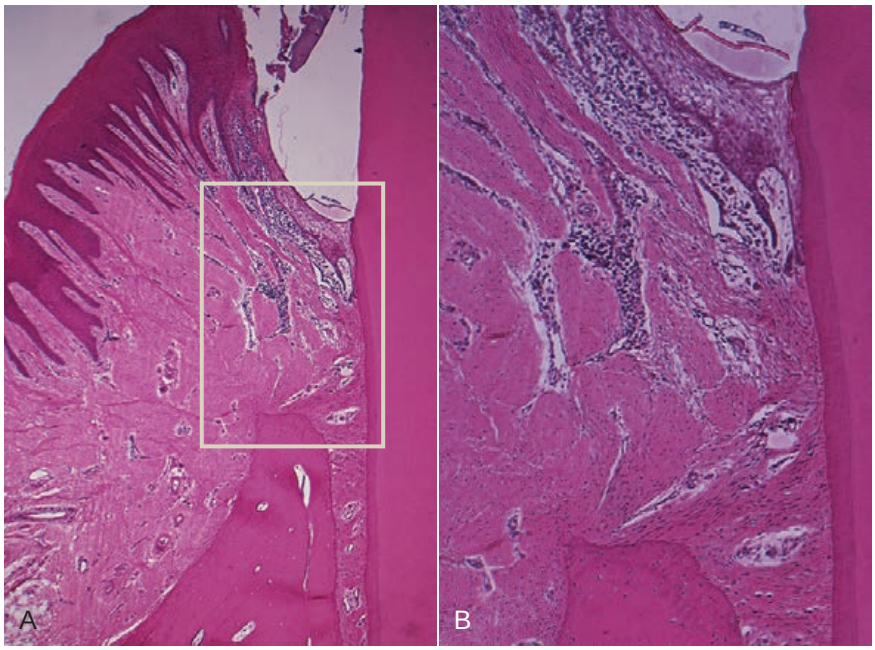

Figure 14-1 **A**, Area of inflammation extending from the gingiva into the suprabony area. **B,** Extension of inflammation along blood vessels and between collagen bundles.

Heijl and coworkers[22] were able to convert, in experimental animals, a confined, naturally occurring chronic gingivitis into a progressive periodontitis by placing a silk ligature into the sulcus and tying it around the neck of the tooth. This induced ulceration of the sulcular epithelium, a shift in the inflammatory infiltrate from predominantly plasma cells to predominantly polymorphonuclear leukocytes, and osteoclastic resorption of the alveolar crest. The recurrence of episodes of acute destruction over time may be one mechanism leading to progressive bone loss in marginal periodontitis.

The extension of inflammation to the supporting structures of a tooth may be modified by the pathogenic potential of plaque and the resistance of the host. The latter includes immunologic activity and other tissue-related mechanisms, such as the degree of fibrosis of the gingiva, probably the width of the attached gingiva, and the reactive fibrogenesis and osteogenesis, that occur peripheral to the inflammatory lesion.[52]

Histopathology Gingival inflammation extends along the collagen fiber bundles and follows the course of the blood vessels through the loosely arranged tissues around them into the alveolar bone[67] (Figure 14-1). Although the inflammatory infiltrate is concentrated in the marginal periodontium, the reaction is a much more diffuse one, often reaching the bone and eliciting a response before evidence of crestal resorption or loss of attachment exists.[41] In the upper molar region, inflammation can extend to the maxillary sinus, resulting in thickening of the sinus mucosa.[40]

Interproximally, inflammation spreads to the loose connective tissue around the blood vessels, through the fibers, and then into the bone through vessel channels that perforate the crest of the interdental septum at the center of the crest (Figure 14-2), toward the side of the crest (Figure 14-3), or at the angle of the septum. Also, inflammation may enter the bone through more than one channel. Less frequently, the inflammation spreads from the gingiva directly into the periodontal ligament and from there into the interdental septum[1] (Figure 14-4).

Figure 14-2 Inflammation extending from the pocket area *(top)* between the collagen fibers, which are partially destroyed.

Facially and lingually, inflammation from the gingiva spreads along the outer periosteal surface of the bone (see Figure 14-4) and penetrates into the marrow spaces through vessel channels in the outer cortex.

Along its course from the gingiva to the bone, the inflammation destroys the gingival and transseptal fibers, reducing them to disorganized granular fragments interspersed among the inflammatory cells and edema.[45] However, there is a continuous tendency to recreate transseptal fibers across the crest of the interdental septum farther along the root as the bone destruction progresses (Figure 14-5). As a result, transseptal fibers are present, even in cases of extreme periodontal bone loss.

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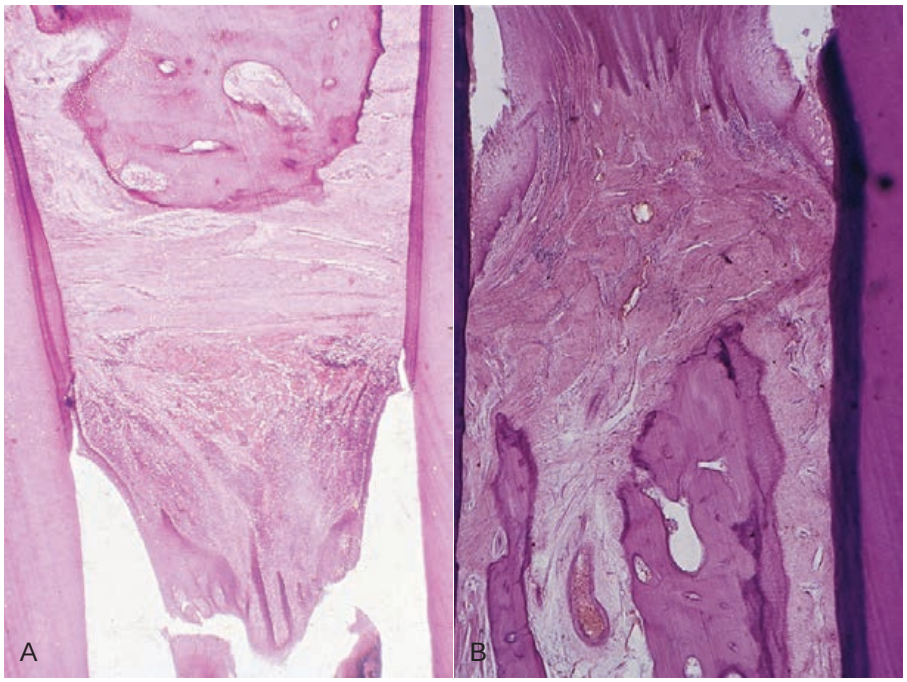

Figure 14-3 **A,** Extension of inflammation into the center of the interdental septum. Inflammation from the gingiva penetrates the transseptal fibers and enters the bone around blood vessel in the center of the septum. **B,** Cortical layer at the top of the septum has been destroyed, and the inflammation penetrates into the marrow spaces.

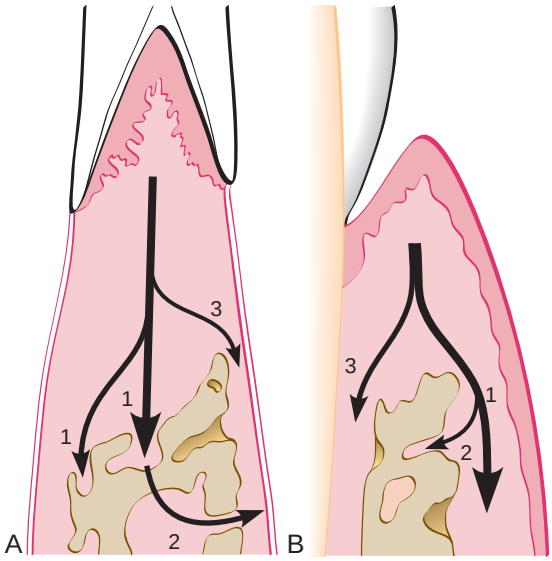

Figure 14-4 Pathways of inflammation from the gingiva into the supporting periodontal tissues in periodontitis. **A,** Interproximally, from the gingiva into the bone *(1),* from the bone into the periodontal ligament *(2),* and from the gingiva into the periodontal ligament *(3).* **B,** Facially and lingually, from the gingiva along the outer periosteum *(1),* from the periosteum into the bone *(2),* and from the gingiva into the periodontal ligament *(3).*

Figure 14-5 Re-formation of transseptal fibers. Mesiodistal section through interdental septum showing gingival inflammation and bone loss. Recreated transseptal fibers can be seen above the bone margin, partially infiltrated by the inflammatory process.

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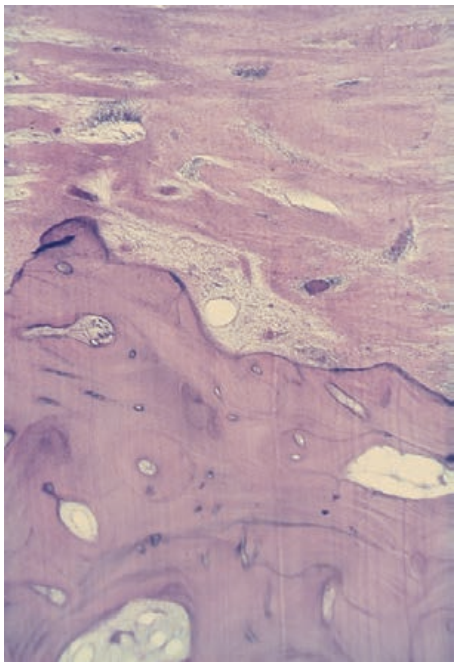

Figure 14-6 Extension of inflammation has reached the crestal bone surface.

Figure 14-7 Interdental septum in human autopsy section. Extensive inflammatory infiltrate has invaded the marrow spaces, entering from both the mesial and the distal aspect. Fatty bone marrow has been replaced by inflammatory cells and fibrous marrow.

The dense transseptal fibers form a firm covering over the bone, which is encountered during periodontal flap surgery after the superficial granulation tissue is removed.[50]

After inflammation reaches the bone by extension from the gingiva (Figure 14-6), it spreads into the marrow spaces and replaces the marrow with a leukocytic and fluid exudate, new blood vessels, and proliferating fibroblasts (Figure 14-7). Multinuclear osteoclasts and mononuclear phagocytes increase in number, and the bone surfaces appear lined with Howship lacunae (Figure 14-8).

In the marrow spaces, resorption proceeds from within, causing a thinning of the surrounding bony trabeculae and enlargement of the marrow spaces, followed by destruction of the bone and a reduction in bone height. Normally, fatty bone marrow is partially or totally replaced by a fibrous type of marrow in the vicinity of the resorption.

Bone destruction in periodontal disease is not a process of bone necrosis.[27] It involves the activity of living cells along viable bone. When tissue necrosis and pus are present in periodontal disease, they occur in the soft tissue walls of periodontal pockets, not along the resorbing margin of the underlying bone.

The amount of inflammatory infiltrate correlates with the degree of bone loss but not with the number of osteoclasts. However, the distance from the apical border of the inflammatory infiltrate to the alveolar bone crest correlates with both the number of osteoclasts on the alveolar crest and the total number of osteoclasts.[51] Similar findings have been reported in experimentally induced periodontitis in animals.[32]

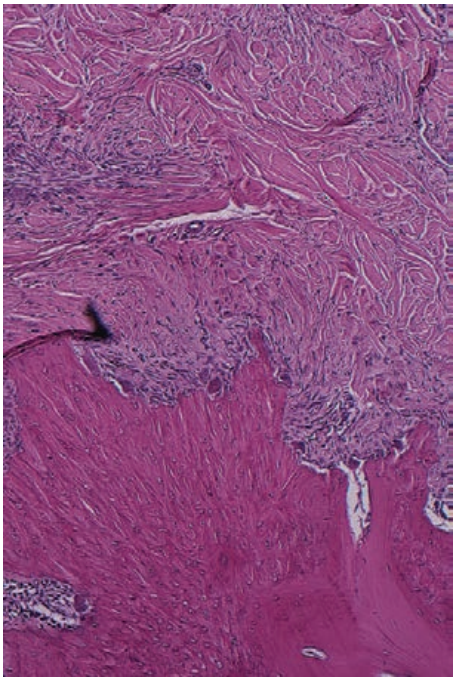

Figure 14-8 Osteoclasts and Howship lacunae in the resorption of crestal bone.

Radius of Action

Garant and Cho[10] suggested that locally produced bone resorption factors may need to be present in the proximity of the bone surface to exert their action. Page and Schroeder,[49] on the basis of Waerhaug's measurements made on human autopsy specimens,[64,65] postulated a range of effectiveness of about 1.5 to 2.5 mm in which bacterial plaque can induce loss of bone. Beyond 2.5 mm there is no effect; interproximal angular defects can appear only in spaces that are wider than 2.5 mm because narrower spaces would be

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destroyed entirely. Tal[61] corroborated this with measurements in human patients.

Large defects greatly exceeding a distance of 2.5 mm from the tooth surface (as described in aggressive types of periodontitis) may be caused by the presence of bacteria in the tissues.[6,10,54]

Rate of Bone Loss

In a study of Sri Lankan tea laborers with no oral hygiene and no dental care, Löe et al[36] found the rate of bone loss to average about 0.2 mm a year for facial surfaces and about 0.3 mm a year for proximal surfaces when periodontal disease was allowed to progress untreated. However, the rate of bone loss may vary, depending on the type of disease present. Löe et al[35] identified the following three subgroups of patients with periodontal disease based on interproximal loss of attachment* and tooth mortality:

1. Approximately 8% of persons had rapid progression of periodontal disease, characterized by a yearly loss of attachment of 0.1 to 1.0 mm.
2. Approximately 81% of individuals had moderately progressive periodontal disease, with a yearly loss of attachment of 0.05 to 0.5 mm.
3. The remaining 11% of persons had minimal or no progression of destructive disease (0.05 to 0.09 mm yearly).

Periods of Destruction

Periodontal destruction occurs in an episodic, intermittent manner, with periods of inactivity or quiescence, and destructive periods that result in loss of collagen and alveolar bone and deepening of the periodontal pocket.

Periods of destructive activity are associated with subgingival ulceration and an acute inflammatory reaction, resulting in rapid loss of alveolar bone[49,56]; it was hypothesized that this coincides with the conversion of a predominantly T-lymphocyte lesion to one with a predominantly B-lymphocyte–plasma cell infiltrate.[59] Microbiologically they are associated with an increase of the loose, unattached, motile, gram-negative, anaerobic pocket flora, while periods of remission coincide with the formation of a dense, unattached, nonmotile, gram-positive flora with a tendency to mineralize.[43]

It has also been suggested that the onset of periods of destruction coincide with tissue invasion by one or several bacterial species and is followed by an advanced local host defense that controls the attack.[54]

Mechanisms of Bone Destruction

The factors involved in bone destruction in periodontal disease are bacterial and host mediated. Bacterial plaque products induce the differentiation of bone progenitor cells into osteoclasts and stimulate gingival cells to release mediators that have the same effect.[21,57] Plaque products and inflammatory mediators can also act directly on osteoblasts or their progenitors, inhibiting their action and reducing their numbers.

In addition, in rapidly progressing diseases such as aggressive periodontitis, bacterial microcolonies or single bacterial cells have been found between collagen fibers and over the bone surface, suggesting a direct effect.[6,9,57]

Several host factors released by inflammatory cells are capable of inducing bone resorption in vitro and play a role in periodontal disease. These include host-produced prostaglandins and their precursors, interleukin-1α (IL-1α) and IL-β, and tumor necrosis factor alpha (TNF-α).

When injected intradermally, prostaglandin E_2 (PGE_2) induces the vascular changes seen in inflammation; when injected over a bone surface, PGE_2 induces bone resorption in the absence of inflammatory cells and with few multinucleated osteoclasts.[18,26] In addition, nonsteroidal antiinflammatory drugs (NSAIDs), such as flurbiprofen and ibuprofen, inhibit PGE_2 production, slowing bone loss in naturally occurring periodontal disease in beagle dogs and humans. This effect occurs without changes in gingival inflammation and rebounds 6 months after cessation of drug administration.[25,68] (For more information on the host-mediated mechanisms of bone destruction, see Chapter 25.)

Bone Formation in Periodontal Disease

Areas of bone formation are also found immediately adjacent to sites of active bone resorption and along trabecular surfaces at a distance from the inflammation, in an apparent effort to reinforce the remaining bone (buttressing bone formation). This osteogenic response is clearly found in experimentally produced periodontal bone loss in animals.[7] In humans, it is less obvious but has been confirmed by histometric[4,5] and histologic studies.[13]

Autopsy specimens from individuals with untreated disease occasionally show areas in which bone resorption has ceased and new bone is being formed on previously eroded bone margins. This confirms the intermittent character of bone resorption in periodontal disease and is consistent with the varied rates of progression observed clinically in untreated periodontal disease.

The periods of remission and exacerbation (or inactivity and activity, respectively) appear to coincide with the quiescence or exacerbation of gingival inflammation, manifested by changes in the extent of bleeding, amount of exudate, and composition of bacterial plaque (see Chapter 23).

The presence of bone formation in response to inflammation, even in active periodontal disease, has an effect on the outcome of treatment. The basic aim of periodontal therapy is the elimination of inflammation to remove the stimulus for bone resorption and therefore allow the inherent constructive tendencies to predominate.

BONE DESTRUCTION CAUSED BY TRAUMA FROM OCCLUSION

Another cause of bone destruction in periodontal disease is trauma from occlusion, which can occur in the absence or presence of inflammation (see Chapter 15).

In the absence of inflammation, the changes caused by trauma from occlusion vary from increased compression and tension of the periodontal ligament and increased osteoclasis of alveolar bone to necrosis of the periodontal ligament and bone and the resorption of bone and tooth structure. These changes are reversible in that they can be repaired if the offending forces are removed. However, persistent trauma from occlusion results in funnel-shaped widening of the crestal portion of the periodontal ligament, with resorption of the adjacent bone.[33] These changes, which may cause the bony crest to have an angular shape, represent adaptation of the periodontal tissues aimed at "cushioning" increased occlusal forces, but the modified bone shape may weaken tooth support and cause tooth mobility.

When combined with inflammation, trauma from occlusion aggravates the bone destruction caused by the inflammation[33] and results in bizarre bone patterns.

*Loss of attachment can be equated with loss of bone, although attachment loss precedes bone loss by about 6 to 8 months.[17]

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BONE DESTRUCTION CAUSED BY SYSTEMIC DISORDERS

Local and systemic factors regulate the physiologic equilibrium of bone. When a generalized tendency toward bone resorption exists, bone loss initiated by local inflammatory processes may be magnified.

This systemic influence on the response of alveolar bone, as envisioned by Glickman[12] in the early 1950s, considers a systemic regulatory influence in all cases of periodontal disease. In addition to the virulence of plaque bacteria, the nature of the systemic component, not its presence or absence, influences the severity of periodontal destruction. This concept of a role played by systemic defense mechanisms has been validated by the studies on immune deficiencies and host modulation in severely destructive types of periodontitis (see Chapters 25 and 37).

In recent years, interest has increased in the possible relationship between periodontal bone loss and *osteoporosis*.[11] Osteoporosis is a physiologic condition of postmenopausal women, resulting in loss of bone mineral content and structural bone changes. Periodontitis and osteoporosis share a number of risk factors (e.g., aging, smoking, diseases, or medications that interfere with healing). Some studies show a relationship between skeletal density and oral bone density and between crestal height and residual ridge resorption, as well as a relationship between osteopenia and periodontitis, tooth mobility, and tooth loss[19,23,24,55,63] (see Chapters 18 and 38).

Periodontal bone loss may also occur in generalized skeletal disturbances (e.g., hyperparathyroidism, leukemia, or histiocytosis X) by mechanisms that may be totally unrelated to the usual periodontal problem.

FACTORS DETERMINING BONE MORPHOLOGY IN PERIODONTAL DISEASE

Normal Variation in Alveolar Bone

Considerable normal variation exists in the morphologic features of alveolar bone (see Chapter 2), which affect the osseous contours produced by periodontal disease. The anatomic features that substantially affect the bone-destructive pattern in periodontal disease include the following:

- Thickness, width, and crestal angulation of the interdental septa
- Thickness of the facial and lingual alveolar plate
- Presence of fenestrations and dehiscences
- Alignment of the teeth
- Root and root trunk anatomy
- Root position within the alveolar process
- Proximity with another tooth surface

For example, angular osseous defects cannot form in thin facial or lingual alveolar plates, which have little or no cancellous bone between the outer and inner cortical layers. In such cases the entire crest of the plate is destroyed, and the height of the bone is reduced (see Figure 14-9).

Figure 14-9 can be found on the Expert CONSULT companion website at www.expertconsult.com.

Exostoses

Exostoses are outgrowths of bone of varied size and shape. Palatal exostoses have been found in 40% of human skulls.[46] They can occur as small nodules, large nodules, sharp ridges, spikelike projections, or any combination of these (Figure 14-10).[42] Exostoses have been described in rare cases as developing after the placement of free gingival grafts.[46]

Trauma from Occlusion

Trauma from occlusion may be a factor in determining the dimension and shape of bone deformities. It may cause a thickening of the cervical margin of alveolar bone or a change in bone morphology (e.g., angular defects, buttressing bone) on which inflammatory changes will later be superimposed.

Buttressing Bone Formation

Bone formation sometimes occurs in an attempt to buttress bony trabeculae weakened by resorption. When it occurs within the jaw, it is termed *central buttressing bone formation*. When it occurs on the external surface, it is referred to as *peripheral buttressing bone formation*.[13] The latter may cause bulging of the bone contour, which sometimes accompanies the production of osseous craters and angular defects (Figure 14-11).

Food Impaction

Interdental bone defects often occur where proximal contact is abnormal or absent. Pressure and irritation from food impaction contribute to the inverted bone architecture. In some cases, the poor proximal relationship may result from a shift in tooth position because of extensive bone destruction preceding food impaction. In such patients, food impaction is a complicating factor rather than the cause of the bone defect.

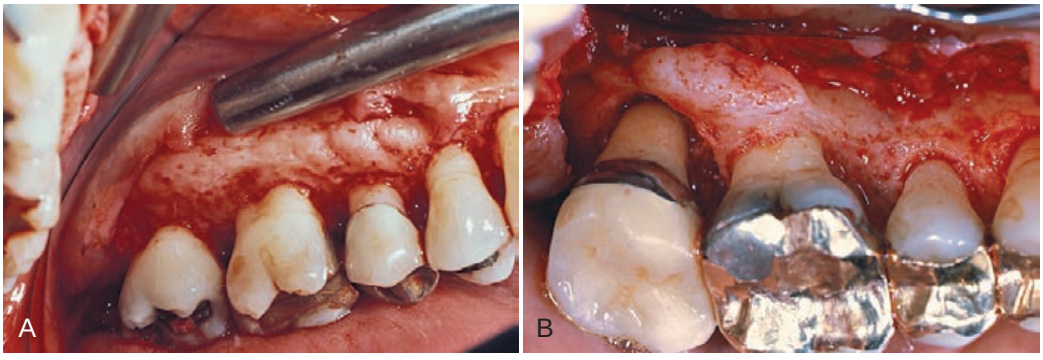

Figure 14-10 **A,** Exostosis in the facial aspect of upper second premolar and molars. **B,** Exostosis in the palatal aspect of first and second molars. Note also the circumferential defect in the second molar *(left)*.

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Aggressive Periodontitis

A vertical or angular pattern of alveolar bone destruction is found around the first molars in aggressive periodontitis. The cause of the localized bone destruction in this type of periodontal disease is unknown (see Chapter 18).

BONE DESTRUCTION PATTERNS IN PERIODONTAL DISEASE

Periodontal disease alters the morphologic features of the bone in addition to reducing bone height. An understanding of the nature and pathogenesis of these alterations is essential for effective diagnosis and treatment.[48]

Horizontal Bone Loss

Horizontal bone loss is the most common pattern of bone loss in periodontal disease. The bone is reduced in height, but the bone margin remains approximately perpendicular to the tooth surface. The interdental septa and facial and lingual plates are affected, but not necessarily to an equal degree around the same tooth (Figure 14-12, *A*).

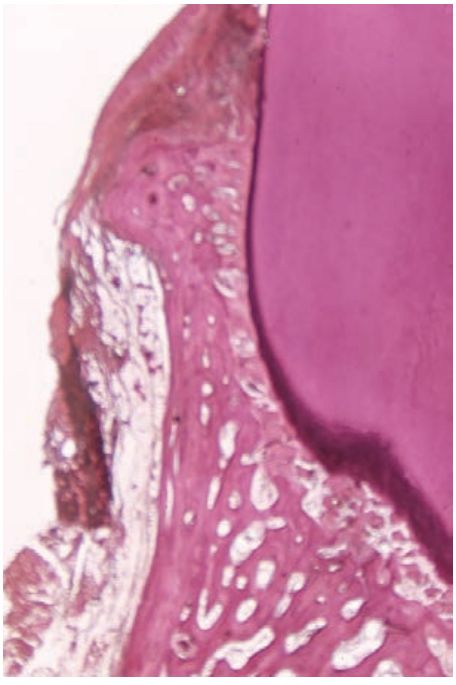

Figure 14-11 Lipping of facial bone. Peripheral buttressing bone formation along external surface of the facial bony plate and at the crest. Note the deformity in the bone produced by the buttressing bone formation and the bulging of the mucosa.

Bone Deformities (Osseous Defects)

Different types of bone deformities can result from periodontal disease. These usually occur in adults but have also been reported in human skulls with deciduous dentitions.[28] Their presence may be suggested on radiographs, but careful probing and surgical exposure of the areas are required to determine their exact conformation and dimensions.

Vertical or Angular Defects

Vertical or angular defects are those that occur in an oblique direction, leaving a hollowed-out trough in the bone alongside the root; the base of the defect is located apical to the surrounding bone (Figures 14-12, *B*; 14-13; and 14-14). In most instances, angular defects have accompanying infrabony periodontal pockets; infrabony pockets, on the other hand always must have an underlying angular defect.

Angular defects were classified by Goldman and Cohen on the basis of the number of osseous walls.[16] Angular defects may have one, two, or three walls (Figures 14-15 to 14-18). The number of walls in the apical portion of the defect is often greater than that in its occlusal portion, in which case the term *combined osseous defect* is used (Figure 14-19).

Vertical defects occurring interdentally can generally be seen on the radiograph, although thick, bony plates sometimes may obscure them. Angular defects can also appear on facial and lingual or palatal surfaces, but these defects are not seen on radiographs. Surgical exposure is the only sure way to determine the presence and configuration of vertical osseous defects.

Vertical defects increase with age.[44,47,69] Approximately 60% of persons with interdental angular defects have only a single defect.[44] Vertical defects detected radiographically have been reported to appear most often on the distal[44] and mesial surfaces.[47] However, three-wall defects are more frequently found on the mesial surfaces of upper and lower molars.[29]

Osseous Craters

Osseous craters are concavities in the crest of the interdental bone confined within the facial and lingual walls (Figure 14-20). Craters have been found to make up about one-third (35.2%) of all defects and about two-thirds (62%) of all mandibular defects

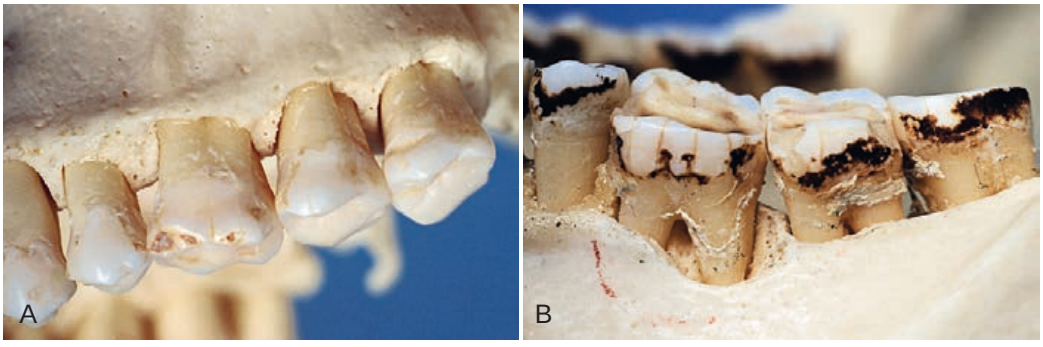

Figure 14-12 **A,** Horizontal bone loss. Note the reduction in height of the marginal bone, exposing cancellous bone and reaching the furcation of the second molar. **B,** Vertical (angular) bone loss on the distal root of the first molar.

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Figure 14-13 Angular (vertical) defects of different depths.

Figure 14-14 Angular defect on mesial surface of the first molar. Note also the furcation involvement.

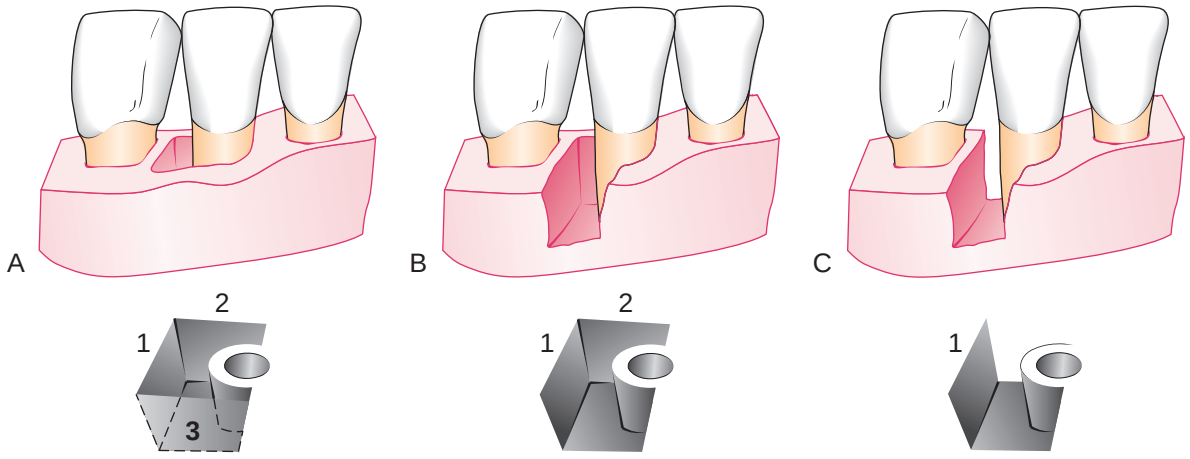

Figure 14-15 One-, two-, and three-walled vertical defects on right lateral incisor. **A,** Three bony walls: distal *(1),* lingual *(2),* and facial *(3).* **B,** Two-wall defect: distal *(1)* and lingual *(2).* **C,** One-wall defect: distal wall only *(1).*

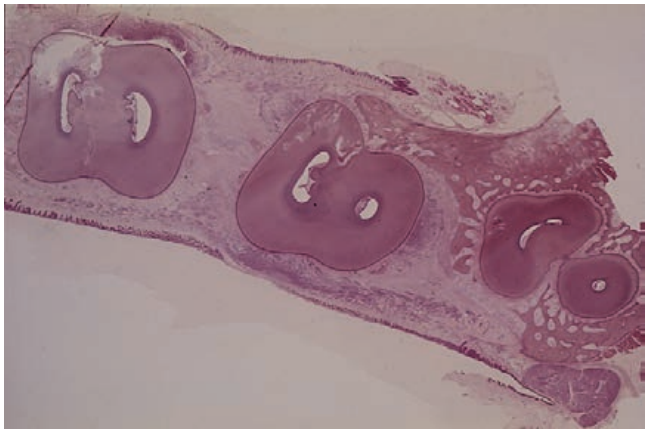

Figure 14-16 Horizontal section of lower molars at midroot level, showing a two-wall osseous defect distal to second molar.

Figure 14-17 One-wall vertical defect on mesial surface of lower first molar.

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Bone loss patterns associated with periodontal disease are varied, and the type of loss can be different in various areas in the same patient. Vertical bone loss is amenable to regenerative periodontal surgery using a variety of bone graft materials, bioactive molecules, and membranes. Often, more than one material may need to be used with larger wide defects to obtain good results. Horizontal bone loss and bone craters generally cannot be treated with regeneration, thus these lesions require flap surgery combined with osseous surgery.

Furcation bone loss is more difficult to treat than interproximal bone loss, and in advanced lesions of grade III furcation involvement, the prognosis may be so compromised that extraction and tooth replacement with dental implants should be done as soon as possible to maintain as much bone to support the implants.

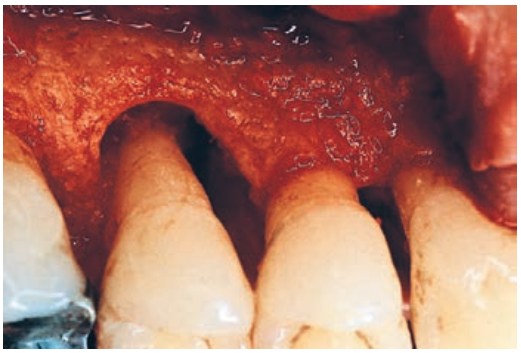

Figure 14-18 Circumferential vertical defect in relation to the upper premolar.

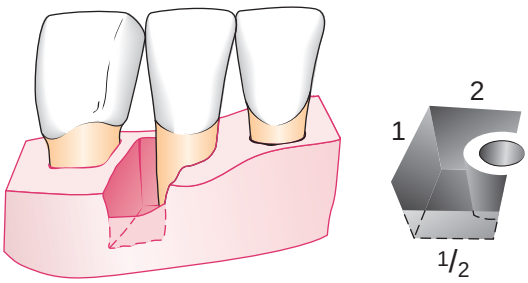

Figure 14-19 Combined type of osseous defect. Because the facial wall is half the height of the distal *(1)* and lingual *(2)* walls, this is an osseous defect with three walls in its apical half and two walls in the occlusal half.

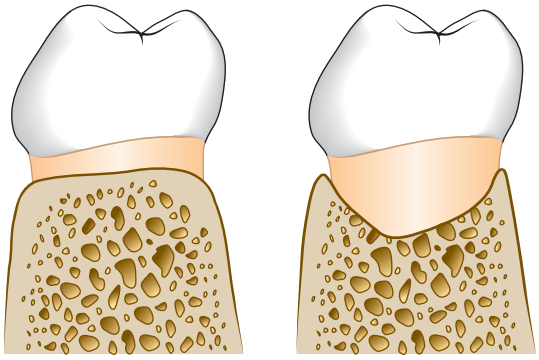

Figure 14-20 Diagrammatic representation of an osseous crater in a faciolingual section between two lower molars. *Left,* Normal bone contour. *Right,* Osseous crater.

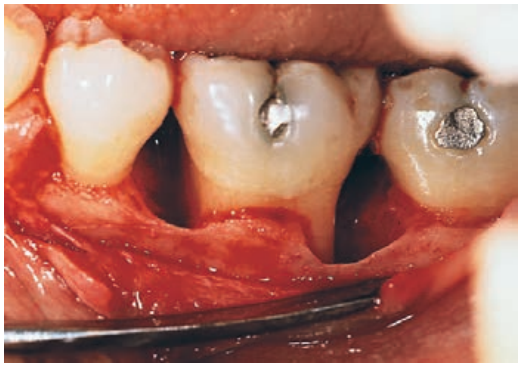

Figure 14-21 Reversed architecture. Elevated flap shows irregular bone margin.

and to occur twice as often in posterior segments as in anterior segments.[37,38]

The heights of the facial and lingual crests of a crater have been found to be identical in 85% of cases, with the remaining 15% being almost equally divided between higher facial crests and higher lingual crests.[53]

The following reasons for the high frequency of interdental craters have been suggested[37,38,53]:

- The interdental area collects plaque and is difficult to clean.
- The normal flat or even slightly concave faciolingual shape of the interdental septum in lower molars may favor crater formation.
- Vascular patterns from the gingiva to the center of the crest may provide a pathway for inflammation.

Bulbous Bone Contours

Bulbous bone contours are bony enlargements caused by exostoses (see Figure 14-10), adaptation to function, or buttressing bone formation. They are found more frequently in the maxilla than in the mandible.

Reversed Architecture

Reversed architecture defects are produced by loss of interdental bone, including the facial plates and lingual plates, without concomitant loss of radicular bone, thereby reversing the normal architecture (Figure 14-21). Such defects are more common in the maxilla.[44]

Ledges

Ledges are plateaulike bone margins caused by resorption of thickened bony plates (Figure 14-22).

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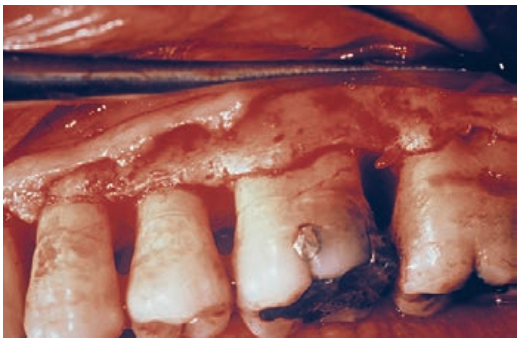

Figure 14-22 Ledge produced by interproximal resorption.

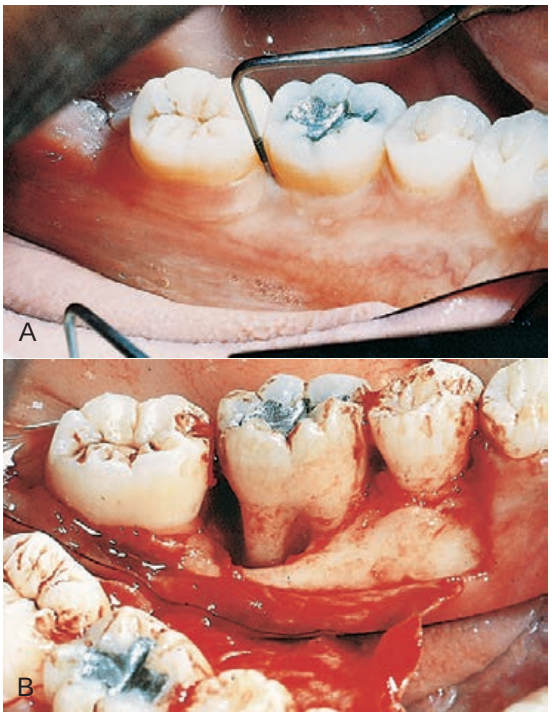

Figure 14-23 **A,** Molar with slightly inflamed gingiva clinically; however, it has a deep distal pocket. **B,** Flap elevation reveals extensive bone loss and furcation involvement. *(Courtesy Dr. Terry Fiori, Palo Alto, CA.)*

Furcation Involvement

The term *furcation involvement* refers to the invasion of the bifurcation and trifurcation of multirooted teeth by periodontal disease. The prevalence of furcation-involved molars is not clear.[8,47] Although some reports indicate that the mandibular first molars are the most common sites and the maxillary premolars are the least common,[30] other studies have found higher prevalence in upper molars.[69] The number of furcation involvements increases with age.[30,31]

The denuded furcation may be visible clinically or covered by the wall of the pocket. The extent of involvement is determined by exploration with a blunt probe, along with a simultaneous blast of warm air to facilitate visualization (Figure 14-23).

Furcation involvements have been classified as grades I, II, III, and IV according to the amount of tissue destruction. Grade I is incipient bone loss, grade II is partial bone loss (cul-de-sac), and grade III is total bone loss with through-and-through opening of the furcation. Grade IV is similar to grade III but with gingival recession exposing the furcation to view.

Figure 14-24 Different degrees of furcation involvement in human autopsy specimen. Furcation involvement is found in all three molars, with advanced lesion in the second molar and an extremely severe lesion in the first molar, exposing almost the entire mesial root.

Microscopically, furcation involvement presents no unique pathologic features. It is simply a phase in the rootward extension of the periodontal pocket. In its early stages, a widening of the periodontal space occurs, with cellular and fluid inflammatory exudation, followed by epithelial proliferation into the furcation area from an adjoining periodontal pocket. Extension of the inflammation into the bone leads to resorption and reduction in bone height. The bone-destructive pattern may produce horizontal loss, or angular osseous defects associated with intrabony pockets may exist (Figure 14-24). Plaque, calculus, and bacterial debris occupy the denuded furcation space.

The destructive pattern in a furcation involvement varies in different cases and with the degree of involvement. Bone loss around each individual root may be horizontal or angular, and frequently a crater develops in the interradicular area (Figure 14-25). Probing to determine the presence of these destructive patterns must be done horizontally and vertically around each involved root and in the crater area to establish the depth of the vertical component.

Furcation involvement is a stage of progressive periodontal disease and has its same etiology. The difficulty and sometimes the impossibility[2,3] of controlling plaque in furcations are responsible for the presence of extensive lesions in this area.[66]

The role of trauma from occlusion in the etiology of furcation lesions is controversial. Some assign a key role to trauma, believing that furcation areas are most sensitive to injury from excessive occlusal forces.[15] Others deny the initiating effect of trauma and consider that inflammation and edema caused by plaque in the furcation area tend to extrude the tooth, which then becomes traumatized and sensitive.[60,66]

Other factors that may play a role are the presence of enamel projections into the furcation,[39] which occurs in about 13% of multirooted teeth, and the proximity of the furcation to the cementoenamel junction, which occurs in about 75% of cases of furcation involvement.[31]

The presence of accessory pulpal canals in the furcation area may extend pulpal inflammation to the furcation.[20] This possibility should be carefully explored, particularly when mesial and distal bone retains its normal height. Accessory canals connecting the pulp chamber floor to the furcation have been found in 36% of

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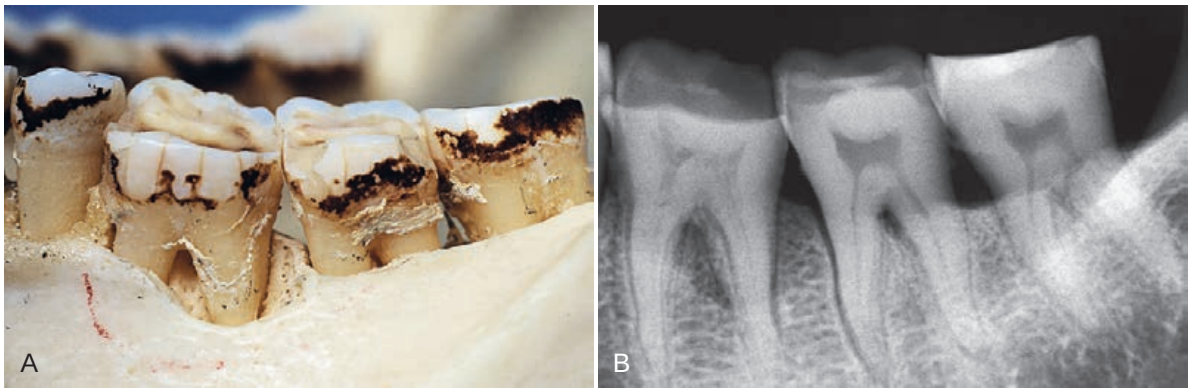

Figure 14-25 Photograph **(A)** and radiograph **(B)** of different degrees of bone loss in a skull. Furcation involvements in the first and second molar; deep angular bone loss in the distal root of the first molar; and interradicular and interdental craters in the second molar and between the second and third molars, respectively.

maxillary first molars, 12% of maxillary second molars, 32% of mandibular first molars, and 24% of mandibular second molars.[62]

The diagnosis of furcation involvement is made by clinical examination and careful probing with a specially designed probe (see Chapter 30). Radiographic examination of the area is helpful, but lesions can be obscured by angulation of the beam and the radiopacity of neighboring structures (see Chapter 31).

For more detailed clinical considerations in the diagnosis and treatment of furcation involvement, see Chapter 63.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 15

Periodontal Response to External Forces

Fermin A. Carranza

CHAPTER OUTLINE

ADAPTIVE CAPACITY OF THE PERIODONTIUM TO OCCLUSAL FORCES
TRAUMA FROM OCCLUSION
Acute and Chronic Trauma
Primary and Secondary Trauma from Occlusion
STAGES OF TISSUE RESPONSE TO INCREASED OCCLUSAL FORCES
Stage I: Injury
Stage II: Repair
Stage III: Adaptive Remodeling of the Periodontium
EFFECTS OF INSUFFICIENT OCCLUSAL FORCE
REVERSIBILITY OF TRAUMATIC LESIONS
EFFECTS OF EXCESSIVE OCCLUSAL FORCES ON DENTAL PULP
INFLUENCE OF TRAUMA FROM OCCLUSION ON PROGRESSION OF MARGINAL PERIODONTITIS
Clinical and Radiographic Signs of Trauma from Occlusion Alone
PATHOLOGIC TOOTH MIGRATION
Pathogenesis

ADAPTIVE CAPACITY OF THE PERIODONTIUM TO OCCLUSAL FORCES

The periodontium attempts to accommodate to the forces exerted on the crown. This adaptive capacity varies in different persons and in the same person at different times. The effect of occlusal forces on the periodontium is influenced by the magnitude, direction, duration, and frequency of the forces.

When the *magnitude* of occlusal forces is increased, the periodontium responds with a widening of the periodontal ligament space, an increase in the number and width of periodontal ligament fibers, and an increase in the density of alveolar bone.

Changing the *direction* of occlusal forces causes a reorientation of the stresses and strains within the periodontium (Figure 15-1).[24] The principal fibers of the periodontal ligament are arranged so that they best accommodate occlusal forces along the long axis of the tooth. *Lateral* (horizontal) and *torque* (rotational) forces are more likely to injure the periodontium.

The response of alveolar bone is also affected by the *duration* and *frequency* of occlusal forces. Constant pressure on the bone is more injurious than intermittent forces. The more frequent the application of an intermittent force, the more injurious is the force to the periodontium.

TRAUMA FROM OCCLUSION

An inherent "margin of safety" common to all tissues permits some variation in occlusion without adversely affecting the periodontium. However, when occlusal forces exceed the *adaptive capacity* of the tissues, tissue injury results.[44,45] The resultant injury is termed *trauma from occlusion*, also known as *traumatism* or *occlusal trauma*.

Thus trauma from occlusion refers to the *tissue injury* not the *occlusal force*. An occlusion that produces such an injury is called a *traumatic occlusion*.[2] Excessive occlusal forces may also disrupt the function of the masticatory musculature and cause painful spasms, injure the temporomandibular joints, or produce excessive tooth wear. However, the term *trauma from occlusion* is generally used in connection with injury in the periodontium.

Acute and Chronic Trauma

Trauma from occlusion may be acute or chronic. *Acute trauma* from occlusion results from an abrupt occlusal impact such as that produced by biting on a hard object (e.g., olive pit). Restorations or prosthetic appliances that interfere with or alter the direction of occlusal forces on the teeth may also induce acute trauma.

Acute trauma results in tooth pain, sensitivity to percussion, and increased tooth mobility. If the force is dissipated by a shift in the position of the tooth or by wearing away or correction of the restoration, the injury heals and the symptoms subside. Otherwise, periodontal injury may worsen and develop into necrosis, accompanied by periodontal abscess formation, or may persist as a symptom-free, chronic condition. Acute trauma can also produce cementum tears (Figure 15-2) (see Chapter 2).

Chronic trauma from occlusion is more common than the acute form and is of greater clinical significance. It most often develops from gradual changes in occlusion produced by tooth wear, drifting

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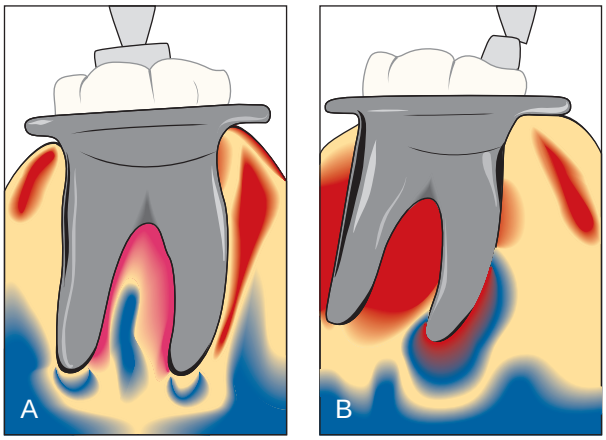

Figure 15-1 Stress patterns around the roots changed by shifting the direction of occlusal forces (experimental model using photoelastic analysis). **A,** Buccal view of an ivorine molar subjected to an axial force. The shaded fringes indicate that the internal stresses are at the root apices. **B,** Buccal view of ivorine molar subjected to a mesial tilting force. The shaded fringes indicate that the internal stresses are along the mesial surface and at the apex of the mesial root.

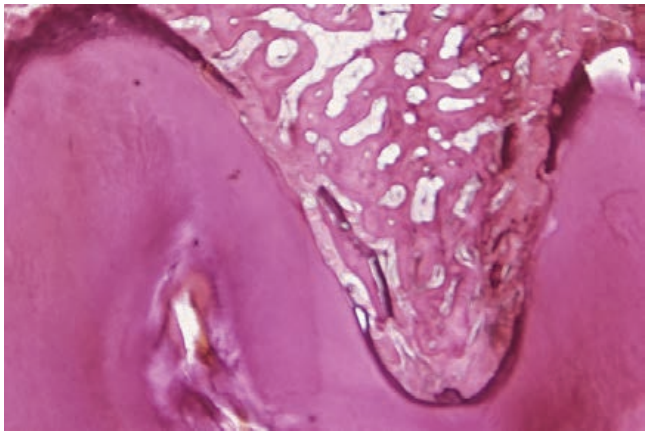

Figure 15-2 Cemental tear presumably caused by acute trauma from occlusion in human autopsy specimen. Note the repair process depositing bone on the displaced, torn cementum and recreating a periodontal ligament.

movement, and extrusion of teeth, combined with parafunctional habits, such as bruxism and clenching, rather than as a sequela of acute periodontal trauma (see Chapter 20). The features of chronic trauma from occlusion and their significance are discussed next.

The criterion that determines if an occlusion is traumatic is whether it produces periodontal injury, *not* how the teeth occlude. Any occlusion that produces periodontal injury is traumatic. Malocclusion is not necessary to produce trauma; periodontal injury may occur when the occlusion appears normal. The dentition may be anatomically and aesthetically acceptable but functionally injurious. Similarly, not all malocclusions are necessarily injurious to the periodontium. Traumatic occlusal relationships are referred to by such terms as *occlusal disharmony, functional imbalance,* and *occlusal dystrophy.* These terms refer to the effect of the occlusion on the periodontium, not to the position of the teeth. Because trauma

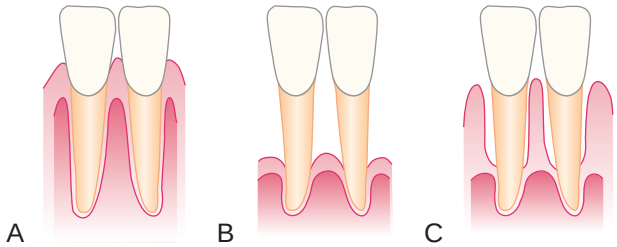

Figure 15-3 Traumatic forces can occur on **A,** normal periodontium with normal height of bone; **B,** normal periodontium with reduced height of bone; or **C,** marginal periodontitis with reduced height of bone.

from occlusion refers to the tissue injury rather than the occlusion, an increased occlusal force is not traumatic if the periodontium can accommodate it.

Primary and Secondary Trauma from Occlusion

Trauma from occlusion may be caused by alterations in occlusal forces, reduced capacity of the periodontium to withstand occlusal forces, or both. When trauma from occlusion is the result of alterations in occlusal forces, it is called *primary trauma from occlusion.* When it results from reduced ability of the tissues to resist the occlusal forces, it is known as *secondary trauma from occlusion.*

Primary trauma from occlusion occurs if trauma from occlusion is considered the primary etiologic factor in periodontal destruction and if the only local alteration to which a tooth is subjected is from occlusion. Examples include periodontal injury produced around teeth with a previously healthy periodontium after the (1) insertion of a "high filling," (2) insertion of a prosthetic replacement that creates excessive forces on abutment and antagonistic teeth, (3) drifting movement or extrusion of teeth into spaces created by unreplaced missing teeth, or (4) orthodontic movement of teeth into functionally unacceptable positions. Most studies on experimental animals of the effect of trauma from occlusion have examined the primary type of trauma. Changes produced by primary trauma do not alter the level of connective tissue attachment and do not initiate pocket formation. This is probably because the supracrestal gingival fibers are not affected and therefore prevent apical migration of the junctional epithelium.[49]

Secondary trauma from occlusion occurs when the adaptive capacity of the tissues to withstand occlusal forces is impaired by bone loss resulting from marginal inflammation. This reduces the periodontal attachment area and alters the leverage on the remaining tissues. The periodontium becomes more vulnerable to injury, and previously well tolerated occlusal forces become traumatic.

Figure 15-3 depicts three different situations on which excessive occlusal forces can be superimposed, as follows:

1. Normal periodontium with normal height of bone
2. Normal periodontium with reduced height of bone
3. Marginal periodontitis with reduced height of bone

The first case is an example of primary trauma from occlusion, whereas the last two represent secondary trauma from occlusion. The effects of trauma from occlusion in these different situations are analyzed in the following discussion.

It has been found in experimental animals that systemic disorders can reduce tissue resistance and that previously tolerable forces may become excessive.[22,52,62] This could theoretically represent another mechanism by which tissue resistance to increased forces is lowered, resulting in secondary trauma from occlusion.

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STAGES OF TISSUE RESPONSE TO INCREASED OCCLUSAL FORCES

Tissue response occurs in three stages[4,8]: injury, repair, and adaptive remodeling of the periodontium.

Stage I: Injury

Tissue injury is produced by excessive occlusal forces. The body then attempts to repair the injury and restore the periodontium. This can occur if the forces are diminished or if the tooth drifts away from them. If the offending force is chronic, however, the periodontium is remodeled to cushion its impact. The ligament is widened at the expense of the bone, resulting in angular bone defects without periodontal pockets, and the tooth becomes loose.

Under the forces of occlusion, a tooth rotates around a fulcrum or axis of rotation, which in single-rooted teeth is located in the junction between the middle third and the apical third of the clinical root and in multirooted teeth in the middle of the interradicular bone (Figure 15-4). This creates areas of pressure and tension on opposite sides of the fulcrum. Different lesions are produced by different degrees of pressure and tension. If jiggling forces are exerted, these different lesions may coexist in the same area.

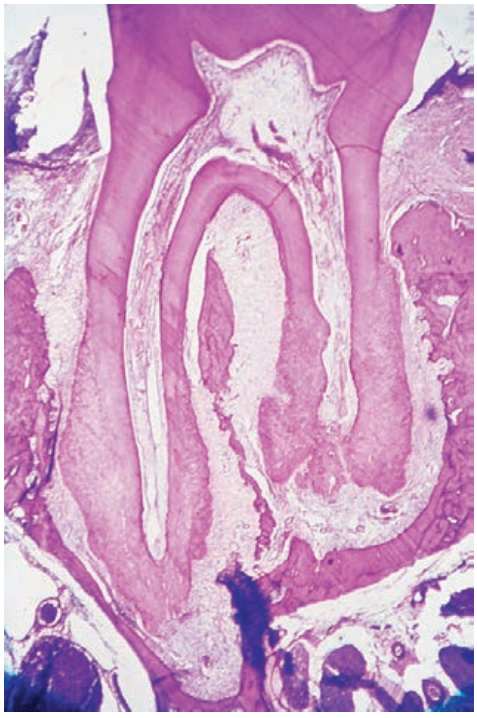

Figure 15-4 Areas of tension and pressure in opposite sites of the periodontal ligament caused by experimentally induced orthodontic movement in a rat molar.

Slightly excessive pressure stimulates resorption of the alveolar bone, with a resultant widening of the periodontal ligament space. *Slightly excessive tension* causes elongation of the periodontal ligament fibers and apposition of alveolar bone. In areas of increased pressure, the blood vessels are numerous and reduced in size; in areas of increased tension, they are enlarged.[68]

Greater pressure produces a gradation of changes in the periodontal ligament, starting with compression of the fibers, which produces areas of hyalinization.[55-57] Subsequent injury to the fibroblasts and other connective tissue cells leads to necrosis of areas of the ligament.[53,57] Vascular changes are also produced: within 30 minutes, impairment and stasis of blood flow occur; at 2 to 3 hours, blood vessels appear to be packed with erythrocytes, which start to fragment; and between 1 and 7 days, disintegration of the blood vessel walls and release of the contents into the surrounding tissue occur.[54,64] In addition, increased resorption of alveolar bone and resorption of the tooth surface occur.[30,35]

Severe tension causes widening of the periodontal ligament, thrombosis, hemorrhage, tearing of the periodontal ligament, and resorption of alveolar bone.

Pressure severe enough to force the root against bone causes necrosis of the periodontal ligament and bone. The bone is resorbed from viable periodontal ligament adjacent to necrotic areas and from marrow spaces, a process called *undermining resorption.*[26,44]

The areas of the periodontium most susceptible to injury from excessive occlusal forces are the furcations.[23]

Injury to the periodontium produces a temporary depression in mitotic activity and the rate of proliferation and differentiation of fibroblasts,[63] in collagen formation, and in bone formation.[30,59,61,63] These return to normal levels after dissipation of the forces.

Stage II: Repair

Repair is constantly occurring in the normal periodontium, and trauma from occlusion stimulates increased reparative activity.

The damaged tissues are removed, and new connective tissue cells and fibers, bone, and cementum are formed in an attempt to restore the injured periodontium (Figure 15-5). Forces remain traumatic only as long as the damage produced exceeds the reparative capacity of the tissues.

SCIENCE TRANSFER

Periodontal tissues within the bony housing of the socket transmit forces to the surrounding bone and cause osseous changes such as resorption in regions of compression and bone apposition in regions of tension. The periodontal ligament provides a viscoelastic gel–like milieu that protects the dentition from excessive forces by its cushioning or dampening effect.

Occlusal trauma first results in vasodilation with associated extrusion and pain on biting. This will resolve quickly if the occlusal trauma in eliminated by therapy such as occlusal adjustment. These changes can also be associated with increased pulpal sensitivity and pain with hot and cold from vascular events in the periodontium causing vasodilation of the pulp, and these patients can get almost immediate relief of pain by occlusal adjustment.

Trauma from occlusion is not the initial cause of plaque-induced periodontal pockets and bone loss, but patients with occlusal trauma and periodontitis may have accentuation of bone loss. Many patients will benefit when occlusal therapy is part of the overall periodontal treatment protocol.

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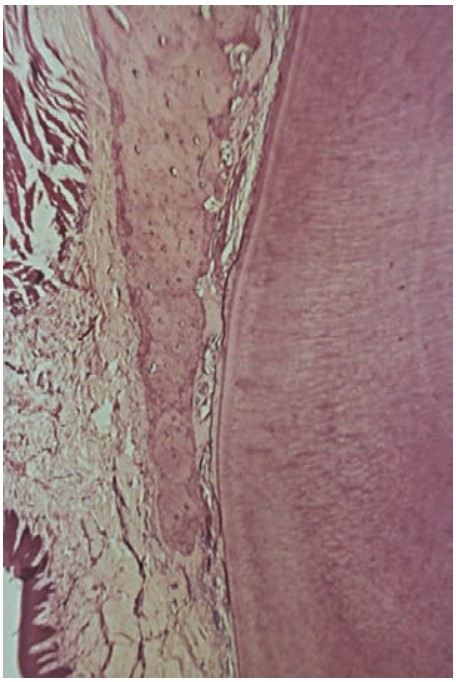

Figure 15-5 Experimental occlusal trauma in rats. Area of necrosis of the marginal periodontal ligament and resorption and remodeling in more apical periodontal sites.

When bone is resorbed by excessive occlusal forces, the body attempts to reinforce the thinned bony trabeculae with new bone (Figure 15-6). This attempt to compensate for lost bone is called *buttressing bone formation* and is an important feature of the reparative process associated with trauma from occlusion.[17] It also occurs when bone is destroyed by inflammation or osteolytic tumors.

Buttressing bone formation occurs within the jaw (central buttressing) and on the bone surface (peripheral buttressing). In *central buttressing* the endosteal cells deposit new bone, which restores the bony trabeculae and reduces the size of the marrow spaces. *Peripheral buttressing* occurs on the facial and lingual surfaces of the alveolar plate. Depending on its severity, peripheral buttressing may produce a shelflike thickening of the alveolar margin, referred to as *lipping* (Figure 15-7), or a pronounced bulge in the contour of the facial and lingual bone[8,17] (see Chapter 14).

Cartilage-like material sometimes develops in the periodontal ligament space as an aftermath of the trauma.[14] Formation of crystals from erythrocytes has also been shown.[58]

Stage III: Adaptive Remodeling of the Periodontium

If the repair process cannot keep pace with the destruction caused by the occlusion, the periodontium is remodeled in an effort to create a structural relationship in which the forces are no longer injurious to the tissues.[19] *This results in a widened periodontal ligament, which is funnel shaped at the crest, and angular defects in the bone, with no pocket formation. The involved teeth become loose.*[68] Increased vascularization has also been reported.[9]

The three stages in the evolution of traumatic lesions have been differentiated histometrically by the relative amounts of periodontal bone surface undergoing resorption or formation[5,8] (Figure 15-8). The injury phase shows an increase in areas of resorption and a decrease in bone formation, whereas the repair phase demonstrates decreased resorption and increased bone formation. After adaptive remodeling of the periodontium, resorption and formation return to normal.

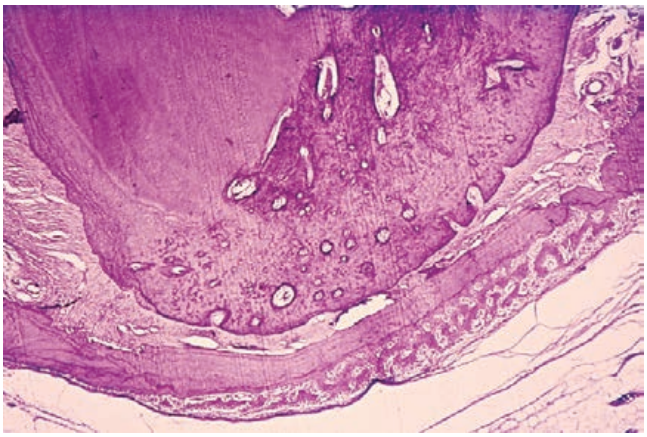

Figure 15-6 Apical area of a premolar subjected to experimental occlusal trauma in a dog, causing intrusion of the tooth and areas of necrosis in the periodontal ligament. Note the active bone formation on the outer aspect of the bone and the resorptive activity in the periphery of the necrotic site.

EFFECTS OF INSUFFICIENT OCCLUSAL FORCE

Insufficient occlusal force may also be injurious to the supporting periodontal tissues.[6,37] Insufficient stimulation causes thinning of the periodontal ligament, atrophy of the fibers, osteoporosis of the alveolar bone, and reduction in bone height. Hypofunction can result from an open-bite relationship, an absence of functional antagonists, or unilateral chewing habits that neglect one side of the mouth.

REVERSIBILITY OF TRAUMATIC LESIONS

Trauma from occlusion is reversible. When trauma is artificially induced in experimental animals, the teeth move away or intrude into the jaw. When the impact of the artificially created force is relieved, the tissues undergo repair. Although trauma from occlusion is reversible under such conditions, it does not always correct itself, and therefore it is not always temporary or of limited clinical significance. The injurious force must be relieved for repair to occur.[23,50] If conditions in humans do not permit the teeth to escape from or adapt to excessive occlusal force, periodontal damage persists and worsens.

The presence of inflammation in the periodontium as a result of plaque accumulation may impair the reversibility of traumatic lesions.[31,50]

EFFECTS OF EXCESSIVE OCCLUSAL FORCES ON DENTAL PULP

The effects of excessive occlusal forces on the dental pulp have not been established. Some clinicians report the disappearance of pulpal symptoms after correction of excessive occlusal forces. Pulpal reactions have been noted in animals subjected to increased occlusal forces[7,36] but did not occur when the forces were minimal and occurred over short periods.[36]

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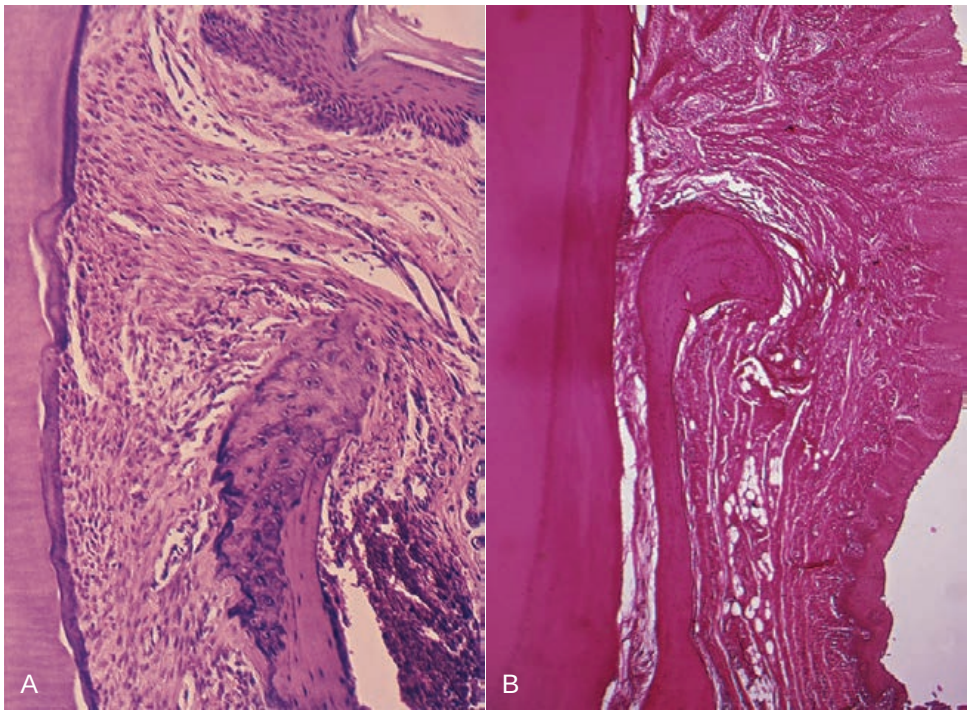

Figure 15-7 **A,** Widening of the periodontal ligament space in cervical area and a change in the shape of marginal alveolar bone as a result of chronic prolonged trauma from occlusion in rats. **B,** Comparable change in shape of marginal bone found in a human autopsy case.

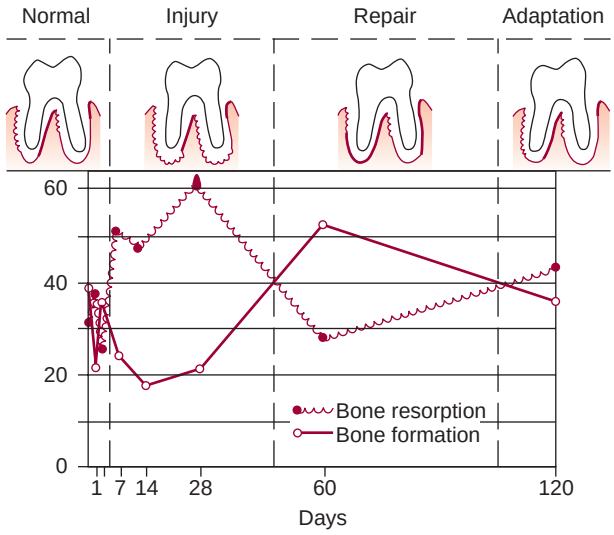

Figure 15-8 Evolution of traumatic lesions as depicted experimentally in rats by variations in relative amounts of areas of bone formation and bone resorption in periodontal bone surfaces. Horizontal axis, days after initiation of traumatic interference. Vertical axis, percentage of bone surface undergoing resorption or formation. The stages in the evolution of the lesions are represented in the top drawings, which show the average amount of bone activity for each group.[4]

INFLUENCE OF TRAUMA FROM OCCLUSION ON PROGRESSION OF MARGINAL PERIODONTITIS

The clinical impressions of early investigators and clinicians assigned an important role to trauma from occlusion in the etiology of periodontal lesions. Since then, numerous studies have been performed attempting to determine the mechanisms by which trauma from occlusion may affect periodontal disease.

Initial studies involved the placement of high crowns or restorations on the teeth of dogs or monkeys, resulting in a continuous or intermittent force in one direction.[2,21] These investigations provided an orthodontic type of force and gave clear descriptions of changes occurring in pressure zones and tension zones. These procedures usually resulted in tooth displacement and consolidation in a new, nontraumatized position.

Trauma from occlusion in humans, however, is the result of forces that act alternatively in opposing directions. These were analyzed in experimental animals with "jiggling forces," usually produced by a high crown combined with an orthodontic appliance that would bring the traumatized tooth back to its original position when the force was dissipated by separating the teeth. In another method, the teeth were separated by wooden or elastic material wedged interproximally to displace a tooth toward the opposite proximal side. After 48 hours, the wedge was removed and the procedure was repeated on the opposite side.

These studies resulted in a combination of changes produced by pressure and tension on both sides of the tooth, with an increase in the width of the ligament and increased tooth mobility. None of these methods caused gingival inflammation or pocket formation, and the results essentially represented different degrees of functional adaptation to increased forces.[49,68]

To mimic the problem in humans more closely, studies were then conducted on the effect produced by jiggling trauma and simultaneous plaque-induced gingival inflammation.

The accumulation of bacterial plaque that initiates gingivitis and results in periodontal pocket formation affects the marginal gingiva, but trauma from occlusion occurs in the supporting tissues and does not affect the gingiva (Figure 15-9). The marginal gingiva is unaffected by trauma from occlusion because its blood supply is not affected, even when the vessels of the periodontal ligament are obliterated by excessive occlusal forces.[25] It has been repeatedly proved that trauma from occlusion does not cause pockets or gingivitis* nor does it increase gingival fluid flow.† Furthermore,

*References 2, 21, 51, 66, 67, 69.
†References 28, 34, 37, 42, 43, 51.

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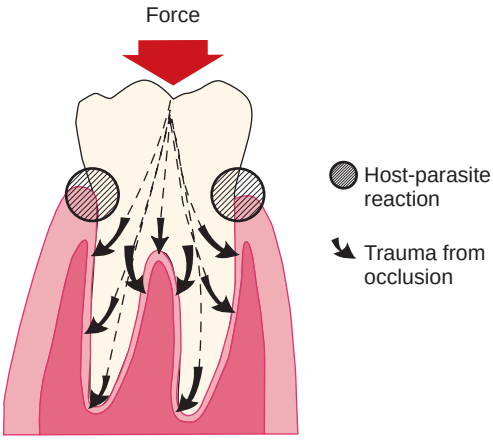

Figure 15-9 The reaction between dental plaque and the host takes place in the gingival sulcus region. Trauma from occlusion appears in the tissues supporting the tooth.

experimental trauma in dogs does not influence bacterial repopulation of pockets after scaling and root planing.[32] However, mobile teeth in humans harbor significantly higher proportions of *Campylobacter rectus* and *Peptostreptococcus micros* than nonmobile teeth.[27]

As long as inflammation is confined to the gingiva, the inflammatory process is not affected by occlusal forces.[33] When inflammation extends from the gingiva into the supporting periodontal tissues (i.e., when gingivitis becomes periodontitis), plaque-induced inflammation enters the zone influenced by occlusion, which Glickman has called the *zone of co-destruction.*[15,16,18]

Two groups have studied this topic experimentally with conflicting results, probably because of the different methods used. The Eastman Dental Center group in Rochester, NY, used squirrel monkeys, produced trauma by repetitive interdental wedging, and added mild-to-moderate gingival inflammation; experimental times were up to 10 weeks. They reported that the presence of trauma did not increase the loss of attachment induced by periodontitis.[43,46-48] The University of Gothenburg group in Sweden used beagle dogs, produced trauma by placing cap splints and orthodontic appliances, and induced severe gingival inflammation; experimental times were up to 1 year. This group found that occlusal stresses increase the periodontal destruction induced by periodontitis.[11,12,40]

When trauma from occlusion is eliminated, a substantial reversal of bone loss occurs, except in the presence of periodontitis. This indicates that inflammation inhibits the potential for bone regeneration.[31,39,49,50] *Thus it is important to eliminate the marginal inflammatory component in cases of trauma from occlusion because the presence of inflammation affects bone regeneration after the removal of the traumatizing contacts.*[31] It also has been shown in experimental animals that trauma from occlusion does not induce progressive destruction of the periodontal tissues in regions kept healthy after the elimination of preexisting periodontitis.[11]

Trauma from occlusion also tends to change the shape of the alveolar crest. The change in shape consists of a widening of the marginal periodontal ligament space, a narrowing of the interproximal alveolar bone, and a shelflike thickening of the alveolar margin.[9,40,43] Therefore, although trauma from occlusion does not alter the inflammatory process, it changes the architecture of the area around the inflamed site.[18,40] *Thus, in the absence of inflammation, the response to trauma from occlusion is limited to adaptation to the increased forces. In the presence of inflammation, however, the changes in the shape of the alveolar crest may be conducive to angular bone loss, and existing pockets may become intrabony.*

Other theories that have been proposed to explain the interaction of trauma and inflammation include the following:

- Trauma from occlusion may alter the pathway of extension of gingival inflammation to the underlying tissues. This may be favored by the reduced collagen density and increased number of leukocytes, osteoclasts, and blood vessels in the coronal portion of increasingly mobile teeth.[3] Inflammation then may proceed to the periodontal ligament rather than to the bone. Resulting bone loss would be angular, and pockets could become intrabony.[1,18,20,41]
- Trauma-induced areas of root resorption uncovered by apical migration of the inflamed gingival attachment may offer a favorable environment for the formation and attachment of plaque and calculus and therefore may be responsible for the development of deeper lesions.[60]
- Supragingival plaque can become subgingival if the tooth is tilted orthodontically or migrates into an edentulous area, resulting in the transformation of a suprabony pocket into an intrabony pocket.[10,12,18]
- Increased mobility of traumatically loosened teeth may have a pumping effect on plaque metabolites, increasing their diffusion.[65]

Clinical and Radiographic Signs of Trauma from Occlusion Alone

The most common clinical sign of trauma to the periodontium is *increased tooth mobility.* In the injury stage of trauma from occlusion, destruction of periodontal fibers occurs, which increases tooth mobility. In the final stage the accommodation of the periodontium to increased forces entails a widening of the periodontal ligament, which also leads to increased tooth mobility. Although this tooth mobility is greater than the so-called normal mobility, it cannot be considered pathologic because it is an adaptation and not a disease process. If it does become progressively worse, then it can be considered pathologic.

Other causes of increased tooth mobility include advanced bone loss, inflammation of the periodontal ligament of periodontal or periapical origin, and some systemic causes (e.g., pregnancy). The destruction of surrounding alveolar bone, such as occurs in osteomyelitis or jaw tumors, may also increase tooth mobility (see Chapter 30).

Radiographic signs of trauma from occlusion may include the following:

1. Increased width of the periodontal space, often with thickening of the lamina dura along the lateral aspect of the root, in the apical region, and in bifurcation areas. These changes do not *necessarily* indicate destructive changes because they may result from thickening and strengthening of the periodontal ligament and alveolar bone, constituting a favorable response to increased occlusal forces.
2. A "vertical" rather than "horizontal" destruction of the interdental septum.
3. Radiolucency and condensation of the alveolar bone.
4. Root resorption (see Chapter 13).

In summary, trauma from occlusion does not initiate gingivitis or periodontal pockets, but it may constitute an additional risk factor for the progression and severity of the disease. An understanding of the effect of trauma from occlusion on the periodontium is useful in the clinical management of periodontal problems.

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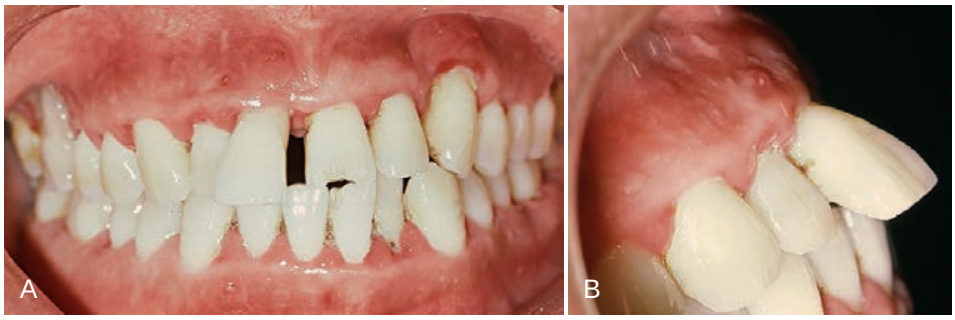

Figure 15-10 Labial migration of maxillary central incisors, especially the right incisor. **A,** Frontal view. **B,** Lateral view.

PATHOLOGIC TOOTH MIGRATION

Pathologic migration refers to tooth displacement that results when the balance among the factors that maintain physiologic tooth position is disturbed by periodontal disease. Pathologic migration is relatively common and may be an early sign of disease, or it may occur in association with gingival inflammation and pocket formation as the disease progresses.

Pathologic migration occurs most frequently in the anterior region, but posterior teeth may also be affected. The teeth may move in any direction, and the migration is usually accompanied by mobility and rotation. Pathologic migration in the occlusal or incisal direction is termed *extrusion.* All degrees of pathologic migration are encountered, and one or more teeth may be affected (Figure 15-10). It is important to detect migration in its early stages and prevent more serious involvement by eliminating the causative factors. Even in the early stage, some degree of bone loss occurs.

Pathogenesis

Two major factors play a role in maintaining the normal position of the teeth: the health and normal height of the periodontal attachment apparatus and the forces exerted on the teeth. The latter includes the forces of occlusion and pressure from the lips, cheeks, and tongue. Factors that are important in relation to the forces of occlusion include (1) tooth morphologic features and cuspal inclination; (2) the presence of a full complement of teeth; (3) a physiologic tendency toward mesial migration; (4) the nature and location of contact point relationships; (5) proximal, incisal, and occlusal attrition; and (6) the axial inclination of the teeth. Alterations in any of these factors start an interrelated sequence of changes in the environment of a single tooth or group of teeth that may result in pathologic migration. Thus pathologic migration occurs under conditions that weaken the periodontal support, increase or modify the forces exerted on the teeth, or both.

Weakened Periodontal Support. The inflammatory destruction of the periodontium in periodontitis creates an imbalance between the forces maintaining the tooth in position and the occlusal and muscular forces the tooth ordinarily needs to bear. The tooth with weakened support is unable to maintain its normal position in the arch and moves away from the opposing force unless it is restrained by proximal contact. The force that moves the weakly supported tooth may be created by factors such as occlusal contacts or pressure from the tongue.

It is important to understand that the abnormality in pathologic migration rests with the weakened periodontium; the force itself is not necessarily abnormal. Forces that are acceptable to an intact periodontium become injurious when periodontal support is reduced, as in the tooth with abnormal proximal contacts. Abnormally located proximal contacts convert the normal anterior component of force to a wedging force that moves the tooth occlusally or incisally. The wedging force, which can be withstood by the intact periodontium, causes the tooth to extrude when the periodontal support is weakened by disease. *As its position changes, the tooth is subjected to abnormal occlusal forces, which aggravate the periodontal destruction and the tooth migration.*

Figure 15-11 Calculus and bone loss on the mesial surface of a canine that has drifted distally.

Pathologic migration may continue after a tooth no longer contacts its antagonist. Pressures from the tongue, the food bolus during mastication, and proliferating granulation tissue provide the force.

Pathologic migration is also an early sign of localized aggressive periodontitis. Weakened by loss of periodontal support, the maxillary and mandibular anterior incisors drift labially and extrude, creating diastemata between the teeth (see Chapter 18).

Changes in the Forces Exerted on the Teeth. Changes in the magnitude, direction, or frequency of the forces exerted on the teeth can induce pathologic migration of a tooth or group of teeth. These forces do not have to be abnormal to cause migration if the periodontium is sufficiently weakened. Changes in the forces may result from unreplaced missing teeth, or other causes.

Unreplaced Missing Teeth. Drifting of teeth into the spaces created by unreplaced missing teeth often occurs. Drifting differs from pathologic migration in that it does not result from destruction of the periodontal tissues. However, it usually creates conditions that lead to periodontal disease, and thus the initial tooth movement is aggravated by loss of periodontal support (Figure 15-11).

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Figure 15-12 Maxillary first molar tilted and extruded into the space created by a missing mandibular tooth.

Figure 15-13 No drifting or extrusion despite 4 years' absence of mandibular teeth.

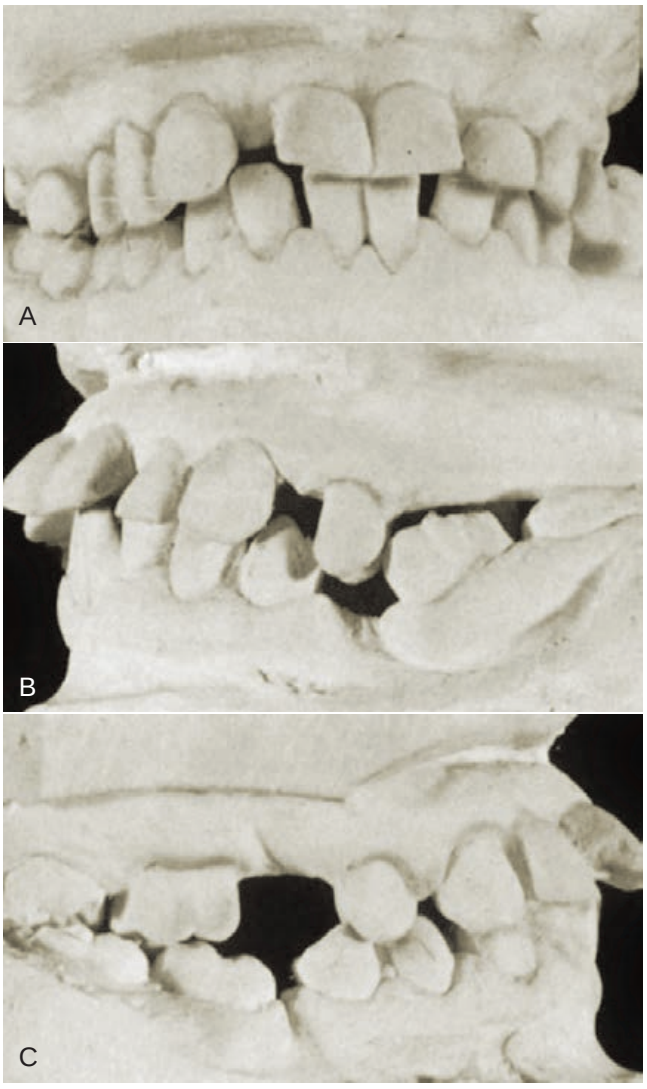

Figure 15-14 Examples of mutilation of occlusion associated with unreplaced missing teeth. Note pronounced pathologic migration, disturbed proximal contacts, and functional relationships with closing of the bite.

Drifting generally occurs in a mesial direction, combined with tilting or extrusion beyond the occlusal plane. The premolars frequently drift distally (Figure 15-12). Although drifting is a common sequela when missing teeth are not replaced, it does not always occur (Figure 15-13).

Failure to Replace First Molars. The pattern of changes that may follow failure to replace missing first molars is characteristic. In extreme cases it consists of the following:

1. The second and third molars tilt mesially, resulting in a decrease in vertical dimension (Figure 15-14).
2. The premolars move distally, and the mandibular incisors tilt or drift lingually. While drifting distally, the mandibular premolars lose their intercuspating relationship with the maxillary teeth and may tilt distally.
3. Anterior overbite is increased. The mandibular incisors strike the maxillary incisors near the gingiva or traumatize the gingiva.
4. The maxillary incisors are pushed labially and laterally (Figure 15-15).
5. The anterior teeth extrude because the incisal apposition has largely disappeared.
6. Diastemata are created by the separation of the anterior teeth (see Figure 15-14).

Figure 15-15 Maxillary incisors pushed labially in patient with bilateral unreplaced mandibular molars. Note extrusion of the maxillary molars.

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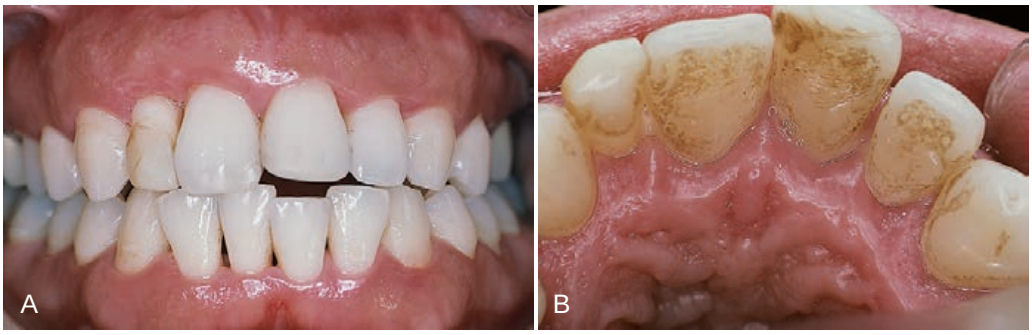

Figure 15-16 Pathologic migration associated with tongue pressure. **A,** Facial view. **B,** Palatal view.

The disturbed proximal contact relationships lead to food impaction, plaque accumulation resulting in gingival inflammation, and pocket formation, followed by bone loss and tooth mobility. Occlusal disharmonies created by the altered tooth positions traumatize the supporting tissues of the periodontium and aggravate the destruction caused by the inflammation. Reduction in periodontal support leads to further migration of the teeth and mutilation of the occlusion.

Other Causes. *Trauma from occlusion* may cause a shift in tooth position either by itself or in combination with inflammatory periodontal disease. The direction of movement depends on the occlusal force.

Pressure from the tongue may cause drifting of the teeth in the absence of periodontal disease or may contribute to pathologic migration of teeth with reduced periodontal support (Figure 15-16).

In tooth support weakened by periodontal destruction, *pressure from the granulation tissue of periodontal pockets* has been mentioned as contributing to pathologic migration.[29,38] The teeth may return to their original positions after the pockets are eliminated, but if more destruction has occurred on one side of a tooth than on the other, the healing tissues tend to pull in the direction of the lesser destruction.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 16

Chronic Periodontitis

M. John Novak and Karen F. Novak

CHAPTER OUTLINE

CLINICAL FEATURES
- General Characteristics
- Disease Distribution
- Disease Severity
- Symptoms
- Disease Progression
- Prevalence

RISK FACTORS FOR DISEASE
- Prior History of Periodontal Disease
- Local Factors
- Systemic Factors
- Environmental and Behavioral Factors
- Genetic Factors

Chronic periodontitis, formerly known as *adult periodontitis* or *chronic adult periodontitis,* is the most prevalent form of periodontitis. It is generally considered to be a slowly progressing disease. However, in the presence of systemic or environmental factors that may modify the host response to plaque accumulation, such as diabetes, smoking, or stress, disease progression may become more aggressive. Although chronic periodontitis is most frequently observed in adults, it can occur in children and adolescents in response to chronic plaque and calculus accumulation. This observation underlies the recent name change from "adult" periodontitis, which suggests that chronic, plaque-induced periodontitis is only observed in adults, to a more universal description of "chronic" periodontitis, which can occur at any age (see Chapter 4).

Chronic periodontitis has been defined as "an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss, and bone loss."[3] This definition outlines the major clinical and etiologic characteristics of the disease: (1) microbial plaque formation, (2) periodontal inflammation, and (3) loss of attachment and alveolar bone. Periodontal pocket formation is usually a sequela of the disease process unless gingival recession accompanies attachment loss, in which case pocket depths may remain shallow, even in the presence of ongoing attachment loss and bone loss.

CLINICAL FEATURES

General Characteristics

Characteristic clinical findings in patients with untreated chronic periodontitis may include supragingival and subgingival plaque accumulation (frequently associated with calculus formation), gingival inflammation, pocket formation, loss of periodontal attachment, loss of alveolar bone, and occasional suppuration (Figure 16-1). In patients with poor oral hygiene, the gingiva typically may be slightly to moderately swollen and exhibit alterations in color ranging from pale red to magenta. Loss of gingival stippling and changes in the surface topography may include blunted or rolled gingival margins and flattened or cratered papillae.

In many patients, especially those who perform regular home care measures, the changes in color, contour, and consistency frequently associated with gingival inflammation may not be visible on inspection, and inflammation may be detected only as bleeding of the gingiva in response to examination of the periodontal pocket with a periodontal probe (see Figures 16-2, *A,* and 16-3, *A*). Gingival bleeding, either spontaneous or in response to probing, is common, and inflammation-related exudates of crevicular fluid and suppuration from the pocket also may be found. In some cases, probably as a result of long-standing, low-grade inflammation, thickened, fibrotic marginal tissues may obscure the underlying inflammatory changes. Pocket depths are variable, and both horizontal and vertical bone loss can be found. Tooth mobility often appears in advanced cases with extensive attachment loss and bone loss.

Chronic periodontitis can be clinically diagnosed by the detection of chronic inflammatory changes in the marginal gingiva, presence of periodontal pockets, and loss of clinical attachment. It is diagnosed radiographically by evidence of bone loss. These findings may be similar to those seen in aggressive disease. A differential diagnosis is based on the age of the patient, rate of disease progression over time, familial nature of aggressive disease, and relative absence of local factors in aggressive disease compared with the presence of abundant plaque and calculus in chronic periodontitis.

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Disease Distribution

Chronic periodontitis is considered a *site-specific* disease. The clinical signs of chronic periodontitis—inflammation, pocket formation, attachment loss, and bone loss—are believed to be caused by the direct, site-specific effects of subgingival plaque accumulation. As a result of this local effect, pocket formation and attachment and bone loss may occur on one surface of a tooth while other surfaces maintain normal attachment levels. For example, a proximal surface with chronic plaque accumulation may have loss of attachment, whereas the plaque-free facial surface of the same tooth may be free of disease.

In addition to being site specific, chronic periodontitis may be described as being *localized,* when few sites demonstrate attachment and bone loss, or *generalized,* when many sites around the mouth are affected, as follows:

Localized periodontitis: Periodontitis is considered localized when less than 30% of the sites assessed in the mouth demonstrate attachment loss and bone loss (Figure 16-2).

Generalized periodontitis: Periodontitis is considered generalized when 30% or more of the sites assessed in the mouth demonstrate attachment loss and bone loss (Figure 16-3).

The pattern of bone loss observed in chronic periodontitis may be *vertical* (angular), when attachment and bone loss on one tooth surface is greater than that on an adjacent surface (see Figure 16-2, *C*), or *horizontal,* when attachment and bone loss proceeds at a uniform rate on the majority of tooth surfaces (see Figure 16-3, *B*). Vertical bone loss is associated with intrabony pocket formation. Horizontal bone loss is usually associated with suprabony pockets.

Figure 16-1 Clinical features of chronic periodontitis in 45-year-old patient with poor oral home care and no previous dental treatment. Abundant plaque and calculus are associated with redness, swelling, and edema of the gingival margin. Gingival recession is evident, resulting from loss of attachment and alveolar bone. Spontaneous bleeding is present, and there is visible exudate of gingival crevicular fluid. Gingival stippling has been lost.

Disease Severity

The severity of destruction of the periodontium that occurs as a result of chronic periodontitis is generally considered a function of time. With increasing age, attachment loss and bone loss become more prevalent and more severe because of an accumulation of destruction. Disease severity may be described as *slight (mild), moderate,* or *severe* (see Chapter 4). These terms may be used to describe the disease severity of the entire mouth or part of the mouth (e.g., quadrant, sextant) or the disease status of an individual tooth, as follows.

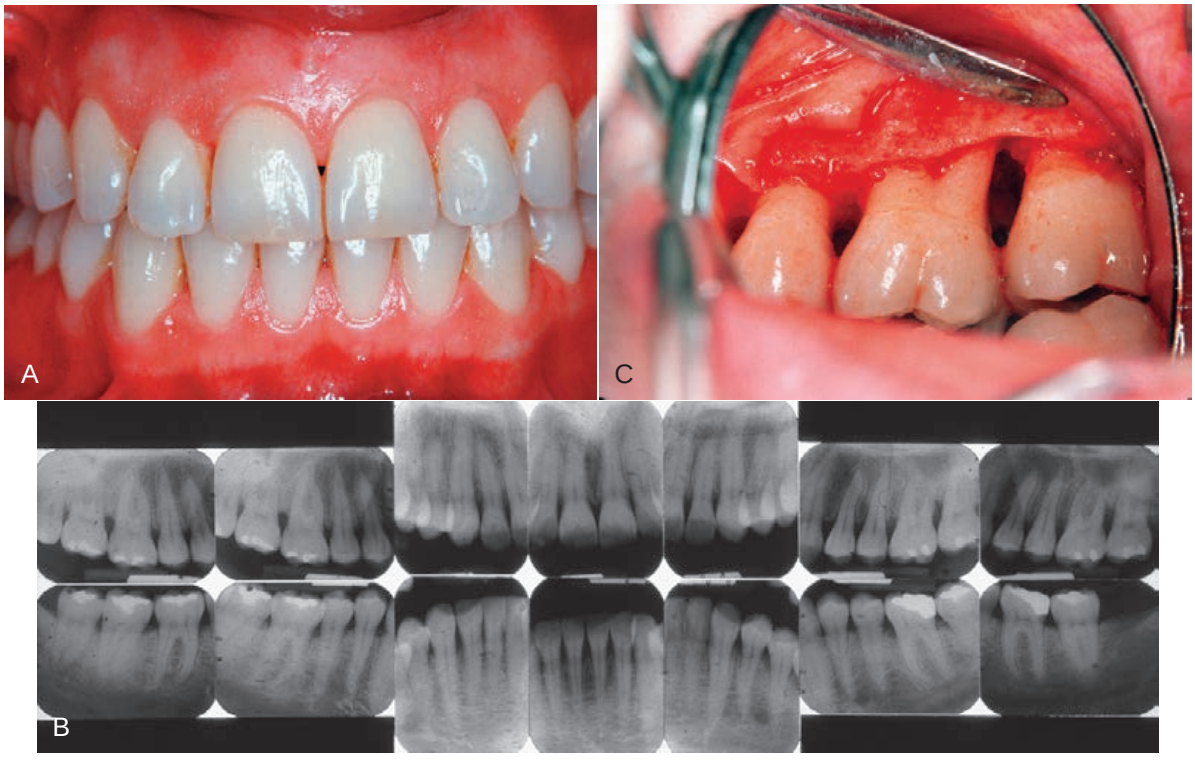

Figure 16-2 Localized chronic periodontitis in 42-year-old female. **A,** Clinical view of anterior teeth showing minimal plaque and inflammation. **B,** Radiographs showing presence of localized, vertical, angular bone loss on the distal side of the maxillary left first molar. **C,** Surgical exposure of the vertical (angular) defect associated with the chronic plaque accumulation and inflammation in the distobuccal furcation.

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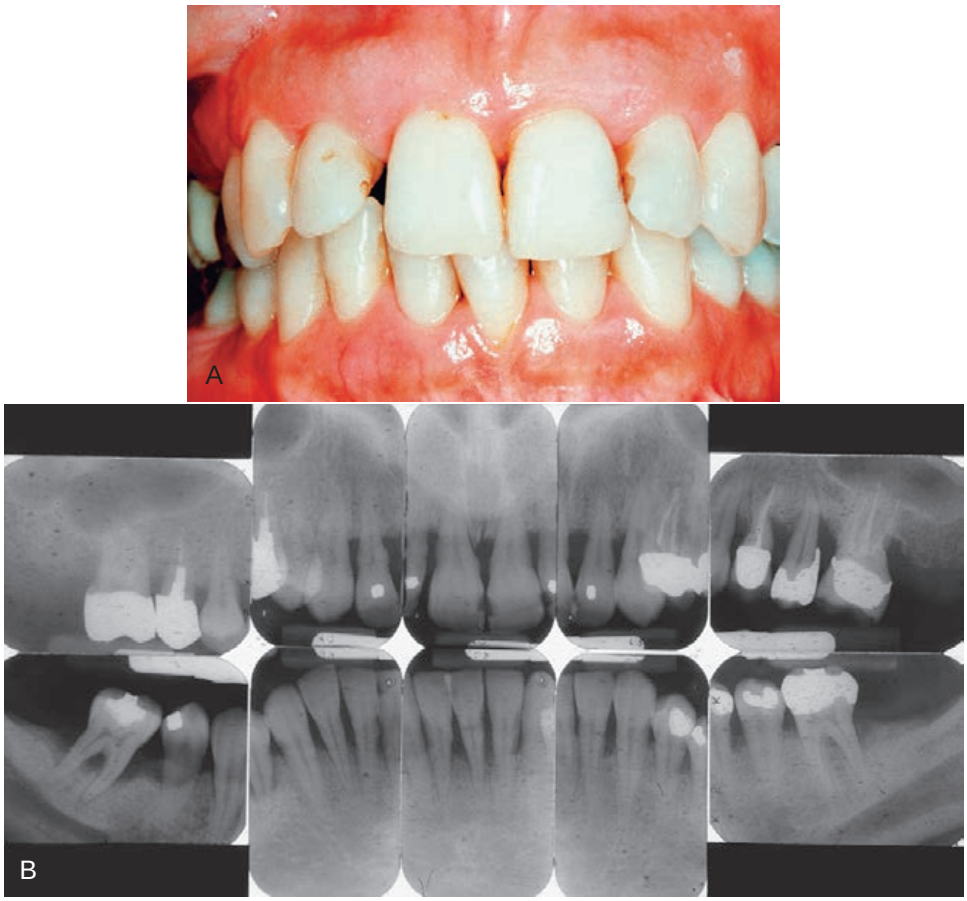

Figure 16-3 Generalized chronic periodontitis in 38-year-old female with 20-year history of smoking at least one pack of cigarettes per day. **A,** Clinical view showing minimal plaque and inflammation. Probing produced negligible bleeding, which is common with smokers. Patient complained of spacing between the right maxillary incisors, which was associated with advanced attachment and bone loss. **B,** Radiographs showing severe, generalized, horizontal pattern of bone loss. Maxillary and mandibular molars have already been lost through advanced disease and furcation involvement.

Slight (mild) periodontitis: Periodontal destruction is generally considered slight when no more than 1 to 2 mm of clinical attachment loss has occurred.

Moderate periodontitis: Periodontal destruction is generally considered moderate when 3 to 4 mm of clinical attachment loss has occurred.

Severe periodontitis: Periodontal destruction is considered severe when 5 mm or more of clinical attachment loss has occurred.

Symptoms

Patients may first become aware that they have chronic periodontitis when they notice that their gums bleed when brushing or eating; that spaces occur between their teeth as a result of tooth movement; or that teeth have become loose. Because chronic periodontitis is usually painless, however, patients may be totally unaware that they have the disease and may be less likely to seek treatment and accept treatment recommendations. In addition, a negative response to questions such as, "Are you in pain?" is not sufficient to eliminate suspicion of periodontitis. Occasionally, pain may be present in the absence of caries caused by exposed roots that are sensitive to heat, cold, or both. Areas of localized dull pain, sometimes radiating deep into the jaw, have been associated with periodontitis. The presence of areas of food impaction may add to the patient's discomfort. Gingival tenderness or "itchiness" may also be found.

Disease Progression

Patients appear to have the same susceptibility to plaque-induced chronic periodontitis throughout their lives. The rate of disease progression is usually slow but may be modified by systemic or environmental and behavioral factors. Onset of chronic periodontitis can occur at any time, and the first signs may be detected during adolescence in the presence of chronic plaque and calculus accumulation. Because of its slow rate of progression, however, chronic periodontitis usually becomes clinically significant in the mid-thirties or later.

Chronic periodontitis does not progress at an equal rate in all affected sites throughout the mouth. Some involved areas may remain static for long periods,[6] whereas others may progress more rapidly. More rapidly progressive lesions occur most frequently in interproximal areas[5,7] and may also be associated with areas of greater plaque accumulation and inaccessibility to plaque control measures (e.g., furcation areas, overhanging margins of restorations, sites of malposed teeth, or areas of food impaction).

Several models have been proposed to describe the rate of disease progression.[10] In these models, progression is measured by determining the amount of attachment loss during a given period, as follows:

- The *continuous model* suggests that disease progression is slow and continuous, with affected sites showing a constantly progressive rate of destruction throughout the duration of the disease.

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SCIENCE TRANSFER

Patients with chronic periodontitis most often show slow progressive attachment and bone loss that extends over decades. This bone loss is initiated by specific groups of periodontal pathogenic gram negative anaerobic bacteria and therefore control of the subgingival plaque biofilm is an essential part of therapy. Some patients are susceptible to more rapid bone and attachment loss including those with a history of smoking, diabetes, or a genetic profile that increases the production of interleukin-1, a potent inflammatory cytokine that plays a pivotal role in bone and tissue destruction.

Attachment loss of 2 mm or more per year is an indicator of accentuated disease progression and these patients should be treated quickly to change the bacterial milieu by pocket depth reduction, and improved daily oral hygiene followed by maintenance recall visits ever 3 or 4 months.

- The *random* or *episodic-burst model,* proposes that periodontal disease progresses by short bursts of destruction followed by periods of no destruction. This pattern of disease is random with respect to the tooth sites affected and the chronology of the disease process.
- The *asynchronous, multiple-burst model* of disease progression suggests that periodontal destruction occurs around affected teeth during defined periods of life and that these bursts of activity are interspersed with periods of inactivity or remission. The chronology of these bursts of disease is asynchronous for individual teeth or groups of teeth.

Prevalence

Chronic periodontitis increases in prevalence and severity with age, generally affecting both genders equally. Periodontitis is an *age-associated,* not an age-related, disease. In other words, it is not the age of the individual that causes the increase in disease prevalence, but rather the length of time that the periodontal tissues are challenged by chronic plaque accumulation.

RISK FACTORS FOR DISEASE

Prior History of Periodontal Disease

Although not a true risk factor for disease but rather a disease *predictor,* a prior history of periodontal disease puts patients at greater risk for developing loss of attachment and bone, given a challenge from bacterial plaque accumulation.[8,9]

This means that a patient who presents with persistent gingivitis or periodontitis with pocketing, attachment loss, and bone loss may continue to lose periodontal support if not successfully treated. In addition, a patient with chronic periodontitis who has been successfully treated will develop continuing disease if plaque is allowed to accumulate. This emphasizes the need for continuous monitoring, treatment, and maintenance of patients with persistent gingivitis or periodontitis to prevent recurrence of disease. The risk factors that contribute to patient susceptibility are discussed in the following sections.

Local Factors

Plaque accumulation on tooth and gingival surfaces at the dentogingival junction is considered the primary initiating agent in the etiology of gingivitis and chronic periodontitis.[5] Attachment and bone loss are associated with an increase in the proportion of gram-negative organisms in the subgingival plaque biofilm, with specific increases in organisms known to be exceptionally pathogenic and virulent. *Porphyromonas gingivalis* (formerly *Bacteroides gingivalis*), *Tannerella forsythia* (formerly *Bacteroides forsythus*), and *Treponema denticola,* otherwise known as the "red complex," are frequently associated with ongoing attachment and bone loss in chronic periodontitis (see Chapter 23).

Chronic periodontitis is generally slowly progressive, with some patients having increased susceptibility to attachment and bone loss and pocketing. Some patients who have a genetic profile that accentuates interleukin-1 (IL-1) production may have an increased risk of tooth loss, and if these patients are also smokers, their risk increases. Diabetes is another factor that often leads to severe and extensive periodontal destruction. Also, a specific group of microorganisms is seen in the subgingival biofilm of patients with ongoing bone loss associated with chronic periodontitis, including *Porphyromonas gingivalis, Tannerella forsythia,* and *Treponema denticola.*

The identification and characterization of these and other pathogenic microorganisms and their association with attachment and bone loss have led to the *specific plaque hypothesis* for the development of chronic periodontitis. This hypothesis implies that although a general increase occurs in the proportion of gram-negative microorganisms in the subgingival plaque in periodontitis, it is the presence of increased proportions of members of the red complex, and perhaps other microorganisms, that precipitates attachment and bone loss. The mechanisms by which this occurs have not been clearly delineated, but these bacteria may impart a local effect on the cells of the inflammatory response and the cells and tissues of the host, resulting in a local, site-specific disease process. The interactions between pathogenic bacteria and the host and their potential effects on disease progression are discussed in detail in Part 4.

Because plaque accumulation is the primary initiating agent in periodontal inflammation and destruction, anything that facilitates plaque accumulation or prevents plaque removal by oral hygiene procedures can be detrimental to the patient. *Plaque-retentive factors* are important in the development and progression of chronic periodontitis because they retain plaque microorganisms in proximity to the periodontal tissues, providing an ecologic niche for plaque growth and maturation. *Calculus* is considered the most important plaque-retentive factor because of its ability to retain and harbor plaque bacteria on its rough surface. As a result, calculus removal is essential for the maintenance of a healthy periodontium. Other factors that are known to retain plaque or prevent its removal are subgingival and overhanging margins of restorations; carious lesions that extend subgingivally; furcations exposed by loss of bone; crowded and malaligned teeth; and root grooves and concavities. These potential risk factors for periodontitis are discussed further in Chapter 32, and their impact on the prognosis of periodontal treatment is discussed in Chapter 33.

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Systemic Factors

The rate of progression of plaque-induced chronic periodontitis is generally considered to be slow. However, when chronic periodontitis occurs in a patient who also has a systemic disease that influences the effectiveness of the host response, the rate of periodontal destruction may be significantly increased.

Diabetes is a systemic condition that can increase the severity and extent of periodontal disease in an affected patient. Type 2 diabetes, or non–insulin-dependent diabetes mellitus (NIDDM), is the most prevalent form of diabetes and accounts for 90% of diabetic patients.[1]

In addition, type 2 diabetes is most likely to develop in an adult population at the same time as chronic periodontitis. The synergistic effect of plaque accumulation and modulation of an effective host response through the effects of diabetes can lead to severe and extensive periodontal destruction that may be difficult to manage with standard clinical techniques without controlling the systemic condition. An increase in type 2 diabetes in teenagers and young adults has been observed and may be associated with an increase in juvenile obesity.

In addition, type 1 diabetes, or insulin-dependent diabetes mellitus (IDDM), is observed in children, teenagers, and young adults and may lead to increased periodontal destruction when it is uncontrolled. It is likely that chronic periodontitis, aggravated by the complications of type 1 and type 2 diabetes, will increase in prevalence in the near future and will provide therapeutic challenges to the clinician.

Environmental and Behavioral Factors

Smoking has been shown to increase the severity and extent of periodontal disease. When combined with plaque-induced chronic periodontitis, an increase in the rate of periodontal destruction may be observed in patients who smoke and have chronic periodontitis. As a result, smokers with chronic periodontitis have more attachment and bone loss, more furcation involvements, and deeper pockets (see Figure 16-3). In addition, they appear to form more supragingival and less subgingival calculus and demonstrate less bleeding on probing than nonsmokers. Initial evidence to explain these effects suggests changes in the subgingival microflora of smokers compared with nonsmokers, in addition to the effects of smoking on the host response. The clinical, microbiologic, and immunologic effects of smoking also appear to influence the response to therapy and the frequency of recurrent disease (see Chapter 26).

Emotional stress has previously been associated with necrotizing ulcerative disease, perhaps because of the effects of stress on immune function. Increasing evidence suggests that emotional stress also may influence the extent and severity of chronic periodontitis, probably through the same mechanisms.

Genetic Factors

Periodontitis is considered to be a multifactorial disease in which the normal balance between microbial plaque and host response is disrupted. This disruption, as described previously, can occur through changes in the plaque composition, changes in the host response, or environmental and behavioral influences on both plaque response and host response. In addition, periodontal destruction is frequently seen among family members and across different generations within a family, suggesting a genetic basis for the susceptibility to periodontal disease. Recent studies have demonstrated a familial aggregation of localized and generalized aggressive periodontitis. In addition, studies of monozygotic twins suggest a genetic component to chronic periodontitis, but the influences of bacterial transmission among family members and environmental effects make it difficult to interpret a complex interaction (see Chapters 24 and 27).

Although no clear genetic determinants have been described for patients with chronic periodontitis, a genetic predisposition to more aggressive periodontal breakdown in response to plaque and calculus accumulation may exist. Studies indicate that a genetic variation or polymorphism in the genes encoding IL-1α and IL-1β is associated with an increased susceptibility to a more aggressive form of chronic periodontitis in subjects of Northern European origin,[4] although more recent studies have disputed this association.[2] In addition, smokers demonstrating the composite IL-1 genotype are at even greater risk for severe disease. One study suggested that patients with the IL-1 genotype increased the risk for tooth loss by 2.7 times; those who were heavy smokers and IL-1 genotype negative increased the risk for tooth loss by 2.9 times. The combined effect of the IL-1 genotype and smoking increased the risk of tooth loss by 7.7 times.[2] With increased characterization of genetic polymorphisms that may exist in other target genes, a complex genotype may be identified for many different clinical forms of periodontitis. However, given the multifactorial nature of periodontal disease, the confounding influence of multiple local, systemic, and environmental conditions and our inability to clearly define the different types of periodontitis, it is unlikely that a clear genetic predisposition to periodontal disease will be found.

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CHAPTER 17

Necrotizing Ulcerative Periodontitis

Perry R. Klokkevold

CHAPTER OUTLINE

NECROTIZING ULCERATIVE PERIODONTITIS
Clinical Features
Microscopic Findings
HIV/AIDS Patients
Etiology of Necrotizing Ulcerative Periodontitis
Malnutrition
SUMMARY

Necrotizing ulcerative periodontitis (NUP) may be an extension of *necrotizing ulcerative gingivitis* (NUG) into the periodontal structures, leading to periodontal attachment and bone loss. On the other hand, NUP and NUG may be different diseases. To date, there is little evidence to support the progression of NUG to NUP or to establish a relationship between the two conditions as a single disease entity. However, numerous clinical descriptions and case reports of NUP clearly demonstrate many clinical similarities between the two conditions. A recent article reports the clinical and microscopic findings from 45 patients seen between 1965 and 2000. In this article, the authors suggest that NUG may be a precursor to NUP, citing one case of a 9-year-old malnourished male presenting with three distinct lesions that were consistent with a diagnosis of NUG, NUP, and noma.[16] Until a distinction between NUG and NUP can be proved or disproved, it has been suggested that NUG and NUP be classified together under the broader category of *necrotizing periodontal diseases,* although with differing levels of severity.[1,24]

NUG has been recognized and described in the literature for centuries.[27] The features of NUG are presented in Chapter 10 and briefly reviewed here. Clinically they consist of areas of ulceration and necrosis of the interdental papilla covered by a whitish yellow soft layer, or pseudomembrane, and surrounded by an erythematous halo. Lesions are typically painful and bleed easily, often without provocation. Patients may also present with oral malodor, localized lymphadenopathy, fever, and malaise.

Microscopically, NUG lesions demonstrate a nonspecific necrotizing inflammation that presents with a predominant polymorphonuclear leukocyte (PMN, neutrophil) infiltrate in the ulcerated areas and an abundant chronic infiltrate of lymphocytes and plasma cells in the peripheral and deeper areas.[35]

The bacterial flora associated with NUG is well known. The constant cultivable flora consists of *Prevotella intermedia* and *Fusobacterium* species, whereas the constant microscopic observations reveal the presence of *Treponema* and *Selenomonas* species. The association of these bacteria with NUG is compelling. However, the bacterial etiology has not been proven because the bacteria have not been able to transfer the disease between healthy animals (i.e., have not been able to fulfill one of Koch's postulates). Interestingly, bacterial isolates have transmitted NUG from animal to animal in the beagle dog with a steroid-induced immunosuppression.[20–22] The ability to transmit NUG with bacteria in an immunosuppressed animal (but not in immunocompetent animals) suggests that the host response or resistance is an important factor in the pathogenesis of NUG.

The lesions of NUG are confined to the gingiva without loss of periodontal attachment or alveolar bone support, a feature that distinguishes this condition from NUP. In contrast to this view, MacCarthy and Claffey[19] suggested that periodontal attachment loss *is* one of the consequences of NUG lesions. In their evaluation of 13 patients with NUG, the mean probing attachment level for NUG-affected sites (2.2 ± 0.9 mm) was greater than control sites (0.8 ± 0.7 mm). This finding supports the concept that NUG and NUP are similar (or identical) diseases, with differences in host response or resistance rather than differences in bacterial etiology and pathogenesis.

NECROTIZING ULCERATIVE PERIODONTITIS

The term "necrotizing ulcerative periodontitis" was first adopted at the 1989 World Workshop in Clinical Periodontics.[3] It was changed from the 1986 term of "necrotizing ulcerative gingivoperiodontitis," which represented the condition of recurrent NUG progressing to a chronic form of periodontitis with attachment and bone loss. The 1989 adoption of NUP as a disease entity occurred when there was a heightened awareness and an increase in the number of necrotizing periodontitis cases being diagnosed and described in the literature. Specifically, more cases of NUP were being described in immunocompromised patients, especially those who were human immunodeficiency virus (HIV) positive or had acquired immunodeficiency syndrome (AIDS). In 1999 the subclassifications of NUG and NUP were included as separate

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diagnoses under the broader classification of "necrotizing ulcerative periodontal diseases."[1] Again, a distinction between the two conditions as separate diseases has not been clarified, but they are distinguished by the presence or absence of attachment and bone loss.

Clinical Features

Similar to NUG, clinical cases of NUP are defined by necrosis and ulceration of the coronal portion of the interdental papillae and gingival margin, with a painful, bright red marginal gingiva that bleeds easily.

The distinguishing feature of NUP is the *destructive progression* of the disease that includes periodontal attachment and bone loss. Deep interdental osseous craters typify periodontal lesions of NUP (Figure 17-1). However, "conventional" periodontal pockets with deep probing depth are not found because the ulcerative and necrotizing nature of the gingival lesion destroys the marginal epithelium and connective tissue, resulting in gingival recession. Periodontal pockets are formed because the junctional epithelial cells remain viable and can therefore migrate apically to cover areas of connective tissue loss. The necrosis of the junctional epithelium in NUG and NUP creates an ulcer that prevents this epithelial migration, and a pocket cannot form. Advanced lesions of NUP lead to severe bone loss, tooth mobility, and ultimately tooth loss. In addition to these manifestations, as previously mentioned, NUP patients may present with oral malodor, fever, malaise, or lymphadenopathy.

Microscopic Findings

In a study using transmission (TEM) and scanning electron microscopy (SEM) of the microbial plaque overlying the necrotic gingival papillae, Cobb et al[4] demonstrated striking histologic similarities between NUP in HIV-positive patients and previous descriptions of NUG lesions in non-HIV patients. Biopsies of involved posterior papillae from 10 male and 6 female HIV-positive patients with NUP were evaluated. Microscopic examination revealed a surface biofilm composed of a mixed microbial flora with different morphotypes and a subsurface flora with dense aggregations of spirochetes (bacterial zone). Below the bacterial layers were dense aggregations of PMNs (neutrophil-rich zone) and necrotic cells (necrotic zone). The biopsy technique used in this study did not allow observation of the deepest layer and thus was not able to identify the spirochetal infiltration zone, which is classically described in NUG lesions. In addition to the NUG-like microscopic features of NUP described in this study, high levels of yeasts and herpes-like viruses were observed. This latter finding is most likely indicative of the conditions afforded to opportunistic microbes in the immunocompromised host (HIV-positive patients).

HIV/AIDS Patients

Gingival and periodontal lesions with distinctive features are frequently found in patients with HIV infection and AIDS. Many of these lesions are atypical manifestations of inflammatory periodontal diseases that arise in the course of HIV infection and the patient's concomitant immunocompromised state. Linear gingival erythema (LGE), NUG, and NUP are the most common HIV-associated periodontal conditions reported in the literature.[25] Chapter 19 provides detailed descriptions of these and other atypical periodontal diseases that occur in the HIV-infected patient.

NUP lesions found in HIV-positive/AIDS patients can present with similar features to those seen in HIV-negative patients. On the other hand, NUP lesions in HIV-positive/AIDS patients can be much more destructive and frequently result in complications that are extremely rare in non-HIV/AIDS patients. For example, periodontal attachment and bone loss associated with HIV-positive NUP may be extremely rapid. Winkler et al[37] reported cases of NUP in HIV-positive patients (formerly referred to as "HIV-P") with teeth that lost more than 90% of periodontal attachment and

Figure 17-1 Necrotizing ulcerative periodontitis in 45-year-old, HIV-negative, white male patient. **A,** Buccal view of maxillary cuspid-bicuspid area. **B,** Palatal view of same area. **C,** Buccal view of mandibular anteriors. Note the deep craters associated with bone loss.

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10 mm of bone over a 3- to 6-month period. Ultimately, many of these lesions resulted in tooth loss. Other complications reported in this population include a progression of the lesions to involve large areas of soft tissue necrosis, with exposure of bone and sequestration of bone fragments. This type of severe, progressive lesion with extension into the vestibular area and the palate is referred to as *necrotizing ulcerative stomatitis* (see Figure 19-31).

The reported prevalence of NUP in HIV-infected patients varies.[6,13,25,27] Riley et al[28] reported only two cases of NUP in 200 HIV-positive patients (1%), whereas Glick et al[13] found a prevalence of 6.3% for NUP cases in a prospective study of 700 HIV-positive patients. Variations in reported findings may be related to differences in the populations (e.g., intravenous drug users versus homosexuals versus patients with hemophilia) and differences in the immune status of the study subjects.

Necrotizing forms of periodontitis appear to be more prevalent in patients with more severe immunosuppression.[25,26] Case reports have depicted NUP as a progressive extension of HIV periodontitis (i.e., chronic to necrotic progression).[29] Glick et al[13,14] found a high correlation between the diagnosis of NUP and immunosuppression in HIV-positive patients. Those patients presenting with NUP were 20.8 times more likely to have CD4+ counts below 200 cells/mm^3 compared with HIV-positive patients without NUP. The authors consider a diagnosis of NUP to be a marker for immune deterioration and a predictor for the diagnosis of AIDS.[13] Others have suggested that NUP may be used as an indicator of HIV infection in undiagnosed patients. Shangase et al[32] reported that a diagnosis of NUG or NUP in systemically healthy, asymptomatic South Africans was strongly correlated with HIV infection. Of patients presenting with NUG or NUP, 39 of 56 (69.6%) were subsequently found to be HIV positive (see Chapter 19).

Etiology of Necrotizing Ulcerative Periodontitis

The etiology of NUP has not been determined, although a mixed fusiform-spirochete bacterial flora appears to play a key role. Because bacterial pathogens are not solely responsible for causing the disease, some predisposing "host" factor(s) may be necessary. Numerous predisposing factors have been attributed to NUG, including poor oral hygiene, preexisting periodontal disease, smoking, viral infections, immunocompromised status, psychosocial stress, and malnutrition.

NUP is frequently associated with a diagnosis of AIDS or a positive HIV status. Therefore clinicians should check all patients presenting with NUP to ascertain their HIV status. NUP can progress rapidly and lead to tooth exfoliation, so treatment should include local debridement, local antiplaque agents, and systemic antibiotics. Early diagnosis and treatment of NUP are crucial because the osseous defects that occur in the late stages of the disease are extremely difficult to resolve, even with extensive regenerative surgical procedures. If a child presents with NUP, severe systemic abnormalities, such as advanced malnutrition, are often present.

Microbial Flora. Assessment of the microbial flora of NUP lesions is almost exclusively limited to studies involving HIV-positive and AIDS patients, with some conflicting evidence. Murray et al[24] reported that cases of NUP in HIV-positive patients demonstrated significantly greater numbers of the opportunistic fungus *Candida albicans* and a higher prevalence of *Actinobacillus (Aggregatibacter) actinomycetemcomitans, Prevotella intermedia, Porphyromonas gingivalis, Fusobacterium nucleatum,* and *Campylobacter* species compared with HIV-negative controls. Further, they reported a low or variable level of spirochetes, which is inconsistent with the flora associated with NUG. Citing differences in microbial flora, they refuted the notion that the destructive lesions seen in HIV-positive patients were related to NUG lesions; they suggested that the flora of NUP lesions in HIV-positive patients is comparable to that of chronic periodontitis lesions, thus supporting their concept that necrotizing periodontitis in the HIV-positive patient is an aggressive manifestation of chronic periodontitis in the immunocompromised host.

In contrast to these findings, Cobb et al[4] reported that the microbial composition of NUP lesions in HIV-positive patients was very similar to that of NUG lesions, as discussed earlier. Using electron microscopy, they described a mixed microbial flora with various morphotypes in 81.3% of specimens. The subsurface microbial flora featured dense aggregations of spirochetes in 87.5% of specimens. They also reported opportunistic yeasts and herpes-like viruses in 65.6% and 56.5% of NUP lesions, respectively. The differences between these reports may be explained by the limitations in obtaining viable cultures of spirochetes[24] compared with the more definitive electron microscopic observation of spirochetes.[4]

In a recent review article, Feller and Lemmer suggested that spirochetes, herpesviruses, candida, and HIV have a potential pathogenic role in NUP lesions in the HIV-seropositive individual.[12] Spirochetes have the ability to modulate host innate and adaptive immune responses and to stimulate host inflammatory reactions,[8] which may reduce the local immune competence and facilitate the development of necrotizing disease.[12] Activated herpesviruses have the capacity to deregulate the host immune system, which may lead to an increase in the colonization and activity of other pathogenic microorganisms. *Candida albicans* has been reported to produce eicosanoids leading to the release of

SCIENCE TRANSFER

Necrotizing ulcerative gingivitis and necrotizing ulcerative periodontitis are more prevalent and more severe in patients who are HIV positive. These patients require urgent treatment as untreated lesions can progress rapidly and in a few days, severe bone loss around affected teeth can be seen.

Smoking, malnutrition, and high plaque levels all increase the risk of necrotizing ulcerative gingivitis and need to be changed so that treatment success is obtained.

Many necrotizing ulcerative gingivitis lesions respond well to initial therapy and the gingival tissues can heal and return to health. Patients need to be thoroughly reevaluated 4 to 6 weeks after managing the acute stages of necrotizing ulcerative gingivitis to ascertain if additional periodontal surgical treatment is needed to treat residual soft tissue and bony defects.

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proinflammatory mediators, which may facilitate spirochete colonization and invasion, promoting the development of necrotizing periodontal diseases.[11,12]

Immunocompromised Status. Clearly, both NUG and NUP lesions are more prevalent in patients with compromised or suppressed immune systems. Numerous studies, particularly those evaluating HIV-positive and AIDS patients, support the concept that a diminished host response is present in those individuals diagnosed with necrotizing ulcerative periodontal diseases.[37] Whereas a compromised immune system ("immune compromise") in the HIV-infected patient is driven by impaired T-cell function and altered T-cell ratios, evidence indicates that other forms of compromised immunity predispose individuals to NUG and NUP as well.

Cutler et al[6] described impaired bactericidal activity of PMNs in two children with NUP. In a comparative assay of PMNs against periodontal pathogens, two brothers (ages 9 and 14 years) showed significant depression of PMN phagocytosis and killing function compared with gender- and age-matched controls. Further, Batista et al[2] reported periodontal findings and NUP in an adolescent with a rare genetic disease (multifactorial congenital immunodeficiency [CVID]) that causes impaired secretion of immunoglobulin; the oral lesions resolved with administration of intravenous immunoglobulin (IVIG).

Psychologic Stress. Most clinical and animal studies evaluating the role of stress on necrotizing periodontal disease have evaluated subjects with NUG[7,15,33,34] and thus have not specifically addressed the role of stress on NUP.

NUG patients have been found to have had significantly more anxiety, higher depression scores, a greater magnitude of recent stressful events, more overall distress and adjustments related to these events, and more negative life events.[5,14] Although the role of stress in the development of NUP has not been reported specifically, the many similarities between NUG and NUP would suggest that similar relationships to stress may exist.

The mechanisms that predispose an individual with stress to necrotizing ulcerative periodontal diseases have not been established. However, it is well known that stress increases systemic cortisol levels, and sustained increases in cortisone have a suppressive effect on the immune response. In an investigation of 474 military personnel, Shannon et al[33] found that urinary levels of 17-hydroxycorticosteroid were higher in subjects with NUG than in all other subjects diagnosed with periodontal health, gingivitis, or periodontitis. Experimentally, noma-like lesions have been produced in rats by administering cortisone and causing mechanical injury to the gingiva[30] and in hamsters by total body irradiation.[20] Thus, stress-induced immunosuppression may be one mechanism that impairs the host response and leads to necrotizing periodontal disease. The scientific evidence supporting an etiologic role of stress in chronic periodontitis is not as clear (see Chapter 27).

Malnutrition

Direct evidence of the relationship between malnutrition and necrotizing periodontal disease is limited to descriptions of necrotizing infections in severely malnourished children. Lesions resembling NUG but with progression to become *gangrenous stomatitis,* or noma, have been described in children with severe malnutrition in underdeveloped countries. Jimenez and Baer[17] reported cases of NUG in children and adolescents ages 2 to 14 years with malnutrition in Colombia. In the advanced stages, NUG lesions extended from the gingiva to other areas of the oral cavity, becoming gangrenous stomatitis (noma) and causing exposure, necrosis, and sequestration of the alveolar bone. Later, Jimenez et al reported that 44 of the 45 cases of necrotizing disease (NUG = 29, NUP = 7, noma = 9) documented from 1965 to 2000 were from a low socioeconomic group and that malnutrition was associated with nearly all of the necrotizing conditions (29/29 NUG, 6/7 NUP and 9/9 noma cases).[16] In a study of socioeconomically deprived Nigerian children with NUG (153 cases), Enwonwu et al confirmed malnutrition by measuring circulating micronutrients.[10] Compared with neighborhood counterparts, the children with NUG and micronutrient deficiencies demonstrated dysregulated cytokine production with a complex interplay of elevated proinflammatory and antiinflammatory mediators.

A plausible explanation is that malnutrition, particularly when extreme, contributes to a diminished host resistance to infection and necrotizing disease. It is well documented that many of the host defenses, including phagocytosis; cell-mediated immunity; and complement, antibody, and cytokine production and function, are impaired in malnourished individuals.[9] Depletion of nutrients to cells and tissues results in immunosuppression and increases disease susceptibility. Thus it is reasonable to conclude that malnutrition can predispose an individual to opportunistic infections or intensify the severity of existing oral infections.

SUMMARY

NUP and NUG share many clinical and microbiologic features, but NUP is distinguished by a more severe condition with periodontal attachment and bone loss. Indeed, some patients with NUP, particularly those with compromised immunity, can have severe and rapidly progressive disease. It appears that an impaired immune response and lowered host resistance to infection are significant factors in the onset and progression of NUP. The best example of an immunocompromised host with a predisposition for NUP is the HIV-positive/AIDS patient. As with the other infection-related complications of HIV/AIDS, the immunocompromised status of these patients renders them vulnerable to opportunistic periodontal infections, including NUP. Several other factors have been identified, specifically in cases of NUG, that may also play a role in NUP, including smoking, viral infections, psychosocial stress, and malnutrition. Although none of these factors alone is sufficient to cause necrotizing disease, in combination with other immunocompromising conditions, they undoubtedly have the potential to adversely influence the host response or resistance to infection.

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CHAPTER 18

Aggressive Periodontitis

Karen F. Novak and M. John Novak

CHAPTER OUTLINE

LOCALIZED AGGRESSIVE PERIODONTITIS
Historical Background
Clinical Characteristics
Radiographic Findings
Prevalence and Distribution by Age and Gender
GENERALIZED AGGRESSIVE PERIODONTITIS
Clinical Characteristics
Radiographic Findings
Prevalence and Distribution by Age and Gender
RISK FACTORS FOR AGGRESSIVE PERIODONTITIS
Microbiologic Factors
Immunologic Factors
Genetic Factors
Environmental Factors

Aggressive periodontitis generally affects systemically healthy individuals less than 30 years of age, although patients may be older. Aggressive periodontitis may be universally distinguished from *chronic periodontitis* by the age of onset, the rapid rate of disease progression, the nature and composition of the associated subgingival microflora, alterations in the host's immune response, and a familial aggregation of diseased individuals.[17] In addition, a strong racial influence is observed in the United States (US); the disease is more prevalent among African Americans.[26]

Aggressive periodontitis describes three of the diseases formerly classified as "early-onset periodontitis." They are *localized aggressive periodontitis* (LAP), which was formerly termed *localized juvenile periodontitis* (LJP), and *generalized aggressive periodontitis* (GAP), which encompasses the diseases previously classified as *generalized juvenile periodontitis* (GJP) and *rapidly progressive periodontitis* (RPP).

LOCALIZED AGGRESSIVE PERIODONTITIS

Historical Background

In 1923, Gottlieb[13] reported a patient with a fatal case of epidemic influenza and a disease that Gottlieb called "diffuse atrophy of the alveolar bone." This disease was characterized by a loss of collagen fibers in the periodontal ligament and their replacement by loose connective tissue and extensive bone resorption, resulting in a widened periodontal space. The gingiva apparently was not involved. In 1928, Gottlieb[14] attributed this condition to the inhibition of continuous cementum formation, which he considered essential for maintenance of the periodontal fibers. He then termed the disease "deep cementopathia" and hypothesized that this was a "disease of eruption" and that cementum initiated a foreign body response. As a result, it was postulated that the host attempted to exfoliate the tooth, resulting in the observed bone resorption and pocket formation.[14]

In 1938, Wannenmacher[49] described incisor–first molar involvement and called the disease "parodontitis marginalis progressiva." Several explanations evolved for the etiology and pathogenesis of this type of disease. Many authors considered this to be a degenerative, noninflammatory disease process[12,34,45] and therefore gave it the name "periodontosis." Other investigators denied the existence of a degenerative type of periodontal disease and attributed the changes observed to trauma from occlusion.[6,33] Finally, in 1966, the World Workshop in Periodontics concluded that the concept of "periodontosis" as a degenerative entity was unsubstantiated and that the term should be eliminated from periodontal nomenclature.[38] The committee did recognize that a clinical entity different from "adult periodontitis" might occur among adolescents and young adults.

The term "juvenile periodontitis" was introduced by Chaput and colleagues in 1967[8a] and by Butler[5] in 1969. In 1971, Baer[2] defined it as "a disease of the periodontium occurring in an otherwise healthy adolescent which is characterized by a rapid loss of alveolar bone about more than one tooth of the permanent dentition. The amount of destruction manifested is not commensurate with the amount of local irritants." In 1989 the World Workshop in Clinical Periodontics categorized this disease as LJP, a subset of the broad classification of *early-onset periodontitis* (EOP).[8] Under this classification system, age of onset and distribution of lesions were of primary importance when making a diagnosis of LJP. Most recently, disease with the characteristics of LJP has been renamed *localized aggressive periodontitis* (LAP).

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Localized aggressive periodontitis (LAP) affects primarily first molars and incisor teeth in adolescents with deep pockets and advanced bone loss. It occurs in less than 1% of adolescents. The exact etiology is unknown, but there may be a connection between the disease and the presence of the anaerobic gram-negative bacteria, *Aggregatibacter actinomycetemcomitans,* in the subgingival plaque biofilm. The rate of bone loss can be 3 to 4 times faster than that seen in chronic periodontitis, although in many cases the rate of bone loss is dramatically reduced when the patients reach 20 years of age or older.

Generalized aggressive periodontitis (GAP) usually occurs first in young adults and may be present in some populations in up to 8% of the adult population. Smoking may play a role because smokers with GAP have more teeth involved and more advanced pockets than nonsmokers.

Figure 18-1 Localized aggressive periodontitis in 15-year-old black female patient who had a twin with a similar disease. **A,** Clinical view showing minimal plaque and inflammation except for localized inflammation on the distal side of the maxillary left central incisor and the mandibular right central incisor. **B,** Radiographs showing localized, vertical, angular bone loss associated with the maxillary and mandibular first molars and the mandibular central incisors. The maxillary incisors show no apparent involvement. **C,** Surgical appearance of the localized, vertical, angular bony defects affecting the mandibular incisors. Note the wide circumferential nature of the defects and the lack of calculus on the root surfaces.

Clinical Characteristics

LAP usually has an age of onset at about puberty.[22] Clinically, it is characterized as having "localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors"[22] (Figure 18-1). The localized distribution of lesions in LAP is characteristic but as yet unexplained. The following possible reasons for the limitation of periodontal destruction to certain teeth have been suggested:

1. After initial colonization of the first permanent teeth to erupt (the first molars and incisors), *Aggregatibacter* (formerly *Actinobacillus) actinomycetemcomitans* evades the host defenses by different mechanisms, including production of PMN chemotaxis-inhibiting factors, endotoxin, collagenases, leukotoxin, and other factors that allow the bacteria to colonize the pocket and initiate the destruction of the periodontal tissues. After this initial attack, adequate immune defenses are stimulated to produce opsonic

antibodies to enhance the clearance and phagocytosis of invading bacteria and neutralize leukotoxic activity. In this manner, colonization of other sites may be prevented.[51] A strong antibody response to infecting agents is one characteristic of LAP.[22]

2. Bacteria antagonistic to *A. actinomycetemcomitans* may colonize the periodontal tissues and inhibit *A. actinomycetemcomitans* from further colonization of periodontal sites in the mouth. This would localize *A. actinomycetemcomitans* infection and tissue destruction.[19]
3. *A. actinomycetemcomitans* may lose its leukotoxin-producing ability for unknown reasons.[44] If this happens, the progression of the disease may become arrested or impaired, and colonization of new periodontal sites may be averted.
4. A defect in cementum formation may be responsible for the localization of the lesions.[25,35] Root surfaces of teeth extracted from patients with LAP have been found to have hypoplastic or aplastic cementum. This was true not only of root surfaces exposed to periodontal pockets but also of roots still surrounded by their periodontium.

A striking feature of LAP is the *lack of clinical inflammation* despite the presence of deep periodontal pockets and advanced bone loss (see Figure 18-1). Furthermore, in many cases the amount of plaque on the affected teeth is *minimal,* which seems inconsistent with the amount of periodontal destruction present.[22] The plaque that is present forms a thin biofilm on the teeth and rarely mineralizes to form calculus.[48] Although the quantity of plaque may be limited, it often contains elevated levels of *A. actinomycetemcomitans,* and in some patients, *Porphyromonas gingivalis.* The potential significance of the qualitative composition of the microbial flora in LAP is discussed later in the section on risk factors.

As the name suggests, LAP *progresses rapidly.* Evidence suggests that the rate of bone loss is about three to four times faster than in chronic periodontitis.[2] Other clinical features of LAP may include (1) distolabial migration of the maxillary incisors with concomitant diastema formation, (2) increasing mobility of the maxillary and mandibular incisors and first molars, (3) sensitivity of denuded root surfaces to thermal and tactile stimuli, and (4) deep, dull, radiating pain during mastication, probably caused by irritation of the supporting structures by mobile teeth and impacted food. Periodontal abscesses may form at this stage, and regional lymph node enlargement may occur.[30]

Not all cases of LAP progress to the degree just described. In some patients the progression of attachment loss and bone loss may be self-arresting.[22]

Radiographic Findings

Vertical loss of alveolar bone around the first molars and incisors, beginning around puberty in otherwise healthy teenagers, is a classic diagnostic sign of LAP. Radiographic findings may include an "arc-shaped loss of alveolar bone extending from the distal surface of the second premolar to the mesial surface of the second molar"[33] (see Figure 18-1, *B*). Bone defects are usually wider than usually seen with chronic periodontitis (see Figure 18-1, *C*).

Prevalence and Distribution by Age and Gender

The prevalence of LAP in geographically diverse adolescent populations is estimated at less than 1%. Most reports suggest a low prevalence, about 0.2%.[26] Two independent radiographic studies of 16-year-old adolescents, one in Finland[41] and the other in Switzerland,[21] followed the strict diagnostic criteria delineated by Baer[2] and reported a prevalence rate of 0.1%. A clinical and radiographic study of 7266 English adolescents 15 to 19 years old also showed a prevalence rate of 0.1%.[40] In the US, a national survey of adolescents ages 14 to 17 reported that 0.53% had LAP.[26] Blacks were at much higher risk for LAP, and black male teenagers were 2.9 times more likely to have the disease than black female adolescents. In contrast, white female teenagers were more likely to have LAP than white male adolescents. Several other studies have found the highest prevalence of LAP among black males,[4,32,40] followed in descending order by black females, white females, and white males.[32]

LAP affects both males and females and is seen most frequently in the period between puberty and 20 years of age. Some studies have suggested a predilection for female patients, particularly in the youngest age groups,[20] whereas others report no male-female differences in incidence when studies are designed to correct for ascertainment bias.[18] (For additional epidemiologic data on LAP, see Chapter 5.)

GENERALIZED AGGRESSIVE PERIODONTITIS

Clinical Characteristics

GAP usually affects individuals under age 30, but older patients also may be affected.[22] In contrast to LAP, evidence suggests that individuals affected with GAP produce a poor antibody response to the pathogens present. Clinically, GAP is characterized by "generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors."[22] The destruction appears to occur episodically, with periods of advanced destruction followed by stages of quiescence of variable length (weeks to months or years). Radiographs often show bone loss that has progressed since the radiographic examination.

As seen in LAP, patients with GAP often have small amounts of bacterial plaque associated with the affected teeth.[22] Quantitatively, the amount of plaque seems inconsistent with the amount of periodontal destruction. Qualitatively, *P. gingivalis, A. actinomycetemcomitans,* and *Tannerella forsythia* (formerly *Bacteroides forsythus*) frequently are detected in the plaque that is present.[46]

Two gingival tissue responses can be found in cases of GAP. One is a severe, acutely inflamed tissue, often proliferating, ulcerated, and fiery red. Bleeding may occur spontaneously or with slight stimulation. Suppuration may be an important feature. This tissue response is believed to occur in the destructive stage, in which attachment and bone are actively lost.

In other cases the gingival tissues may appear pink, free of inflammation, and occasionally with some degree of stippling, although stippling may be absent (Figure 18-2, *A*). However, despite the apparently mild clinical appearance, deep pockets can be demonstrated by probing. Page and Schroeder[36] believe that this tissue response coincides with periods of quiescence in which the bone level remains stationary.

Some patients with GAP may have systemic manifestations, such as weight loss, mental depression, and general malaise.[37] Patients with a presumptive diagnosis of GAP must have their medical histories updated and reviewed. These patients should receive medical evaluations to rule out possible systemic involvement. As seen with LAP, cases of GAP may be arrested spontaneously or after therapy, whereas others may continue to progress inexorably to tooth loss despite intervention with conventional treatment.

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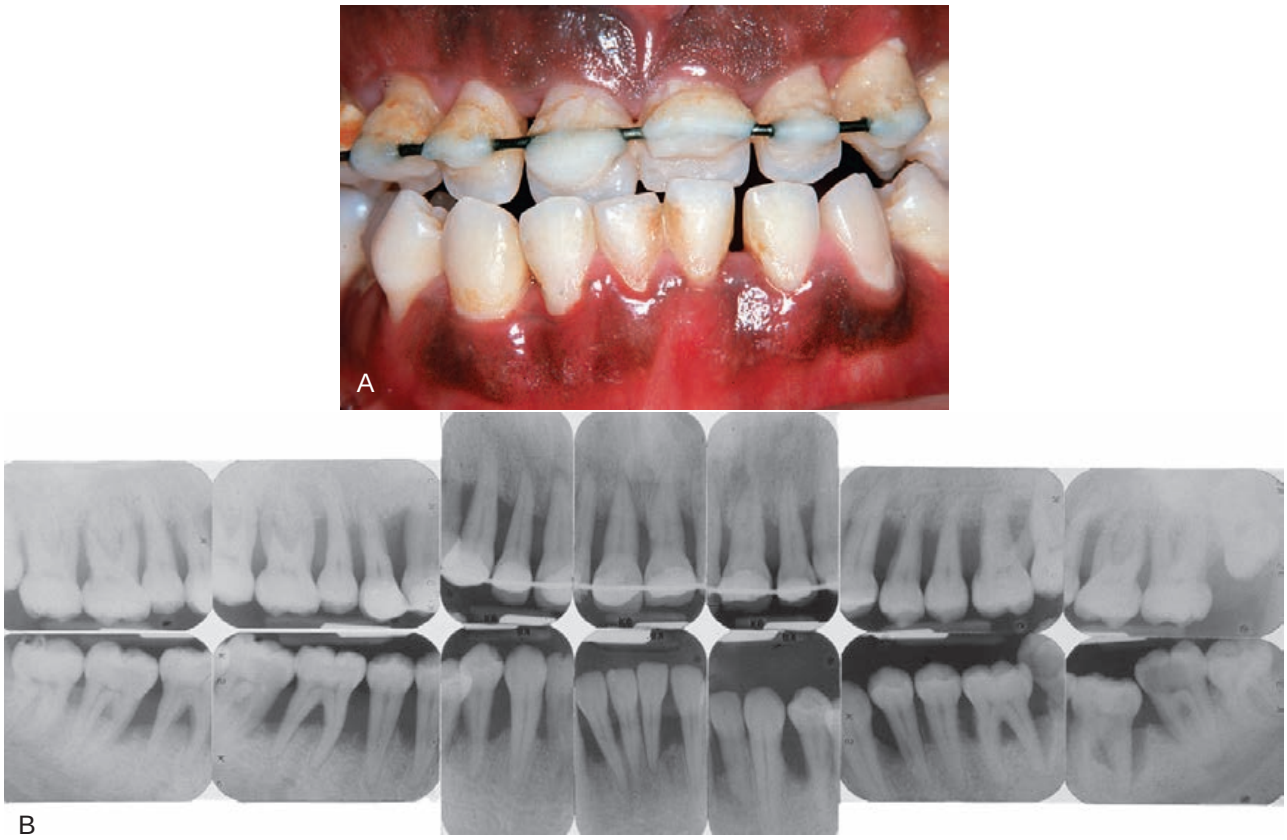

Figure 18-2 Severe generalized aggressive periodontitis in 22-year-old black male patient with a family history of early tooth loss through periodontal disease. **A,** Clinical view showing minimal plaque and inflammation. A provisional wire-and-resin splint had been placed by the general-practice dentist to stabilize the teeth. **B,** Radiographs showing the severe, generalized nature of the disease with all erupted teeth affected.

Radiographic Findings

The radiographic picture in GAP can range from severe bone loss associated with the minimal number of teeth, as described previously, to advanced bone loss affecting the majority of teeth in the dentition (see Figure 18-2, *B*). A comparison of radiographs taken at different times illustrates the aggressive nature of this disease. Page et al[37] described sites in patients with GAP that demonstrated osseous destruction of 25% to 60% during a 9-week period. Despite this extreme loss, other sites in the same patients showed no bone loss.

Prevalence and Distribution by Age and Gender

In a study of untreated periodontal disease conducted in Sri Lanka by Löe et al,[27] 8% of the population had rapid progression of periodontal disease, characterized by a yearly loss of attachment of 0.1 to 1.0 mm. A US national survey of adolescents ages 14 to 17 reported that 0.13% had GAP.[26] In addition, blacks were at much higher risk than whites for all forms of aggressive periodontitis, and male teenagers were more likely to have GAP than female adolescents (see Chapter 5).

RISK FACTORS FOR AGGRESSIVE PERIODONTITIS

Microbiologic Factors

Although several specific microorganisms frequently are detected in patients with LAP (*A. actinomycetemcomitans, Capnocytophaga* spp., *Eikenella corrodens, Prevotella intermedia,* and *Campylobacter rectus*), *A. actinomycetemcomitans* has been implicated as the primary pathogen associated with LAP. As summarized by Tonetti and Mombelli,[46] this link is based on the following evidence:

1. *A. actinomycetemcomitans* is found in high frequency (approximately 90%) in lesions characteristic of LAP.
2. Sites with evidence of disease progression often show elevated levels of *A. actinomycetemcomitans.*
3. Many patients with the clinical manifestations of LAP have significantly elevated serum antibody titers to *A. actinomycetemcomitans.*
4. Clinical studies show a correlation between reduction in the subgingival load of *A. actinomycetemcomitans* during treatment and a successful clinical response.
5. *A. actinomycetemcomitans* produces a number of virulence factors that may contribute to the disease process.

Not all reports support the association of *A. actinomycetemcomitans* and LAP. In some studies, *A. actinomycetemcomitans* either could not be detected in patients with this form of disease or could not be detected at the previously reported frequencies. Another study found elevated levels of *P. gingivalis, Prevotella intermedia, Fusobacterium nucleatum, C. rectus,* and *Treponema denticola* in patients with either localized or generalized aggressive disease, but no significant association was found between the presence of aggressive disease and *A. actinomycetemcomitans.*

In addition, *A. actinomycetemcomitans* often can be detected in periodontally healthy subjects, suggesting that this microorganism may be part of the normal flora in many individuals.

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Electron microscopy studies of LAP have revealed bacterial invasion of connective tissue[9,11] that reaches the bone surface.[7] The invading flora has been described as morphologically mixed but composed mainly of gram-negative bacteria, including cocci, rods, filaments, and spirochetes.[11] Using different methods, including immunocytochemistry, several tissue-invading microorganisms have been identified as *A. actinomycetemcomitans, Capnocytophaga sputigena, Mycoplasma* species, and spirochetes.[39]

Immunologic Factors

Some immune defects have been implicated in the pathogenesis of aggressive periodontitis. The human leukocyte antigens (HLAs), which regulate immune responses, have been evaluated as candidate markers for aggressive periodontitis. Although the findings with many HLAs have been inconsistent, HLA A9 and B15 antigens are consistently associated with aggressive periodontitis.

Several investigators[10,23,24] have shown that patients with aggressive periodontitis display functional defects of polymorphonuclear leukocytes (PMNs), monocytes, or both. These defects can impair either the chemotactic attraction of PMNs to the site of infection or their ability to phagocytose and kill microorganisms. Current studies have also demonstrated a hyperresponsiveness of monocytes from LAP patients involving their production of prostaglandin E_2 (PGE_2) in response to lipopolysaccharide (LPS).[43] This hyperresponsive phenotype could lead to increased connective tissue or bone loss caused by excessive production of these catabolic factors. Also, poorly functional inherited forms of monocyte FcγRII, the receptor for human immunoglobulin G2 (IgG2) antibodies, have been shown to be disproportionately present in patients with LAP.[50] These PMN and monocyte defects may be induced by the bacterial infection or may be genetic in origin. Further studies are needed to characterize the origin of these cellular alterations.

Autoimmunity has a role in GAP, according to Anusaksathien and Dolby,[1] who found host antibodies to collagen, deoxyribonucleic acid (DNA), and IgG. Possible immune mechanisms include an increase in the expression of major histocompatibility complex (MHC) class II molecules, HLA DR4,[3] altered helper or suppressor T-cell function, polyclonal activation of B cells by microbial plaque, and genetic predisposition.

Genetic Factors

Results from several studies support the concept that all individuals are not equally susceptible to aggressive periodontitis.[46] Specifically, several authors have described a familial pattern of alveolar bone loss and have implicated genetic factors in aggressive periodontitis.[5,28,29,31] Segregational and linkage analyses of families with a genetic predisposition for LAP suggest that a major gene or set of genes play a role in LAP and are transmitted through an autosomal dominant mode of inheritance in US populations.[31] It should be noted that most of the segregational studies were conducted in African-American populations, therefore other modes of inheritance may exist in different populations.

Evidence suggests that some immunologic defects associated with aggressive periodontitis may be inherited. For example, Van Dyke et al[47] reported a familial clustering of the neutrophil abnormalities seen in LAP. This clustering suggests that the defect(s) may be inherited.[46] Studies also have demonstrated that the antibody response to periodontal pathogens, particularly *A. actinomycetemcomitans,* is under genetic control and that the ability to mount high titers of specific, protective antibody (primarily IgG2) against *A. actinomycetemcomitans* may be race dependent.[15]

In summary, data support the concept that a gene or genes of major effect exists for aggressive periodontitis. Data also support a genetic basis for some of the immunologic defects seen in patients with aggressive periodontitis. However, it is unlikely that all patients affected with aggressive periodontitis have the same genetic defect. As summarized by Tonetti and Mombelli,[46] "it seems that specific genes may be different in various populations and/or ethnic groups and therefore true heterogeneity in disease susceptibility may be present. The role of specific genes remains to be elucidated" (see Chapter 24).

Environmental Factors

The amount and duration of smoking are important variables that can influence the extent of destruction seen in young adults.[46] Patients with GAP who smoke have more affected teeth and more loss of clinical attachment than nonsmoking patients with GAP.[16] However, smoking may not have the same impact on attachment levels in younger patients with LAP.[42]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 19

Pathology and Management of Periodontal Problems in Patients with HIV Infection

Terry D. Rees

CHAPTER OUTLINE

PATHOGENESIS (online only)
EPIDEMIOLOGY AND DEMOGRAPHICS (online only)
CLASSIFICATION AND STAGING (online only)
- CDC Surveillance Case Classification (online only)
- Antiretroviral Therapy (online only)
- Highly Active Antiretroviral Therapy (online only)

ORAL AND PERIODONTAL MANIFESTATIONS OF HIV INFECTION
- Oral Candidiasis
- Oral Hairy Leukoplakia
- Kaposi's Sarcoma and Other Malignancies
- Non-Hodgkin's Lymphoma
- Bacillary (Epithelioid) Angiomatosis
- Oral Hyperpigmentation
- Atypical Ulcers
- Salivary Gland Disorders and Xerostomia

DENTAL TREATMENT COMPLICATIONS
- Adverse Drug Effects (online only)

GINGIVAL AND PERIODONTAL DISEASES
- Linear Gingival Erythema
- Necrotizing Ulcerative Gingivitis
- Necrotizing Ulcerative Periodontitis
- Necrotizing Ulcerative Stomatitis
- Chronic Periodontitis

PERIODONTAL TREATMENT PROTOCOL
- Health Status
- Infection Control Measures
- Goals of Therapy
- Maintenance Therapy
- Psychologic Factors

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some text and figures may be out of numeric order in this printed chapter.

Acquired immunodeficiency syndrome (AIDS) is characterized by profound impairment of the immune system. The condition was first reported in 1981, and a viral pathogen, the *human immunodeficiency virus* (HIV), was identified in 1984.[179] The condition was originally believed to be restricted to male homosexuals. Subsequently, it was also identified in male and female heterosexuals and bisexuals who participated in unprotected sexual activities or abused injected drugs.[153] Currently, sexual activity and drug abuse remain the primary means of transmission.

HIV has a strong affinity for cells of the immune system, most specifically those that carry the CD4 cell surface receptor molecule. Thus helper T lymphocytes (T4 cells) are most profoundly affected, but monocytes, macrophages, Langerhans cells, and some neuronal and glial brain cells may also be involved.[102] Viral replication occurs continuously in the lymphoreticular tissues of lymph nodes, spleen, gut-associated lymphoid cells, and macrophages.[231,232]

In recent years, combined therapeutic regimens consisting of antiretroviral agents and protease-inhibiting drugs have resulted in marked improvement in the health status of HIV-infected individuals and occasionally a reduction in viral plasma bioloads below detectable levels (<50 copies per milliliter), although the infection may still be transmissible.[64,91,96,237] Current evidence indicates that the virus is never completely eradicated; rather, it is sequestered at low levels in resting CD4 cells even in individuals with no detectable plasma viral ribonucleic acid (RNA).[40,63] These findings suggest that effective combination drug therapy may be necessary for the lifetime of infected individuals. Long-term control of the infection may be difficult because the antiviral agents currently used have many adverse side effects and readily develop drug-resistant variant strains.[231]

For more information on pathogenesis, epidemiology and demographics, and classification and staging, please visit the companion Expert CONSULT website at www.expertconsult.com.

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SCIENCE TRANSFER

Patients who are positive for the human immunodeficiency virus (HIV) can be managed in the dental office in much the same way as healthy patients with respect to strict infection control measures that follow the guidance of the American Dental Association and the Centers for Disease Control and Prevention. There may be an increased risk of periodontal disease in HIV positive patients, and these patients can exhibit an exaggerated acute inflammation of the gingiva. Necrotizing lesions of the gingiva and periodontium can progress dramatically in HIV positive patients, thus it is necessary to utilize thorough local therapy combined with systemic antibiotics and local use of antimicrobial mouthwashes such as chlorhexidine and meticulous oral hygiene by the patient. HIV positive patients with these lesions should be seen daily until the tissues heal to ensure that the tissue destruction is controlled.

HIV patients can be generally managed with nonsurgical periodontal treatment. In patients with low viral loads and near normal CD4 lymphocyte count, periodontal surgery and implant placement is possible after detailed consultation and clearance from the patient's physician.

ORAL AND PERIODONTAL MANIFESTATIONS OF HIV INFECTION

Oral lesions are common in HIV-infected patients, although geographic and environmental variables may exist. Previous reports indicated that most AIDS patients have head and neck lesions,[181] whereas oral lesions are quite common in HIV-positive individuals who do not yet have AIDS.[14,76] Several reports have identified a strong correlation between HIV infection and oral candidiasis, oral hairy leukoplakia, atypical periodontal diseases, oral Kaposi's sarcoma, and oral non-Hodgkin's lymphoma.[55,58,126]

Oral lesions less strongly associated with HIV infection include melanotic hyperpigmentation, mycobacterial infections, necrotizing ulcerative stomatitis (NUS), miscellaneous oral ulcerations, and viral infections (e.g., HSV, herpes zoster, and condyloma acuminatum). Lesions seen in HIV-infected individuals but of undetermined frequency include less common viral infections (e.g., CMV, molluscum contagiosum), recurrent aphthous stomatitis, and bacillary angiomatosis (epithelioid angiomatosis).[55]

The advent of HAART has resulted in greatly diminished frequency of oral lesions associated with HIV infection and AIDS.[30,147,212]

For more information on oral lesions, please visit the companion Expert CONSULT website at www.expertconsult.com.

Oral Candidiasis

For more information on oral candidiasis, please visit the companion Expert CONSULT website at www.expertconsult.com.

Candidiasis is the most common oral lesion in HIV diseases and has been found in approximately 90% of AIDS patients.[157,203] It usually has one of four clinical presentations: pseudomembranous, erythematous, or hyperplastic candidiasis or angular cheilitis.[97,146]

Pseudomembranous candidiasis ("thrush") presents as painless or slightly sensitive, yellow-white curdlike lesions that can be readily scraped and separated from the surface of the oral mucosa. This type is most common on the hard and soft palate and the buccal or labial mucosa but can occur anywhere in the oral cavity (Figure 19-2).

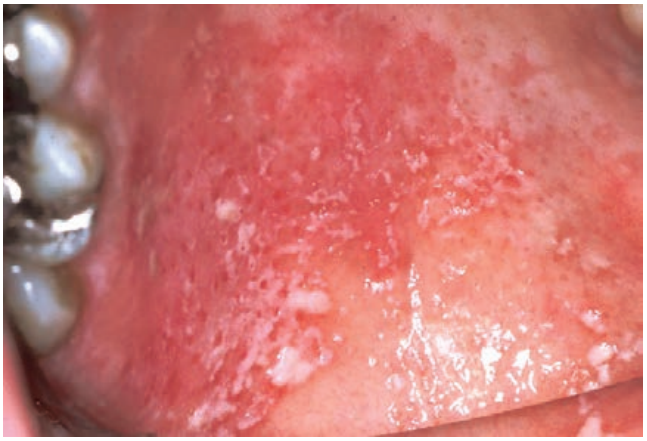

Figure 19-2 Palatal pseudomembranous candidiasis.

Figure 19-3 Palatal erythematous candidiasis.

Erythematous candidiasis may be present as a component of the pseudomembranous type, appearing as red patches on the buccal or palatal mucosa, or it may be associated with depapillation of the tongue. If gingiva is affected, it may be misdiagnosed as desquamative gingivitis (Figures 19-3 to 19-5).

Hyperplastic candidiasis is the least common form and may be seen in the buccal mucosa and tongue. It is more resistant to removal than the other types (Figures 19-6 and 19-7).

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Figure 19-4 Gingival erythematous candidiasis suggestive of desquamative gingivitis.

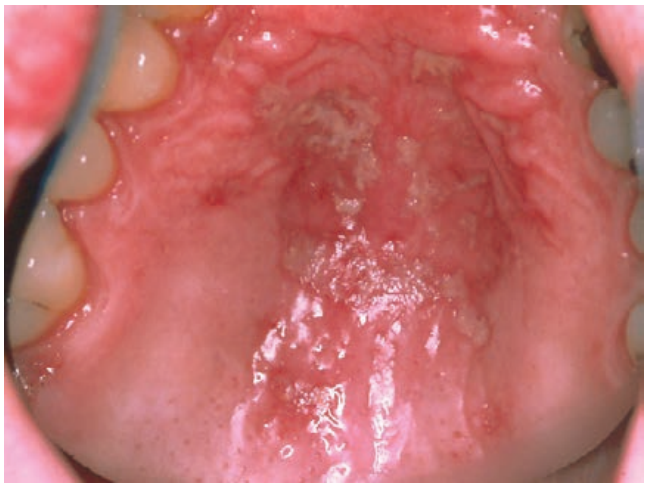

Figure 19-5 Mixed erythematous and pseudomembranous candidiasis of the palate.

In *angular cheilitis* the commissures of the lips appear erythematous with surface crusting and fissuring.

Diagnosis of candidiasis is made by clinical evaluation, culture analysis, or microscopic examination of a tissue sample or smear of material scraped from the lesion, which shows hyphae and yeast forms of the organisms (Figure 19-8). When oral candidiasis appears in patients with no apparent predisposing causes, the clinician should be alerted to the possibility of HIV infection.[55] Many patients at risk for HIV infection who present with oral candidiasis also have esophageal candidiasis, a diagnostic sign of AIDS.[214]

Oral Hairy Leukoplakia

Oral hairy leukoplakia (OHL) primarily occurs in persons with HIV infection.[84,87] Found on the lateral borders of the tongue, OHL frequently has a bilateral distribution and may extend to the ventrum. This lesion is characterized by an asymptomatic, poorly demarcated keratotic area ranging in size from a few millimeters to several centimeters (Figure 19-11, *A*). Often, characteristic vertical striations are present, imparting a corrugated appearance, or the surface may be shaggy and appear "hairy" when dried. The lesion does not rub off and may resemble other keratotic oral lesions.

For more information on oral hairy leukoplakia, please visit the companion Expert CONSULT website at www.expertconsult.com.

Figure 19-6 Mixed erythematous and hyperplastic candidiasis of corner of mouth.

Figure 19-7 Hyperplastic candidiasis in corner of mouth. Lesion has persisted despite use of systemic antifungal drugs.

Kaposi's Sarcoma and Other Malignancies

Oral malignancies occur more frequently in severely immunocompromised individuals than in the general population. An HIV-positive individual with non-Hodgkin's lymphoma (NHL) or Kaposi's sarcoma (KS) is categorized as having AIDS (Figure 19-13). Oral lesions are reported in approximately 4% of individuals with NHL, and the gingiva and palate are common sites. The incidence of oral squamous cell carcinoma may also increase in HIV-infected individuals.[19] KS was a very rare disease until the early 1980s. In its classic form, it is found in older men, occasionally in postorgan transplant individuals, and it is endemic in parts of Africa. An epidemic form has subsequently been described in HIV-positive individuals, and it is an AIDS-defining condition.[156]

KS is the most common oral malignancy associated with AIDS. This angioproliferative tumor is a rare, multifocal, vascular neoplasm; it was originally described in 1872 as occurring in the skin of the lower extremities of older men of Mediterranean origin. Today it is closely associated with homosexual and heterosexual transmission but occurs five to ten times more often in male homosexuals than in other HIV high-risk groups. The causative agent has been identified as human herpesvirus-8 (HHV-8).

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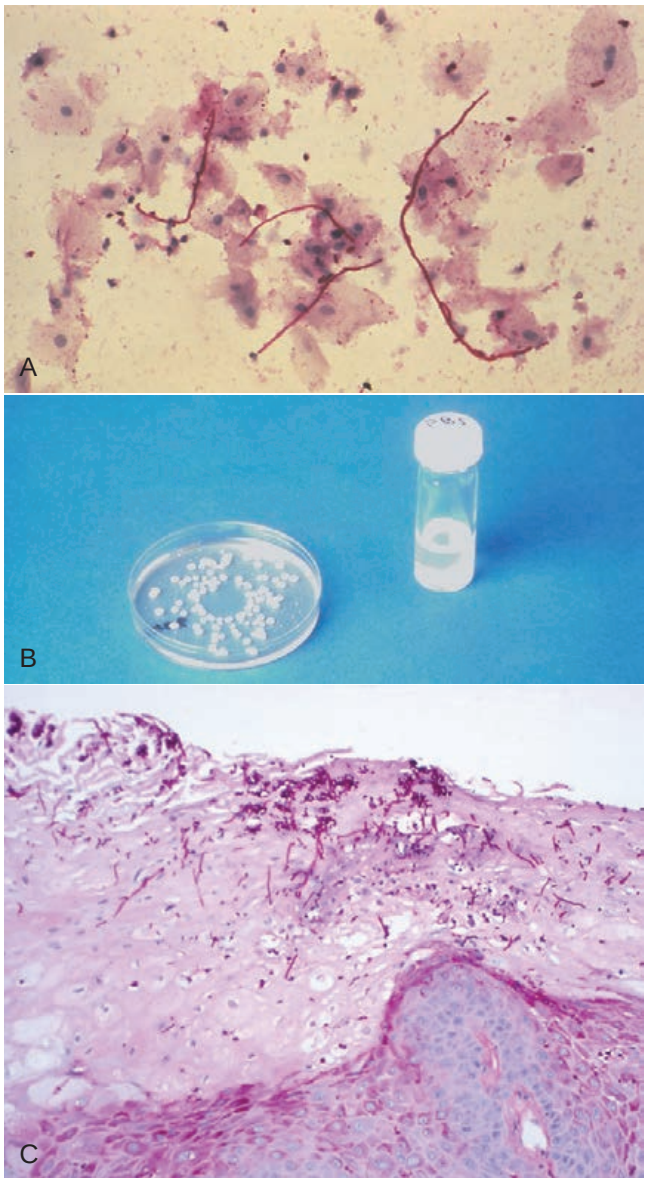

Figure 19-8 Techniques for diagnosis of candidiasis. **A,** Candidal hyphae in tissue smear after periodic acid–Schiff (PAS) staining. **B,** Culture media is specific for *Candida* species. **C,** Candidal hyphae in epithelium of biopsied tissue.

For more information on Kaposi's sarcoma, please visit the companion Expert CONSULT website at www.expertconsult.com.

Microscopically, Kaposi's sarcoma consists of four components: (1) endothelial cell proliferation with the formation of atypical vascular channels; (2) extravascular hemorrhage with hemosiderin deposition; (3) spindle cell proliferation in association with atypical vessels; and (4) a mononuclear inflammatory infiltrate consisting mainly of plasma cells[82] (Figure 19-17).

Non-Hodgkin's Lymphoma

Lymphoma represents a heterogeneous malignancy characterized by proliferation of lymphoid cells. It is broadly classified as

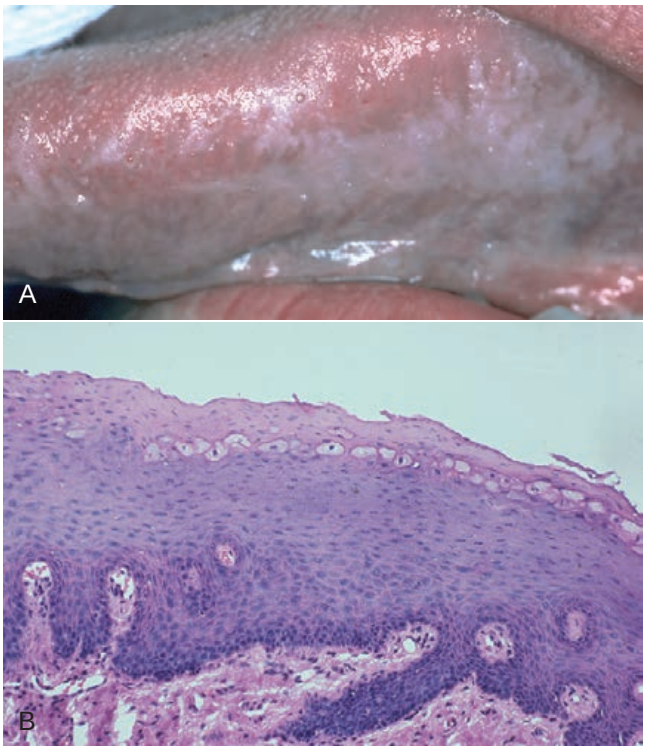

Figure 19-11 Oral hairy leukoplakia on left lateral border of tongue. **A,** Clinical view. **B,** Biopsy confirmation of oral hairy leukoplakia. Note the ballooned epithelial cells near the surface of the epithelium.

Figure 19-13 Non-Hodgkin's lymphoma of anterior mandibular gingiva.

Hodgkin's disease (14%) or non-Hodgkin lymphoma (NHL). NHL in individuals with HIV infection is an AIDS-defining condition, and elevated cumulative viremia may be a strong predictor of AIDS-related lymphoma.[239]

For more information on non-Hodgkin's lymphoma, please visit the companion Expert CONSULT website at www.expertconsult.com.

Oral lesions usually appear as erythematous, painless enlargements that may become ulcerated because of traumatic injury (Figure 19-19). In some cases, bone involvement occurs, although this is rare in the United States. Diagnosis is based on physical examination, complete blood count with differential, imaging studies, and lymph node and tissue biopsy.[141]

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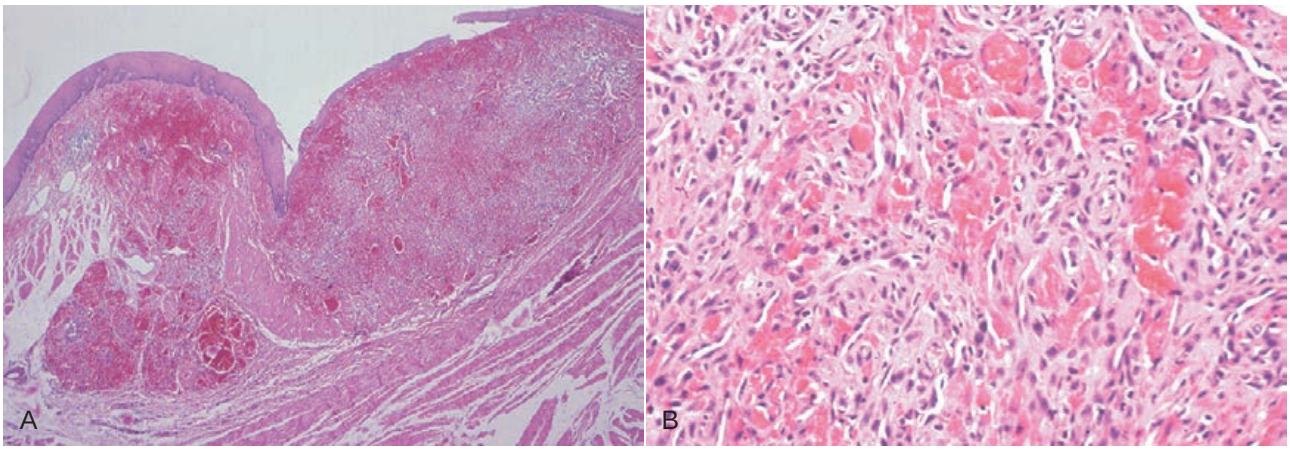

Figure 19-17 **A,** Histologic view of Kaposi's sarcoma. Lesion is exophytic and only minimally inflamed (low magnification). **B,** Note sheets of endothelial cells and numerous small blood vessels (high magnification).

Figure 19-19 Bacillary angiomatosis mimicking Kaposi's sarcoma.

Bacillary (Epithelioid) Angiomatosis

Bacillary (epithelioid) angiomatosis (BA) is an infectious vascular proliferative disease with clinical and histologic features similar to those of KS. BA is believed to be transmitted by a cat scratch and caused by rickettsia-like organisms (e.g., *Bartonella henselae* and *Bartonella quintana*).[55,173,188]

It can occur in immunocompetent persons but is most commonly associated with AIDS. Skin lesions are similar to those seen in KS or cat-scratch disease. Gingival BA manifests as red, purple, or blue edematous soft tissue lesions that may cause destruction of periodontal ligament and bone[74] (see Figure 19-19).

For more information on bacillary (epithelioid) angiomatosis, please visit the companion Expert CONSULT website at www.expertconsult.com.

Oral Hyperpigmentation

An increased incidence of **oral hyperpigmentation** has been described in HIV-infected individuals.[41,114,117,238] Oral pigmented areas often appear as spots or striations on the buccal mucosa, palate, gingiva, or tongue (Figure 19-20).

For more information on oral hyperpigmentation, please visit the companion Expert CONSULT website at www.expertconsult.com.

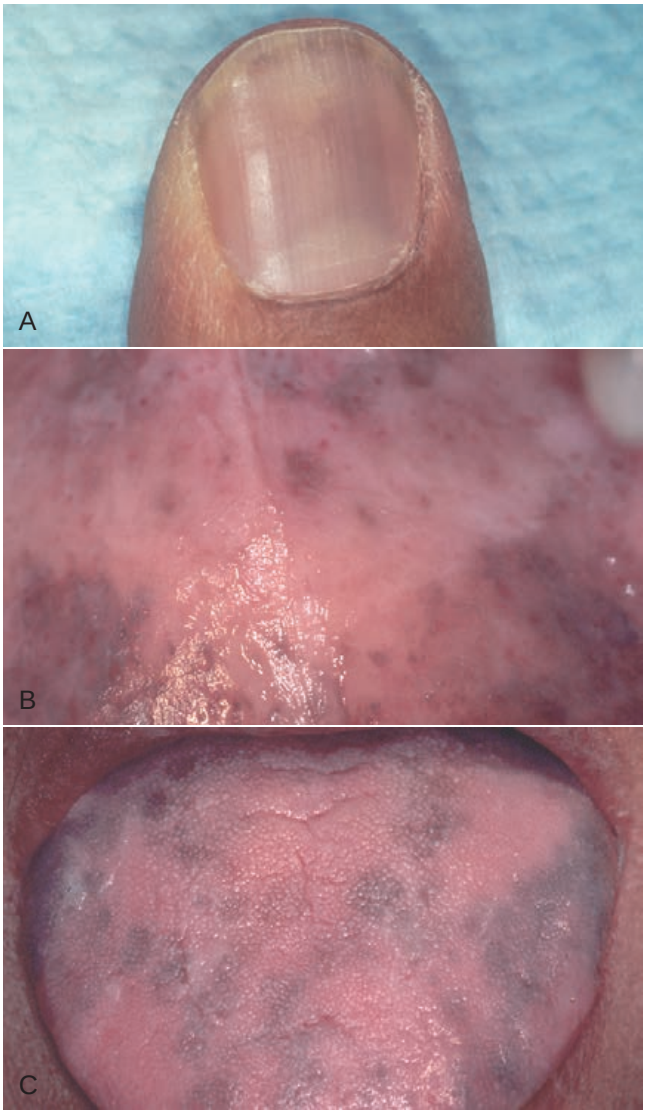

Figure 19-20 Drug-induced hyperpigmentation (zidovudine). **A,** Fingernail bed discoloration. **B,** Same patient, palatal hyperpigmentation. **C,** Same patient, tongue hyperpigmentation. Note similarity between the oral lesions and those caused by adrenal cortex hypofunction (Addison's disease).

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Atypical Ulcers

Atypical ulcers (nonspecific oral ulcerations) in HIV-infected individuals may have multiple etiologies that include neoplasms such as lymphoma, KS, and squamous cell carcinoma. HIV-associated neutropenia may also feature oral ulcerations.

For more information on atypical ulcers, please visit the companion Expert CONSULT website at www.expertconsult.com.

HIV-infected patients have a higher incidence of recurrent herpetic lesions and aphthous stomatitis (Figures 19-21 and 19-22).

Salivary Gland Disorders and Xerostomia

For more information on salivary gland disorders and xerostomia, please visit the companion Expert CONSULT website at www.expertconsult.com.

Salivary gland hypofunction and xerostomia may be most common in HIV-infected men in both early and advanced stages of HIV infection and immunosuppression.

DENTAL TREATMENT COMPLICATIONS

Concerns have been expressed regarding the potential for postoperative complications (hemorrhage, infection, or delayed wound healing) in individuals with HIV/AIDS. Medically compromised patients must be carefully managed in the dental office to avoid undue treatment complications.[48] However, *systematic reviews of the literature indicate that special precautions are usually not necessary based simply on the HIV status of patients when performing periodontal treatment procedures such as dental prophylaxis, scaling and root planing, periodontal surgery, extractions, and placement of implants*. On occasion, however, the poor overall health status of an individual with AIDS may limit periodontal therapy to conservative, minimally invasive procedures, and antibiotic therapy may be required.[8,51,52,73,158] When possible, antibiotics should be avoided in significantly immunocompromised individuals to minimize the risk of OIs (i.e., candidiasis), superinfection, and microorganism drug resistance.[140]

For more information on adverse drug effects, please visit the companion Expert CONSULT website at www.expertconsult.com.

GINGIVAL AND PERIODONTAL DISEASES

Considerable research has focused on the nature and incidence of periodontal diseases in HIV-infected individuals. Some studies suggest that chronic periodontitis is more common in this patient population, but others do not. Periodontal diseases are more common among HIV-infected users of injection drugs, but this may relate to poor oral hygiene and lack of dental care rather than decreased CD4 cell counts.[89,116,161] However, some unusual types of

Figure 19-21 Abnormal herpetic lesions in HIV-positive individuals. **A,** Lip crusting of primary herpetic gingivostomatitis. **B,** Same patient, ulcerations of gingiva, alveolar mucosa, and vestibule. **C,** Severe herpes labialis in commissures of lip. D, Close-up view of herpes labialis. Note fluid-filled vesicles.

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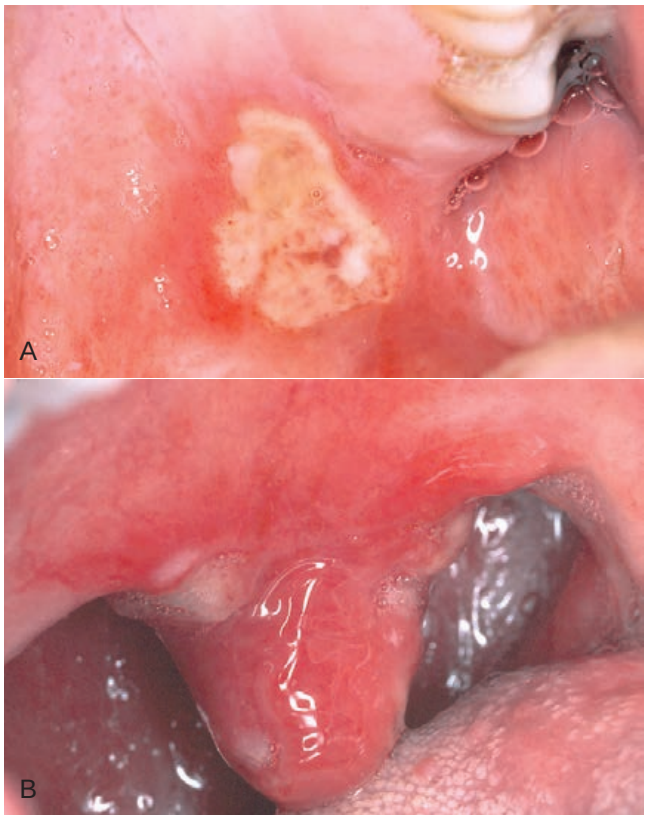

Figure 19-22 Intraoral aphthous stomatitis in AIDS patient. **A,** Major aphthous lesion in left soft palate. **B,** Same patient, ulcerations of uvula.

Figure 19-26 Linear gingival erythema (LGE) and necrotizing ulcerative gingivitis (NUG) in AIDS patient.

periodontal diseases do seem to occur with greater frequency in HIV-positive individuals.

Linear Gingival Erythema

A persistent, linear, easily bleeding, erythematous gingivitis has been described in some HIV-positive patients. Linear gingival erythema (LGE) may or may not serve as a precursor to rapidly progressive NUP[75,77] (Figures 19-26 and 19-27). The microflora of LGE may closely mimic that of periodontitis rather than gingivitis. However, candida infection has been implicated as a major etiologic factor, and human herpesviruses have been proposed as possible triggers or cofactors.[5,44,184,207,218] Linear gingivitis lesions may be localized or generalized in nature. The erythematous gingivitis (1) may be limited to marginal tissue, (2) may extend into attached gingiva in a punctate or a diffuse erythema, or (3) may extend into the alveolar mucosa.

Figure 19-27 Mild LGE. Patient had a T4 count of 9 and a viral load too numerous to count.

LGE is sometimes unresponsive to corrective therapy, but such lesions may undergo spontaneous remission. Concomitant oral candidiasis and LGE lesions have been identified, suggesting a possible etiologic role for candidal species in LGE.[116] In one study, direct microscopic cultures from LGE lesions implicated *Candida dubliniensis* in four patients, all of whom experienced complete or partial remission after systemic antifungal therapy.[223] It is not yet known whether candidal infections are etiologic in all LGE cases. A recent systematic review indicates that LGE is more common among HIV-infected populations but that most individuals who are HIV positive do not experience LGE.[157]

LGE-like lesions can often be adequately managed by following the therapeutic principles associated with marginal gingivitis. However, it has been suggested that gingivitis lesions that respond to conventional therapy do not represent LGE.[215] The affected sites should be scaled and polished. Subgingival irrigation with chlorhexidine or 10% povidone-iodine may be beneficial. The patient should be carefully instructed in performance of meticulous oral hygiene procedures. The condition should be reevaluated 2 to 3 weeks after initial therapy. If the patient is compliant with home care procedures and the lesions persist, the possibility of a candidal infection should be considered. It is doubtful that topical antifungal rinses will reach the base of the gingival crevices. Consequently, the treatment of choice may be the empiric administration of a systemic antifungal agent, such as fluconazole, for 7 to 10 days.[98]

It is important to remember that LGE may be refractory to treatment. If so, the patient should be carefully monitored for developing signs of more severe periodontal conditions (e.g., NUG, NUP, or NUS). The patient should be placed on a 2- to 3-month recall maintenance interval and re-treated as necessary. As mentioned, despite the occasional resistance of LGE to conventional periodontal therapy, spontaneous remission may occur for reasons that are not yet known.

Necrotizing Ulcerative Gingivitis

For more information on NUG and NUP, please visit the companion Expert CONSULT website at www.expertconsult.com.

There is no consensus on whether the incidence of necrotizing ulcerative gingivitis (NUG) increases in HIV-positive patients.[155,158,177,197] However, a recent study evaluated the HIV status of individuals who presented with necrotizing periodontal diseases, and 69% were found to be HIV seropositive.[197] Treatment of NUG in these patients does not differ from that in HIV-negative individuals (see Chapter 41).

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Necrotizing Ulcerative Periodontitis

A necrotizing, ulcerative, rapidly progressive form of periodontitis occurs more frequently among HIV-positive individuals, although such lesions were described long before the onset of the AIDS epidemic. Necrotizing ulcerative periodontitis (NUP) may represent an extension of NUG in which bone loss and periodontal attachment loss occur.[148,197]

NUP is characterized by soft tissue necrosis, rapid periodontal destruction, and interproximal bone loss[77,166] (Figures 19-28, 19-29, and 19-30). Lesions may occur anywhere in the dental arches and are usually localized to a few teeth, although generalized NUP is sometimes present after marked CD4+ cell depletion. Bone is often exposed, resulting in necrosis and subsequent sequestration. NUP is severely painful at onset, and immediate treatment is necessary. Occasionally, however, patients undergo spontaneous resolution of the necrotizing lesions, leaving painless, deep interproximal craters that are difficult to clean and that may lead to conventional periodontitis.[75]

Therapy for NUP includes local debridement, scaling and root planing, in-office irrigation with an effective antimicrobial agent, such as chlorhexidine gluconate or povidone-iodine (Betadine), and establishment of meticulous oral hygiene, including home use of antimicrobial rinses or irrigation.[158,165,187]

In severe NUP, antibiotic therapy may be necessary but should be used with caution in HIV-infected patients to avoid an opportunistic and potentially serious, localized candidiasis or even candidal septicemia.[166] If an antibiotic is necessary, metronidazole (250 mg, with 2 tablets taken immediately and then 2 tablet 4 times daily for 5 to 7 days) is the drug of choice. Prophylactic prescription of a topical or systemic antifungal agent is prudent if an antibiotic is used.

Figure 19-28 Necrotizing ulcerative periodontitis (NUP). Note adjacent LGE.

For more information on NUS, please visit the companion Expert CONSULT website at www.expertconsult.com.

Chronic Periodontitis

A number of longitudinal or prevalence studies have suggested that HIV-positive individuals are more likely to experience chronic periodontitis than the general population.[10,134,143,174] Most studies, however, do not take into account the level of oral hygiene or the degree of immunodeficiency in the population studied or whether

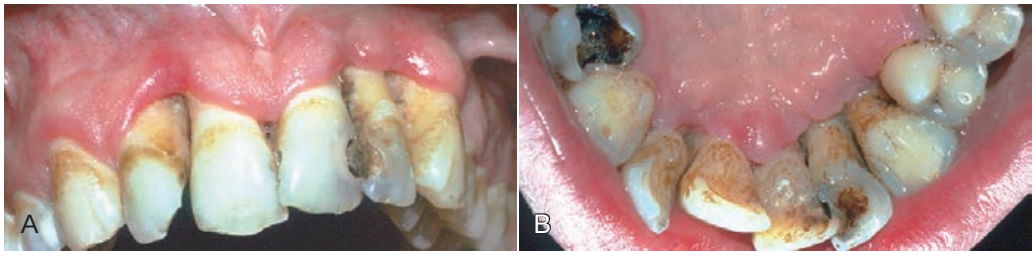

Figure 19-29 NUP in otherwise healthy 19-year-old male patient who was HIV negative. **A,** Anterior maxilla. **B,** Palatal view.

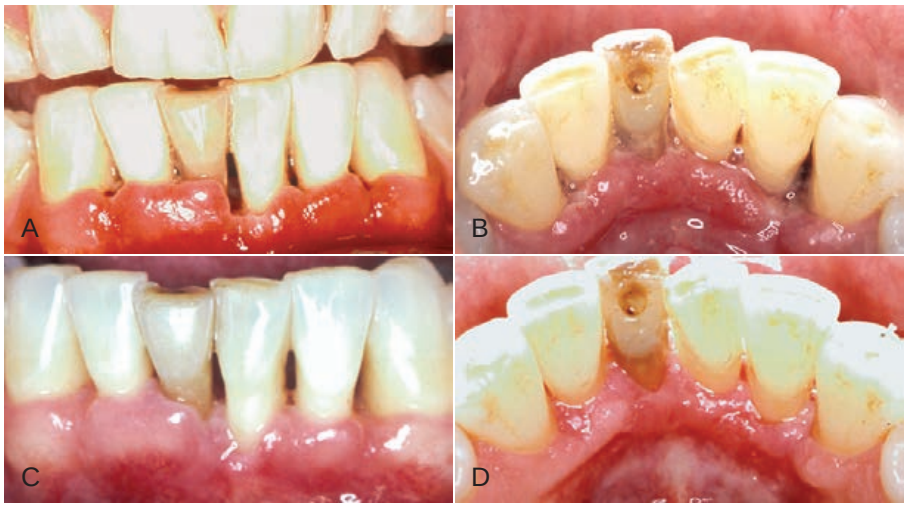

Figure 19-30 Early NUP in patient with AIDS. **A,** Facial view. **B,** Lingual view. **C,** Complete resolution of NUP after treatment, facial view. **D,** Lingual view.

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Figure 19-32 Periodontal health in an individual with advanced AIDS.

the individuals in the study are injection drug users (IDUs); these confounding factors cloud the issue.

For more information on chronic periodontitis, please visit the companion Expert CONSULT website at www.expertconsult.com.

With proper home care and appropriate periodontal treatment and maintenance, HIV-positive individuals can anticipate reasonably good periodontal health throughout the course of their disease.[110] The median period between initial HIV infection and outright AIDS is approximately 15 years, and the life expectancy of persons living with AIDS has been significantly prolonged with current anti-HIV drug therapy.[20,34]

This indicates that HIV-infected patients are potential candidates for conventional periodontal treatment procedures including periodontal surgery and implant placement. Treatment decisions should be based on the overall health status of the patient, the degree of periodontal involvement, and the motivation and ability of the patient to perform effective oral hygiene (Figure 19-32).

Clearly, some less common periodontal diseases do occur more frequently in HIV-positive individuals, but these same conditions are also reported among HIV-negative persons. Consequently, definitions for these conditions and discussion of their management should not be construed as limiting them to individuals with HIV or AIDS.

PERIODONTAL TREATMENT PROTOCOL

It is imperative that medically compromised patients, including those with HIV or AIDS, be safely and effectively managed in dental practice.[198] Several universal treatment considerations are important to ensure that this is achieved.

Health Status

The patient's health status should be determined from the health history, physical evaluation, and consultation with the patient's physician. Treatment decisions will vary depending on the patient's state of health. For example, delayed wound healing and increased risk of postoperative infection are possible complicating factors in AIDS patients, but neither concern should significantly alter treatment planning in an otherwise healthy, asymptomatic, HIV-infected patient with a normal or near-normal CD4 count and a low viral bioload.[68,92,126,168]

For more information on health status, please visit the companion Expert CONSULT website at www.expertconsult.com.

Infection Control Measures

Clinical management of HIV-infected periodontal patients requires strict adherence to established methods of infection control, based on guidance from the American Dental Association (ADA) and the CDC. Compliance, especially with universal precautions, will eliminate or minimize risks to patients and the dental staff.[68,123,224] Immunocompromised patients are potentially at risk for acquiring as well as transmitting infections in the dental office or other health care facility.[119,139,149]

Goals of Therapy

For more information on goals of therapy, please visit the companion Expert CONSULT website at www.expertconsult.com.

The primary goals of dental therapy should be the restoration and maintenance of oral health, comfort, and function. At the very least, periodontal treatment goals should be directed toward control of HIV-associated mucosal diseases such as chronic candidiasis and recurrent oral ulcerations. Acute periodontal and dental infections should be managed, and the patient should receive detailed instructions in performance of effective oral hygiene procedures.[73,98]

Conservative, nonsurgical periodontal therapy should be a treatment option for virtually all HIV-positive patients, and performance of elective surgical periodontal procedures, including implant placement, has been reported.[72,187] NUP or NUS can be severely destructive to periodontal structures, but a history of these conditions does not automatically dictate extraction of involved teeth unless the patient is unable to maintain effective oral hygiene in affected areas. Decisions regarding elective periodontal procedures should be made with the informed consent of the patient and after medical consultation, when possible.

Maintenance Therapy

It is imperative that the patient maintain meticulous personal oral hygiene. In addition, periodontal maintenance recall visits should be conducted at short intervals (2 to 3 months) and any progressive periodontal disease treated vigorously.[235]

For more information on health maintenance, please visit the companion Expert CONSULT website at www.expertconsult.com.

Psychologic Factors

For more information on psychologic factors, please visit the companion Expert CONSULT website at www.expertconsult.com.

Patients may be greatly concerned with maintenance of medical confidentiality, and such confidentiality must be upheld. Coping with a life-threatening disease may elicit depression, anxiety, and anger in such patients, and this anger may be directed toward the dentist and the staff. It is important to display concern and understanding for the patient's situation. Treatment should be provided in a calm, relaxed atmosphere, and stress to the patient must be minimized.[4]

The dentist should be prepared to advise and counsel patients on their oral health status. Dentists often encounter HIV-infected

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patients who are unaware of their disease status. Early diagnosis and treatment of HIV infection can have a profound effect on the patient's life expectancy and quality of life, and the dentist should be prepared to assist the patient in obtaining testing.[192,199] Any patient with oral lesions suggestive of HIV infection should be informed of the findings and, if appropriate, questioned regarding any previous exposure to HIV. If HIV testing is requested, it must be accompanied by patient counseling; therefore, it might be best to obtain such tests through medical referral. However, if the dentist elects to request testing for HIV antibody, the patient must be informed. In most circumstances, written informed consent is desirable before testing.

References and suggested readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 20

Masticatory System Disorders

Michael J. McDevitt

CHAPTER OUTLINE

TEMPOROMANDIBULAR JOINT
MUSCLES AND NERVES OF THE MASTICATORY SYSTEM
CENTRIC RELATION
BIOMECHANICS OF THE MASTICATORY SYSTEM
DYSFUNCTION AND DETERIORATION
OROFACIAL PAIN
COMPREHENSIVE EVALUATION
Patient History and Interview
Clinical Examination
Imaging
DIAGNOSTIC DECISION MAKING (online only)

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

The masticatory system consists of the temporomandibular joints (TMJs), masticatory muscles, teeth in occlusion, and neurologic and vascular supplies supporting all these structures. Research suggests that masticatory system disorders include many varied conditions with multiple possible contributing factors, rather than different manifestations of a single disease or syndrome.[2,78,123] The ability to understand the anatomy and function of the masticatory system and correctly interpret relevant diagnostic information is a prerequisite to fulfilling comprehensive standards of care. Our diagnostic process must be broad based and inclusive enough to determine the most appropriate cause of masticatory dysfunction.[131]

TEMPOROMANDIBULAR JOINT

Harmonious function of the TMJs is a product of the coordination of the muscles of mastication by intricate mechanisms of neurologic control. Understanding the dynamics and the relationship of the TMJ to the associated muscles and nerves provides the working knowledge required for effective assessment and diagnosis.

The TMJ is one of the most complex joints in the human body. It is capable of providing both hinging (rotation) and gliding (translation) movements and is able to resist incredible forces of mastication. The TMJ is formed by the head of the condyle of the mandible as it fits into the articular fossa of the temporal bone (Figure 20-1). The body of the mandible effectively connects both condyles so that neither condyle functions independently of the other. Interposed between the head of the condyle and the articular surface of the temporal bone is the articular disc, consisting of dense connective tissue, resulting in a compound joint with two joint cavities (Figure 20-2). The articulating surfaces of the osseous structures are essentially convex in a healthy situation, so the biconcave configuration of the articular disc compensates for the opposing convexities. The articular surfaces of the condyles and temporal bones consist of fibrous connective tissue, rendering them resistant to breakdown and capable of repair. Deep to the superficial connective tissue layer, articular cartilage provides the cellular and structural basis for the response to the functional loading and movement of the TMJs.[78,131,177] The discal ligaments and attachments to the capsule, along with the disc itself, become the means of separating the joint into superior and inferior joint spaces (see Figures 20-1 and 20-2). Synovial lubrication of the articular surfaces is a function of synovial fluid production by endothelial cells along the borders of each joint cavity and at the anterior extent of the retrodiscal tissues.*

MUSCLES AND NERVES OF THE MASTICATORY SYSTEM

For more information on muscles and nerves of the masticatory system, please visit the companion Expert CONSULT website at www.expertconsult.com.

The muscles of mastication consist principally of two groups: the *elevator muscles* and *depressor muscles.* The muscles responsible for elevating the mandible are the masseter, internal pterygoid, and

*References 40, 41, 78, 131, 172, 174, and 177.

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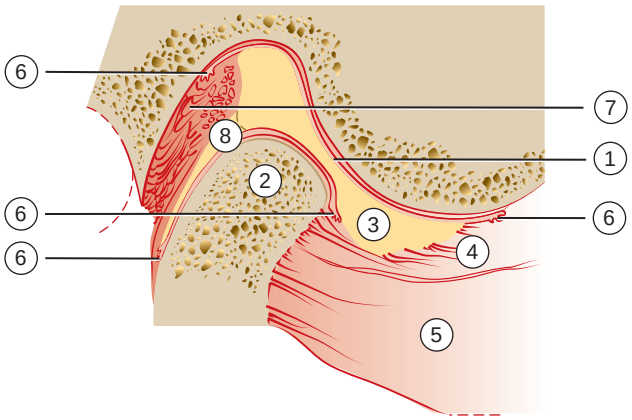

Figure 20-1 Lateral view of cross-section through temporomandibular joint. *1,* Posterior slope of articular eminence of temporal bone; *2,* head of condyle; *3,* articular disc (note biconcave shape); *4,* superior lateral pterygoid muscle (note attachment to both head of condyle and articular disc); *5,* inferior lateral pterygoid muscle; *6,* synovial tissue; *7,* retrodiscal tissue; *8,* discal ligament attachment to the posterior surface of head of condyle. (Modified from Dawson PE: *Evaluation, diagnosis, and treatment of occlusal problems,* ed 2, St Louis, 1989, Mosby.)

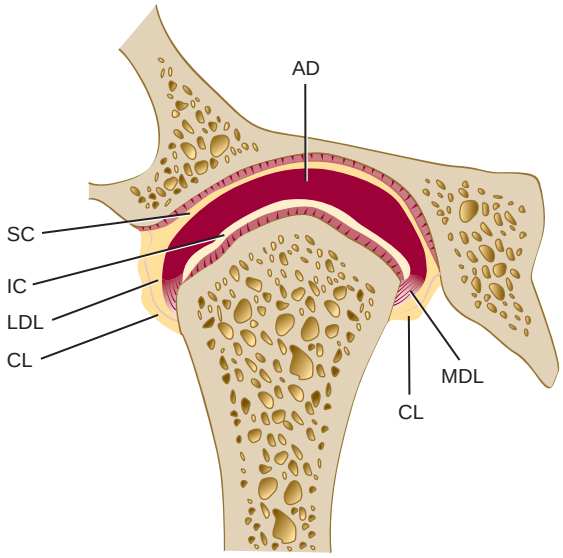

Figure 20-2 Temporomandibular joint (anterior view), showing collateral ligaments. *AD,* articular disc; *CL,* capsular ligament; *IC,* inferior joint cavity; *LDL,* lateral discal ligament; *MDL,* medial discal ligament; *SC,* superior joint cavity. (From Okeson JP: *Management of temporomandibular joint disorders and occlusion,* ed 4, St Louis, 1998, Mosby.)

much of the temporal muscle. The posteriorly oriented fibers of the temporal muscle also retrude the mandible. The superficial muscle bundle of the masseter muscle may also assist in protruding the mandible while the deeper bundle serves to stabilize the condylar head against the articular eminence. Juxtaposed with the masseter muscle, the medial pterygoid forms a muscular support for the mandible at its angle. Although the primary function of elevation of the mandible, it is also active during protrusion.[78,131] The lateral pterygoid muscle is now known to function as two distinct muscles, the inferior and superior lateral pterygoid muscles, with independent and almost opposite functions.[6,14] The inferior lateral pterygoid muscle depresses and protrudes the mandible. The superior lateral pterygoid muscle does not contract during depression of the mandible but rather contracts along with the elevator muscles, bracing the condyle anteromedially.[110,119,129,131,151]

Motor and sensory innervation of the TMJs and the rest of the masticatory system are provided by structures of the trigeminal nerve.

The innervation of both the capsular ligaments and the discal ligaments provide essential proprioceptive input with regard to joint position. Efferent or motor neurons cause muscle contraction in response to central cortical stimulation and in response to afferent stimuli in reflex activity.[40,131,177]

Pain perception causes the nociceptive reflex to open the mouth rapidly through contraction of depressor muscles and suppression of elevator muscles, consistent with other protective reflexes within the musculoskeletal system.[131] Protective reflexes may be suppressed in individuals experiencing chronic occlusal parafunction (clenching or grinding of teeth).[25,49] Pressure perception is a function of the numerous mechanoreceptors within the PDL of teeth in contact. Discrimination within the dentition based on specific teeth in contact, direction of force, and intensity of force and their influence on muscle activity have been demonstrated in human study populations and animal studies.[22,37,99,109,154] Both research and clinical observations suggest that elevator muscle contraction is suppressed when anterior teeth promote disclusion or separation of posterior teeth during excursive mandibular movements.[173]

Patients with significant bone loss, significant inflammatory disruption of the integrity of the PDL, or chronic occlusal parafunction may experience compromised regulation of muscle activity.*

CENTRIC RELATION

The mandible is suspended from the cranial base by ligaments and muscles. Understanding mandibular movement begins with an initial reference point for each condyle, usually referred to as *centric relation;* this clinically determined relationship of the mandible to the maxilla occurs when both condyle-disc assemblies are positioned in their most superior position in the maxillary (or glenoid) fossa and against the slope of the articular eminence of the temporal bone. Verification of centric relation is obtained by loading the TMJs bilaterally with the teeth apart, using the bimanual mandibular manipulation technique advocated by Dawson and others.[40,41,42,43,165] When both condyles are in this relationship, rotation or hinging action occurs around an axis defined by the medial poles of each condyle (Figure 20-3).

For more information on centric relation, please visit the companion Expert CONSULT website at www.expertconsult.com.

For TMJs to maintain orthopedic stability, the condyles must remain fully seated in their respective fossae when the teeth occlude in maximal intercuspation. Orthopedic instability occurs when the occlusal relationships are such that contraction of elevator muscles is required to achieve stable occlusion in maximal intercuspal position resulting in the unseating of one or both condyles from their respective fossae (Figure 20-4).

An individual's susceptibility to masticatory system disorders determines whether that person adapts with minimal consequence or develops dysfunction or degeneration.[24,40,41,131,165]

*References 2, 24, 49, 75, 78, 124, 131, and 149.

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BIOMECHANICS OF THE MASTICATORY SYSTEM

Biomechanics of mandibular movement are a function of neurologic input from cortical and stomatognathic sources acting to initiate or restrict muscular contraction. Muscular action either stabilizes the condyle against the articular eminence or directs its rotational and translational movements relative to each respective temporal bone. The position and functional movement of one condyle always depend on the status or activity of the other.

For more information on biomechanics of the masticatory system, please visit the companion Expert CONSULT website at www.expertconsult.com.

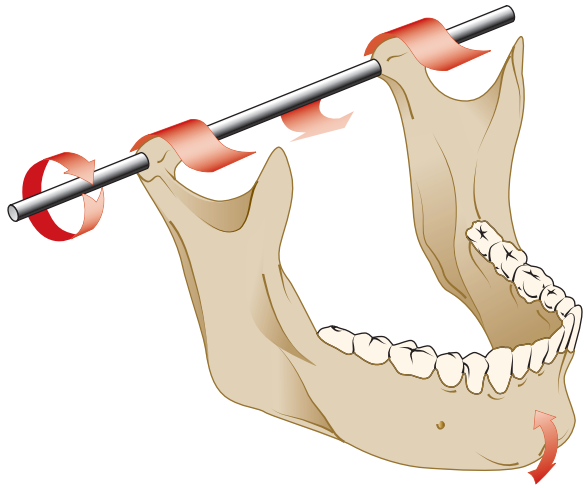

Figure 20-3 In centric relation, condyles can rotate on a fixed axis. As long as the rotational axis stays fixed at the most superior position against the eminentiae, the mandible can open or close and still be in centric relation. If the condyle axis moves forward, it is no longer in centric relation. (From Dawson PE: *Evaluation, diagnosis, and treatment of occlusal problems,* ed 2, St Louis, 1989, Mosby.)

The mandible can move within a range of motion (ROM) that is limited by skeletal, muscular, and ligamentous structures. Pure rotation of up to approximately 25 mm can occur before translation of the condyle is required to continue toward maximal opening of the jaw. Movement of the condyle is relative to the disc, so rotation effectively occurs within the inferior joint space (Figure 20-5, *A*). Strict translation protrudes the mandible while the condyle-disc assembly moves anterior and inferior toward the articular eminence of the temporal bone.

The disc moves relative to the temporal bone, and movement occurs within the upper joint space (see Figure 20-5, *B*). In combination translation/rotation movement of the condyle, the axis of

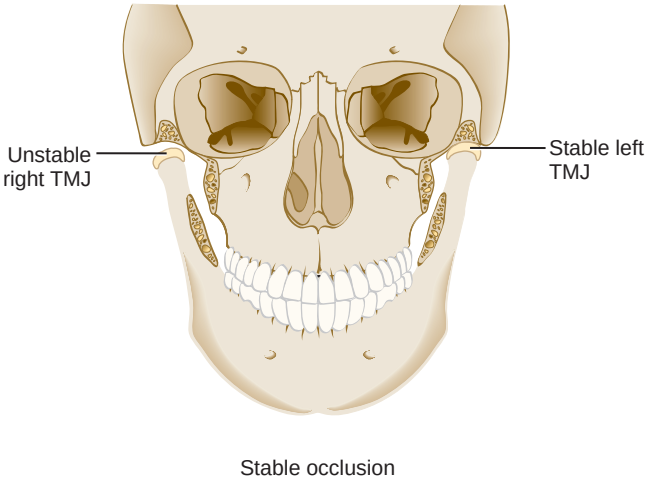

Figure 20-4 Example of orthopedic instability. Note that with the teeth in their stable position (maximum intercuspation), the left temporomandibular joint (TMJ) is in a stable relationship with the fossa. The right TMJ, however, is not in a stable position in the fossa. When the elevator muscles contract, the right condyle moves superiorly, seeking a more stable relationship with the articular disc and fossa (the musculoskeletally stable position). This type of loading can lead to an intracapsular disorder. (From Okeson JP: *Management of temporomandibular joint disorders and occlusion,* ed 4, St Louis, 1998, Mosby.)

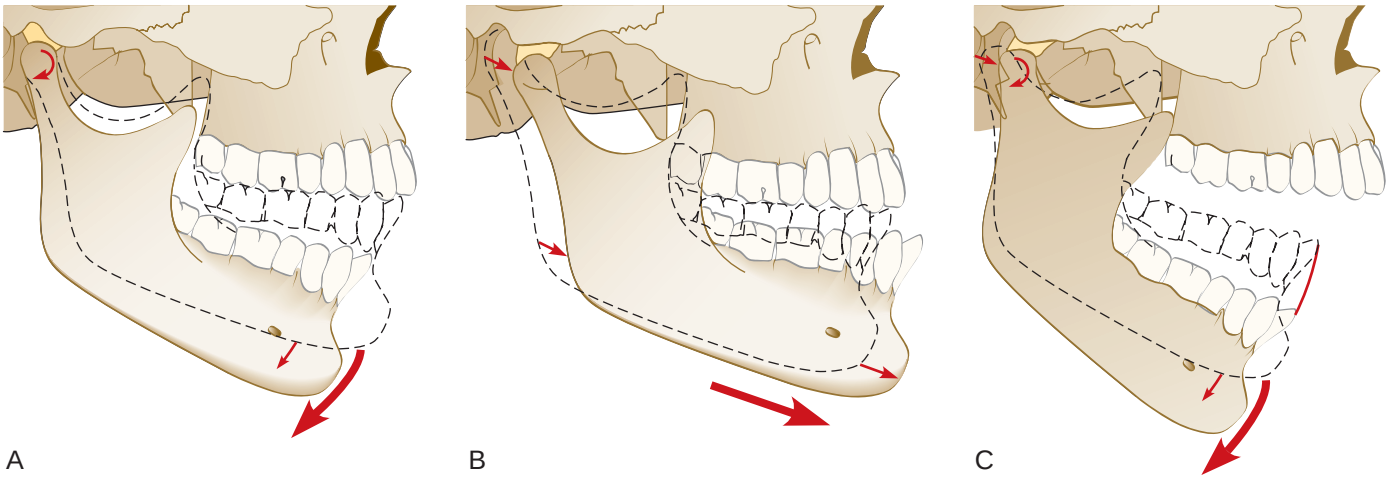

Figure 20-5 **A,** Rotational movement of mandible with condyles in centric relation. This pure rotational opening can occur until the anterior teeth are about 20 to 25 mm apart. **B,** Translational movement of the condyle-disc assembly during protrusion of the mandible. **C,** Second stage of rotational movement during opening. Note the dual activity relative to the articular disc. *1,* Rotation of the condylar head, relative to the disc, occurs in the inferior space. *2,* Movement of the disc anteriorly and inferiorly along the articular surface of the temporal bone. The articular disc moves anteriorly and inferiorly with the head of the condyle, which continues to rotate against the disc. Translation occurs in the superior joint space, and rotation occurs in the inferior joint space. (From Okeson JP: *Management of temporomandibular joint disorders and occlusion,* ed 4, St Louis, 1998, Mosby.)

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rotation for each condyle changes as the condyle translates down the articular eminence to a position inferior to its fossa (see Figure 20-5, *C*). Harmonious muscle function and ligament attachments keep the condyle-disc assembly properly related so that the articular disc remains loaded in its concave, avascular central portion between the condyle and the articular surface of the temporal bone. The elasticity and vascularity of the retrodiscal tissues permit anterior movement of the disc during translation of each respective condyle.

When the teeth are in contact, their ability to influence both the position and the direction of movement of condyle-disc assemblies is defined by the intensity of muscle activity and the steepness of the inclines of those teeth.*

DYSFUNCTION AND DETERIORATION

Ideally, function never exceeds the integrity or adaptive limits of the structural elements of the masticatory system. Clinical experience shows that the tolerance of the components of the masticatory system can be exceeded by both acute trauma and chronic trauma. *Acute trauma* to the head and neck region can range from a distinct event, such as an accident or a blow to the face, to a sustained overuse experience, such as a long dental appointment. Acute trauma can serve as an initiating event leading toward a chronic condition, so accurate documentation and careful monitoring may prove extremely valuable should symptoms or dysfunction persist.[12,19,44]

Chronic trauma is defined as any experience that repeatedly exceeds the tolerances of the affected masticatory system structure. Postural stresses and parafunctional occlusal habits, with or without occlusal discrepancies, may produce musculoskeletal disharmony and orthopedic instability of the TMJ.

For more information on dysfunction and deterioration, please visit the companion Expert CONSULT website at www.expertconsult.com.

Recently completed prospective research found only weak association with occlusal disharmonies, but significant correlations with both reported bruxism and tooth wear index and TMD symptoms were confirmed.[23,112] When TMJ dysfunction could be correlated with specific occlusal relationships, the trend was to recognize that when inclines of posterior teeth dominated occlusal function, masticatory system harmony was disrupted.† Other researchers have found that various occlusal interferences and relationships are common among individuals with and without masticatory system disorders.

The general terms for occlusal parafunction used in this text include *bruxism,* or grinding of the teeth, and *clenching,* in which a person holds the teeth firmly together with significant force. Bruxism is usually confirmed by observing excessive tooth wear. Clenching type of parafunction can be distinguished from grinding the teeth and seems to be more often associated with masticatory system disorders than does bruxism.‡ Sleep bruxism may include both tooth grinding and clenching and seems to occur primarily in stage 1 and stage 2 (non-REM [rapid eye movement]) sleep. These episodes often occur in association with short brain and cardiac reactivations called "micro-arousals." Rhythmic masticatory muscle activity is relatively common among non-bruxers, but the frequency and intensity of the muscle contraction is substantially greater for the sleep bruxer. The central pattern generator of the primate brainstem does not modulate or reduce muscle contraction during sleep as occurs during waking hours. Additionally, amplification of oral parafunction has been reported that is associated with a patient's intake of selective serotonin reuptake inhibitors (SSRIs; e.g., Prozac and others).*

If sufficient evidence exists to suspect that the occlusal relationships in function or parafunction may have exceeded the tolerances of that individual's masticatory system, responsible intervention or monitoring can be initiated.[41,60,134,141]

Disruption of the relationship or alignment of the condyle, disc, and articular surface of the temporal bone is typically called an *intracapsular disorder* or *internal derangement* of the TMJ.

When the disc cannot return to its normal relationship to the condyle on full closure of the mouth, it is considered to be displaced or dislocated. Progressive disc displacement most often begins on its lateral aspect and occurs in an anterior and medial direction because of the insertion of muscle fibers into the anteromedial aspect of the disc and the reported variability in resistance of the attachment of the lateral aspect of the disc.[11,155] Stretching of the retrodiscal tissues and collateral ligaments permits the disc to be displaced and function to be limited because of pain resulting from compressive forces on retrodiscal tissues. At some stage of opening, the remaining elasticity of the retrodiscal tissues and tension of the capsular ligaments can pull the disc onto the head of the condyle, often with a discernible sound. Closing then results in the disc again becoming dislocated anteriorly, with a common joint sound often referred to as a *reciprocal click*† (Figure 20-6).

When the disc remains anterior to the head of the condyle during rotation and the limited range of translation possible, the condition is called *closed lock* or *disc displacement (dislocation) without reduction* (Figure 20-7).[40,131]

When the intensity and duration of the functional and dysfunctional loading of the TMJs result in injury, molecular agents appear to be active in the degeneration of joints. Free radicals, various catabolic enzymes, neuropeptides, estrogen, cytokines, and prostaglandins are implicated in inflammatory reactions that have an impact on the articular surfaces and synovial fluid.‡ Loss of the ability of the synovial fluid to lubricate articular surfaces can result in adherence of the disc. Limitation of rotation occurs with adherence between the disc and the condyle, whereas fixation of the disc against the fossa permits rotation but does not allow the disc to move forward during translation.[131]

OROFACIAL PAIN

Discomfort associated with masticatory system disorders falls under the larger umbrella of orofacial pain. Pain associated with TMJ dysfunction is most frequently muscular in origin[40] and may be amplified by both occlusal parafunction and stress.[58] Although pain itself is a complex entity,[161] working knowledge of even the uncommon sources of pain perceived in the region of the masticatory system is essential to providing comprehensive diagnosis and treatment.

*References 40, 41, 72, 84, 103, and 131.
†References 1, 28, 42, 51, 65, 71, 76, 122, 131, 171, and 173.
‡References 29, 55, 57, 77, 134, and 139.

*References 9, 40, 41, 131, 142, and 167.
†References 34, 79, 80, 97, 98, and 107.
‡References 53, 63, 88, 91, 93, 120, 121, 127, 145, and 157.

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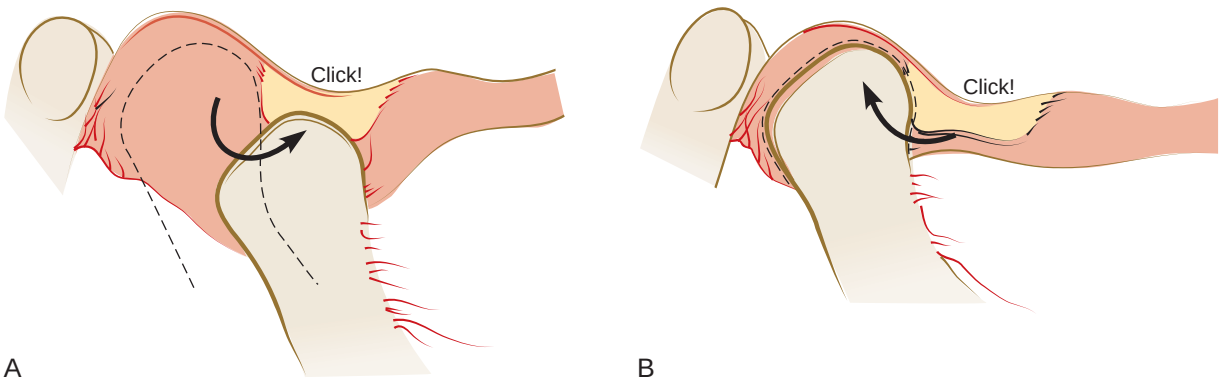

Figure 20-6 Reciprocal click. **A,** Reciprocal click occurs when the condyle moves onto the articular disc from a position behind the posterior band of the disc and then, **B,** clicks off the disc when the condyle moves back. This occurs as the condyle translates forward and back in the opening and closing movements. (From Dawson PE: *Evaluation, diagnosis, and treatment of occlusal problems,* ed 2, St Louis, 1989, Mosby.)

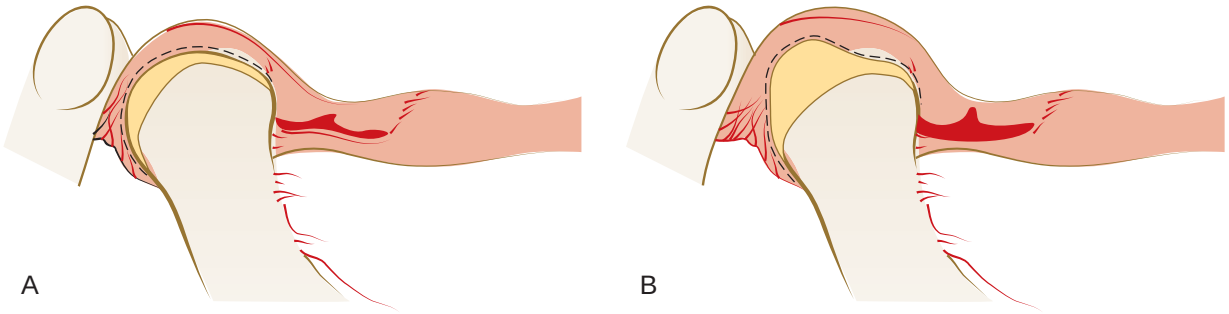

Figure 20-7 Anterior disc displacement without reduction. **A,** Condyle positioned in the fossa on retrodiscal tissues with the disc remaining anterior to head of condyle. **B,** During translation, the disc is further misshapen, restricting full opening of the mandible. (From Dawson PE: *Evaluation, diagnosis, and treatment of occlusal problems,* ed 2, St Louis, 1989, Mosby.)

For more information on orofacial pain, please visit the companion Expert CONSULT website at www.expertconsult.com.

Box 20-1 provides the current list of possible sources of orofacial pain, prepared by the American Academy of Orofacial Pain.[130]

Headache pain is perceived primarily within the trigeminal nerve pathways, although other cranial and cervical nerves may offer painful sensory input.[78,130,152] Pain originating in masticatory system structures, which are also innervated by the trigeminal nerve, requires diagnostic differentiation from headache pain.[153]

Pain of dental and periodontal origin must be clearly defined and differentiated from heart attack, sinus pain, and myofascial pain.[130,131]

Orofacial pain originating in the TMJs or the muscles of mastication can result from neoplasm, macrotrauma, repeated microtrauma, systemic disease, and anatomic predisposition.

Orofacial pain originating in the muscles of mastication may be perceived in that region or may be referred to other structures such as individual teeth.

Cranial nerves experience referral on the same side, whereas skeletal nerve referral can occur to the opposite side; both sources generally refer pain centrally or superiorly.[78,131,158]

The American Academy of Orofacial Pain designates myofascial pain, myositis, muscle spasm (myospasm), local myalgia, and myofibrotic contracture as major categories of these conditions.

Occlusal parafunction, extended dental appointments, metabolic imbalances, and sympathetic nervous system influences have been associated with this painful muscular reaction. An extended period of limited range of mandibular movement can result in fibrosis of the muscle and related attachments, creating a painless condition called myofibrotic contracture.[130]

Otolaryngologic symptoms associated with masticatory system disorders have been reported and include degrees of deafness, tinnitus, and vertigo.* Trauma and postural stress in the cervical spine can be responsible for both the perception and the origin of pain within the masticatory system.[12,19,78,131]

Sensitization of peripheral nociceptors by higher neural centers and inflammation at the site of origin of pain can alter pain perception.[81,113,156] Persistent inflammation therefore may be a contributing factor to chronic pain.[81,113,156] Systemic conditions that may contribute to or predispose an individual to compromised pain regulation include sleep disorders, fibromyalgia, chronic depression, chronic fatigue syndrome, hypothyroidism, insufficient thyroid receptor activity, prolactin feedback disorder, epinephrine sensitivity related to mitral valve prolapse, premenstrual syndrome, androgen excess in women, and posttraumatic stress disorder.

COMPREHENSIVE EVALUATION

Patient History and Interview

The written history and personal interview should be designed to invite open-ended responses and reflection by the patient on past experiences and the current condition.

*References 15, 21, 26, 27, 36, 38, 54, 83, 125, 131, 133, 147, 148, and 175.

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These issues should be documented with regard to timing, duration, frequency, and relationship to any history of trauma.[130]

Clinical Examination

For more information on the clinical examination, please visit the companion Expert CONSULT website at www.expertconsult.com.

BOX 20-1 Differential Diagnosis of Orofacial Pain

Intracranial Pain Disorders[64,70,78,117,130,136]
Neoplasm, aneurysm, abscess, hemorrhage, hematoma, edema

Primary Headache Disorders (Neurovascular Disorders)[86,118]
Migraine, migraine variants, cluster headache, paroxysmal hemicrania, cranial arteritis, carotidynia, tension-type headache[4,87,94,131]

Neurogenic Pain Disorders[117,118]
Paroxysmal neuralgias: Trigeminal, glossopharyngeal, nervus intermedius, and superior laryngeal neuralgias[156]
Continuous pain disorders: Deafferentation pain syndromes (peripheral neuritis, postherpetic neuritis, posttraumatic and postsurgical neuralgia, neuralgia-inducing cavitation osteonecrosis[16,17])
Sympathetically maintained pain[20]

Intraoral Pain Disorders
Dental pulp, periodontium, mucogingival tissues, tongue[13,104,117,176]

Temporomandibular Disorders
Masticatory muscle, temporomandibular joint, associated structures

Associated Structures[36,38,125,130,131]
Ears, eyes, nose, paranasal sinuses, throat, lymph nodes, salivary glands, neck

Axis II Mental Disorders
Somatoform disorders
Pain syndromes of psychogenic origin

Compiled by American Academy of Orofacial Pain.

The physical examination actually begins during the interview, when asymmetries in facial form, head posture, and mandibular movement patterns can be observed. Clinical evaluation of the various structures of the masticatory system, although individual to each practitioner, should afford the patient opportunity for understanding and should include the following[9,40,131]:

1. Observation and measurement of full ROM of the mandible.
2. Auscultation and light palpation of each TMJ in its full ROM.
3. Load testing of each TMJ.
4. Palpitation of each muscle of mastication and related head and neck muscles.
5. Evaluation of all the soft tissues of the face, oral cavity, and oropharynx.
6. Periodontal and dental examinations.
7. Complete occlusal analysis, including accurately mounted diagnostic models.

Evaluation of the TMJ begins with ROM analysis. Observation of departure from a straight path of opening and closing the mandible suggests an intracapsular disorder or masticatory muscle incoordination.[40,41,131] An average maximal opening of 50 mm is common; findings of less than 40 mm of opening suggest limited opening caused by a masticatory system disorder. The range of right and left lateral excursions is usually about 9 mm, and protrusion of mandible is typically 7 mm. Limitation in ROM may be normal for some patients, but for most, these observations are of diagnostic value.[9,47,68,131,160] *Auscultation* of the joint by listening with a stethoscope or Doppler instrument, which amplifies joint sound for both the patient and the dentist, can reveal noises diagnostic for numerous conditions. The intensity and nature of any sounds, clicks, pops, or crepitus (grinding, grating, or rubbing sounds) should be recorded accurately. Any sound detected as part of the initial evaluation should be tracked consistently to detect any change. Diagnostic interpretation and management based on specific sounds correlated with status of the TMJ can be found in other references.[7,9,40,41,131,170]

Firm palpation of the TMJ with the mouth closed can be uncomfortable for the patient with inflammation in joint structures or superficial muscles.

Load testing of the TMJ is essentially a means to palpate the head of the condyle, surface of the glenoid fossa, and tissue interposed between them, except in the case of bone-to-bone contact. With bimanual mandibular manipulation, the dentist loads the joints equally and may detect resistance or tension on either side. The patient is in a supine position in the dental chair to minimize

SCIENCE TRANSFER

Dysfunction of the masticatory system resulting in excessive nonfunctional forces can cause pathologic changes in the temporomandibular joints, masticatory muscles, occlusal tooth surfaces, and in the periodontal support tissues.

In patients with bone loss caused by periodontitis, abnormal occlusal loads can cause pain and add to tooth mobility from the tissue changes associated with traumatic occlusion.

All patients need an evaluation of their masticatory system, particularly those with a predisposition for periodontal disease. The establishment of a stable centric relation together with anterior disclusion will allow for the proper seating of the condyles in the glenoid fossa and reduce the risk of masticatory muscles dysfunction with bruxism, clenching, and pain. This should be considered for all patients, particularly those with existing periodontal disease. Occlusal therapy, including occlusal adjustment and the use of hard acrylic occlusal splints adjusted for a stable centric relation and the absence of posterior guidance in protrusion and balancing movements, should be available as a part of comprehensive periodontal therapy.

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Figure 20-8 Bimanual manipulation load testing in centric relation with the teeth apart.

postural influence on muscle activity. The patient's head is braced by the support of the chair and by being cradled against the dentist's arm or abdomen. The dentist's middle fingers locate the notch in the mandible just anterior to the angle, and the thumbs are placed near the midline in the mental region of the mandible (Figure 20-8). Initially, the dentist provides very gentle guidance to the hinging action of the mandible, with a slight lifting force applied by the fingers and slight depressing force applied with the thumbs. If the patient remains comfortable, increasing force can be applied at both points, ultimately with enough pressure to load-test the joints. With the avascular fibrous discs interposed, the condyles are in centric relation, and the loading of both joints is comfortable. Discomfort may occur with muscle incoordination or bracing or with an anterior displaced disc and attempted loading of vascular retrodiscal tissues of either TMJ.

Muscle palpation is also a learned technique that requires both experience and expertise to derive the most reliable information. Too little pressure is not diagnostic of modest muscle pain or spasm, whereas too much pressure can hurt even when normal musculature is palpated.[56] Externally, the muscles palpated include the anterior, middle, and posterior temporalis; superficial masseter; anterior and posterior digastric; sternocleidomastoid; trapezius; posterior cervical muscles; and insertion of the medial pterygoid muscle. Intraorally, the deep masseter is tested with moderate squeezing pressure, and the medial pterygoid muscle is palpated directly in the general region of the insertion point for local anesthesia with mandibular block. The lateral pterygoid is difficult to palpate because of the dominance of the medial pterygoid in the same region. Its palpation superior and distal to the palpation point for the medial pterygoid can be attempted distal to the maxillary tuberosity. Offering manual resistance to the patient's efforts to protrude the mandible is also a test of the lateral pterygoid muscle. Neither method of evaluation of the possible soreness of the lateral pterygoid muscle is completely reliable, although both provide some insight into muscle status.*

During muscle palpation, the dentist may be able to detect a particularly taut and uncomfortable band of muscle fibers. This condition represents regional myofascial pain or trigger point myalgia, which can be responsible for referral of pain to the teeth and other orofacial regions. Diagnostic injection of local anesthesia can be effective in identifying trigger point pain and pain referral patterns.[131,158]

The *occlusal analysis* is a logical extension of the evaluation of the teeth and periodontium. Tooth mobility is assessed in both static and dynamic modes. Pressure applied to a tooth with a firm object allows detection of movement through both visual and tactile evaluation (see Chapters 30 and 49). Asking the patient to move in excursions of the mandible while maintaining firm contact of opposing teeth also permits visual and tactile assessment.

The physical evaluation of the teeth and any restorations can reflect history of trauma or wear. Visual observation, registration with marking paper or wax, and electronic assessment of tooth contacts as the patient moves in all excursions may reveal disharmonies sufficient to cause orthopedic instability of either TMJ.

While maintaining centric relation, the dentist continues the physical examination by positioning the condyles in a fully seated relationship without tooth-to-tooth contact, using bimanual manipulation or a leaf gauge technique. The mandible is manually guided to close until the first tooth-to-tooth contact is made. If that position is also maximum intercuspation, optimum seating of the condyles is maintained. If the initial contact in centric relation is not maximum intercuspation, the condyles will be directed from a fully seated position to an inferior position relative to their respective fossae, resulting in an orthopedic instability.

To increase the reliability of this evaluation, a muscle-deprogramming effort may be employed. The simplest approach is to use cotton rolls placed between the anterior teeth for 5 to 15 minutes to allow possible muscle relaxation through avoidance of proprioceptive or pressure neurologic input. A prefabricated or directly fabricated acrylic or composite bite stop for anterior teeth offers the same advantage.[9,10,131] A more complex means of confirming the seating of the condyles in centric relation is achieved with a maxillary or mandibular muscle relaxation occlusal appliance. These appliances provide full coverage for the respective arch and occlusal contact for at least one cusp or incisal edge of the opposing teeth so that teeth are protected from spontaneous shifting. The occlusal design provides immediate disclusion of all the posterior teeth in every protrusive or lateral excursion. This allows for the progressive deprogramming of muscles through the advantage of reduced muscle contractions and limited noxious neurologic input.[42,95,172]

Models of the dentition must be accurately mounted to be diagnostic. The facebow transfer relates the maxillary cast to the axis of rotation of the articulator as the maxillary teeth relate to the cranial case. With careful bimanual manipulation or the use of an anterior bite stop, a transfer wafer is generated with the condyles fully seated in their glenoid fossa. The maxillary and mandibular teeth of each diagnostic model then relate to one another in centric relation, as determined by condylar position. They may reveal an occlusal discrepancy in centric relation, which would require compensation by the patient.[9,40]

Imaging

When the clinical evaluation, panoramic radiography, and patient history indicate the possibility of structural masticatory system disorders or the possible presence of pathology, especially neoplasm, appropriate imaging of the TMJ is warranted.[102] The state-of-the-art technique for imaging of soft tissue, especially the articular disc, is magnetic resonance imaging (MRI). The current highest standard for imaging of hard tissue, such as the condyle or the temporal bone, is computed tomography (CT). Cone-beam CT (CBCT) has

*References 3, 9, 40, 41, 50, 52, 78, 131, and 160.

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become much more readily available to dentistry in the last few years, with software systems able to display the data gathered as both anteroposterior and cross-sectional depiction of the condyle and cranial structures with the same or better image quality as spiral CT. Less radiation exposure and lower cost to the patient are both reasons to favor CBCT over conventional CT. The interpretation of MRI and CT images usually requires specialized training for the clinician or access to a radiologist. Arthrography is still being used for certain diagnostic situations, such as suspected perforation of the articular disc, and nuclear medicine has developed protocols to image the TMJ to determine if active deterioration is occurring.*

Although plain-film tomography is occasionally a feature of some of the newer radiographic equipment, the technique most readily available to a majority of practitioners is panoramic radiography. The image produced depicts only general relationships and gross anatomy, so the information provided should be used only for screening purposes. When pathology or marked deformation is suggested by a panoramic radiograph, further diagnostic imaging and procedures may be warranted.[18,78]

*References 9, 18, 66, 69, 82, 96, 114, and 137.

For information on Diagnostic Decision Making, please visit the companion Expert CONSULT website at www.expertconsult.com.

Acknowledgments

I would like to acknowledge the encouragement and the recommendation of references provided by Dr. Henry Gremillion during the revision of this chapter.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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PART 4

Etiology of Periodontal Diseases

Philip Preshaw

Periodontal disease is a common, complex, inflammatory disease characterized by the destruction of tooth-supporting soft and hard tissues of the periodontium, including alveolar bone and periodontal ligament (PDL). Although the inflammation is initiated by bacteria, the tissue breakdown events that lead to the clinical signs of disease result from the host inflammatory response that develops to combat the challenge presented by the subgingival biofilm.

Historically, periodontal disease was regarded as an infection caused by bacterial species that colonize the periodontal pocket. However, it is now clear that the inflammatory responses against these bacteria drive the progression of disease in susceptible individuals. Microbial products trigger the release of pro-inflammatory cytokines and host-derived enzymes, the excessive and/or dysregulated production of which results in tissue breakdown. The impact of microbial products such as lipopolysaccharide (LPS) on induction of immune responses, toll-like receptor (TLR) signaling and cytokine networks is crucial to inflammatory changes that develop in the tissues. Elevated levels of tissue-destructive enzymes such as matrix metalloproteinases (MMPs) and pro-inflammatory cytokines can be detected in the gingival crevicular fluid (GCF) and saliva of patients with periodontitis. As we improve our understanding of the pathogenic inflammatory mechanisms that lead to the development and progression of disease, we will have the potential to develop novel therapies that target different aspects of inflammation and immune functioning and that promote resolution of inflammatory responses.

Periodontal disease and its management must be considered within the context of the patient's general health status and the presence of risk factors for disease. The role of host genes in the pathogenesis of the condition is critically important in determining risk for periodontal breakdown. Identifying the precise impact of specific genotypes on susceptibility to disease is difficult, given the complexity of periodontal pathogenesis and the wide range of molecules that play a role in disease progression. Knowledge of specific genetic risk factors or inflammatory biomarkers could ultimately enable clinicians to direct preventive and treatment approaches to those individuals who are most susceptible to disease. Assessment of risk for periodontal disease must form part of the assessment and treatment of any patient with periodontitis. Smoking is a major risk factor for periodontal disease and smoking cessation should form an integral part of the management strategy for all smokers with periodontitis.

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CHAPTER 21

Periodontal Pathogenesis

Philip M. Preshaw and John J. Taylor

CHAPTER OUTLINE

HISTOPATHOLOGY OF PERIODONTAL DISEASE
- Clinical Healthy Gingival Tissues
- Histopathology of Gingivitis and Periodontitis

INFLAMMATORY RESPONSES IN THE PERIODONTIUM
- Microbial Virulence Factors
- Host-Derived Inflammatory Mediators
- Role of Specific Inflammatory Mediators in Periodontal Disease

LINKING PATHOGENESIS TO CLINICAL SIGNS OF DISEASE
- Alveolar Bone Resorption
- Receptor Activator of Nuclear Factor-κB Ligand and Osteoprotegerin

RESOLUTION OF INFLAMMATION
- Lipoxins
- Resolvins and Protectins

IMMUNE RESPONSES IN PERIODONTAL PATHOGENESIS
- Innate Immunity
- Adaptive Immunity

CONCEPT OF HOST SUSCEPTIBILITY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some text figures, references, and tables may be out of numeric order in this printed chapter.

Understanding periodontal pathogenesis is key to improving management strategies for this common, complex disease. The first challenge is to understand exactly what is meant by the term *pathogenesis.* According to *Merriam Webster's Collegiate Dictionary,* pathogenesis is defined as *the origination and development of a disease.* Essentially, this means the step-by-step processes that lead to the development of the disease, resulting in a series of changes in the structure and function of, in this case, the periodontium. In broad terms, the pathogenesis of a disease is the mechanism by which an etiologic factor (or factors) causes the disease. The word derives from the Greek *pathos* (suffering, which is a now obsolete translation of pathos) and *genesis* (generation/creation).

Our knowledge of periodontal pathogenesis has evolved over the years. It is important to be aware of this, since treatment philosophies have similarly changed in parallel with our improving understanding of disease processes. For example, in the late 1800s, Willoughby D. Miller, the eminent dental researcher who established the important causal role of oral bacteria in the etiology of dental caries, also asserted "during the last few years the conviction has grown continually stronger, among physicians as well as dentists, that the human mouth, as a gathering-place and incubator of diverse pathogenic germs, performs a significant role in the production of varied disorders of the body, and that if many diseases whose origin is enveloped in mystery could be traced to their source, they would be found to have originated in the oral cavity."[115] This marked the beginning of an era of dental treatment strategies that aimed to treat systemic diseases by eliminating so-called foci of infection in the mouth. As a result, many patients underwent dental clearances as a management for their systemic diseases.

By the 1930s, such approaches were beginning to be questioned, as evidenced by a clinical study of 200 patients with rheumatoid arthritis of whom 92 patients had their tonsils removed as treatment for the arthritis (even though only about 15% gave any history of tonsillitis or sore throat) and 52 patients had some or all of their teeth removed.[28] Of the 92 who had their tonsils removed, there was no impact on the arthritis in 86 patients (and two got worse), and of the 52 who had teeth removed, there was no benefit in 47 cases (and three patients reported a worsening of their arthritis after the extractions). The authors wrote that "focal infection is a splendid example of a plausible medical theory which is in danger of being converted by its too enthusiastic supporters into the status of an accepted fact."[28] The end of the focal infection era was signalled by an editorial in the *Journal of the American Medical Association* in 1952 that stated "many patients with diseases presumably caused by foci of infection have not been relieved of their symptoms by removal of the foci, many patients with these same systemic diseases have no evident focus of infection, foci of

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infection are as common in apparently healthy persons as in those with disease."[163]

Advances in the management of periodontitis have been driven by improved knowledge of the epidemiology, etiology, and pathogenesis of the disease.[193] In the 1970s, the role of plaque as the sole etiologic factor for periodontitis was unquestioned. In those days, nonsurgical treatment was in its infancy, and most treatment options involved surgery, for example, gingivectomy in the case of shallower pockets or access flap surgery for treatment of deeper sites. When looking back, it becomes clear that treatment strategies used in a given time period are entirely dependent on the prevailing understanding of pathogenesis at that particular point in time. It is therefore very likely that the management options that we take for granted now will change again in the future. This is to be welcomed because a progressive clinical discipline, such as periodontology, that is well founded in science and with patient benefit as its primary value should strive to improve therapeutic strategies in the light of continued discovery.

Periodontal disease results from a complex interplay between the subgingival biofilm and the host immune-inflammatory events that develop in the gingival and periodontal tissues in response to the challenge presented by the bacteria. It is generally accepted that gingivitis precedes periodontitis, but it is clear that not all cases of gingivitis progress to periodontitis. In gingivitis, the inflammatory lesion is confined to the gingiva, but in periodontitis, the inflammatory processes extend to additionally affect the periodontal ligament and alveolar bone. The net result of these inflammatory changes is breakdown of the fibers of the periodontal ligament, resulting in clinical loss of attachment, together with resorption of the alveolar bone.

In the 1970s and 1980s, bacterial plaque was generally considered to be preeminent as the cause of periodontitis. In that era, it was accepted that poor oral hygiene results in increased plaque accumulation, which in turn results in periodontal disease. However, this model failed to take into account observations such as there are many individuals with poor oral hygiene who do not develop advanced periodontal disease, and conversely, there are unfortunate individuals who, despite good oral hygiene and compliance with periodontal treatment protocols, continue to experience progressive periodontal breakdown and would be considered to have aggressive periodontitis. These findings were confirmed by the work of Löe and colleagues who studied Sri Lankan tea laborers who had no access to dental care and who could be divided into three main categories: (1) individuals (≈8% of the population studied) who had rapid progression of periodontal disease, (2) those (≈81%) who had moderate progression, and (3) those (≈11%) who demonstrated no progression of periodontal disease beyond gingivitis.[106] All patients in this population displayed abundant plaque and calculus deposits. The etiologic role of plaque bacteria is clear in that the bacteria initiate and perpetuate the inflammatory responses that develop in the gingival tissues. However, the major determinant of susceptibility to disease is the nature of the immune-inflammatory responses themselves. It is paradoxical that these defensive processes, which are protective by intent (to prevent ingress of the bacteria and their products into the tissues), result in the majority of tissue damage leading to the clinical manifestations of disease.

Periodontal disease is therefore a unique clinical entity. It is not an infection in the classic sense of the word. In most infections, a single infective organism causes the disease (e.g., human immunodeficiency virus [HIV], syphilis, or tuberculosis), and identification of that organism provides the basis for the diagnosis. In periodontal disease, a large number of species are identifiable in the periodontal pocket, and many more are, as yet, unknown because they have not been cultured. It is impossible to conclude that a single species, or even a group of species, causes periodontal disease. Many of the species that are considered important in periodontal pathogenesis may simply reside in deep pockets because the pocket is a favorable environment for them to survive (e.g., it is warm, moist, and anaerobic, with a ready supply of nutrients). Many of the unique features of periodontitis derive from the anatomy of the periodontium, in which a hard, nonshedding surface (the tooth) is partly embedded within the body (within connective tissue), crosses an epithelial surface, and is partly exposed to the outside world (within the confines of the mouth). The bacteria that colonize this surface are effectively outside the body (even though they are in the subgingival crevice), yet the inflammatory response that develops is located within the body. These factors add complexity to our understanding of the role of the biofilm and the immune-inflammatory responses in periodontal tissue breakdown.

HISTOPATHOLOGY OF PERIODONTAL DISEASE

To better understand periodontal pathogenesis, it is important to have an appreciation of the histologic appearance of clinically healthy tissues, as well as inflamed gingival and periodontal tissues. It is important to note that even in gingival tissues that clinically would be considered to be noninflamed and healthy, there is always evidence of inflammatory responses occurring if they are examined microscopically. This is normal, given that there is a chronic low-grade challenge presented by the subgingival plaque bacteria. The low-grade inflammatory response that results is not detectable macroscopically at the clinical level but is an essential protective mechanism to combat the microbial challenge and to prevent bacteria and their products from infiltrating the tissues and causing tissue damage. Our current understanding of susceptibility to periodontitis suggests that individuals who are more susceptible to the disease mount an excessive, or dysregulated, immune-inflammatory response for a given bacterial challenge, leading to increased tissue breakdown compared to those individuals who have a more normal inflammatory response.

Clinically Healthy Gingival Tissues

Clinically healthy gingival tissues (e.g., those observed in patients with excellent oral hygiene, no visible plaque deposits, and typically who have received regular and meticulous professional cleaning) are pink in appearance, not swollen, not inflamed, and firmly attached to the underlying tooth/bone, with minimal bleeding on probing. The dentogingival junction is a unique anatomic feature whose function is the attachment of the gingiva to the tooth. It comprises an epithelial portion and a connective tissue portion, both of which are of fundamental importance in periodontal pathogenesis. The epithelial portion can be divided into three distinct epithelial structures, the gingival epithelium, sulcular epithelium, and junctional epithelium (Figure 21-1). These epithelial structures are in continuity with each other but have distinct structures and functions, as indicated in Box 21-1.

The junctional epithelium is a particularly unique epithelial structure because the surface cells are specialized for the purpose of attachment to the tooth.[11] Therefore, unlike other epithelial tissues elsewhere in the body, there is no opportunity for sloughing of cells from the surface. Instead, cells at the basal layer continually divide and move to within two or three cell layers of the tooth surface and then migrate coronally, parallel to the tooth surface to eventually reach the floor of the sulcus and be sloughed off into the gingival crevice. The extracellular spaces between the junctional

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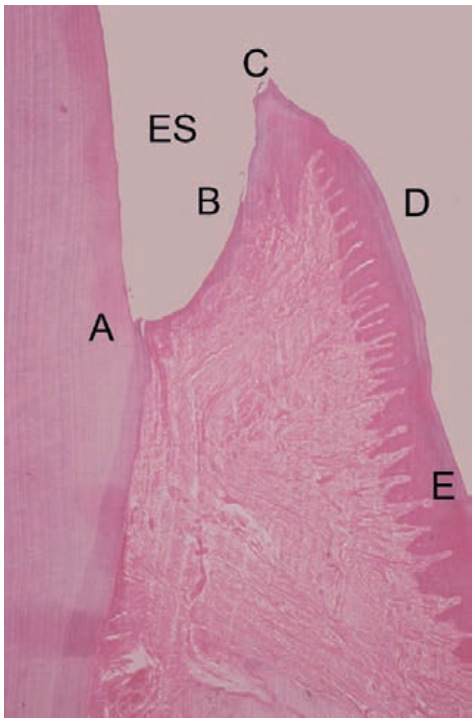

Figure 21-1 Histologic appearance of healthy gingiva. A photomicrograph of a demineralized tooth with the gingival tissues in situ (H&E, low magnification). Amelocemental junction *(A)*. Enamel space *(ES)*. Gingival health is characterized by organization of the epithelium into distinct zones; junctional epithelium *(A-B)*, sulcular epithelium *(B-C)*, free gingiva *(C-D)* and attached gingiva *(D-E)*. The gingival connective tissue is composed of densely packed, organized, and interlacing collagen bundles. There are a few scattered inflammatory cells, but no significant inflammatory cell infiltrate.

BOX 21-1 Characteristics of the Epithelial Component of the Dentogingival Unit

Gingival Epithelium
- Stratified squamous keratinized epithelium.
- Continuous with the sulcular epithelium at the gingival crest/gingival margin.
- Covers the gingiva and forms the clinically visible gingival tissues.
- Covers both the free and attached gingival tissues.

Sulcular Epithelium
- Stratified squamous epithelium.
- Nonkeratinized.
- Faces the tooth surface but is not attached to it.
- Forms the soft tissue lining of the gingival sulcus or periodontal pocket.

Junctional Epithelium
- Forms the epithelial attachment between the gingiva and the tooth.
- Nonkeratinized.
- Forms the floor of the sulcus/pocket.
- Wraps around the tooth like a collar, in health following the morphology of the cementoenamel junction (CEJ).
- Wider at the floor of the sulcus (15-30 cells thick) and tapers apically to 3-4 cells thick.
- Comprised of layers of flattened squamous cells oriented parallel to the tooth surface.
- The surface cells attach to the tooth surface via hemidesmosomes.
- The basal lamina differs from other basal laminae that oppose connective tissue in that type IV collagen is absent.

epithelium are also greater than other epithelial tissues, with intercellular spaces comprising approximately 18% of the volume of the epithelium. This is a result of a lower density of desmosomes in the junctional epithelium compared to the gingival epithelium, and the junctional epithelium is therefore intrinsically "leaky." This has great relevance in periodontal pathogenesis, since the widened intercellular spaces in the junctional epithelium permit migration of neutrophils (polymorphonuclear [PMN] leukocytes) and macrophages from the gingival connective tissues to enter the sulcus to phagocytose bacteria, as well as the ingress of bacterial products and antigens.

The connective tissue component of the dentogingival unit contains densely packed collagen fiber bundles (mixture of type I and III collagen fibers) that are arranged in distinct patterns that maintain the functional integrity of the tissues and tight adaptation of the soft tissues to the teeth. These include the following:

- Dentogingival fibers (extend from the cementum into the free and attached gingiva)
- Alveologingival fibers (extend from the alveolar crest into the free and attached gingiva)
- Circular fibers (wrap around the tooth, maintaining close adaptation of the free gingiva to the tooth, and interweaving with other collagen fiber bundles)
- Dentoperiosteal fibers (run from the cementum, over the alveolar crest, and insert into the alveolar process)
- Transseptal fibers (run interdentally, from the cementum just apical to the junctional epithelium, over the alveolar crest, and insert into the cementum of the neighboring tooth).

It is important to note that even in clinically healthy gingiva, the gingival connective tissue contains at least some inflammatory cells, particularly neutrophils. Neutrophils continually migrate through the connective tissues and pass through the junctional epithelium to enter the sulcus/pocket. These findings were reported in the classic investigations of the histology of periodontal disease reported by Page and Schroeder in 1976.[131] This low-grade inflammation occurs in response to the continued presence of bacteria and their products in the gingival crevice. There is a continuous exudate of fluid from the gingival tissues that enters the crevice and flows out as gingival crevicular fluid (GCF). In addition to the continuous migration of neutrophils through the gingival tissues, lymphocytes and macrophages also accumulate. The presence of leukocytes in the connective tissues results from the chemotactic stimulus created by the subgingival biofilm and bacterial products, as well as chemoattractant factors produced by the host.

In clinically healthy tissues, this steady state equilibrium between low-grade inflammation in the tissues and the continual presence of the subgingival microflora may persist for many years or indeed for the lifetime of the individual. Overt clinical signs of gingivitis (redness, swelling, and bleeding on probing) do not develop because of several innate and structural defense mechanisms, including the following:

- The maintenance of an intact epithelial barrier (the junctional and sulcular epithelium).
- Outflow of GCF from the sulcus (dilution effect and flushing action).

- Sloughing of surface epithelial cells of the junctional and sulcular epithelium.
- Presence of neutrophils and macrophages in the sulcus, phagocytosing bacteria.
- Antibodies in the GCF (although it is not clear whether these are effective).

However, if plaque accumulation increases so that these defense mechanisms are overwhelmed, then inflammation and the classic clinical signs of gingivitis will develop. Even though the development of gingivitis in response to the accumulation of plaque is fairly predictable, research has identified that a spectrum of responses may be observed, with some individuals developing marked gingival inflammation for a given plaque challenge and others developing minimal gingival inflammation.[181] These observations underscore the importance of variations in host responses between individuals in terms of gingival inflammatory responses. Furthermore, many individuals may never develop periodontitis despite having widespread gingivitis. The host's immune-inflammatory response is fundamental in determining which individuals may progress to developing periodontitis, and it is likely that inflammatory responses are markedly different in those individuals who develop periodontitis compared to those who never progress beyond gingivitis. The challenge that this presents clinically is that we do not know (yet) enough about susceptibility to periodontitis to identify these individuals before they actually develop signs of the disease.

Histopathology of Gingivitis and Periodontitis

Development of gingivitis is very clearly observed from a clinical perspective. In addition, the changes that occur within the tissues are very obvious when examined under a microscope. In broad terms, there is infiltration of the connective tissues by numerous defense cells, particularly neutrophils, macrophages, plasma cells, and lymphocytes. As a result of the accumulation of these defense cells and the extracellular release of their destructive enzymes, there is disruption of the normal anatomy of the connective tissues resulting in collagen depletion and subsequent proliferation of the junctional epithelium. Vasodilation and increased vascular permeability lead to increased leakage of fluid out of the vessels and facilitate the passage of defense cells from the vasculature into the tissues, resulting in enlargement of the tissues, which appear erythematous and edematous (i.e., the clinical appearance of gingivitis). These changes are all reversible if the bacterial challenge is substantially reduced by improved oral hygiene.

The landmark studies of Page and Schroeder[131] described the histologic changes that occur in the gingival tissues as the *initial, early, established,* and *advanced* gingival lesions. In broad terms, the initial lesion corresponds to clinically healthy (but nonetheless slightly inflamed) tissues, the early lesion corresponds to the early stages of (clinically evident) gingivitis, the established lesion corresponds to chronic gingivitis, and the advanced lesion marks the transition to periodontitis, with attachment loss and bone resorption. It is important to note that these are *histologic descriptions only,* and they should not form part of a clinical diagnosis. It is not possible to make any statements about the histologic status of a patient's tissues, unless a biopsy is taken and the tissue examined microscopically. It is also important to note that these classic descriptions are primarily based on findings in experimental animals. The histologic stages of gingivitis are described in more detail in Chapter 7, but given their importance for understanding periodontal pathogenesis, these stages are also considered briefly below and summarized in Box 21-2.

BOX 21-2 Key Features of the Histologic Stages of Gingivitis and Periodontitis

Initial Lesion (corresponds to clinically healthy gingival tissues)
- Slightly elevated vascular permeability and vasodilation.
- GCF flows out of the sulcus.
- Migration of leukocytes, primarily neutrophils, in relatively small numbers through the gingival connective tissue, across the junctional epithelium, and into the sulcus.

Early Lesion (corresponds to early gingivitis that is evident clinically)
- Increased vascular permeability, vasodilation, and GCF flow.
- Large numbers of infiltrating leukocytes (mainly neutrophils and lymphocytes)
- Degeneration of fibroblasts.
- Collagen destruction, resulting in collagen depleted areas of the connective tissue.
- Proliferation of the junctional and sulcular epithelium into collagen-depleted areas.

Established Lesion (corresponds to established, chronic gingivitis)
- Dense inflammatory cell infiltrate (plasma cells, lymphocytes, neutrophils).
- Accumulation of inflammatory cells in the connective tissues.
- Elevated release of MMPs and lysosomal contents from neutrophils.
- Significant collagen depletion and proliferation of epithelium.
- Formation of pocket epithelium containing large numbers of neutrophils.

Advanced Lesion (marks the transition from gingivitis to periodontitis)
- Predominance of neutrophils in the pocket epithelium and in the pocket.
- Dense inflammatory cell infiltrate in the connective tissues (primarily plasma cells).
- Apical migration of junctional epithelium to preserve intact epithelial barrier.
- Continued collagen breakdown resulting in large areas of collagen depleted connective tissue.
- Osteoclastic resorption of alveolar bone.

Adapted from Page RC, Schroeder HE: *Lab Invest* 33:235-249, 1976 and linked to the clinical stages of gingivitis and periodontitis.
GCF, Gingival crevicular fluid, *MMPs,* matrix metalloproteinases.

The Initial Lesion. The initial lesion is typically said to develop within 2 to 4 days of accumulation of plaque at a site that was otherwise plaque-free and at which there was no inflammation evident microscopically. However, this situation is probably never encountered in reality, and the gingival tissues always have characteristics of a low-grade chronic inflammatory response as a result of the continual presence of the subgingival biofilm. In other words, the initial lesion corresponds to the histologic picture that is evident in clinically healthy gingival tissues. This low-grade inflammation is characterized by dilation of the vascular network and increased vascular permeability, permitting the neutrophils and monocytes from the gingival vasculature to migrate through the connective tissues toward the source of the chemotactic stimulus—the bacterial products in the gingival sulcus. Upregulation of adhesion molecules, such as intercellular adhesion molecule-1 (ICAM-1) and E-selectin, in the gingival vasculature facilitates the migration of neutrophils from the capillaries into the connective tissues. Increased leakage of fluid from the vessels increases the hydrostatic pressure in the local microcirculation, and as a result, GCF flow increases. Increased GCF flow has the effect of diluting bacterial

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products and also potentially has a flushing action to remove bacteria and their products from the crevice, although given the nature of the bacterial biofilm, it is likely that only planktonic (free-floating) bacteria are removed in this way.

The Early Lesion. The early lesion develops after about 1 week of continued plaque accumulation and corresponds to the early clinical signs of gingivitis. The gingiva are erythematous in appearance as a result of proliferation of capillaries, opening up of microvascular beds, and continued vasodilation.[104] Increasing vascular permeability leads to increased GCF flow, and transmigrating neutrophils increase significantly in number. The predominant infiltrating cell types are neutrophils and lymphocytes (primarily thymic lymphocytes [T-cells]),[135] and the neutrophils migrate through the tissues to the sulcus and phagocytose bacteria. Fibroblasts degenerate, primarily via apoptosis (programmed cell death), which increases the space available for infiltrating leukocytes. Collagen destruction occurs, resulting in collagen depletion in the areas apical and lateral to the junctional and sulcular epithelium. The basal cells of these epithelial structures begin to proliferate to maintain an intact barrier against the bacteria and their products, and as a result the epithelium can be seen proliferating into the collagen depleted areas of the connective tissues (Figure 21-2).[151] As a result of edema of the gingival tissues, the gingiva may appear slightly swollen, and accordingly, the gingival sulcus becomes slightly deeper. The subgingival biofilm exploits this ecologic niche and proliferates apically (thereby rendering effective plaque control more difficult). The early gingival lesion may persist indefinitely or may progress further.

The Established Lesion. The established lesion roughly corresponds with what clinicians would refer to as chronic gingivitis. The progression from the early lesion to the established lesion depends on many factors, including the plaque challenge (the composition and quantity of the biofilm), host susceptibility factors, and risk factors (both local and systemic). In the initial work by Page and Schroeder, the established lesion was defined as being dominated by plasma cells.[131] In human studies, reports have suggested that plasma cells predominate in established gingivitis in older subjects,[51] whereas lymphocytes predominate in younger individuals, although the relevance of these findings is not clear.[23,51] What is clear from all the studies is that there is a significant inflammatory cell infiltrate in established gingivitis that occupies a considerable volume of the inflamed connective tissues. Large numbers of infiltrating cells can be identified adjacent and lateral to the junctional and sulcular epithelium, around blood vessels, and between collagen fiber bundles.[22] Collagen depletion continues, with further proliferation of the epithelium into the connective tissue spaces. Neutrophils accumulate in the tissues and release their lysosomal contents extracellularly (in an attempt to kill bacteria that are not phagocytosed), resulting in the further tissue destruction. Neutrophils are also a major source of MMP-8 (neutrophil collagenase) and MMP-9 (gelatinase B), and these enzymes are produced in large quantities in the inflamed gingival tissues as the neutrophils migrate through the densely packed collagen fiber bundles to enter the sulcus. The junctional and sulcular epithelium form a pocket epithelium that is not firmly attached to the tooth surface and that contains large numbers of neutrophils and is more permeable to the passage of substances into or out of the underlying connective tissue. The pocket epithelium may be ulcerated and is less able to resist the passage of the periodontal probe, so bleeding on probing is a common feature of chronic gingivitis. It is important to remember that these inflammatory changes are still completely reversible if effective plaque control is reinstituted.

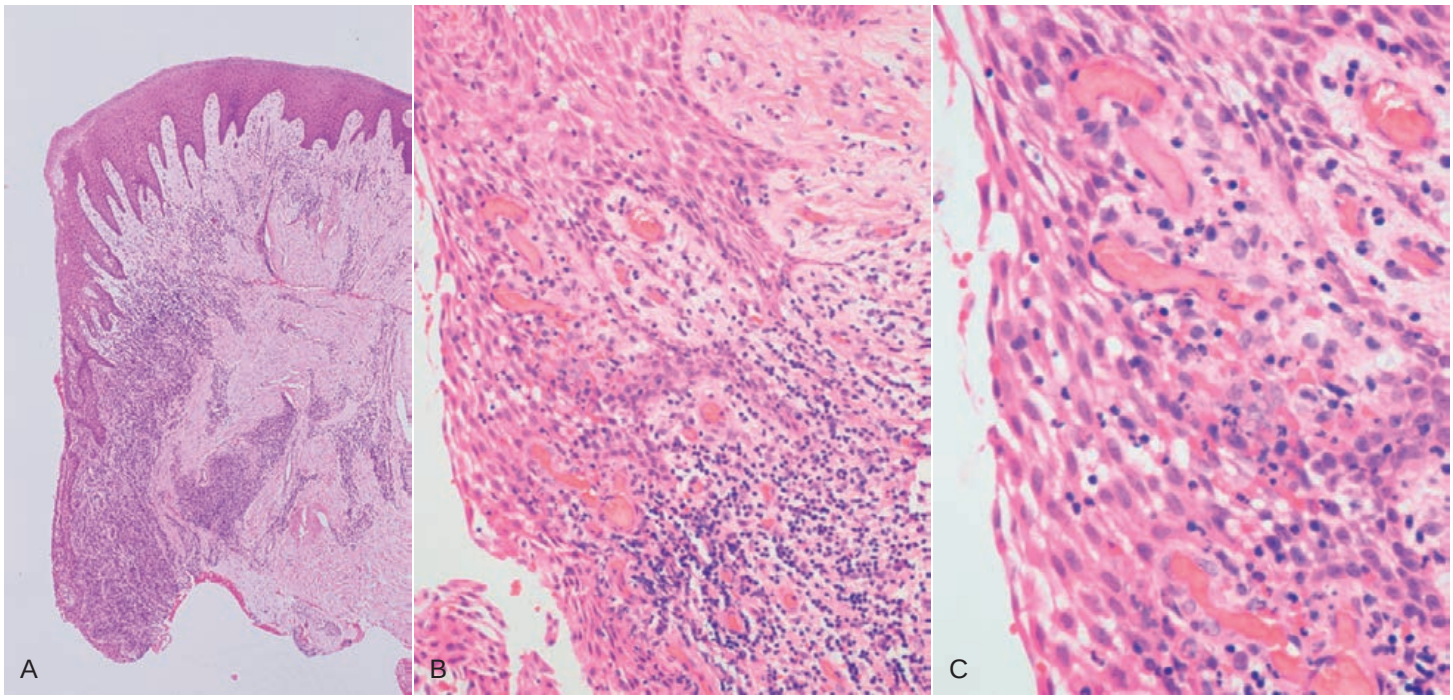

Figure 21-2 Histologic appearance of gingivitis. A series of photomicrographs illustrating gingivitis (H&E). In all cases, the tooth would be to the left side of the image. Low magnification of the gingiva **(A)** demonstrates hyperplastic junctional and sulcular epithelium with a dense inflammatory cell infiltrate in the adjacent connective tissue. Medium magnification of the epithelial-connective tissue interface **(B)** shows numerous intraepithelial inflammatory cells along with intercellular edema. The connective tissue contains dilated capillaries (hyperemia), and there is a dense inflammatory cell infiltrate. High magnification **(C)** shows neutrophils and small lymphocytes transiting the sulcular epithelium.

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The Advanced Lesion. The advanced lesion marks the transition from gingivitis to periodontitis. This transition is determined by many factors, the relative importance of which is, at present, unknown but includes the bacterial challenge (both the composition and the quantity of the biofilm), the host inflammatory response, and susceptibility factors, including environmental and genetic risk factors. Histologic examination reveals continued evidence of collagen destruction (extending now into the periodontal ligament and alveolar bone). Neutrophils predominate in the pocket epithelium and the periodontal pocket, and plasma cells dominate in the connective tissues. The junctional epithelium migrates apically along the root surface into the collagen depleted areas that develop below it to maintain an intact epithelial barrier. Osteoclastic bone resorption commences, and the bone retreats from the advancing inflammatory front as a defense mechanism to prevent spread of bacteria into the bone (Figure 21-3). As the pocket deepens, plaque bacteria proliferate apically into a niche, which is very favorable for many of the species that are regarded as periodontal pathogens. The pocket presents a protected, warm, moist, and anaerobic environment with a ready nutrient supply, and since the bacteria are effectively outside the body (not withstanding that they are in the periodontal pocket), they are not significantly eliminated by the inflammatory response. Thus a cycle develops in which chronic inflammation and associated tissue damage continue; the tissue damage mainly being caused by the inflammatory response, yet the initiating factor, the biofilm, is not eliminated. Destruction of collagen fibers in the periodontal ligament continues, bone resorption progresses, the junctional epithelium migrates apically to maintain an intact barrier, and as a result, the pocket deepens fractionally. This makes it even more difficult to remove the bacteria and disrupt the biofilm through oral hygiene techniques, and thus the cycle perpetuates.

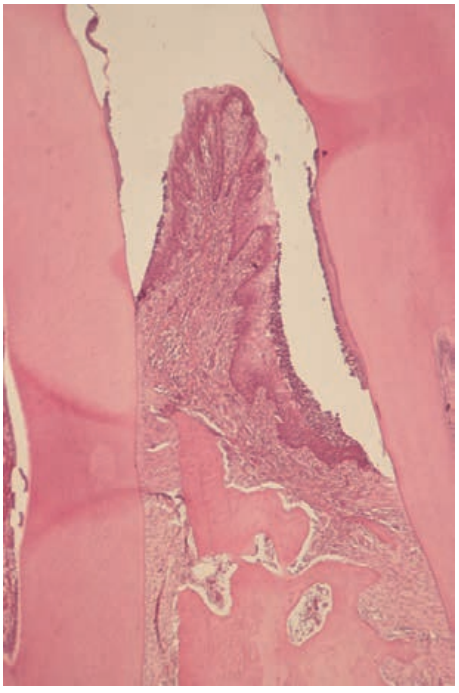

Figure 21-3 Histologic appearance of periodontitis. A photomicrograph of adjacent demineralized teeth with the interproximal gingiva and periodontium in situ (H&E, low magnification). The root of the tooth on the right is coated with a layer of dental plaque/calculus, and there is attachment loss with the formation of a periodontal pocket. The periodontium is densely inflamed and there is alveolar bone loss producing a triangular-shaped defect; vertical bone loss. The base of the pocket is apical to the crest of the alveolar bone and is termed an *infrabony periodontal pocket. (From Soames JV, Southam JC:* Oral pathology, *ed 4, Oxford, 2005, Oxford University Press.).*

INFLAMMATORY RESPONSES IN THE PERIODONTIUM

Now that the histopathology of gingivitis and periodontitis has been reviewed, it is important to consider some of the specific molecules that signal tissue damage as the inflammatory response develops. These can be broadly divided into two main groups: those derived from the subgingival microflora (i.e., microbial virulence factors) and those derived from the host immune-inflammatory response. In terms of the relative importance of each, it is now clear that the great majority of the tissue breakdown results from the host's inflammatory processes. The bacteria are important because they drive and perpetuate the inflammation but are only responsible directly for a relatively small proportion of the tissue damage that occurs.

Microbial Virulence Factors

The subgingival biofilm initiates and perpetuates inflammatory responses in the gingival and periodontal tissues. The subgingival bacteria also contribute directly to tissue damage by the release of noxious substances, but their primary importance in periodontal pathogenesis is that of activating immune-inflammatory responses that in turn result in tissue damage (which may well be beneficial to the bacteria located within the periodontal pocket by providing nutrient sources). Microbial virulence factors that are important in these processes are now discussed in turn.

Lipopolysaccharide. Lipopolysaccharides (LPS) are large molecules composed of a lipid component (lipid A) and a polysaccharide component. They are found in the outer membrane of gram-negative bacteria, they act as endotoxins (LPS is frequently referred to as *endotoxin*), and they elicit strong immune responses in animals. LPS is highly conserved in bacterial species, which reflects its importance in maintaining the structural integrity of the bacterial cells. Immune systems in animals have evolved to recognize LPS via toll-like receptors (TLRs), a family of cell surface molecules that are highly conserved in animal species from *Drosophila* (a genus of fruit flies) to humans, reflecting their importance in innate immune responses. TLRs are also present in lower animals and are in fact more varied than in higher species.[26] TLRs are cell surface receptors that recognize *microbe-associated molecular patterns* (MAMPs), which are conserved molecular structures located on diverse pathogens. TLR-4 recognizes LPS from gram-negative bacteria and functions as part of a complex of cell surface molecules, including CD14 and MD-2 (also known as *lymphocyte antigen 96*). Interaction of this CD14/TLR-4/MD-2 complex with LPS triggers a series of intracellular events, the net result of which is increased production of inflammatory mediators (most notably cytokines) and the differentiation of immune cells (e.g., dendritic cells) for the development of effective immune responses against the pathogens. It is particularly interesting to the periodontist that the pathogen *Porphyromonas gingivalis* has an atypical form of LPS and is recognized by both TLR-2 and TLR-4.[38,45]

It is important to remember that a component of gram-positive cell walls, lipoteichoic acid (LTA), also stimulates immune responses, although less potently than LPS. LTA signals through TLR-2. Both LPS and LTA are released from the bacteria present in the

biofilm and stimulate inflammatory responses in the tissues, resulting in increased vasodilation and vascular permeability, recruitment of inflammatory cells by chemotaxis, and release of proinflammatory mediators by the leukocytes that are recruited to the area. LPS in particular is of key importance in initiating and sustaining inflammatory responses in the gingival and periodontal tissues.

Bacterial Enzymes and Noxious Products. Plaque bacteria produce a number of metabolic waste products that contribute directly to tissue damage. These include noxious agents, such as ammonia (NH_3) and hydrogen sulfide (H_2S), and short-chain carboxylic acids, such as butyric acid and propionic acid. These acids are detectable in GCF and are found in increasing concentrations as the severity of periodontal disease increases. These substances have profound effects on host cells (e.g., butyric acid induces apoptosis in T-cells, B-cells, fibroblasts, and gingival epithelial cells).[94,95,164] The short-chain fatty acids may aid *P. gingivalis* infection through tissue destruction and may also create a nutrient supply for the organism by increasing bleeding into the periodontal pocket. The short-chain fatty acids also influence cytokine secretion by immune cells and may potentiate inflammatory responses after exposure to proinflammatory stimuli such as LPS, interleukin-1 beta (IL-1β), and tumor necrosis factor alpha (TNF-α).[122]

Plaque bacteria produce proteases, which are capable of breaking down structural proteins of the periodontium such as collagen, elastin, and fibronectin. Bacteria produce these proteases to digest proteins and thereby provide peptides for bacterial nutrition. Bacterial proteases disrupt host responses, compromise tissue integrity, and facilitate microbial invasion of the tissues. *P. gingivalis* produces two classes of cysteine proteases that have been implicated in periodontal pathogenesis. These are known as *gingipains* and include the lysine specific gingipain Kgp and the arginine specific gingipains RgpA and RgpB. The gingipains can modulate the immune system and disrupt immune-inflammatory responses, potentially leading to increased tissue breakdown.[137] Gingipains can reduce the concentrations of cytokines in cell culture systems[7] and they digest and inactivate TNF-α.[25] The gingipains can also stimulate cytokine secretion via activation of protease-activated receptors (PARs). For example, RgpB activates two different PARs (PAR-1 and PAR-2), thereby stimulating cytokine secretion[107] and both Rgp and Kgp gingipains stimulate IL-6 and IL-8 secretion by monocytes via activation of PAR-1, PAR-2 and PAR-3.[182]

Microbial Invasion. Microbial invasion of the periodontal tissues has long been a contentious topic. In histologic specimens, bacteria, including cocci, filaments, and rods, have been identified in the intercellular spaces of the epithelium.[50] Periodontal pathogens such as *P. gingivalis* and *Aggregatibacter actinomycetemcomitans* have been reported to invade the gingival tissues,[30,76,146] including the connective tissues.[145] *Fusobacterium nucleatum* can invade oral epithelial cells, and bacteria that routinely invade host cells may facilitate the entry of noninvasive bacteria by coaggregating with them (Figure 21-4).[47] It has also been shown that *A. actinomycetemcomitans* can invade epithelial cells and persist intracellularly.[49] The clinical relevance of these findings is unclear, however. Some investigators have suggested that tissue invasion by subgingival bacteria is an active process, whereas others have considered it to be an artifact, or simply a passive translocation process.

The reports of bacteria present in the tissues have sometimes been used to justify the use of antibiotics in the treatment of periodontitis as a means of attempting to eliminate those organisms that are located in the tissues and that are therefore "protected" from mechanical disruption by root surface debridement. It has also been reported that bacteria in the tissues represent a "reservoir for reinfection" after nonsurgical management. However, until the clinical relevance of bacteria being present in the tissues is better defined, it is inappropriate to make clinical treatment decisions (e.g., whether to use adjunctive systemic antibiotics) on this premise alone.

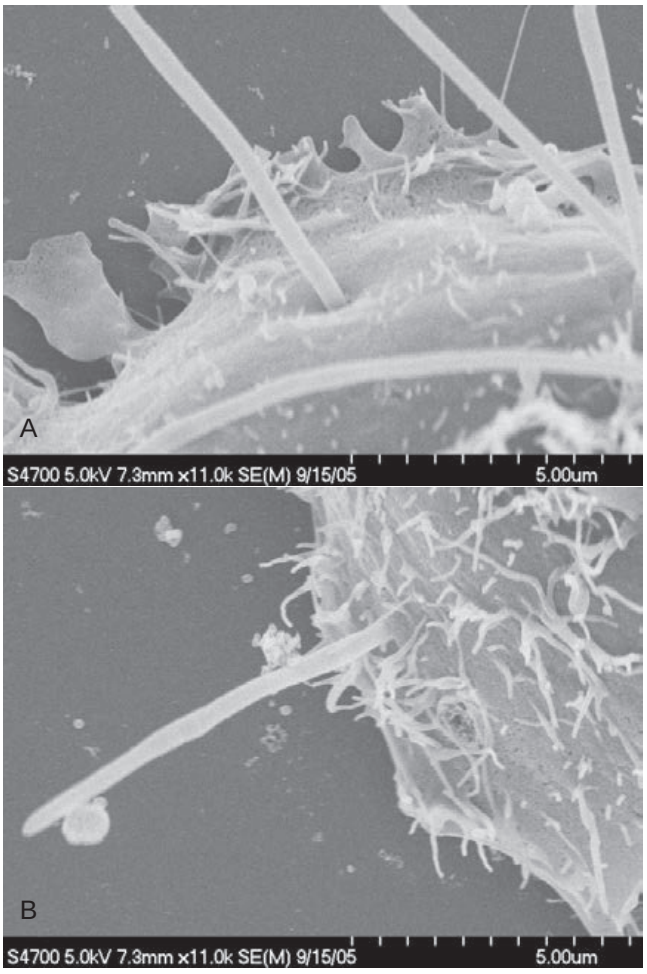

Figure 21-4 Invasion of epithelial cells by *Fusobacterium nucleatum*. In both images, a single epithelial cell is shown, being penetrated by invading *F. nucleatum* bacteria (3-4 bacteria are evident in **A** and 1 bacterium is evident in **B**). The ruffled surface of the epithelial cells (multiple small fingerlike projections, much smaller than the *F. nucleatum* bacteria) is likely to be an artifact. In **B,** *F. nucleatum* may facilitate the colonization of epithelial cells by bacteria unable to adhere or invade directly as evidenced by the single coccoid bacterium (*Streptococcus cristatus*) that has coaggregated with the *F. nucleatum* bacterium as it penetrates the epithelial cell. *(Images courtesy Dr. A.E. Edwards, Dr. J.D. Rudney, and Dr. T.J. Grossman, Bath University, United Kingdom, and the University of Minnesota.).*

Fimbriae

For more information on fimbriae, please visit the companion Expert CONSULT website at www.expertconsult.com.

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Bacterial Deoxyribonucleic Acid and Extracellular Deoxyribonucleic Acid

For more information on bacterial DNA and eDNA, please visit the companion Expert CONSULT website at www.expertconsult.com.

Host-Derived Inflammatory Mediators

The inflammatory and immune processes that develop in the periodontal tissues in response to the long-term presence of the subgingival biofilm are protective by intent but result in considerable tissue damage. This has sometimes been referred to as *bystander damage,* denoting that the host response is mainly responsible for the tissue damage that occurs, leading to the clinical signs and symptoms of periodontal disease. It is paradoxical that the host response causes most of the tissue damage, although this is by no means unique to periodontal disease. For example, the tissue damage that occurs in the joints in rheumatoid arthritis results from prolonged and excessive inflammatory responses and is characterized by increased production of many of the cytokines known to be important in periodontal pathogenesis. In the case of rheumatoid arthritis, the initiating factor is an autoimmune response to structural components of the joint, whereas in periodontitis, the initiating factor is the subgingival biofilm. In both cases, however, the destructive inflammatory events are remarkably similar, although the pathogenesis varies as a result of the different anatomy.

Having understood that the majority of the tissue damage in periodontitis derives from the excessive and dysregulated production of a variety of inflammatory mediators and destructive enzymes in response to the presence of the subgingival plaque bacteria, it is important to review the key types of mediators that orchestrate the host responses. These can be broadly divided into the cytokines, the prostanoids, and the matrix metalloproteinases (MMPs).

Cytokines. Cytokines play a fundamental role in inflammation and are key inflammatory mediators in periodontal disease.[159] They are soluble proteins and act as messengers to transmit signals from one cell to another. Cytokines bind to specific receptors on target cells and initiate intracellular signalling cascades resulting in phenotypic changes in the cell via altered gene regulation.[17,174] Cytokines are effective in very low concentrations, are produced transiently in the tissues, and primarily act locally in the tissues in which they are produced. Cytokines are able to induce their own expression either in an autocrine or paracrine fashion and have pleiotropic effects (i.e., multiple biologic activities) on a large number of cell types. (Autocrine signalling means that the autocrine agent, in this case cytokines, binds to receptors on the cell that secreted the agent, whereas paracrine signalling affects other nearby cells.) Simply, cytokines bind to cell surface receptors, trigger a sequence of intracellular events that lead ultimately to the production of protein by the target cell, which alters that cell's behavior, and could result in, for example, increased secretion of more cytokines in a positive feedback cycle leading to inflammation.

Cytokines are produced by a large number of cell types, including infiltrating inflammatory cells such as neutrophils, macrophages, and lymphocytes, and also by resident cells in the periodontium, including fibroblasts and epithelial cells.[170] Cytokines signal, broadcast, and amplify immune responses and are fundamentally important in regulating immune-inflammatory responses and in combating infections. However, they also have profound biologic effects that lead to tissue damage in chronic inflammation, and prolonged and excessive production of cytokines and other inflammatory mediators in the periodontium leads to the tissue damage that characterizes the clinical signs of the disease. For example, cytokines mediate connective tissue and alveolar bone destruction through the induction of fibroblasts and osteoclasts to produce proteolytic enzymes (i.e., MMPs) that break down structural components of these connective tissues.[12]

There is significant overlap and redundancy between the function of individual cytokines, and cytokines do not act in isolation, but rather in flexible and complex networks that involve both proinflammatory and antiinflammatory effects and that bring together aspects of both innate and acquired immunity.[8] Cytokines play a key role at all stages of the immune response in periodontal disease. Among the most studied (and probably the most important) cytokines in periodontal pathogenesis are the proinflammatory cytokines IL-1β and TNF-α. Both of these cytokines play a key role in the initiation, regulation, and perpetuation of innate immune responses in the periodontium, resulting in vascular changes and migration of effector cells such as neutrophils into the periodontium as part of a normal immune response to the presence of subgingival bacteria.[57]

Prostaglandins. The prostaglandins (PGs) are a group of lipid compounds derived from arachidonic acid, a polyunsaturated fatty acid found in the plasma membrane of most cells. Arachidonic acid is metabolized by cyclooxygenase-1 and -2 (COX-1 and COX-2) to generate a series of related compounds called the *prostanoids,* which includes the PGs, thromboxanes, and prostacyclins. PGs are important mediators of inflammation, particularly prostaglandin E_2 (PGE_2), which results in vasodilation and induces cytokine production by a variety of cell types. COX-2 is upregulated by IL-1β, TNF-α, and bacterial LPS, resulting in increased production of PGE_2 in inflamed tissues. PGE_2 is produced by various types of cells and most significantly in the periodontium by macrophages and fibroblasts. PGE_2 results in induction of MMPs and osteoclastic bone resorption and has a major role in contributing to the tissue damage that characterizes periodontitis.

Matrix Metalloproteinases. MMPs are a family of proteolytic enzymes that degrade extracellular matrix molecules such as collagen, gelatin, and elastin. They are produced by a variety of cell types, including neutrophils, macrophages, fibroblasts, epithelial cells, osteoblasts, and osteoclasts. The names and functions of key MMPs are shown in Table 21-1. The nomenclature of MMPs has been based on the perception that each enzyme has its own specific substrate, for example, MMP-8 and MMP-1 are both collagenases (i.e., they break down collagen). However, it is now appreciated that MMPs usually degrade multiple substrates, with significant substrate overlap between individual MMPs.[70] The substrate-based classification is still used, however, and MMPs can be divided into collagenases, gelatinases/type IV collagenases, stromelysins, matrilysins, membrane-type metalloproteinases, and others.

MMPs are secreted in a latent form (inactive) and are activated by the proteolytic cleavage of a portion of the latent enzyme. This is achieved by proteases, such as cathepsin G, produced by neutrophils. MMPs are inhibited by proteinase inhibitors, which have antiinflammatory properties. Key inhibitors of MMPs found in the serum include the glycoprotein α_1-antitrypsin and α_2-macroglobulin, a large plasma protein produced by the liver that is capable of inactivating a wide variety of proteinases. Inhibitors of MMPs that

TABLE 21-1 Classification of Matrix Metalloproteinases

Group	Enzyme	Name
Collagenases	MMP-1	Collagenase 1, fibroblast collagenase
	MMP-8	Collagenase 2, neutrophil collagenase
	MMP-13	Collagenase 3
Gelatinases	MMP-2	Gelatinase A
	MMP-9	Gelatinase B
Stromelysins	MMP-3	Stromelysin 1
	MMP-10	Stromelysin 2
	MMP-11	Stromelysin 3
Matrilysins	MMP-7	Matrilysin 1, pump-1
	MMP-26	Matrilysin 2
Membrane-type MMPs	MMP-14	MT1-MMP
	MMP-15	MT2-MMP
	MMP-16	MT3-MMP
	MMP-17	MT4-MMP
	MMP-24	MT5-MMP
	MMP-25	MT6-MMP
Others	MMP-12	Macrophage elastase
	MMP-19	—
	MMP-20	Enamelysin

MMPs, Matrix metalloproteinases; *MT,* membrane type.
Adapted from Hannas AR, Pereira JC, Granjeiro JM, et al: *Acta Odontol Scand* 65:1-13, 2007.

are found in the tissues include the tissue inhibitors of metalloproteinases (TIMPs), which are produced by many cell types; the most important in periodontal disease is TIMP-1.[18] MMPs are also inhibited by the tetracycline class of antibiotics, which has led to the development of sub-antimicrobial formulation of doxycycline as a licensed systemic adjunctive drug treatment for periodontitis that exploits the anti-MMP properties of this molecule (see Chapter 48).

Role of Specific Inflammatory Mediators in Periodontal Disease

Interleukin-1 Family Cytokines. The IL-1 family of cytokines comprises at least 11 members, including IL-1α, IL-1β, IL-1 receptor antagonist (IL-1Ra), IL-18, and IL-33.

IL-1β plays a key role in inflammation and immunity, is closely linked to the innate immune response, and induces the synthesis and secretion of other mediators that contribute to inflammatory changes and tissue damage. For example, IL-1β stimulates the synthesis of PGE_2, platelet-activating factor (PAF), and nitrous oxide (NO), resulting in vascular changes associated with inflammation, increasing blood flow to the site of infection or tissue injury. IL-1β is mainly produced by monocytes, macrophages, and neutrophils and also by other cell types such as fibroblasts, keratinocytes, epithelial cells, B-cells, and osteocytes.[40] IL-1β increases the expression of ICAM-1 on endothelial cells and stimulates secretion of the chemokine CXCL8 (which is IL-8), thereby stimulating and facilitating the infiltration of neutrophils into the affected tissues. IL-1β also synergizes with other proinflammatory cytokines and PGE_2 to induce bone resorption. IL-1β has a role in adaptive immunity, regulates the development of antigen-presenting cells, such as dendritic cells, stimulates IL-6 secretion by macrophages (which in turn activates B-cells), and has been shown to enhance antigen-mediated stimulation of T-cells.[13] GCF concentrations of IL-1β are increased at sites affected by gingivitis[73] and periodontitis,[98] and tissue levels of IL-1β correlate with clinical periodontal disease severity.[165] Studies in experimental animals have shown that IL-1β exacerbates inflammation and alveolar bone resorption.[87] It is clear from the multiplicity of studies that have investigated this cytokine that IL-1β plays a fundamental role in the pathogenesis of periodontal disease.[90]

IL-1α is primarily an intracellular protein that is not normally secreted and therefore is not usually found in the extracellular environment or in the circulation.[43] Unlike IL-1β, biologically active IL-1α is constitutively expressed and likely mediates inflammation only when released from necrotic cells, acting as an "alarmin" to signal the immune system during cell and tissue damage.[16] The precise role of IL-1α in periodontal pathogenesis is not well defined, although studies have reported elevated IL-1α levels in GCF and gingival tissues in patients with periodontitis.[139] IL-1α is a potent bone resorbing factor involved in the bone loss that is associated with inflammation.[172] It is possible that the measured IL-1α in gingival tissues represents intracellular IL-1α that has been released from damaged or necrotic cells. It is probable that IL-1α plays a role in periodontal pathogenesis, possibly as a signalling cytokine (signalling tissue damage) and contributing to bone resorptive activity.

IL-1Ra has structural homology to IL-1β, and binds to the IL-1 receptor (IL-1R1). However, binding of IL-1Ra does not result in signal transduction, therefore IL-1Ra antagonizes the action of IL-1β.[42] IL-1Ra is important in regulating inflammatory responses and can be considered to be an antiinflammatory cytokine. IL-1Ra levels have been reported to be elevated in the GCF and tissues of patients with periodontal disease, suggesting a role in immunoregulation in periodontitis.[142]

IL-18 interacts with IL-1β and shares many of the proinflammatory effects of IL-1β. It is mainly produced by stimulated monocytes and macrophages.[63] There is increasing evidence to suggest that IL-18 plays a significant role in inflammation and immunity. IL-18 results in proinflammatory responses, including activation of neutrophils.[101] It is chemoattractant for T-cells,[88] and it interacts with IL-12 and IL-15 to induce interferon gamma (IFN-γ), thereby inducing T-helper (Th1) cells, which activate cell-mediated immunity.[197] Interestingly, in the absence of IL-12, IL-18 induces IL-4 and a Th2 response, which regulates humoral (antibody-mediated) immunity.[198] There is very limited direct evidence for a role of IL-18 in periodontal pathogenesis. Oral epithelial cells secrete IL-18 in response to stimulation with LPS,[143] and a correlation between GCF IL-18 levels and sulcus depth has been reported.[82] IL-18 levels have been reported to be higher than those of IL-1β in patients with periodontitis, suggesting that IL-18, along with IL-1β is predominant in periodontitis lesions.[129] Since IL-18 has the ability to induce either Th1 or Th2 differentiation, it is likely to play an important role in periodontal disease pathogenesis.[130]

Other Interleukin-1 Family Cytokines

For more information on other IL-1 family cytokines, please visit the companion Expert CONSULT website at www.expertconsult.com.

Tumor Necrosis Factor Alpha. TNF-α is a key inflammatory mediator in periodontal disease and shares many of the cellular actions of IL-1β.[64] It plays a fundamental role in immune responses, increases neutrophil activity, and mediates cell and tissue turnover

by inducing MMP secretion. TNF-α stimulates the development of osteoclasts and limits tissue repair by induction of apoptosis in fibroblasts. TNF-α is secreted by activated macrophages, as well as other cell types, particularly in response to bacterial LPS. Proinflammatory effects of TNF-α include stimulation of endothelial cells to express selectins that facilitate leukocyte recruitment, activation of macrophage IL-1β production, and induction of PGE_2 by macrophages and gingival fibroblasts.[133] TNF-α, although possessing similar activity to IL-1β, has a less potent effect on osteoclasts, and is present at lower levels in inflamed gingival tissues than IL-1β.[166] GCF levels of TNF-α increase as gingival inflammation develops, and higher levels are found in periodontitis.[64,73] The importance of TNF-α (and IL-1β) in periodontal pathogenesis is unquestioned and is highlighted, particularly by studies showing that application of antagonists to IL-1β and TNF-α resulted in an 80% reduction in recruitment of inflammatory cells in proximity to the alveolar bone and a 60% reduction in bone loss.[6]

Interleukin-6 and Related Cytokines. The cytokines in this group, which include IL-6, IL-11, leukemia-inhibitory factor (LIF), and oncostatin M, share common signalling pathways via signal transducers glycoprotein (gp) 130.[74] IL-6 is the most extensively studied of this group and has pleiotropic proinflammatory properties.[86] IL-6 secretion is stimulated by cytokines such as IL-1β and TNF-α and it is produced by a range of immune cells, including T-cells, B-cells, macrophages, and dendritic cells, as well as resident cells such as keratinocytes, endothelial cells, and fibroblasts.[186] IL-6 is also secreted by osteoblasts and stimulates bone resorption and development of osteoclasts.[81,93] IL-6 is elevated in the cells, tissues, and GCF of patients with periodontal disease.[56,103] IL-6 may have an influence on monocyte differentiation into osteoclasts and a role in bone resorption in periodontal disease.[128] IL-6 also has a key role in the regulation of proliferation and differentiation of B-cells and T-cells (in particular the Th17 subset).[86] IL-6 therefore has an important role in periodontal pathogenesis, although less than that of IL-1β or TNF-α.

For more information on interleukin-6 and related cytokines, please visit the companion Expert CONSULT website at www.expertconsult.com.

Prostaglandin E_2. The cells primarily responsible for PGE_2 production in the periodontium are macrophages and fibroblasts. PGE_2 levels are increased in the tissues and in GCF at sites undergoing periodontal attachment loss. PGE_2 induces the secretion of MMPs and osteoclastic bone resorption and contributes significantly to the alveolar bone loss seen in periodontitis. PGE_2 release from monocytes from patients with severe or aggressive periodontitis is greater than that from monocytes from patients who are periodontally healthy.[55,125] A large body of evidence has demonstrated the importance of PGE_2 in periodontal pathogenesis, and given that prostaglandins are inhibited by nonsteroidal antiinflammatory drugs (NSAIDs), researchers have investigated the use of NSAIDs as potential host-response modulators in the management of periodontal disease.[191,192] However, daily administration for extended periods is necessary for the periodontal benefits to become apparent, and NSAIDs are associated with significant unwanted side effects, including gastrointestinal problems, hemorrhage (from impaired platelet aggregation resulting from inhibition of thromboxane formation), and renal and hepatic impairment. NSAIDs are therefore not indicated as adjunctive treatments for the management of periodontitis.

For more information on prostaglandin E_2, please visit the companion Expert CONSULT website at www.expertconsult.com.

Matrix Metalloproteinases. MMPs are a family of zinc-dependent enzymes that are capable of degrading extracellular matrix molecules, including collagens.[18,144] MMPs play a key role in periodontal tissue destruction and are secreted by the majority of cell types in the periodontium, including fibroblasts, keratinocytes, endothelial cells, osteoclasts, neutrophils, and macrophages. In healthy tissues, MMPs are mainly produced by fibroblasts, which produce MMP-1 (also known as *collagenase-1*), and these have a role in the maintenance of the periodontal connective tissues. Transcription of genes coding for MMPs is upregulated by cytokines such as IL-1β and TNF-α.[108] MMP activity is regulated by specific endogenous tissue inhibitors of metalloproteinases (TIMPs) and serum glycoproteins such as α-macroglobulins, which form complexes with active MMPs and their latent precursors.[141] TIMPs are produced by fibroblasts, macrophages, keratinocytes, and endothelial cells and are specific inhibitors that bind to MMPs in a 1:1 stoichiometry.[70] MMPs are also produced by some periodontal pathogens, such as *A. actinomycetemcomitans* and *P. gingivalis*, but the relative contribution of these bacterially-derived MMPs to periodontal pathogenesis is small. The great majority of MMP activity in the periodontal tissues derives from infiltrating inflammatory cells.

In healthy periodontal tissues, collagen homeostasis is a controlled process that is mediated extracellularly by MMP-1 (expressed by resident cells, primarily fibroblasts) and intracellularly by a variety of lysosomal acid–dependent enzymes. In inflamed periodontal tissues, excessive quantities of MMPs are secreted by resident cells and the large numbers of infiltrating inflammatory cells, particularly neutrophils, as they migrate through the tissues. As a result, the balance between MMPs and their inhibitors is disrupted, resulting in breakdown of the connective tissue matrix,[18,175] and leading to the development of collagen depleted areas within the connective tissues, as described earlier. Neutrophils are key infiltrating cells in periodontitis that accumulate in large numbers in inflamed periodontal tissues (see Figure 21-2). Neutrophils have evolved to respond rapidly and aggressively to external stimuli, such as bacterial LPS, and they release large quantities of destructive enzymes very rapidly. The predominant MMPs in periodontitis, MMP-8 and MMP-9, are secreted by neutrophils[62] and are very effective at degrading type 1 collagen, the most abundant collagen type in the periodontal ligament.[110] MMP-8 and MMP-9 levels increase with increasing severity of periodontal disease and decrease after treatment.[61,62,85] The prolonged and excessive release of large quantities of MMPs in the periodontium leads to significant breakdown of structural components of the connective tissues, contributing to the clinical signs of disease.

MMPs play a fundamental role in connective tissue homeostasis and also disease pathogenesis and possess a wide range of biologic effects that are relevant in periodontitis (Table 21-3). MMPs are important in alveolar bone destruction and are expressed by osteoclasts, which also express cathepsin K. Cathepsin K is a lysosomal cysteine protease that is mainly expressed in osteoclasts and plays a key role in bone resorption and remodelling. This enzyme can catabolize collagen, gelatin, and elastin and can therefore contribute to the breakdown of bone and cartilage.

TABLE 21-3 Biologic Activities of Selected MMPs Relevant to Periodontal Disease

MMP Type	Enzyme	Biologic Activity
Collagenases	All	Degrade interstitial collagen (type I, II, and III) Digest ECM and non-ECM molecules
	MMP-1	Keratinocyte migration and re-epithelialization Platelet aggregation
	MMP-13	Osteoclast activation
Gelatinases	All	Degrade denatured collagens and gelatin
	MMP-2	Differentiation of mesenchymal cells with inflammatory phenotype Epithelial cell migration Increased bioavailability of MMP-9
Stromelysins	All	Digest ECM molecules
	MMP-3	Activates pro-MMPs Disrupted cell aggregation Increased cell invasion
Matrilysins	MMP-7	Disrupted cell aggregation Increased cell invasion
Membrane-type MMPs	All	Digest ECM molecules Activate pro-MMP-2 (except MT4-MMP)
	MT1-MMP	Epithelial cell migration Degrade collagen types I, II and III

MMPs, Matrix metalloproteinases; *MT,* membrane type; *ECM,* extracellular matrix. Adapted from Hannas AR, Pereira JC, Granjeiro JM, et al: *Acta Odontol Scand* 65:1-13, 2007.

For more information on matrix metalloproteinases, please visit the companion Expert CONSULT website at www.expertconsult.com.

Chemokines

For more information on chemokines, please visit the companion Expert CONSULT website at www.expertconsult.com.

Antiinflammatory Cytokines

For more information on antiinflammatory cytokines, please visit the companion Expert CONSULT website at www.expertconsult.com.

LINKING PATHOGENESIS TO CLINICAL SIGNS OF DISEASE

Advanced forms of periodontal disease are characterized by the distressing symptoms of tooth mobility and tooth migration. These result from the loss of attachment between the tooth and its supporting tissues following breakdown of the inserting fibers of the periodontal ligament and resorption of alveolar bone. Having reviewed the histopathology and the inflammatory processes that develop in the periodontal tissues as a result of prolonged accumulation of dental plaque, it is now necessary to link these changes to the structural damage that occurs in the periodontium, leading to the well-defined signs of disease.

It is important to note that even clinically healthy tissues demonstrate signs of inflammation when histologic sections are examined. Thus transmigrating neutrophils are evident in the gingival tissues, moving toward the sulcus for the purpose of eliminating bacteria. If the inflammation becomes more extensive, for example, because of an increase in the bacterial challenge, vasodilation and increased vascular permeability lead to edema of the tissues (as well as erythema), causing gingival swelling, a slight deepening of the sulcus, and further compromising plaque removal. Increased infiltration of inflammatory cells, particularly neutrophils, results in development of collagen-depleted areas below the epithelium and as a result, the epithelium proliferates to maintain tissue integrity.

The epithelium provides a physical barrier to impede the ingress of bacteria and their products and disruption of the epithelial barrier can lead to further bacterial invasion and inflammation. Antimicrobial peptides, termed *defensins,* are expressed by epithelial cells, and gingival epithelial cells express two human β-defensins (hBD-1 and hBD-2). Furthermore, a cathelicidin class antimicrobial peptide, LL-37, which is found in the lysosomes of neutrophils, is also expressed in skin and gingiva. These antimicrobial peptides are important in determining the outcomes of the host-pathogen interactions at the epithelial barrier.[190] The epithelium is therefore more than simply a passive barrier, it also has an active role in innate immunity. [37] Epithelial cells in the junctional and sulcular epithelium are in constant contact with bacterial products and respond to these by secreting chemokines (such as IL-8, CXCL8) to attract neutrophils, which migrate up the chemotactic gradient toward the pocket. Epithelial cells are therefore active in responding to infection and signalling further host responses.

If the bacterial challenge persists, the cellular and fluid infiltration continues to develop and neutrophils and other inflammatory cells soon occupy a significant volume of the inflamed gingival tissues. Neutrophils are key components of the innate immune system and play a fundamental role in maintaining periodontal health despite the constant challenge presented by the plaque biofilm. Neutrophils are protective leukocytes that phagocytose and kill bacteria, and deficiencies in neutrophil functioning result in increased susceptibility to infections in general, as well as periodontal disease.[99] Neutrophils also release large quantities of destructive enzymes, such as MMPs, as they migrate through the tissues (particularly MMP-8 and MMP-9), resulting in the breakdown of structural components of the periodontium and development of collagen depleted areas. Neutrophils release their potent lysosomal enzymes, cytokines, and reactive oxygen species (ROS) extracellularly, causing further tissue damage.[83] Neutrophil hyperactivity in periodontitis has also been suggested, leading to overproduction of damaging ROS and other mediators.[53] Patients with periodontitis have been reported to have neutrophils that demonstrate enhanced enzymatic activity and produce increased levels of ROS.[112,113]

However, it is not yet clear whether enhanced responsiveness of neutrophils is due to an innate properties of the neutrophils in certain individuals or results from priming by cytokines or bacteria, or a combination of these factors.

It is certainly clear, however, that extracellular release of lysosomal enzymes contributes to continued tissue damage and collagen depletion in the periodontal tissues. Degeneration of fibroblasts limits opportunities for repair, and the epithelium continues to proliferate apically, deepening the pocket further, which is rapidly colonized by the subgingival bacteria. The very first steps in the development of the pocket result from a combination of factors, including detachment of cells at the coronal aspect of the junctional epithelium as those at the apical aspect migrate apically into the collagen-depleted areas and intraepithelial cleavage within the junctional epithelium.[105,150,171] Epithelial tissues do not have their own blood supply and must rely on diffusion of nutrients from the underlying connective tissues. Thus, as the epithelium proliferates and thickens, necrosis of epithelial cells that are more distant from the connective tissues can lead to intraepithelial clefts and splits, also contributing to the first stages of pocket formation.

A cycle of chronic inflammation is therefore established in which the presence of subgingival bacteria drives inflammatory responses in the periodontal tissues, characterized by infiltration by leukocytes, release of inflammatory mediators and destructive enzymes, connective tissue breakdown, and breakdown and proliferation of the epithelium in an apical direction. The junctional and pocket epithelium becomes thin and ulcerated and bleeds more readily. The bacteria in the pocket are never fully eliminated, as they are effectively outside the body, but their continued presence drives the destructive inflammatory response. Attempts at effective oral hygiene are rendered more difficult by the deepening of the pocket, and the cycle continues.

Alveolar Bone Resorption

As the advancing inflammatory front approaches the alveolar bone, osteoclastic bone resorption commences.[31] This is a protective mechanism to prevent bacterial invasion of the bone, but it ultimately leads to tooth mobility and even tooth loss. Resorption of alveolar bone occurs simultaneously with breakdown of periodontal ligament (PDL) in the inflamed periodontal tissues. There are two critical factors that determine whether bone loss occurs: first, the concentration of inflammatory mediators in the gingival tissues must be sufficient to activate pathways that lead to bone resorption, and second, the inflammatory mediators must penetrate to within a critical distance of the alveolar bone.[64]

Histologic studies have confirmed that the bone resorbs so that there is always a width of noninfiltrated connective tissue of about 0.5 to 1.0 mm overlying the bone.[188] It has also been demonstrated that bone resorption ceases when there is at least a 2.5-mm distance between the site of bacteria in the pocket and the bone.[132] Osteoclasts are stimulated by proinflammatory cytokines and other mediators of inflammation to resorb the bone, and the alveolar bone "retreats" from the advancing inflammatory front. Osteoclasts are multinucleate cells formed from osteoclast progenitor cells/macrophages, and osteoclastic bone resorption is activated by a variety of mediators such as IL-1β, TNF-α, IL-6, and PGE_2.[120] Other mediators that also stimulate bone resorption include LIF, oncostatin M, bradykinin, thrombin, and various chemokines.[100]

Receptor Activator of Nuclear Factor-κB Ligand/Osteoprotegerin

A key system for controlling bone turnover is the receptor activator of nuclear factor-κB (RANK)/RANK ligand (RANKL)/osteoprotegerin (OPG) system (see Figure 25-3). RANK is a cell surface receptor expressed by osteoclast progenitor cells as well as mature osteoclasts. RANKL is a ligand that binds to RANK and is expressed by bone marrow stromal cells, osteoblasts, and fibroblasts. Binding of RANKL to RANK results in osteoclast differentiation and activation and thus bone resorption. Another ligand that binds to RANK is OPG, produced by bone marrow stromal cells, osteoblasts, and PDL fibroblasts. Thus RANKL and OPG are cytokines that bind to RANK, resulting in cellular responses. However, although RANKL promotes activation and differentiation of osteoclasts, OPG has the opposite effect, inhibiting differentiation of osteoclasts. The balance between OPG and RANKL activity can therefore drive bone resorption or bone formation.

IL-1β and TNF-α regulate the expression of RANKL and OPG, and T-cells express RANKL, which binds directly to RANK on the surfaces of osteoclast progenitors and osteoclasts, resulting in cell activation and differentiation to form mature osteoclasts. In periodontitis, elevated levels of proinflammatory cytokines, such as IL-1β and TNF-α, and increasing numbers of infiltrating T-cells result in activation of osteoclasts via RANK, resulting in alveolar bone loss. It has been reported that levels of RANKL are higher and levels of OPG are lower in sites with active periodontal breakdown compared to sites with healthy gingiva,[35] and GCF RANKL:OPG ratios are higher in periodontitis than health.[21] It is clear that alterations in the relative levels of these key regulators of osteoclasts play a key role in the bone loss that characterizes periodontal disease.

RESOLUTION OF INFLAMMATION

Inflammation is an important defense mechanism to combat the threat of bacterial infection, but inflammatory mechanisms are also key in the development and progression of most chronic diseases associated with aging, including periodontal disease.[183] It is also becoming evident that resolution of inflammation (i.e., turning off inflammation) is an active process, regulated by specific mechanisms that restore homeostasis. Furthermore, controlling or augmenting these mechanisms may lead to development of novel treatment strategies for managing chronic diseases such as periodontitis. Therefore, although immune-inflammatory responses to infection and injury are necessary for the survival of the host, inflammatory processes can also lead to tissue damage and chronic disease when they persist inappropriately, or are dysregulated or maladaptive.[185]

Most pharmacologic approaches to control inflammation that have been investigated to date have focussed on inhibiting inflammation. Rather than attempting to inhibit inflammation it is possible that using agonists to stimulate key mechanisms that resolve inflammation may offer new perspectives for controlling chronic diseases. Recently, the mechanisms that regulate inflammation have begun to be identified.[84] Resolution of inflammation is an active process that results in a return to homeostasis, and is mediated by specific molecules including a class of endogenous, proresolving lipid mediators, the lipoxins, resolvins, and protectins.[155] These molecules are actively synthesized during the resolution phases of acute inflammation, they are antiinflammatory, and they inhibit neutrophil infiltration. They are also chemoattractants but do not cause inflammation. For example, lipoxins stimulate infiltration by monocytes but without stimulating release of inflammatory cytokines.

Lipoxins

The lipoxins include lipoxin A_4 (LXA_4) and lipoxin B_4 (LXB_4). The appearance of these molecules signals the resolution of

inflammation.[153] Lipoxins are lipoxygenase (LO) -derived eicosanoids and are generated from arachidonic acid. They are highly potent, possessing biologic activity at very low concentrations, and inhibit neutrophil recruitment, chemotaxis, and adhesion.[169] Lipoxins also signal macrophages to phagocytose the remnants of apoptotic cells at sites of inflammation, without generating an inflammatory response. Pro-inflammatory cytokines such as IL-1β released during acute inflammation can induce expression of lipoxins, which promote resolution of the inflammatory response.[114]

Resolvins and Protectins

Resolvins (resolution phase interaction products) are derived from the omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) and are classified as the E series resolvins (RvE) and D series resolvins (RvD), respectively.[154] Resolvins inhibit neutrophil infiltration and transmigration, they inhibit the production of proinflammatory mediators, and they have potent antiinflammatory and immunoregulatory effects.[156] Resolvins are highly potent and have been shown to reduce neutrophil transmigration by around 50% at concentrations as low as 10 nM.[168] Protectins are also derived from DHA. They are produced by glial cells and reduce cytokine expression.[80] They also inhibit neutrophil infiltration and have been reported to reduce retinal injury[118] and stroke damage.[109]

Neutrophils play a key role in initiating acute inflammation in response to injury or infection, but uncontrolled, or excessive, or persistent inflammatory responses can lead to chronic disease. Release of proinflammatory mediators such as cytokines and prostanoids exacerbates the tissue damage. The release of endogenous proresolving molecules that "switch off" inflammation and act as a braking signal for neutrophil activity indicates that control of inflammation is an active process, rather than a passive dwindling of proinflammatory signals. These molecules could potentially offer benefit in the management of chronic diseases such as periodontitis. This concept has been tested in animal models of periodontitis.[184] In a rabbit model of *P. gingivalis*/ligature-induced experimental periodontitis, periodontal inflammation was clearly evident after 6 weeks, characterized by collagen breakdown and resorption of alveolar bone. As the experiment progressed beyond 6 weeks, topical resolvin E1 (RvE1), 4 μg/tooth, was applied 3 times per week for a further 6 weeks, whereas the control group continued to receive applications of topical *P. gingivalis*. In the control group, inflammation continued, leading to increased alveolar bone loss, with large increases in the numbers of osteoclasts, infiltrating neutrophils, and significant collagen breakdown. However, in the animals that received the topical RvE1, the progression of periodontitis was prevented, resolution of inflammation occurred, and the bone loss that had occurred in the first 6 weeks of the study was reversed, with evidence of bone gain in the RvE1-treated animals.[72] These experiments suggest potential for a new area of research that could have promise for developing exciting new strategies for managing periodontal disease.

Whereas plaque bacteria have a primary etiologic role in initiating and perpetuating periodontal disease, the dysregulated or excessive inflammatory responses that develop as a result are the primary determinants of the progression of the disease and likely explain most of the inter-individual variation that we see in clinical presentation of disease. Inadequate resolution of inflammation is likely to be an important component of the pathogenesis of periodontitis.[184] The endogenous proresolving lipid mediators that resolve inflammation could offer potential for the development of powerful and effective new adjunctive treatments for the management of periodontitis. This would represent yet another paradigm shift in the treatment of this complex disease, with a shift in focus toward resolving inflammation rather than inhibiting aspects of the inflammatory response.

IMMUNE RESPONSES IN PERIODONTAL PATHOGENESIS

The immune system is essential for maintenance of periodontal health and is central to the host response to periodontal pathogens. However, if the immune response is dysregulated, inappropriate, persistent, and/or excessive, then damaging chronic inflammatory responses, such as those observed in periodontal disease, can ensue. The immune response to pathogenic microorganisms involves the integration at the molecular, cellular, and organ level of elements often categorized as being part of the innate immune system or the adaptive immune system. Furthermore, host responses in periodontal disease (and other major human diseases) were until recently represented as a linear progression leading from host recognition of microbial pathogens to innate immune responses dominated by the action of phagocytic neutrophils, culminating in the establishment of adaptive immune responses led by antigen specific effector functions such as cytotoxic T-cells and antibodies. Now, it is widely appreciated that immune responses are examples of complex biologic networks in which pathogen recognition, innate immunity, and adaptive immunity are integrated and mutually dependent.[52] This complex network is flexible and dynamic with aspects of positive and negative regulation, as well as feedback control; signals are amplified and broadcast leading to diverse effector functions. Furthermore the immune system is integrated with other systems, including the nervous system, hematopoiesis, and hemostasis, as well as elements of tissue repair and regeneration.[121]

Observational studies of periodontal tissue inform clinical studies and investigations of animal models and isolated cell and tissue systems have allowed us to identify the elements of the immune response relevant to periodontal disease and to relate these to general principles of immunology.[90,131] It is important to appreciate that immune responses, which underpin periodontal disease, have unique facets that must be considered before we can truly rationalize the detailed information we have on individual immune cell functions and their responses to specific periodontal pathogens. Thus we need to understand how the polymicrobial plaque biofilm (as opposed to individual species of periodontal pathogens) interacts with host immune defenses. We need also to appreciate specific immunologic properties that relate to the unique anatomy of the periodontium and the oral mucosal tissue that is integrated with it. We also need to understand how immune responses contribute to the dynamic aspects of periodontal disease, the varying clinical courses and presentations thereof, and how elements of host immunity contribute to tissue destruction, resolution, repair, and regeneration.

Innate Immunity

Defenses against infection comprise a wide range of mechanical, chemical, and microbiologic barriers that prevent pathogens invading the cells and tissues of the body. Saliva, GCF, and the epithelial keratinocytes of the oral mucosa all protect the underlying tissues of the oral cavity and in particular the periodontium. The commensal microflora (e.g., in dental plaque) may also be important in providing protection against infection by pathogenic microorganisms through effective competition for resources and ecologic niches and also by stimulating protective immune responses. The complex microanatomy of the periodontium, including the diversity of specialized epithelial tissues, presents many interesting

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challenges for the study of the immunopathogenesis of periodontal disease.

If these primary defenses are breached, then the cellular and molecular elements of the innate immune response are activated. Innate immunity refers to the elements of the immune response that are determined by inherited factors (and therefore "innate"), have limited specificity, and are "fixed," in as much as they do not change or improve during an immune response or as the result of previous exposure to a pathogen. Recognition of pathogenic microorganisms and recruitment of effector cells (e.g., neutrophils) and molecules (e.g., the complement system) are central to effective innate immunity. We now have much information about the specific recognition of periodontal pathogens and the events that lead to activation of neutrophils in the periodontium, which are orchestrated by a diverse range of cytokines, chemokines, and cell surface receptors. In pathologic terms, stimulation of innate immunity leads to a state of inflammation, and an important area of periodontal research is to understand the relationship between innate immunity and periodontal disease as a chronic inflammatory disorder.

If innate immune responses fail to eliminate infection (e.g., in the susceptible host), then the effector cells of adaptive immune response (lymphocytes) are activated. It is important to note that whereas historically the adaptive immune system has been the focus of much research in immunology and biomedical science, more recently the innate immune system has enjoyed something of a renaissance, fueled by an explosion in knowledge of pathogen recognition systems such as the TLRs. In particular, how the innate immune response signals adaptive immunity is the subject of intense research, not least in the discipline of periodontology. Thus it is increasingly appreciated that the immune response functions as a network of interacting molecular and cellular elements in which innate immunity and adaptive (antigen-specific) immunity work together toward a common purpose. Aspects of innate immunity that are relevant to periodontal disease are now considered.

Saliva. Saliva secreted from the three major salivary glands (parotid, submandibular, and sublingual), as well as from the numerous minor salivary glands, has an important role in maintaining oral and dental health. The action of shearing forces associated with saliva flow is important in preventing the attachment of bacteria to the dentition and the oral mucosal surfaces. Human saliva also contains numerous molecular components that contribute to host defenses against bacterial colonization and periodontal disease (Table 21-4). These components include molecules that nonspecifically inhibit formation of the plaque biofilm by inhibiting adherence to oral surfaces and promoting agglutination (e.g., mucins), those that inhibit specific virulence factors (e.g., histatins that neutralize LPS), and those that inhibit bacterial cell growth (e.g., lactoferrin) and may induce cell death.[60,97] Saliva also contains specific immunoglobulin A (IgA) antibodies to periodontal pathogens that target specific antigens and inhibit bacterial adherence. Patients with periodontal disease have elevated levels of specific IgA, as well as IgG and IgM, antibodies to periodontal pathogens. However, tooth surfaces coated with a salivary pellicle can provide attachment opportunities for plaque bacteria, thus *P. gingivalis* can attach to the salivary pellicle via fimbriae.

TABLE 21-4 Constituents of Saliva that Contribute to Innate Immunity

Saliva Constituent	Host Defense Function
Antibodies (e.g., IgA)	Inhibit bacterial adherence, promote agglutination
Histatins	Neutralize LPS, inhibit destructive enzymes
Cystatins	Inhibit bacterial growth
Lactoferrin	Inhibits bacterial growth
Lysozyme	Lyses bacterial cell walls
Mucins	Inhibits bacterial adherence, promotes agglutination
Peroxidase	Neutralizes bacterial hydrogen peroxide

IgA, Immunoglobulin A; *LPS,* lipopolysaccharides.

Epithelial Tissues. The epithelial tissues play a key role in host defenses as they are the main site of initial interaction between plaque bacteria and the host and also are the site of invasion of microbial pathogens. The keratinized epithelium of the sulcular and gingival epithelial tissues not only provides a protection for the underlying periodontal tissue but also acts as a barrier against bacteria and their products.[11,152] In contrast, the unique microanatomic structure of the junctional epithelium has significant intercellular spaces, is not keratinized, and exhibits a higher cellular turnover rate. These properties render the junctional epithelium permeable, allowing for inward movement of microbes and their products and outward movement of GCF and the cells and molecules of innate immunity. Furthermore, the spaces between the cells of the junctional epithelium widen with inflammation, resulting in increased GCF flow.[152]

Some species of periodontal bacteria invade host epithelial tissues; at the molecular level the processes of adhesion and invasion are coupled. Studies of the invasion of gingival epithelial cells by *P. gingivalis* have served as a paradigm for the study of this process; infection of host cells by *P. gingivalis* involves the action of proteases and cell surface fimbriae.[3,96] Invasion by *P. gingivalis* is initiated through signalling via interaction of bacterial components with surface integrins, PAR-1 and PAR-2 and TLR.[67,96,196] This in turn activates intracellular signalling pathways (e.g., mitogen-activated protein kinase [MAPK]) and results in the reorganization of actin filaments and microtubules and a modulation of Ca^{2+} influx. It is thought that inhibition of host cell apoptosis may facilitate survival of intracellular bacteria and that bacteria in this compartment are afforded protection from elements of the host immune response. Invasion of host cells could therefore be relevant in the spread and persistence of certain periodontal bacteria. Also, in vivo analysis and studies of a three-dimensional (3D)-engineered human oral mucosa model demonstrate that *P. gingivalis* can migrate through the basement membrane of epithelial layers and invade connective tissue.[3] Histologic analysis reveals that, in periodontitis, epithelial cells become more rounded and tend to detach from the underlying connective tissue.[152] Proteases break down cell-cell junctions in epithelial tissues by digesting transmembrane proteins and adhesion molecules (e.g., E-cadherin). Microanatomic changes associated with the onset of periodontitis, such as the widening of the spaces between the cells of the junctional epithelium and the development of the pocket epithelium, further facilitate bacterial invasion. Infection through the basement membrane and into the underlying connective tissues is facilitated by bacterial-derived proteases and host proteases derived from infiltrating neutrophils.

At the cellular and molecular level, most in vitro studies of epithelial cell responses to periodontal bacteria have been carried out in primary gingival epithelial cells or various immortalized cell lines derived from oral epithelial tissue, and these studies have

provided insight into host-cell responses to periodontal bacteria.[3,67,69,196] Epithelial cells also constitutively express antimicrobial peptides (e.g., hBDs and LL-37) and the synthesis and secretion of these molecules is upregulated in response to periodontal bacteria. Neutrophils are also a source of antimicrobial peptides (α-defensins). Antimicrobial peptides are small, polycationic peptides that disrupt bacterial cell membranes and thereby directly kill bacteria with broad specificity.

For more information on epithelial tissues, please visit the companion Expert CONSULT website at www.expertconsult.com.

Epithelial cells stimulated with bacterial components and cytokines directly produce MMPs, which contribute to loss of connective tissue. Epithelial cells also secrete a range of cytokines in response to periodontal bacteria (such as *P. gingivalis, A. actinomycetemcomitans, F. nucleatum,* and *P. intermedia*), which signal immune responses. These include the proinflammatory cytokines IL-1β, TNF-α, and IL-6, as well as the chemokines IL-8 (CXCL8) and monocyte chemoattractant protein-1 (MCP-1), which serve to signal neutrophil and monocyte migration from the vasculature into periodontal tissue.

In some (but not all) experimental systems, *P. gingivalis* has been shown to inhibit IL-8; it is suggested that in vivo this effect may have a temporary local immune suppression in the periodontium and facilitate the accumulation and invasion of pathogenic periodontal bacteria and the initiation of periodontitis.[39,67] *P. gingivalis* is an example of one periodontal pathogen with a range of virulence factors that affect host immune defenses,[67,96] as indicated in Table 21-5.

Gingival Crevicular Fluid. GCF originates from the postcapillary venules of the gingival plexus. It has a flushing action in the gingival crevice but also likely functions to bring the blood components (e.g., neutrophils, antibodies, and complement components) of the host defenses into the sulcus.[65] The flow of GCF increases in inflammation, and neutrophils are an especially important element of GCF in health and disease.[90]

Pathogen Recognition and Activation of Cellular Innate Responses. If plaque bacteria and their products penetrate the periodontal tissues, then specialized "sentinel cells" of the immune system can recognize their presence and signal protective immune responses. Thus macrophages and dendritic cells express a range of pattern recognition receptors (PRRs) that interact with specific molecular structures on microorganisms called *microbe-associated molecular patterns* (MAMPs) to signal immune responses. Thus innate immune responses are activated that provide immediate protection, and adaptive immunity is also activated with the aim of establishing a sustained antigen-specific defense. Excessive and inappropriate immune responses lead to chronic inflammation and the concomitant tissue destruction associated with periodontal disease. A glossary of terms relevant to periodontal immunobiology is presented in Table 21-6.

The best studied of the signalling systems involved in recognition of plaque bacteria is the interaction of bacterial LPS with TLRs: *P. gingivalis, A. actinomycetemcomitans, F. nucleatum* all possess LPS molecules that interact with TLR-4 to activate myeloid immune cells. However, individual species of plaque bacteria have a wide variety of MAMPs, which may interact with PRRs. Studies of *P. gingivalis* have served as a paradigm for investigations of host-bacteria interactions in periodontal disease at the molecular level. Thus *P. gingivalis* LPS signal via TLR (predominantly TLR-2), and fimbriae, proteases, and DNA from *P. gingivalis* are all recognized by host cells through interaction with specific PRRs. A number of nonimmune cells in the periodontium (epithelial cells, fibroblasts) also express PRRs and may recognize and respond to MAMPs from plaque bacteria.

Although the signalling pathways activated by PRRs may be diverse, in general terms they converge to elicit similar host-cell responses in the form of upregulation of cytokine secretion and in the case of antigen-presenting cells, such as dendritic cells, cell differentiation leading to enhanced signalling of the adaptive immune response. Dendritic cells also have C-type lectin receptors (e.g., mannose receptor, langerin, and DC-SIGN) which recognize glycans on pathogens but the role of these interactions in periodontal disease is not known.

Signalling of cytokine responses via PRRs influences innate immunity (e.g., neutrophil activity), adaptive immunity (T-cell effector phenotype), and the development of destructive inflammation (e.g., activation of fibroblasts and osteoclasts). A number of cytokines are particularly important in innate immune signalling, and there is now good evidence that these have a role in immune responses in the periodontium. The archetypal proinflammatory cytokine is IL-1β, which exerts its action directly by activating other cells that express the IL-1R1 receptor (e.g., endothelial cells) or by stimulating the synthesis and secretion of other, secondary mediators such as PGE_2 and NO. The effect of IL-1β is amplified via a synergistic action with other cytokines such as TNF-α. Upregulation of ICAM-1 and E-selectin on endothelial cells is central to the migration of neutrophils into the periodontium, and this is stimulated by IL-1β and TNF-α. IL-1β also stimulates the secretion of the chemokine IL-8, which stimulates neutrophil chemotaxis (see next section). IL-1β and TNF-α also activate MMP secretion from fibroblasts and osteoclasts; this may facilitate the movement of neutrophils in the connective tissues and thus protective innate responses but may also ultimately contribute to tissue destruction associated with periodontal disease, along with MMPs from neutrophils.

For more information on pathogen recognition and activation of cellular innate responses, please visit the companion Expert CONSULT website at www.expertconsult.com.

Neutrophil Function. Although macrophages have phagocytic capability, neutrophils are the "professional" phagocytes

TABLE 21-5 Virulence Factors of *P. gingivalis* that Interact with the Immune System

Virulence Factor	Effect on Immune System
Proteases (gingipains)	Degradation of signalling molecules (CD14) and cytokines (IL-1β, IL-6)
Cell invasion capability	Inhibition of IL-8 secretion
LPS	Antagonism of the stimulatory effects of LPS from other species; no upregulation of E-selectin
Fimbriae	Inhibition of IL-12 secretion in macrophages
Cell surface polysaccharide	Resistance to complement
Short-chain fatty acids	Induction of apoptosis in host cells

IL, Interleukin; *LPS,* lipopolysaccharides.

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TABLE 21-6 Glossary of Terms Relevant to Periodontal Immunobiology

Term	Definition
Cluster of differentiation (CD)	Molecules that define different immune cell lineages, functional phenotypes, and stages of development.
CD4	Characteristic coreceptor molecule of T-helper cells.
CD8	Characteristic coreceptor molecule of cytotoxic T-cells.
CD14	Binds to LPS, facilitating interaction with TLR, exists in soluble and membrane-bound forms.
Cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2)	Enzymes responsible for synthesis of inflammatory mediators (e.g., PGE_2) from arachidonic acid.
Complement receptor-3 (CR-3)	Cellular receptor for complement that interacts with *P. gingivalis* fimbriae in signalling cytokines.
CXCL and CCL	Two structurally distinct groups of chemokines, which are small proteins that activate and stimulate the movement of leukocytes.
Dendritic cell-specific ICAM-3-grabbing nonintegrins (DC-SIGN)	Cell surface molecule on dendritic cells that has a role in cell-cell interactions and antigen presentation.
Extracellular DNA (eDNA)	A component of bacterial biofilms that may activate innate immunity.
Gingival crevicular fluid (GCF)	Serum exudate that appears in the gingival sulcus (particularly in periodontitis) and that contains molecular and cellular elements of the immune response.
GroEl	Bacterial heat shock proteins that stimulate immune responses.
Human β-defensins (hBD)	Antimicrobial peptides synthesized by epithelial cells that kill bacterial cells but have other functions in immune responses.
Intercellular adhesion molecule-1 (ICAM-1)	Expressed on endothelial molecules during inflammation and interacts with integrins on neutrophils to facilitate neutrophil movement.
Interleukin (IL)	Generic term for cytokines produced by leukocytes that are grouped into families based on structural and functional similarity (e.g., IL-1 family).
Lysine-specific gingipain A (KgpA)	Enzyme secreted by the periodontal pathogen *P. gingivalis,* which is an important virulence factor.
Leukemia-inhibitory factor (LIF)	Cytokine of the IL-6 family.
Peptide cleavage product of human cathelicidin (LL-37)	An antimicrobial peptide widely expressed in tissues, which kills bacterial cells and has other functions in immune responses.
Lipopolysaccharide (LPS)	A bacterial cell surface molecule characteristic of gram-negative bacteria which stimulates innate cells via TLR.
Lipoteichoic acid (LTA)	Characteristic molecule of gram-positive bacterial cell walls, which activates innate immune cells via TLR.
Lipoxin A_4 (LXA_4), lipoxin B_4 (LXB_4)	Arachidonic acid derivatives involved in resolution of inflammation.
Microbe-associated molecular pattern (MAMP)	Molecules associated with microorganisms, which activate innate immunity through specific signalling pathways.
MD-2	Part of the LPS receptor complex; binds to TLR as a prerequisite for binding LPS.
Matrix metalloproteinases (MMPs)	Family of proteolytic enzymes that degrade extracellular matrix molecules.
Natural killer (NK) cells	Large granular lymphocytes, distinct from T-cells and B-cells that are important in innate immunity to intracellular pathogens.
Nucleotide oligomerization domain receptors 1 (NOD 1) and (NOD 2) 2	Intracellular PRRs that interact with intracellular bacteria and stimulate innate immunity.
Osteoprotegerin (OPG)	Cytokine-like molecule that binds to RANK but antagonizes the action of RANKL.
Protease activated receptors 1 (PAR-1) and 2 (PAR-2)	Cellular receptors, which when activated by bacterial enzymes can signal cytokine secretion.
Prostaglandin E_2 (PGE_2)	Inflammatory mediator derived from arachidonic acid.
Pattern-recognition receptors (PRRs)	Integrated membrane proteins and intracellular molecules of immune cells that interact with MAMPs and signal innate immunity.
Receptor activator of nuclear factor-κB (RANK)	Osteoclast cell surface receptor important in bone regulation.
Receptor activator of nuclear factor-κB ligand	Cytokine-like molecule that is the ligand for RANK.
Arginine-specific gingipains A (RgpA) and B (RgpB)	Enzymes secreted by the periodontal pathogen *P. gingivalis,* which are important virulence factors.
T-helper 1 subset (Th1 cells)	A subset of $CD4^+$ T-cells that are characterized by their cytokine secretion profile and that activate cellular immunity.
T-helper 2 subset (Th2 cells)	A subset of $CD4^+$ T-cells that are characterized by their cytokine secretion profile and that activate humoral (antibody) immunity.
T-helper 17 subset (Th17 cells)	A subset of $CD4^+$ T-cells that are characterized by the secretion of IL-17 and that activate inflammatory responses (e.g., neutrophils).
Tissue inhibitors of matrix metalloproteinases (TIMPs)	Molecules concerned with the regulation of MMPs.
Toll-like receptors (TLRs)	A class of PRRs that recognize MAMPs, such as LPS, and signal cytokine secretion in innate cells.
Tumour necrosis factor alpha (TNF-α)	Classic proinflammatory cytokine that activates innate immunity and shares many actions with IL-1β.
Regulatory T-cell subset (T_{reg} cells)	A subset of $CD4^+$ T-cells that are characterized by the secretion of TGF-α and IL-10 and that inhibit immune responses.

critical to clearance of bacteria that may invade host tissues. Neutrophils are a feature of healthy gingival tissues, and there is a significant migration of these cells in the absence of clinical signs of inflammation through the intercellular spaces of the junctional epithelium.[90,152] This is part of a "low grade defense" against plaque bacteria and is necessary to prevent inflammation and periodontal tissue damage.[152] The importance of neutrophils in maintaining periodontal health is demonstrated clinically by the observations of periodontitis in patients with neutrophil defects[71] and the association of periodontitis with experimental immunosuppression in animal models.[90]

Small foci of other leukocytes (lymphocytes, plasma cells, or macrophages) are also found in the healthy gingiva. A small proportion (1% to 2%) of the intercellular spaces in healthy junctional epithelium are occupied by neutrophils (and other leukocytes at various stages of differentiation); but this can increase to 30% with even modest inflammation. In the inflammatory state there are changes to the local vasculature in the gingiva: high endothelial venules develop from the postcapillary venules of the gingival plexus, which facilitates leukocyte emigration and increases flow of GCF into the pocket.[152]

Neutrophils migrate from the gingival plexus to the extravascular connective tissue and then into the junctional epithelium via the basement membrane. The presence of a layer of neutrophils in the junctional epithelium forms a host defense barrier between subgingival plaque and gingival tissue. At the molecular level, the interaction of adhesion molecules (e.g., ICAM-1 and LFA-3) on endothelial and epithelial cells with β2 integrins on neutrophils facilitates neutrophil migration.

Indeed there is some evidence from immunohistochemistry for the existence of gradients of IL-8 (a "chemotactic gradient"), as well as gradients of ICAM-1, which purportedly direct the neutrophils from the vasculature into the tissues and to the junctional epithelium.[179] Increased leukocytes in the periodontium contribute to the disruption of the junctional epithelium by degradation of basement membrane through protease release and the action of reactive oxygen species. Increased expression of IL-8 and adhesion molecules in inflammation may be the result of direct signalling by bacterial products or by signals from cytokines (e.g., IL-1β and TNF-α) upregulated by plaque bacteria. Acute phase proteins, such as α2-macroglobulin, are increased in periodontal tissues as a result of increased vascular leakage in the inflammatory state, resulting in complement and plasmin activation, which also contributes to host defenses.

The neutrophils that infiltrate the periodontium express F_c receptors and complement receptors commensurate with their function in phagocytosis of opsonized bacteria and bacterial antigens. Significantly, the function of neutrophils is not obviously affected by the anaerobic environment within the periodontal pocket. Within the sulcus itself, 95% of the cells are neutrophils, and since neutrophils are present in both health and disease, the transmigration is thought to be a process distinct from that of GCF flow. The remaining 5% of cells are other leukocyte subtypes possibly passively carried into the sulcus by the flow of GCF. The relative proportions of the different leukocytes are highly variable, possibly reflecting the fact that their presence is the result of a passive process and/or a reflection of the natural fluctuations of different cell types during an immune response. Also, the distribution of leukocytes in periodontal tissues is not even; mononuclear cells predominate in the connective tissue and neutrophils in sulcus. This may be the result of selective mechanism mediated by specific chemokine responses and adhesion molecule interactions.

Adaptive Immunity

Adaptive immunity has evolved to provide a focussed and intense defense against infections that overwhelm innate immune responses in the tissues. Adaptive immunity is particularly important as ecologic, social, and demographic changes, which alter susceptibility to existing and emerging infective microorganisms, outpace the natural evolution of biologic systems. Furthermore, the development of effective vaccination is, along with the identification of antibiotics, perhaps one of the greatest triumphs of medical science; this success is based on knowledge of the elements and principles of adaptive immunity. Although the use of effective vaccines against periodontal pathogens remains a topic of research rather than a therapeutic application, it is widely accepted that understanding of immune responses against periodontal pathogens in its widest sense will eventually yield novel therapeutic strategies.

Adaptive immunity contrasts with innate immunity in the dynamic of the underlying cellular and molecular responses: adaptive immunity is slower and reliant on complex interactions between antigen-presenting cells and T- and B-lymphocytes. A key element is the antigen specificity of the responses that facilitates specific targeting of a diverse range of effector elements, including cytotoxic T-cells and antibodies. Another facet is the ability of adaptive immune responses to improve during exposure to antigen and on subsequent infection events. Our current understanding suggests that the cellular and molecular elements of adaptive immunity are more diverse than those of innate immunity, and although a role for many of these factors in periodontal disease has been identified, our knowledge is far from complete. Innate recognition systems interface with periodontal pathogens in the initial stages of pathogenesis, whereas adaptive immune responses predominate in established disease; however, it is widely accepted that the clinical ebb and flow of periodontal disease is a reflection of fluctuations in adaptive immunity.

The importance of adaptive immune responses in periodontal pathogenesis is endorsed by histologic studies of established lesions in periodontal disease.[90,131] The population of leukocytes in the periodontium in gingivitis (i.e., the early stages of responses to the plaque biofilm) and in stable periodontal lesions (i.e., those at which tissue destruction is apparently not progressing) is dominated by T-cells, and these cells are clustered mainly around blood vessels. Cell surface marker studies suggest these cells are activated but not proliferating.[59] Also, there is a predominance of the helper T-cell subset (i.e., CD4 expressing T-cells) over the cytotoxic T-cell subset (i.e., CD8 expressing T-cells). These T-cells are considered to be proactively maintaining tissue homeostasis in the face of the microbial challenge of the plaque biofilm.[59] In contrast, in active periodontitis, B-cells and plasma cells predominate and are associated with pocket formation and progression of disease.

Antigen-Presenting Cells. A central element of the activation and function of T-cells and B-cells is the presentation of antigen by specialized antigen-presenting cells to T-cells and the development of a specific cytokine milieu that influences the development of T-cells with a particular effector function. Antigen-presenting cells are sentinel cells in mucosal tissues such as the periodontium. These cells detect and take up microorganisms and their antigens, after which they may migrate to lymph nodes and interact with T-cells to present antigen. The periodontium is often compared to other mucosal tissues and the skin in terms of its repertoire of immune cells, and it contains a number of "professional" antigen-presenting cells.[36] These include B-cells, macrophages, and at least two types of dendritic cells (dermal dendritic cells and Langerhans cells). These cells naturally express major

histocompatibility complex (MHC) Class II molecules necessary for antigen presentation to cognate T-cell receptors and may take up specific antigens and transport them to local lymph nodes, thereby facilitating the activation of specific effector T-cells and the generation of an antigen-specific immune response to periodontal pathogens. Although these cells have been identified in periodontal tissues, have been shown to stimulate antigen specific T-cell responses in experimental systems, and are generally increased in periodontitis, their relative contribution to antigen presentation in vivo remains to be determined. Expression of MHC Class II molecules may be induced in other cells present in the periodontium (e.g., fibroblasts and epithelial cells), which then also take up antigen and present antigen locally in the periodontium.

For more information on antigen-presenting cells, please visit the companion Expert CONSULT website at www.expertconsult.com.

T-Cells. There are a number of different subsets of thymic lymphocytes (T-cells) that develop in the bone marrow and thymus and migrate to the peripheral tissues to participate in adaptive immune responses. The expression of the cell surface molecules (CD4 or CD8) or particular T-cell antigen receptors (αβ or γδ) broadly defines functional T-cell subsets that emerge from the thymus. The role of T-cells in periodontal disease has been established through immunohistologic studies of disease tissues.[157] $CD4^+$ helper T-cells are the predominant phenotype in the stable periodontal lesion, and it is thought that alterations in the balance of effector T-cell subsets within the $CD4^+$ population may lead to progression toward a destructive, B-cell–dominated lesion.[59] $CD4^+$ T-cell subsets are defined on the basis of their phenotypic characteristics and effector functions. The nature of the antigen-presenting cells, which present antigen to cognate T-cell receptors on T-cells, and the presence of specific combinations of cytokines and chemokines influence the nature of the $CD4^+$ T-cell effector subset, which develops from naive T-cells (Figure 21-5). $CD4^+$ T-cell subsets are defined by the expression of specific transcription factors and their functional characteristics are associated with their cytokine secretion profile.

The best-defined functional subsets of $CD4^+$ T-cells are the Th1 and Th2 cells, and a dynamic interaction between Th1 and Th2 cells may provide an explanation for fluctuations in disease activity and progression of periodontal disease (Box 21-3). Th1 cells secrete IFNγ, which activates cell-mediated immunity (macrophages, natural killer (NK) cells, and $CD8^+$ cytotoxic T-cells) against pathogenic microorganisms. Activation of macrophages promotes phagocytosis and killing of microbial pathogens, whereas NK cells and $CD8^+$ T-cells are cytotoxic T-cells that kill infected host cells. Conversely, Th2 cells regulate humoral (antibody-mediated) immunity and mast cell activity through the secretion of the cytokines IL-4, IL-5, and IL-13. Thus predominance of Th2 cells leads to a B-cell response. The B-cell response may be protective, for example, by the production of specific antibodies that would serve to clear tissue infections through interaction with the complement system and by enhancing neutrophil phagocytosis. However, B-cells are also a source of proinflammatory cytokines that contribute to tissue destruction.

For more information on T-cells, please visit the companion Expert CONSULT website at www.expertconsult.com.

Cytokines produced by differentiated T-cell subsets feedback to stimulate differentiation and sustain the activity of the cells from which they are derived (i.e., in a positive feedback loop). Simultaneously, they inhibit the development of other, competing subsets. For example, IL-4 from Th2 cells inhibits the development of Th1 cells, and IFNγ from Th1 cells inhibits Th2 cells.

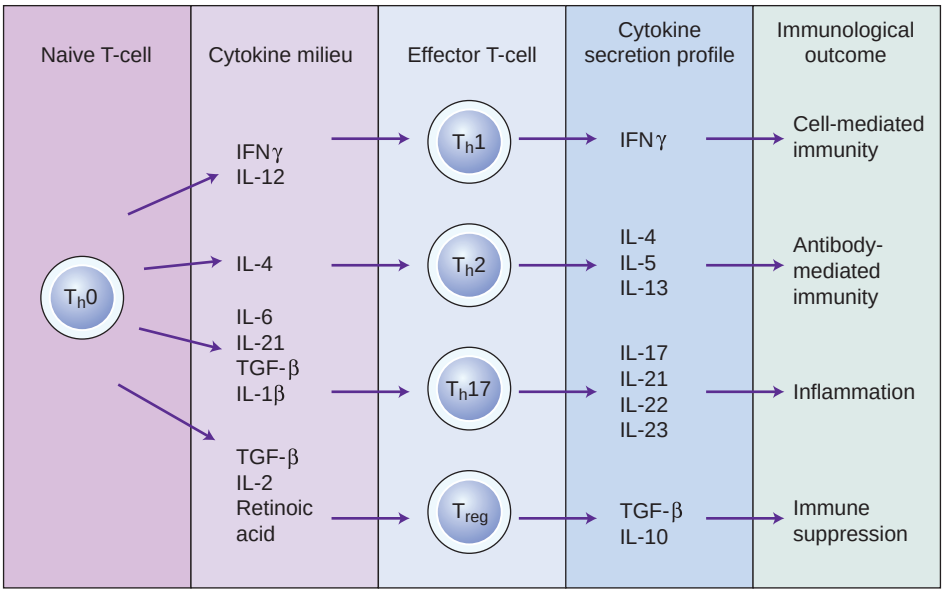

Figure 21-5 $CD4^+$ T-cell subsets and immune responses to microorganisms. Antigen-presenting cells (dendritic cells, macrophages, and B-cells) present specific antigen to naive $CD4^+$ T-cells (Th0 cells), which then differentiate into $CD4^+$ effector T-cells (i.e., Th1, Th2, Th17, and T_{reg} cells) and regulate different aspects of the immune response. Cytokines produced by innate cells in response to microbe-associated molecular patterns (MAMPs) signalling via pattern-recognition receptors (PRRs) influence the nature of the $CD4^+$ effector T-cells that develop from activated Th0 cells. The nature of the antigen-presenting cell and the nature of the antigen itself are also factors that influence effector T-cell function. *IL*, Interleukin; *IFNγ*, interferon gamma; *TGF-β*, transforming growth factor beta.

It is increasingly appreciated that individual CD4+ T-cell clones, once they have encountered antigen and differentiated in response under the influence of a specific cytokine environment, may not be terminally differentiated cells but rather there is functional flexibility between T-cell subsets and in particular within the memory T-cells population (Figure 21-6). Thus Th17 and T_{reg} cells can interconvert, depending on the local concentrations of IL-6, IL-23, and TGF-β.[173] Therefore a picture of functional plasticity in effector T-cells subsets associated with regulating specific immune responses to pathogens is emerging. It is thought that the plethora of functional subsets of T-cells, their anatomic location, and their ability to switch phenotype is a reflection of the requirement for effective responses against diverse pathogens, and this is certainly likely to be a highly relevant facet of immune responses in polymicrobial diseases such as periodontal disease. Other immune cells also have subsets that are defined by expression of cell surface markers and diverse functional and anatomic locations (e.g., myeloid immune cells and NK cells, but these are less well defined than CD4+ T-cell subsets).

Emerging evidence indicates therefore that periodontal pathogenesis involves complex interactions between a number of interacting T-cell subsets that not only modulate adaptive immune function (Th1, Th2 and T_{reg} cells) but also feedback to modify and enhance innate function (Th17 cells).[54] The balance of these different subsets may very well shift and alter longitudinally in periodontal disease. However, the direct relationship (if any) between different T-cell subsets and different clinical presentations remains to be determined.

BOX 21-3 The T-Helper 1/T-Helper 2 Concept of Periodontal Disease Progression

A dynamic interaction between T-helper 1 (Th1) and T-helper 2 (Th2) cells represents a possible explanation for aspects of the fluctuations in disease activity and clinical progression seen in periodontal disease. It has been hypothesized that a strong innate response results in interleukin-12 (IL-12) synthesis (e.g., by tissue macrophages), leading to a Th1 response providing protective cell-mediated immunity that would be manifested as a "stable" periodontal lesion. Conversely, a poor innate response would lead to reduced IL-12, which would permit the development of Th2 responses, leading to activation of B-cells that in turn would mediate a destructive lesion possibly through enhanced B-cell–derived IL-1β.[59,158] Definitive evidence for the existence of Th1 and Th2 cells and any association with different clinical presentations of periodontal disease has proved difficult to obtain, however. It has been suggested that this is because of the variation in the features of experimental studies, which have differed with respect to the material that has been used (and in particular the definitions of disease stage), the experimental design, and analytic methods employed.[54,59,158] Also, in general terms, the Th1/Th2 dichotomy does not explain all aspects of regulation of adaptive immune responses. More recently, other T-cell subsets have been identified and defined. For example, regulatory T-cells (T_{reg}) secrete IL-10 and transforming growth factor beta (TGF-β) and thereby suppress immune responses. Th17 cells have a proinflammatory action via secretion of IL-17, a cytokine that synergizes with IL-1β and tumor necrosis factor alpha (TNF-α). Therefore, although it is widely accepted that Th1 and Th2 cells are likely to be important in the immunopathogenesis of periodontal disease, it is increasingly recognized that the Th1/Th2 model alone is probably inadequate to explain the role of T-cells in this process.

Antibodies. Specific antibodies are produced in response to an increasing bacterial challenge in periodontal disease and are the endpoint of B-cell activation. Circulating antibodies may be more important than locally produced antibodies, but even so, these generally appear in a high titer but have low biologic activity so there is some doubt as to their effectiveness. Commensurate with the appearance of antibodies against plaque bacterial antigens is the appearance of differentiated plasma cells that characterize the established lesion in periodontal disease. High levels of antibodies appear in GCF (in addition to those in the circulation), and these are produced locally by plasma cells in periodontal tissues.[9] Antibodies to periodontal pathogens are primarily IgG with few IgM or IgA types produced.

Many species of oral bacteria elicit a polyclonal B-cell response (with the consequent production of specific antibodies against those bacteria). However, these responses augment responses against nonoral bacteria and may lead to production of autoantibodies (e.g., antibodies against collagen and connective tissue proteins), which may contribute to tissue destruction in periodontal

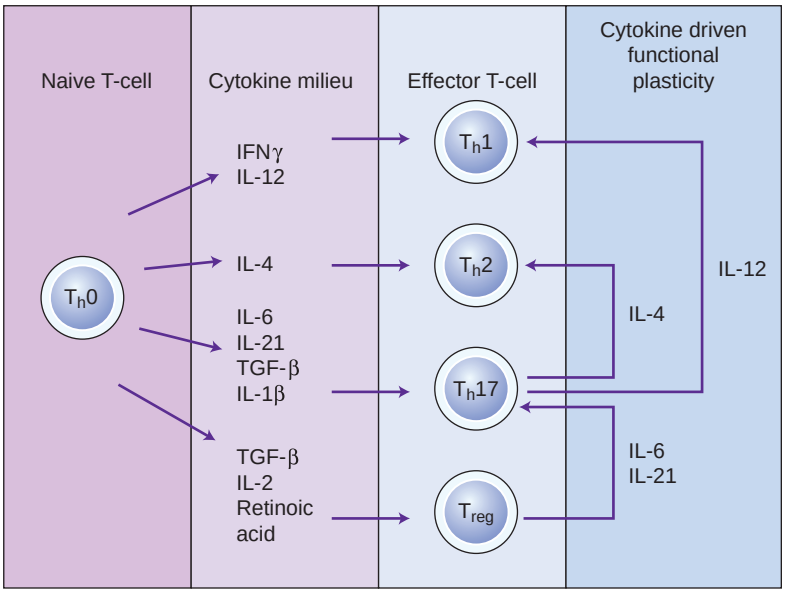

Figure 21-6 Plasticity of T-cell subsets. Although Th1 and Th2 cells have a relatively stable phenotype, other T-cell subsets are functionally "plastic" and can alter phenotype under the influence of different cytokine environments. Thus Th17 cells can develop into Th1 and Th2 cells under the influence of IL-12 and IL-4, respectively. T_{reg} cells can convert into Th17 cells in the presence of IL-6 and IL-21. Other effector T-cell subsets with unique cytokine secretion profiles have been identified (e.g., Th9 and Th22 cells), although these may be transitory and their in vivo relevance remains to be determined. *IL,* Interleukin; *IFNγ,* interferon gamma; *TGF-β,* transforming growth factor beta.

disease.[9,59] The incidence and levels of specific serum and GCF IgG antibodies are raised in chronic periodontitis, which suggests that local and peripheral generation of antibodies may be important in the immune response to periodontal pathogens. Antibodies (IgA) to periodontal pathogens are also found in saliva. Variations in the levels of specific antibodies to different species in different clinical presentations suggest differences in pathogenesis. For example, antibodies to *A. actinomycetemcomitans* of IgG_2 subclass predominate in aggressive periodontitis.[148]

Other *P. gingivalis* molecules (fimbriae and hemagglutinin) also act as antigens. Specific antibodies are also generated by serotype specific carbohydrate antigens (e.g., capsular polysaccharide of *P. gingivalis* and carbohydrate of *A. actinomycetemcomitans* LPS). The subclass distribution of antibodies is influenced by cytokines derived from monocytes.[148] For example, IgG_2 production is regulated by IL-1α, IL-1β, and PGE_2 from monocytes, as well as PAF from neutrophils. PGE_2 and PAF indirectly induce Th1 responses and therefore IFNγ, which stimulates IgG_2 production. Individuals with aggressive periodontitis have monocytes that are hyperresponsive to LPS and produce elevated quantities of PGE_2.[9] *A. actinomycetemcomitans* is commonly associated with aggressive periodontitis, which induces IL-12 production that regulates NK cells and Th1 cells. These cells are a source of IFNγ, which in turn regulates IgG_2.

A number of studies have reported an effect of treatment on levels of specific antibodies to periodontal pathogens. For example, plaque removal reduces the titers of antibodies to *P. gingivalis* and *A. actinomycetemcomitans* in serum, GCF, and saliva.[9] Some studies have observed a transient increase in antibody titers after treatment, which may be due to the release of antigens into the tissue and circulation.

The significance of antibodies in periodontitis is not clear. It is not known if these antibodies have a protective function and whether they participate in disease pathogenesis. Although there is some evidence for a correlation between clinical parameters of disease and titer of specific antibodies to periodontal pathogens, other studies report an inverse correlation of antibody levels and avidity with periodontal destruction. Also, specific antibodies to periodontal pathogens are found in healthy individuals, as well as in those with periodontal disease.

For more information on antibodies, please visit the companion Expert CONSULT website at www.expertconsult.com.

CONCEPT OF HOST SUSCEPTIBILITY

The immune and inflammatory processes that result from the challenge presented by the subgingival biofilm are complex, mediated by a large number of proinflammatory and antiinflammatory cytokines and enzymes that function as a network of mediators with overlapping roles and activity. Immune responses to the bacterial challenge do not occur in isolation but take place in the context of other host and environmental factors that influence these responses and thereby determine the progression of disease. A number of risk factors increase susceptibility to periodontal disease, including smoking, systemic diseases such as diabetes, nutritional factors, and stress, and these are considered in detail elsewhere in this book.

A feature of human development and evolution has been that quantitative and qualitative differences exist in immune responses between individuals.[75] Indeed, infectious agents (e.g., bacteria) exert evolutionary selection pressures on the species that they infect. This may be relevant in periodontal disease, and a large number of studies have confirmed that immune cells from patients with periodontal disease secrete higher quantities of proinflammatory cytokines than those who are periodontally healthy.[174] Cytokine profiles are also different in those individuals with immune-mediated diseases compared to healthy controls.

These observations have led to the development of the concept of the "hyperinflammatory trait," in which certain individuals possess a hyperinflammatory phenotype and that this accounts for their increased susceptibility to chronic inflammatory conditions such as periodontitis. Such a trait may also underpin shared susceptibility between conditions such as periodontitis and cardiovascular disease or diabetes. Researchers have focused on genetic polymorphisms that may result in this hyperinflammatory trait, and many studies have investigated associations between the presence of single nucleotide polymorphisms (SNPs) in the IL-1 genes and periodontal disease (see Chapter 24). These studies suggest that genetic variants may be identified that confer increased susceptibility to periodontitis. Periodontal disease is a polygenic disease in which many interacting gene variants contribute to disease susceptibility. Given the critical role of cytokines in periodontal pathogenesis, investigation of the cytokine genes and immune regulation continues to be an important area of study.

Therefore, at present, it is not possible to identify with certainty those patients who may possess some form of hyperinflammatory trait. The concept of the periodontal hyper-responder (together with other risk factors) could explain increased susceptibility to periodontal disease.[29] The hyper-responder concept was originally proposed in the context of responsiveness of monocytes to LPS challenge, suggesting that patients with disease possess an individual hyper-responsive monocytic trait, characterized by elevated levels of inflammatory mediators released from monocytes in response to bacterial challenge.[125] It likely that there are many reasons contributing to disease variation between individuals, such as variations in immune responses, pathogenesis, and the plaque biofilm, resulting in an uneven disease experience in the population.

Figure 21-7 is a schematic illustration of how increasing bacterial (LPS) challenge can result in differing levels of inflammatory response according to the response profile of an individual patient.[125] Most individuals would be considered normal and for a given bacterial challenge would produce a certain level of inflammatory mediators in the periodontal tissues. For those who are hyperresponders, the same bacterial challenge results in a greater inflammatory response, which over time would result in increased tissue breakdown, earlier presentation of the clinical signs of disease, and a clinical interpretation of having increased susceptibility to periodontitis. Those individuals who are hypo-responsive produce low levels of inflammatory mediators and are therefore somewhat resistant to developing periodontitis, notwithstanding the fact that plaque may be present and they may have widespread gingivitis. The nature of the inflammatory response will be governed by genetic factors and environmental factors and may vary over time within the same individual (e.g., if environmental factors, such as smoking status, stress, or systemic disease, should change).

A similar dose-response curve can also be expressed in the context of stable or progressing disease and as shown in Figure 21-8, a certain level of bacterial challenge results in a moderate release of inflammatory cytokines, mediators, and enzymes. These mediators, together with the infiltrating defense cells have a protective role to eliminate bacteria in the sulcus and do not trigger periodontal disease breakdown. Such a steady-state scenario may

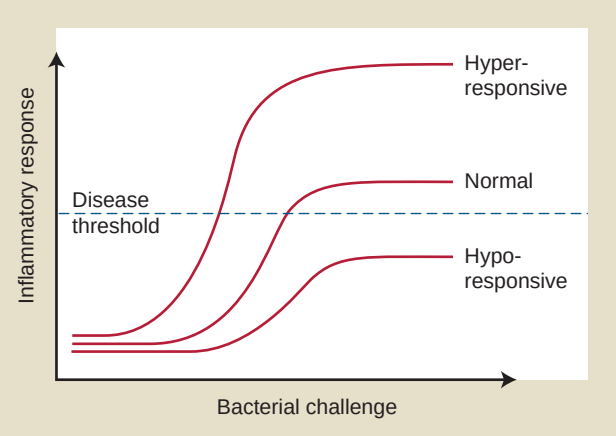

Figure 21-7 Inflammatory response characteristics in relation to bacterial challenge. A given bacterial challenge results in differing levels of inflammatory response according to the response profile of an individual. Most people are close to normal and produce a certain level of inflammatory mediators for a given challenge. Those who are hyper-responders generate an excessive inflammatory response for the same bacterial challenge and cross the threshold into active disease at an earlier stage. Those who are hypo-responsive produce lower levels of inflammatory mediators and despite a significant bacterial challenge, may never develop periodontitis. *(Modified from Champagne CM, Buchanan W, Reddy MS, et al:* Periodontol 2000 *31:167-180, 2003.)*

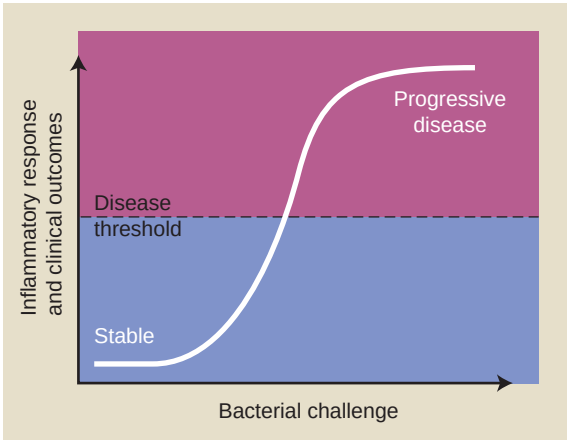

Figure 21-8 Inflammatory response characteristics in relation to threshold for periodontitis. A certain level of bacterial challenge results in a moderate inflammatory response, which is protective by intent, and may not be sufficient to transition to periodontal disease. This stable condition may persist for many years or forever. Changes in the bacterial burden (qualitative and/or quantitative) or changes in the host response (e.g., as a result of change in an environmental exposure), could result in an upregulated inflammatory response characterized by a marked cellular infiltrate and increased secretion of inflammatory mediators leading to tissue damage and a transition from the stable situation to periodontitis. The location of the threshold between stable and active disease varies from person to person. Also, the dose-response curve for any individual can shift to the left or the right according to environmental changes. A shift to the left would result in an increased inflammatory response to a given bacterial challenge and potentially an exacerbation of disease. A shift to the right would have the opposite effect. *(Modified from Champagne CM, Buchanan W, Reddy MS, et al:* Periodontol 2000 *31:167-180, 2003.)*

persist indefinitely. However, if something changes, such as the quantity or quality of the biofilm alters or the host defenses alter (e.g., as a result of a change in an environmental exposure), then increased secretion of cytokines, prostanoids, MMPs, and other mediators may increase in the tissues, leading to the histopathologic changes described earlier and a transition to periodontitis. There is a threshold therefore between stable and active disease, and this will vary from person to person. The dose-response curve for any individual can shift to the left or the right according to environmental changes. A shift to the left would result in an increase in the quantities of inflammatory mediators produced for a given bacterial challenge and potentially an exacerbation of disease. A shift to the right would have the opposite effect. In all cases, an increase in the LPS challenge would have the tendency to increase the production of inflammatory mediators, which may tip the site from a stable to a progressing periodontal lesion.

These are, of course, simplistic models to explain a highly complex phenomenon, and it is clear that cytokines and inflammatory mediators function in complicated networks. Therefore, although increases and decreases in the absolute levels of cytokines have been reported in disease states, it is clear that dysregulation of cytokine networks and other mediators is the key determinant of disease progression. Thus, the relative proportions of mediators within inflammatory networks are fundamental to determining disease progression, and changes in these proportions are driven by the inflammatory challenge and the genetic and environmental factors that govern how the host responds to this challenge. The development and application of techniques to study global gene expression is beginning to provide information to help us understand immune responses on a more holistic basis.[69]

Schematic illustrations to explain the pathogenesis of periodontal disease are useful, although, given the complexity of the disease processes, they are inevitably simplistic. However, a useful conceptual model of periodontitis was introduced in 1997 (Figure 21-9).[134] Earlier models were overly simplistic and were essentially linear, in the first instance suggesting that periodontitis resulted directly from the microbial challenge.[91] This concept has influenced periodontology over the decades, resulting in treatment concepts that focussed primarily on the biofilm. Increasing awareness of the importance of host factors in determining inter-individual differences in disease progression led to the model depicted in Figure 21-9, in which, although plaque bacteria initiate the inflammatory response, most of the tissue damage results from the host response, which is influenced by genetic factors and environmental and acquired risk factors. These factors, such as smoking, or genetic risk factors (which are not yet clearly defined) alter the progression of the immune-inflammatory response and shift the balance toward increased periodontal breakdown. This model implies that the presence of plaque bacteria does not inevitably lead to tissue destruction and is supported by a large number of epidemiologic studies, which confirm that more advanced disease is usually confined to a minority of the population.[106]

The model presented in Figure 21-9 continues to be refined and developed, as new knowledge is obtained about (1) microbial ecologic complexes in the subgingival biofilm and their interactions with the host, (2) links between periodontitis and other complex inflammatory diseases, and (3) increased awareness of the importance of risk factors such as smoking and diabetes. This has led to the development of a biologic systems model for representing periodontal pathogenesis, which involves bacterial components, environmental factors, specific inflammatory mechanisms, and host-genetic variations associated with disease.[91] A biologic systems approach provides a framework for viewing the contributions and

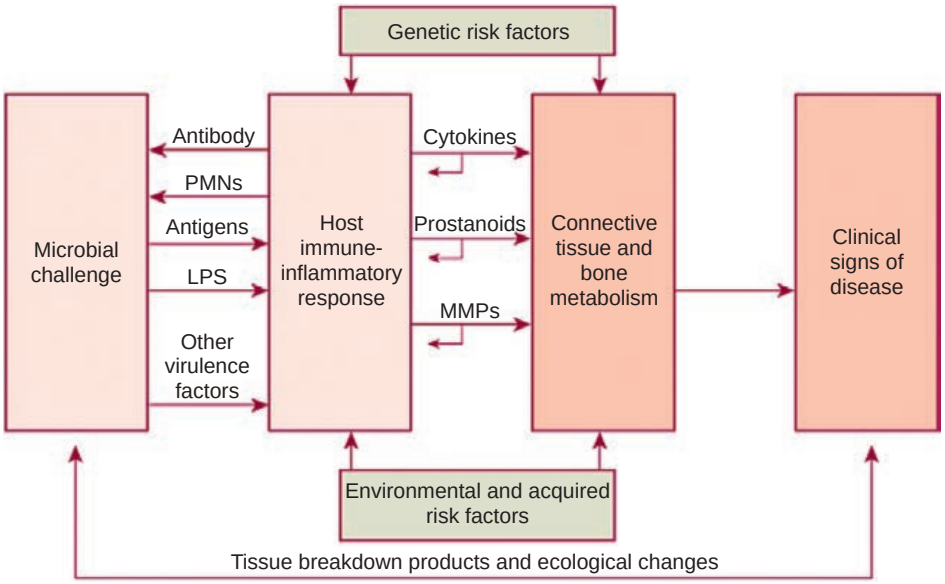

Figure 21-9 Schematic illustration of the pathogenesis of periodontitis. The microbial challenge presented by subgingival plaque bacteria results in an upregulated host immune-inflammatory response in the periodontal tissues that is characterized by the dysregulated and increased production of inflammatory cytokines (e.g., interleukins and tumor necrosis factor alpha), prostanoids (e.g., prostaglandin E_2) and enzymes, including the matrix metalloproteinases *(MMPs)*. These upregulated proinflammatory mediators are responsible for the majority of periodontal tissue breakdown that occurs, including alveolar bone resorption via activation of osteoclasts. These changes result over time in the clinical signs of periodontal disease developing. The process is modified by environmental factors such as smoking, and by genetic susceptibility. *PMNs*, Polymorphonuclear leukocytes; *LPS*, lipopolysaccharide. *(Modified from Page RC, Kornman KS:* Periodontol 2000 *14:9-11, 1997.)*

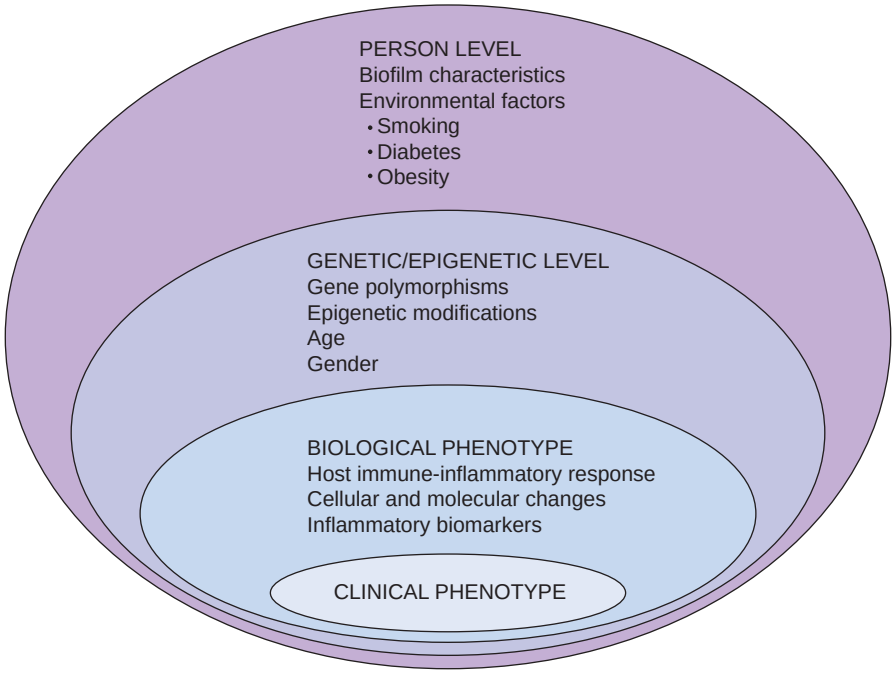

Figure 21-10 A biologic systems model for representing periodontitis. The outermost level of this model is the *Person Level*, representing an individual's unique characteristics as they impact on periodontitis. These include the compositional characteristics of the subgingival biofilm, and known risk factors and environmental exposures such as smoking and diabetes. The *Person Level* characteristics interact with the *Genetic/Epigenetic Level* characteristics, which include nonmodifiable factors such as age, gender, and genetic composition. Gene polymorphisms are known to be associated with periodontal disease, and epigenetics refers to changes in phenotype (i.e., clinical disease expression) caused by mechanisms other than changes in the underlying DNA sequence. Epigenetics can be defined as all the meiotically and mitotically inherited changes in gene expression that are not encoded in the DNA sequence itself, and epigenetic modifications are important permissive and suppressive factors in controlling the expressed genome via gene transcription. Two major epigenetic mechanisms are posttranslational modification of histone proteins in chromatin and methylation of DNA. The *Genetic/Epigenetic Level* characteristics influence the *Biological Phenotype*, which is characterized by the specific immune-inflammatory responses (cellular and molecular events and production of inflammatory mediators) that are associated with the *Clinical Phenotype* (i.e., the clinical presentation of the disease). This model reflects how different individuals with the same presentation (e.g., periodontitis) may have very different predisposing and risk factors. The model depicts the different biologic factors that underpin the development of periodontal disease in different individuals and ultimately may be used to classify disease according to the contribution provided to the clinical phenotype at each level. *(Modified from Offenbacher S, Barros SP, Beck JD:* J Periodontol *79:1577-1584, 2008.)*

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relative importance of all the components that contribute to the clinical presentation of disease. Thus, in the context of periodontal disease, such a system would include a person level, a genetic/epigenetic level, the biologic phenotype, and ultimately the clinical phenotype (Figure 21-10).[126] Such systems will revolutionize conceptual models by creating a more comprehensive view of the disease as a complex regulatory network, in which aspects of the specific genetic factors, environmental exposures, and other modifying factors that an individual is exposed to determine the development of the disease state.

It is clear that the subgingival bacteria initiate and perpetuate the immune-inflammatory responses in the periodontal tissues. These responses are characterized by classic signs of inflammation that are modified as the result of the unique anatomy of the periodontium and the dentogingival apparatus. The inflammatory events that develop in response to the bacterial challenge are protective by intent but result in the majority of tissue damage and breakdown that lead to the clinical signs of periodontitis. Individuals vary in their susceptibility to periodontal disease and also in the threshold level at which a stable periodontal site progresses to an active site. Such variations are genetically determined and influenced by environmental risk factors, some of which are modifiable and some of which are not. The challenge for the future is to identify at-risk individuals who possess the hyperinflammatory trait so that disease can be prevented by careful management strategies before tissue loss has occurred.

References and suggested reading can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

The pathogenesis of gingivitis and periodontitis is a complex molecular phenomenon with many varieties. Thus the exact step-by-step changes that lead from healthy gingiva to gingivitis and periodontitis in any given patient have not been fully elucidated, even though there are accumulated data on many of the theoretical pathways of tissue damage. Gingivitis precedes periodontitis, but not all cases of gingivitis proceed to further destruction of the periodontal tissues, pocket formation, and bone loss.

In the past, the major focus for understanding the pathogenesis of periodontal disease has been the role of anaerobic gram-negative bacteria found in the subgingival biofilms, and periodontal therapy has centered on removing and controlling plaque formation and on using procedures, such as periodontal surgery, to change the milieu so that there are no deep pockets to harbor bacteria and so protect them from oral hygiene plaque removal techniques. Now, it is agreed that the host response involving a variety of cells and inflammatory modulators can be another contributor to the disease process.

Gingival inflammation is based on an initial acute inflammatory reaction coupled with a long-term chronic inflammation. In patients in whom the acute reaction is predominant, there can be a dramatic improvement in redness, swelling, and bleeding on probing and pocket depth when antisubgingival plaque initial therapy is coupled with a high level of oral hygiene. Those patients with more emphasis on chronic inflammatory changes will not show such an obvious clinical improvement with therapy.

Bleeding on probing is often an early sign of gingivitis and continues to be seen as the disease progresses to periodontitis. It is an indication of ongoing connective tissue destruction coupled with vasodilation and ulceration of the gingival lining of the pocket. In the early stages of the disease, plaque control can reverse these inflammatory changes and so patients can reduce and eliminate gingival bleeding in 10 to 14 days. If plaque control becomes inadequate, gingival bleeding can appear as early as 2 days.

New approaches to periodontal treatment couple antibacterial plaque therapy with modulation of the host response. An example of this is the use of low-dose systemic doxycycline coupled with conventional initial therapy. This gives a modest additional therapeutic advantage over conventional treatment, but future host modulation treatment based on counteracting specific tissue destructive molecules may widen and improve the way clinicians can treat these ubiquitous diseases.

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CHAPTER 22

The Role of Dental Calculus and Other Local Predisposing Factors

James E. Hinrichs

CHAPTER OUTLINE

CALCULUS
- Supragingival and Subgingival Calculus
- Prevalence
- Composition
- Attachment to the Tooth Surface
- Formation
- Etiologic Significance
- Materia Alba, Food Debris, and Dental Stains

OTHER PREDISPOSING FACTORS
- Iatrogenic Factors
- Malocclusion
- Periodontal Complications Associated with Orthodontic Therapy
- Extraction of Impacted Third Molars
- Habits and Self-Inflicted Injuries
- Smokeless Tobacco
- Radiation Therapy

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The primary cause of gingival inflammation is bacterial plaque. Other predisposing factors include calculus, faulty restorations, complications associated with orthodontic therapy, self-inflicted injuries, use of tobacco, and others. These will be discussed in turn.

CALCULUS

Calculus consists of mineralized bacterial plaque that forms on the surfaces of natural teeth and dental prostheses.

Supragingival and Subgingival Calculus

Supragingival calculus is located coronal to the gingival margin and therefore is visible in the oral cavity. It is usually white or whitish yellow in color, hard with claylike consistency, and easily detached from the tooth surface. After removal, it may rapidly recur, especially in the lingual area of the mandibular incisors. The color is influenced by contact with such substances as tobacco and food pigments. It may localize on a single tooth or group of teeth, or it may be generalized throughout the mouth.

The two most common locations for supragingival calculus to develop are the buccal surfaces of the maxillary molars (Figure 22-1) and the lingual surfaces of the mandibular anterior teeth (Figure 22-2).[32] Saliva from the parotid gland flows over the facial surfaces of upper molars via the parotid duct, whereas the submandibular duct and lingual duct empty onto the lingual surfaces of the lower incisors from the submaxillary and sublingual glands, respectively. In extreme cases, calculus may form a bridgelike structure over the interdental papilla of adjacent teeth or cover the occlusal surface of teeth lacking functional antagonists.

Subgingival calculus is located below the crest of the marginal gingiva and therefore is not visible on routine clinical examination. The location and extent of subgingival calculus may be evaluated by careful tactile perception with a delicate dental instrument such as an explorer. Clerehugh et al[29] used a World Health Organization #621 probe to detect and score subgingival calculus. Subsequently, these teeth were extracted and visually scored for subgingival calculus. An agreement of 80% was found between these two scoring methods. Subgingival calculus is typically hard and dense and frequently appears dark brown or greenish black in color (Figure 22-3) and is firmly attached to the tooth surface. Supragingival calculus and subgingival calculus generally occur together, but one may be present without the other. Microscopic studies demonstrate that deposits of subgingival calculus usually extend nearly to the base of periodontal pockets in chronic periodontitis but do not reach the junctional epithelium.

When the gingival tissues recede, subgingival calculus becomes exposed and is therefore reclassified as supragingival (Figure 22-4). Thus supragingival calculus can be composed of both supragingival calculus and previous subgingival calculus. A reduction in gingival

Figure 22-1 Supragingival calculus is depicted on the buccal surfaces of maxillary molars adjacent to orifice for parotid duct.

Figure 22-2 Extensive supragingival calculus is present on the lingual surfaces of lower anterior teeth.

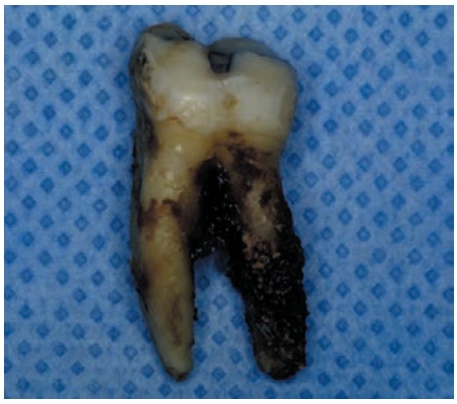

Figure 22-3 Dark pigmented deposits of subgingival calculus are shown on the distal root of an extracted lower molar.

inflammation and probing depths with a gain in clinical attachment can be observed after the removal of subgingival plaque and calculus (Figure 22-5) (see Chapter 45).

Prevalence

Anerud and co-workers[4] observed the periodontal status of a group of Sri Lankan tea laborers and a group of Norwegian academicians for a 15-year period. The Norwegian population had ready access to preventive dental care throughout their lives, whereas the Sri Lankan tea laborers did not. The formation of supragingival calculus was observed early in life in the Sri Lankan individuals, probably shortly after the teeth erupted. The first areas to exhibit calculus deposits were the facial aspects of maxillary molars and the lingual surfaces of mandibular incisors. Deposition of supragingival calculus continued as individuals aged, reaching a maximal calculus score around 25 to 30 years of age. At this time, most of the teeth

Figure 22-4 A 31-year-old white male is shown with extensive supragingival and subgingival calculus deposits throughout his dentition.

Figure 22-5 The same patient shown in Figure 22-4, 1 year after receiving thorough scaling and root planing to remove supragingival and subgingival calculus deposits, followed by restorative care. Note the substantial reduction in gingival inflammation.

were covered by calculus, although the facial surfaces had less calculus than the lingual or palatal surfaces. Calculus accumulation appeared to be symmetric, and by age 45 only a few teeth, typically the premolars, were without calculus. Subgingival calculus appeared first either independently or on the interproximal aspects of areas where supragingival calculus already existed.[4] By age 30, all surfaces of all teeth had subgingival calculus without any pattern of predilection.

The Norwegian academicians received oral hygiene instructions and frequent preventive dental care throughout their lives. The Norwegians exhibited a marked reduction in the accumulation of calculus as compared with the Sri Lankan group. However, despite the fact that 80% of teenagers formed supragingival calculus on the facial surfaces of the upper molars and the lingual surfaces of lower incisors, no additional calculus formation occurred on other teeth, nor did it increase with age.[4]

Both supragingival calculus and subgingival calculus may be seen on radiographs (see Chapter 31). Highly calcified interproximal calculus deposits are readily detectable as radiopaque projections that protrude into the interdental spaces in Figure 22-6. However, the sensitivity level of detecting calculus by radiographs is inconsistent.[23] The location of calculus does not indicate the bottom of the periodontal pocket because the most apical plaque is not sufficiently calcified to be visible on radiographs.

Composition

Inorganic Content. Supragingival calculus consists of inorganic (70% to 90%[50]) and organic components. The major inorganic

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Figure 22-6 A bitewing radiograph illustrating subgingival calculus deposits that are depicted as interproximal spurs *(arrows).*

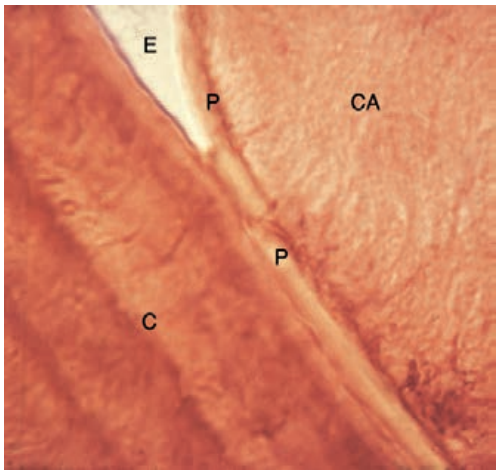

Figure 22-7 Calculus attached to pellicle on enamel surface and cementum. An enamel void *(E)* has been created in the preparation of the specimen. *CA,* Calculus*; P,* pellicle*; C,* cementum.

proportions of calculus have been reported as approximately 76% calcium phosphate, $Ca_3(PO_4)_2$; 3% calcium carbonate, $CaCO_3$; and traces of magnesium phosphate, $Mg_3(PO_4)_2$, and other metals.[169] The percentage of inorganic constituents in calculus is similar to that in other calcified tissues of the body. The principal inorganic components have been reported as approximately 39% calcium, 19% phosphorus, 2% carbon dioxide, and 1% magnesium and trace amounts of sodium, zinc, strontium, bromine, copper, manganese, tungsten, gold, aluminum, silicon, iron, and fluorine.[107]

At least two-thirds of the inorganic component is crystalline in structure.[85] The four main crystal forms and their approximate percentages are as follows: hydroxyapatite 58%, magnesium whitlockite 21%, octacalcium phosphate 12%, and brushite 9%.

Generally, two or more crystal forms are typically found in a sample of calculus. Hydroxyapatite and octacalcium phosphate are detected most frequently (i.e., in 97% to 100% of all supragingival calculus) and constitute the bulk of the specimen. Brushite is more common in the mandibular anterior region and magnesium whitlockite in the posterior areas. The incidence of the four crystal forms varies with the age of the deposit.[15]

Organic Content. The organic component of calculus consists of a mixture of protein-polysaccharide complexes, desquamated epithelial cells, leukocytes, and various types of microorganisms.[94]

For more information on organic content, please visit the Expert CONSULT companion website at www.expertconsult.com.

The composition of subgingival calculus is similar to that of supragingival calculus, with some differences. It has the same hydroxyapatite content, more magnesium whitlockite, and less brushite and octacalcium phosphate.[136,158] The ratio of calcium to phosphate is higher subgingivally, and the sodium content increases with the depth of periodontal pockets.[89] These altered compositions may be attributed to the origin of subgingival calculus being plasma, whereas supragingival calculus is partially composed of saliva constituents. Salivary proteins present in supragingival calculus are not found subgingivally.[10] Dental calculus, salivary duct calculus, and calcified dental tissues are similar in inorganic composition.

Attachment to the Tooth Surface

Differences in the manner in which calculus is attached to the tooth surface affect the relative ease or difficulty encountered in its removal. Four modes of attachment have been described.[81,138,145,173] Attachment by means of an organic pellicle on cementum is depicted in Figure 22-7, and attachment on enamel is shown in Figure 22-8. Mechanical locking into surface irregularities, such as caries lesions or resorption lacunae, is illustrated in Figure 22-9. The fourth mode of attachment consists of close adaptation of the undersurface of calculus to depressions or gently sloping mounds of the unaltered cementum surface,[154] as shown in Figure 22-10, and penetration of bacterial calculus into cementum, as shown in Figures 22-11 and 22-12.

Formation

Calculus is *mineralized dental plaque.* The soft plaque is hardened by the precipitation of mineral salts, which usually starts between the first and fourteenth days of plaque formation. Calcification has been reported to occur in as little as 4 to 8 hours.[159] Calcifying plaques may become 50% mineralized in 2 days and 60% to 90% mineralized in 12 days.[106,139,149] All plaque does not necessarily undergo calcification. Early plaque contains a small amount of inorganic material, which increases as the plaque develops into calculus. Plaque that does not develop into calculus reaches a plateau of maximal mineral content within 2 days.[140] Microorganisms are not always essential in calculus formation because calculus occurs readily in germ-free rodents.[57]

Saliva is the source of mineralization for supragingival calculus, whereas the serum transudate called *gingival crevicular fluid* furnishes the minerals for subgingival calculus.[69,156] The calcium concentration/content in plaque is 2 to 20 times that found in saliva.[15] Early plaque of heavy calculus formers contains more calcium, three times more phosphorus, and less potassium than that

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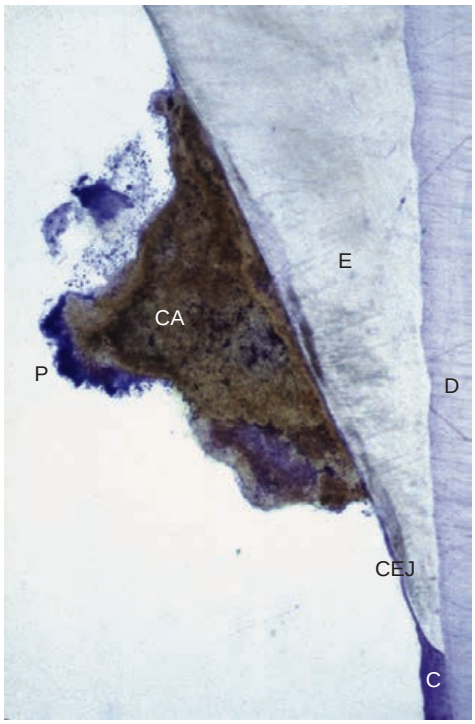

Figure 22-8 Non-decalcified specimen with calculus *(CA)* attached to enamel *(E)* surface just coronal to cementoenamel junction *(CEJ)*. Note plaque *(P)* on the surface of the calculus; dentin *(D)* and cementum *(C)* are identified. *(Courtesy Dr. Michael Rohrer, Minneapolis.)*

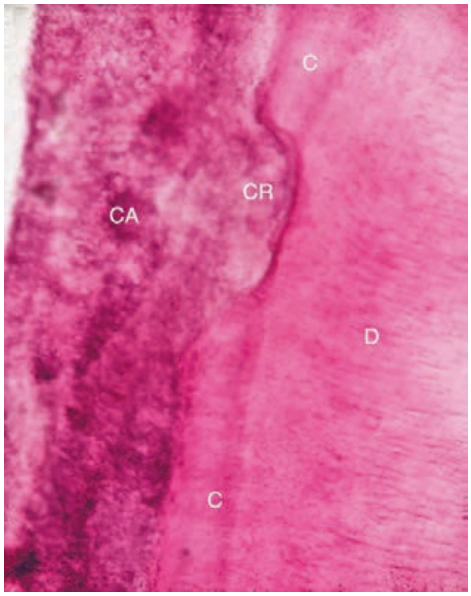

Figure 22-9 Calculus *(CA)* attached to a cemental resorption area *(CR)* with cementum *(C)* adjacent to dentin *(D)*.

Figure 22-10 Undersurface of subgingival calculus *(C)* previously attached to the cementum surface *(S)*. Note impression of cementum mounds in calculus *(arrows)*. *(Courtesy Dr. John Sottosanti, La Jolla, CA.)*

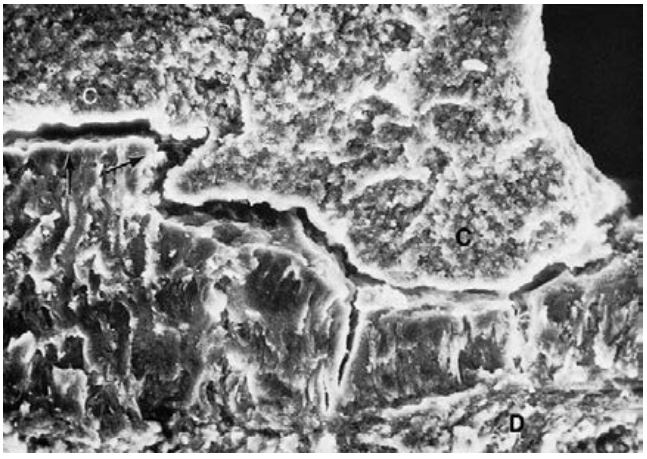

Figure 22-11 Subgingival calculus *(C)* embedded beneath the cementum surface (arrows) and penetrating to the dentin *(D)*, making removal difficult. *(Courtesy Dr. John Sottosanti, La Jolla, CA.)*

of noncalculus formers, suggesting that phosphorus may be more critical than calcium in plaque mineralization.[94] Calcification entails the binding of calcium ions to the carbohydrate-protein complexes of the organic matrix and the precipitation of crystalline calcium phosphate salts.[92] Crystals form initially in the intercellular matrix and on the bacterial surfaces and finally within the bacteria.[51,174]

Calcification of supragingival plaque and in the attached component of subgingival plaque begins along the inner surface adjacent to the tooth. Separate foci of calcification increase in size and coalesce to form solid masses of calculus (see Figure 22-12). Calcification may be accompanied by alterations in the bacterial content and staining qualities of the plaque. As calcification progresses, the number of filamentous bacteria increases and foci of calcification change from basophilic to eosinophilic. There is a reduction in the staining intensity of groups exhibiting a positive periodic acid–Schiff reaction.

For more information on formation, please visit the Expert CONSULT companion website at www.expertconsult.com.

The initiation of calcification and the rate of calculus accumulation vary among individuals, between tooth variety in the same dentition, and at different times in the same person.[107,161] On the basis of these differences, persons may be classified as *heavy, moderate,* or *slight* calculus formers or as noncalculus formers. The average daily increment in calculus formers is from 0.10% to 0.15% of dry weight calculus.[149,161] Calculus formations continue until it reaches a maximum, after which it may be reduced in amount. The time

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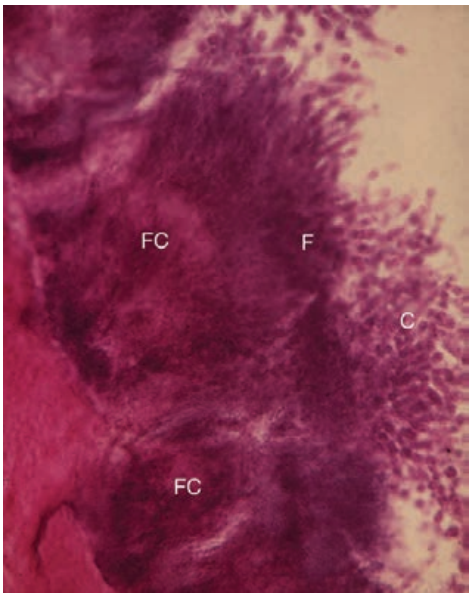

Figure 22-12 Plaque and calculus on tooth surface. Note the spherical areas of focal calcification *(FC)* and the perpendicular alignment of the filamentous *(F)* organisms along the inner surface of plaque and cocci *(C)* on the outer surface.

required to reach the maximal level has been reported between 10 weeks[30] and 6 months.[163]

For more information on formation, please visit the Expert CONSULT companion website at www.expertconsult.com.

Theories Regarding the Mineralization of Calculus. The theoretical mechanisms by which plaque becomes mineralized can be stratified into two categories.[108]

1. Mineral precipitation results from a local rise in the degree of saturation of calcium and phosphate ions, which may be brought about in the following several ways:
   - *A rise in the pH of the saliva causes precipitation of calcium phosphate salts by lowering the precipitation constant.* The pH may be elevated by the loss of carbon dioxide and the formation of ammonia by dental plaque bacteria or by protein degradation during stagnation.[14,64]
   - *Colloidal proteins in saliva bind calcium and phosphate ions and maintain a supersaturated solution with respect to calcium phosphate salts.* With stagnation of saliva, colloids settle out and the supersaturated state is no longer maintained, leading to precipitation of calcium phosphate salts.[124,142]
   - *Phosphatase liberated from dental plaque, desquamated epithelial cells, or bacteria precipitates calcium phosphate by hydrolyzing organic phosphates in saliva, thus increasing the concentration of free phosphate ions.*[168] *Esterase* is another enzyme that is present in the cocci and filamentous organisms, leukocytes, macrophages, and desquamated epithelial cells of dental plaque.[8] Esterase may initiate calcification by hydrolyzing fatty esters into free fatty acids. The fatty acids form soaps with calcium and magnesium that are later converted into the less-soluble calcium phosphate salts.
2. Seeding agents induce small foci of calcification that enlarge and coalesce to form a calcified mass.[109] This concept has been referred to as the *epitactic concept* or more appropriately, *heterogeneous nucleation.* The seeding agents in calculus formation are not known, but it is suspected that the intercellular matrix of plaque plays an active role.[95,106,174] The carbohydrate-protein complexes may initiate calcification by removing calcium from the saliva (chelation) and binding with it to form nuclei that induce subsequent deposition of minerals.[92,164]

Role of Microorganisms in the Mineralization of Calculus. Mineralization of plaque generally starts extracellularly around both gram-positive and gram-negative organisms but may also start intracellularly.[83] Filamentous organisms, diphtheroids, and *Bacterionema* and *Veillonella* species have the ability to form intracellular apatite crystals (see Figure 22-12). Mineralization spreads until the matrix and bacteria are calcified.[51,175]

Bacterial plaque may actively participate in the mineralization of calculus by forming phosphatases, which changes the pH of the plaque and induces mineralization,[38,92] but the prevalent opinion is that these bacteria are only passively involved[51,131,168] and are simply calcified with other plaque components. The occurrence of calculus-like deposits in germ-free animals supports this opinion.[57] However, other experiments suggest that transmissible factors are involved in calculus formation and that penicillin in the diets of some of these animals reduces calculus formation.[9]

Etiologic Significance

Distinguishing between the effects of calculus and plaque on the gingiva is difficult because calculus is always covered with a nonmineralized layer of plaque.[140] A positive correlation between the presence of calculus and the prevalence of gingivitis exists,[126] but this correlation is not as great as that between plaque and gingivitis.[53] The initiation of periodontal disease in young people is closely related to plaque accumulation, whereas calculus accumulation is more prevalent in chronic periodontitis found in older adults.[53,86]

For more information on etiologic significance, please visit the Expert CONSULT companion website at www.expertconsult.com.

Calculus does not contribute directly to gingival inflammation but provides a fixed nidus for the continued accumulation of plaque and its retention in close proximity of the gingiva (Figure 22-13). Subgingival calculus is likely to be the product rather than the cause of periodontal pockets. Plaque initiates gingival inflammation, which leads to pocket formation, and the pocket in turn provides a sheltered area for plaque and bacterial accumulation. The increased flow of gingival fluid associated with gingival inflammation provides the minerals that mineralize the continually accumulating plaque resulting in the formation of subgingival calculus (Figure 22-14). Over a 6-year period, Albandar et al observed 156 teenagers with aggressive periodontitis.[2] They noted that areas with detectable subgingival calculus at the initiation of the study were much more likely to experience loss of periodontal attachment than sites that did not initially exhibit subgingival calculus.

Although the bacterial plaque that coats the teeth is the main etiologic factor in the development of periodontal disease, the removal of subgingival plaque and calculus constitute the cornerstone of periodontal therapy. Calculus plays an important role in maintaining and accentuating periodontal disease by keeping plaque in close contact with the gingival tissue and creating areas where plaque removal is impossible. Therefore the clinician must not only possess the clinical skill to remove the plaque and calculus but also must be very conscientious about performing this task.

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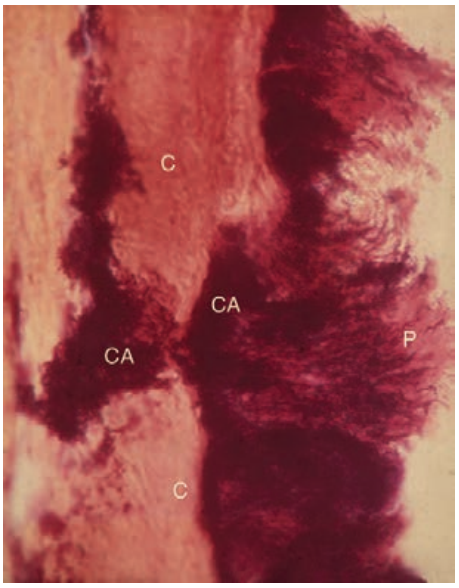

Figure 22-13 Calculus *(CA)* penetrates the tooth surface and is embedded within the cementum *(C)*. Note plaque *(P)* attached to the calculus.

Figure 22-14 Scanning electron microscope view of an extracted human tooth showing a cross-section of subgingival calculus *(C)* separated *(arrows)* from the cemental surface during processing of specimen. Note bacteria *(B)* attached to calculus and cemental surfaces. *(Courtesy Dr. John Sottosanti, La Jolla, CA.)*

Materia Alba, Food Debris, and Dental Stains

Materia alba is an accumulation of microorganisms, desquamated epithelial cells, leukocytes, and a mixture of salivary proteins and lipids, with few or no food particles, and it lacks the regular internal pattern observed in plaque.[141] It is a yellow or grayish-white, soft, sticky deposit and is somewhat less adherent than dental plaque. The irritating effect of materia alba on the gingiva is caused by bacteria and their products.

Most food debris is rapidly liquefied by bacterial enzymes and cleared from the oral cavity by salivary flow and the mechanical action of the tongue, cheeks, and lips. The rate of clearance from the oral cavity varies with the type of food and the individual. Aqueous solutions are typically cleared within 15 minutes, whereas sticky foods may adhere for more than 1 hour.[84,162] Dental plaque is not a derivative of food debris, nor is food debris an important cause of gingivitis.[37,73]

Figure 22-15 Tobacco stains on the apical third of the clinical crown caused by cigarette smoking.

Pigmented deposits on the tooth surface are called *dental stains.* Stains are primarily an aesthetic problem and do not cause inflammation of the gingiva. The use of tobacco products (Figure 22-15), coffee, tea, certain mouthrinses, and pigments in foods can contribute to stain formation.[90,155]

OTHER PREDISPOSING FACTORS

Iatrogenic Factors

Deficiencies in the quality of dental restorations or prostheses are contributing factors to gingival inflammation and periodontal destruction. Inadequate dental procedures that contribute to the deterioration of the periodontal tissues are referred to as *iatrogenic factors*. Iatrogenic endodontic complications that can adversely affect the periodontium include root perforations, vertical root fractures, and endodontic failures that may necessitate tooth extraction.[172,173]

Characteristics of dental restorations and removable partial dentures that are important to the maintenance of periodontal health include the location of the gingival margin for the restoration, the space between the margin of the restoration and the unprepared tooth, the contour of restorations, the occlusion, materials used in the restoration, the restorative procedure itself, and the design of the removable partial denture. These characteristics are described in this chapter as they relate to the etiology of periodontal disease. A more comprehensive review with special emphasis on the interrelationship between restorative procedures and the periodontal status is presented in Chapter 66.

Margins of Restorations. Overhanging margins of dental restorations contribute to the development of periodontal disease by (1) changing the ecologic balance of the gingival sulcus to an area that favors the growth of disease-associated organisms (predominately gram-negative anaerobic species) at the expense of the health-associated organisms (predominately gram-positive facultative species)[84] and (2) inhibiting the patient's access to remove accumulated plaque.

The frequency of overhanging margins on proximal restorations varies in different studies from 16.5% to 75%.[17,49,68,115] A highly significant statistical relationship has been reported between marginal defects and reduced bone height.[17,58,68] Removal of overhangs permits more effective control of plaque, resulting in a reduction of gingival inflammation and a small increase in radiographic alveolar bone support[52,61,152] (Figures 22-16 and 22-17).

The location of the gingival margin of a restoration is directly related to the health status of adjacent periodontal tissues.[150]

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Numerous studies have shown a positive correlation between margins located apical to the marginal gingiva and the presence of gingival inflammation.[49,65,72,129,152] Subgingival margins are associated with large amounts of plaque, more severe gingivitis, and deeper pockets. Even high-quality restorations, if placed subgingivally, will increase plaque accumulation, gingival inflammation,* and the rate of gingival fluid flow.[11,54] Margins placed at the level of the gingival crest induce less severe inflammation, whereas supragingival margins are associated with a degree of periodontal health similar to that seen with nonrestored interproximal surfaces.[44,150]

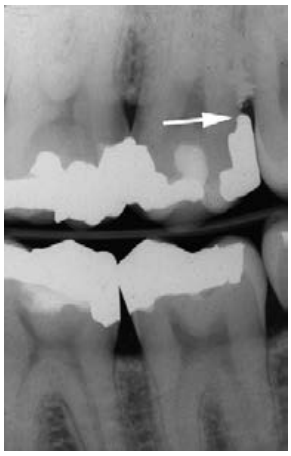

Figure 22-16 Radiograph of amalgam overhang on the distal surface of maxillary second molar that is a source of plaque retention and gingival irritation.

Figure 22-17 Radiograph of the same patient shown in Figure 22-16 after the excessive amalgam has been removed.

Roughness in the subgingival area is considered to be a major contributing factor to plaque buildup and subsequent gingival inflammation.[150] The subgingival zone is composed of the margin of the restoration, the luting material, and the prepared, as well as the unprepared tooth surface. Sources of marginal roughness include grooves and scratches in the surface of acrylic resin, porcelain, or gold restorations (Figure 22-18); separation of the restoration margin and luting material from the cervical finish line, thereby exposing the rough surface of the prepared tooth (Figure 22-19); dissolution and disintegration of the luting material between the preparation and the restoration, leaving a space (Figure 22-20); and inadequate marginal fit of the restoration.[150] Subgingival margins typically have a gap of 20 to 40 µm between the margin of the restoration and the unprepared tooth.[148] Colonization of this gap by bacterial plaque undoubtedly contributes to the detrimental effect of margins placed in a subgingival environment.

Contours/Open Contacts. Overcontoured crowns and restorations tend to accumulate plaque and possibly prevent the self-cleaning mechanisms of the adjacent cheek, lips, and tongue[6,79,104,171] (Figures 22-21 and 22-22). Restorations that fail to reestablish adequate interproximal embrasure spaces are associated with papillary inflammation. Undercontoured crowns that lack a protective height of contour do not retain as much plaque as overcontoured crowns and therefore may not be as detrimental during mastication as once thought.[171]

The contour of the occlusal surface as established by the marginal ridges and related developmental grooves normally serves to deflect food away from the interproximal spaces. The optimal cervico-occlusal location for a posterior contact is at the longest mesiodistal diameter of the tooth, which is generally just apical to the crest of the marginal ridge. The integrity and location of the proximal contacts along with the contour of the marginal ridges and developmental grooves typically prevent interproximal food

*References 82, 84, 105, 111, 130, and 151.

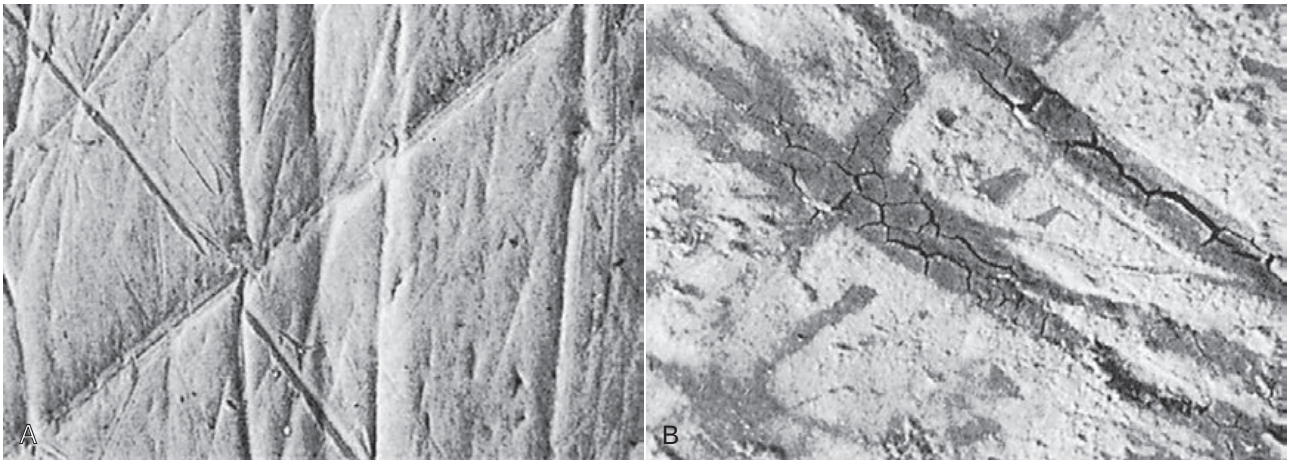

Figure 22-18 **A,** Polished gold alloy crown demonstrates surface scratches. **B,** Gold alloy crown that had been in the mouth for several years has scratches filled with deposits. *(From Silness J:* Dent Clin North Am *24:317, 1980.)*

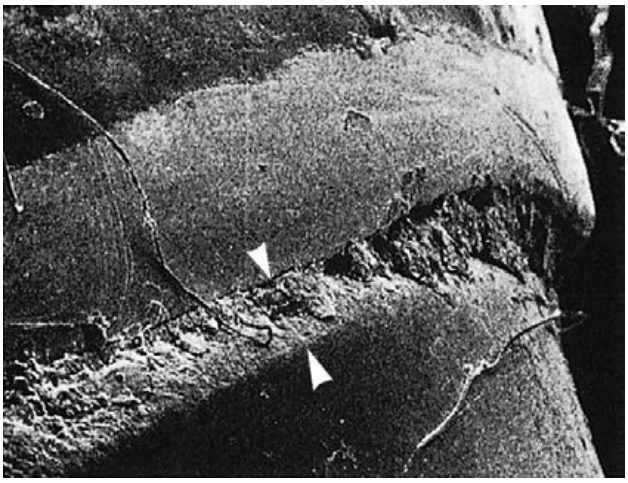

Figure 22-19 After cementation, luting material prevents approximation of the crown margin and the finishing line, leaving part of the prepared tooth uncovered (area between *arrowheads*). *(From Silness J:* Dent Clin North Am *24:317, 1980.)*

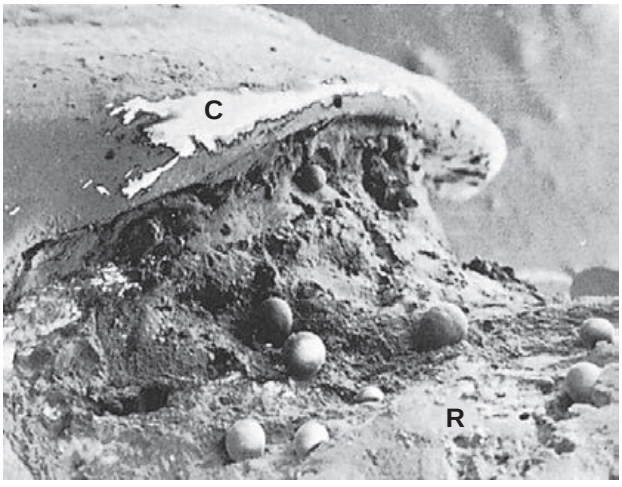

Figure 22-20 Craters have formed after dissolution and disintegration of the luting material. Spherical bodies are not identified. Crown *(C);* root *(R). (From Silness J:* Dent Clin North Am *24:317, 1980.)*

impaction. *Food impaction* is the forceful wedging of food into the periodontium by occlusal forces. As the teeth wear down, their originally convex proximal surfaces become flattened and the wedging effect of the opposing cusp is exaggerated. Cusps that tend to forcibly wedge food into interproximal embrasures are known as *plunger cusps.* The interproximal plunger cusp effect may also be observed when missing teeth are not replaced and the relationship between proximal contacts of adjacent teeth is altered. An intact proximal contact precludes the forceful wedging of food into the interproximal embrasure space, whereas a light or open contact is conducive to impaction.

The classic analysis of the factors leading to food impaction was made by Hirschfeld,[63] who recognized the following factors: uneven occlusal wear, opening of the contact point as a result of loss of proximal support or from extrusion, congenital morphologic abnormalities, and improperly constructed restorations.

The presence of the previously mentioned abnormalities does not necessarily lead to food impaction and periodontal disease. A study of interproximal contacts and marginal ridge relationships[84] in three groups of periodontally healthy males revealed that 0.7%

Figure 22-21 Inflamed marginal and papillary gingiva adjacent to an overcontoured porcelain–fused-to-metal crown on the maxillary left central.

Figure 22-22 Radiograph of ill-fitting porcelain fused to metal crown shown in Figure 22-21.

Figure 22-23 Inflamed palatal gingiva associated with a maxillary provisional acrylic partial denture. Note the substantial difference in color of the inflamed gingiva adjacent to the premolars and first molar compared with the gingiva adjacent to the second molar.

to 76% of the proximal contacts were defective and 33.5% of adjacent marginal ridges were uneven.[114] However, greater probing depth and loss of clinical attachment have been reported for sites that exhibited both an open contact and food impaction as compared with contralateral control sites without open contacts or food impaction.[70] Excessive anterior overbite is a common cause of food impaction on the lingual surfaces of the maxillary anterior teeth and the facial surfaces of the opposing mandibular teeth. These areas may be exemplified by attachment loss with gingival recession.

Materials. In general, restorative materials are not in themselves injurious to the periodontal tissues.[6,75] One exception to this may be self-curing acrylics[166] (Figure 22-23).

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Figure 22-24 Lower incisor showing a prominent root with gingival recession and lack of attached gingiva.

Figure 22-25 The same patient as shown in Figure 22-24 after placement of a soft tissue graft to gain attached gingiva and treat the gingival recession.

Plaque that forms at the margins of restorations is similar to that found on adjacent nonrestored tooth surfaces. The composition of plaque formed on all types of restorative materials is similar, with the exception of that formed on silicate.[109] Although surface textures of restorative materials differ in their capacity to retain plaque,[170] all can be adequately cleaned if they are polished and accessible to methods of oral hygiene.[110,137] The undersurface of pontics in fixed bridges should barely touch the mucosa. Access for oral hygiene is inhibited with excessive pontic to tissue contact, thereby contributing to plaque accumulation that will cause gingival inflammation and possibly formation of pseudopockets.[43,150]

Design of Removable Partial Dentures. Several investigations have shown that after the insertion of partial dentures, the mobility of the abutment teeth, gingival inflammation, and periodontal pocket formation all increase.[16,25,144] This is because partial dentures favor the accumulation of plaque, particularly if they cover the gingival tissue. Partial dentures that are worn during both night and day induce more plaque formation than those worn only during the daytime.[16] These observations emphasize the need for careful and personalized oral hygiene instruction to avoid harmful effects of partial dentures on the remaining teeth and periodontium.[11] The presence of removable partial dentures induces not only quantitative changes in dental plaque[47] but also qualitative changes, promoting the emergence of spirochetal microorganisms.[48]

Restorative Dentistry Procedures. The use of rubber dam clamps, matrix bands, and burs in such a manner as to lacerate the gingiva results in varying degrees of mechanical trauma and inflammation. Although such transient injuries generally undergo repair, they are needless sources of discomfort to the patient. Forceful packing of a gingival retraction cord into the sulcus to prepare subgingival margins on a tooth or for the purpose of obtaining an impression may mechanically injure the periodontium and leave behind impacted debris capable of causing a foreign body reaction.

Malocclusion

Irregular alignment of teeth as found in cases of malocclusion may make plaque control more difficult. Several authors have found a positive correlation between crowding and periodontal disease,[24,117,157] whereas other investigators did not find any correlation.[46] Uneven marginal ridges of contiguous posterior teeth have been found to have a low correlation with pocket depth, loss of attachment, plaque, calculus, and gingival inflammation.[77] Roots of teeth that are prominent in the arch (Figures 22-24 and 22-25), such as in a buccal or lingual version, or that are associated with a high frenal attachment and small quantities of attached gingiva frequently exhibit recession.[1,101]

Figure 22-26 Anterior open bite with flared incisors as observed in association with a habit of tongue thrusting.

For more information on malocclusion, please visit the Expert CONSULT companion website at www.expertconsult.com.

Tongue thrusting exerts excessive lateral pressure on the anterior teeth, which may result in spreading and tilting of the anterior teeth (Figures 22-26 and 22-27). Tongue thrusting is an important contributing factor in tooth migration and the development of an anterior open bite.[26] Mouth breathing may be observed in association with a habit of tongue thrusting and an anterior open bite. Marginal and papillary gingivitis is frequently encountered in the maxillary anterior sextant in cases involving an anterior open bite with mouth breathing. However, the role of mouth breathing as a local etiologic factor is unclear because conflicting evidence has been reported.[3,66,67,157]

Restorations that do not conform to the occlusal pattern of the mouth result in occlusal disharmonies that may cause injury to the supporting periodontal tissues. Teeth with deeper initial probing depths, worse prognoses, and greater mobility have been observed for teeth with occlusal discrepancies than teeth without initial occlusal discrepancies.[60,112] Histologic features of the periodontium for a tooth subjected to traumatic occlusion include a widened subcrestal periodontal ligament space, a reduction in collagen content of the oblique and horizontal fibers, an increase in vascularity and leukocyte infiltration, and an increase in the number of osteoclasts on bordering alveolar bone.[13] However, these observations are generally apical and separate from the bacterial-induced inflammation that occurs at the base of the sulcus. On the basis of current human trials, it is still impossible to definitively answer the question, "Does occlusal trauma modify the progression of periodontal attachment loss associated with periodontal

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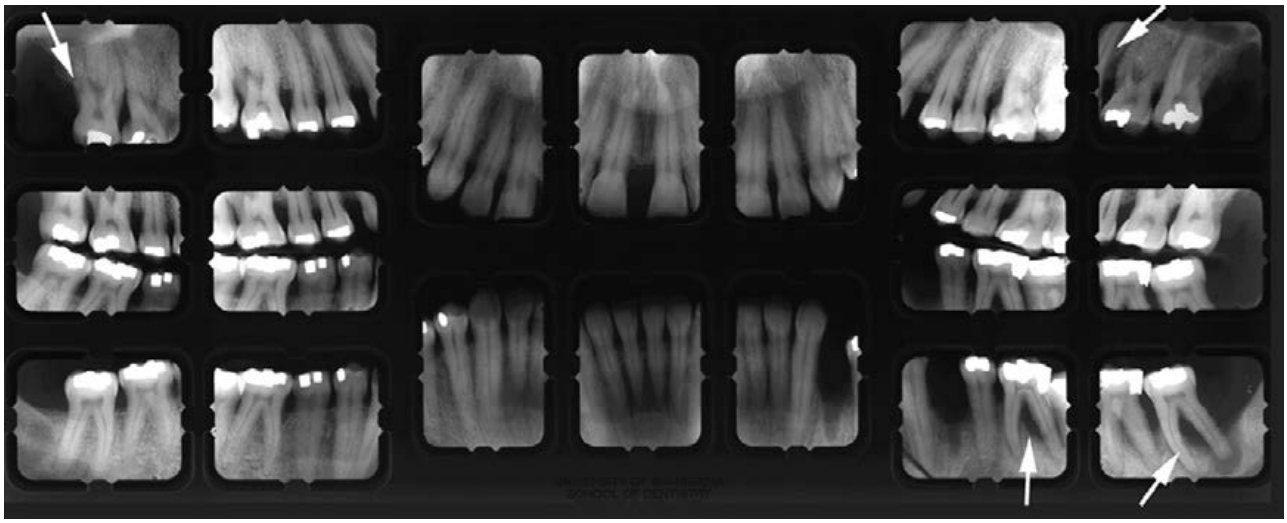

Figure 22-27 Radiographs of patient shown in Figure 22-26 with anterior open bite. Note severe periodontal destruction *(arrows)* in molar regions.

Figure 22-28 Gingival inflammation and enlargement associated with orthodontic appliance and poor oral hygiene.

inflammation?"[147] (See Chapter 49 for a more detailed explanation of periodontal trauma from occlusion and the periodontal response to external forces.)

Periodontal Complications Associated with Orthodontic Therapy

Orthodontic therapy may affect the periodontium by favoring plaque retention, by directly injuring the gingiva as a result of overextended bands, and by creating excessive forces, unfavorable forces, or both on the tooth and supporting structures.

Plaque Retention and Composition. Orthodontic appliances not only tend to retain bacterial plaque and food debris, resulting in gingivitis (Figure 22-28), but also are capable of modifying the gingival ecosystem. An increase in *Prevotella melaninogenica, Prevotella intermedia,* and *Actinomyces odontolyticus* and a decrease in the proportion of facultative microorganisms was detected in the gingival sulcus after the placement of orthodontic bands.[35] More recently, *Aggregatibacter actinomycetemcomitans* was found in at least one site in 85% of children wearing orthodontic appliances.[116] In contrast, only 15% of unbanded control subjects were positive for *A. actinomycetemcomitans*.

Gingival Trauma and Alveolar Bone Height. Orthodontic treatment is often started soon after eruption of the permanent teeth, when the junctional epithelium is still adherent to the enamel surface. Orthodontic bands should not be forcefully placed beyond the level of attachment because this will detach the gingiva from the tooth and result in apical proliferation of the junctional epithelium with an increased incidence of gingival recession.[118]

The mean alveolar bone loss per patient for adolescents who underwent 2 years of orthodontic care during a 5-year observation period ranged between 0.1 and 0.5 mm.[19] This small magnitude of alveolar bone loss was also noted in the control group and therefore considered to be of little clinical significance. However, the degree of bone loss during adult orthodontic care may be higher than that observed in adolescents,[91] especially if the periodontal condition is not treated before initiating orthodontic therapy. *Therefore orthodontic treatment should not be commenced in the presence of uncontrolled periodontal disease.*

Tissue Response to Orthodontic Forces. Orthodontic tooth movement is possible because the periodontal tissues are responsive to externally applied forces.[128,143] Alveolar bone is remodeled by osteoclasts inducing bone resorption in areas of pressure and osteoblasts forming bone in areas of tension. Although moderate orthodontic forces ordinarily result in bone remodeling and repair, excessive force may produce necrosis of the periodontal ligament and adjacent alveolar bone.[121-123] Excessive orthodontic forces also increase the risk of apical root resorption.[21,22] The prevalence of severe root resorption, as indicated by resorption of more than one third of the root length, during orthodontic therapy in adolescents has been reported at 3%.[71] The incidence of moderate to severe root resorption for incisors among adults ranging in age from 20 to 45 years has been reported as 2% pretreatment and 24.5% posttreatment.[91] Risk factors associated with root resorption during orthodontic treatment include duration of treatment, magnitude of the force applied, direction of the tooth movement, and continuous versus intermittent application of forces[120] (Figures 22-29 to 22-31). *It is important to avoid excessive force and/or too rapid of tooth movement in orthodontic treatment.*

The use of elastics to close a diastema may result in severe attachment loss with possible tooth loss as the elastics migrate apically along the root (Figures 22-32 and 22-33). Surgical exposure of impacted teeth and orthodontic-assisted eruption has the potential to compromise the periodontal attachment on adjacent teeth (Figures 22-34 and 22-35). However, the majority of impacted teeth that are surgically exposed and aided in their eruption by

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Figure 22-29 Panoramic radiograph illustrating a limited degree of pretreatment root resorption *(arrows)* existed before orthodontic care was initiated.

Figure 22-30 Panoramic radiograph of the same patient as shown in Figure 22-29 after 4 years of intermittent orthodontic treatment. Note that several roots have undergone severe resorption *(arrows)* during orthodontic care.

Figure 22-31 Panoramic radiograph of the same patient as depicted in Figure 22-30. Note the teeth that had developed extensive root resorption with accompanying hypermobility have been extracted and replaced by implant supported crowns.

orthodontic treatment subsequently exhibited >90% of their attachment intact.[62]

It has been reported that the dentoalveolar gingival fibers that are located within the marginal and attached gingiva are stretched when teeth are rotated during orthodontic therapy.[36] Surgical severing or removal of these gingival fibers in combination with a brief period of retention may reduce the incidence of relapse after orthodontic treatment intended to realign rotated teeth.[20,102]

Figure 22-32 Maxillary central incisors in which an elastic ligature was used to close a midline diastema. Note inflamed gingiva and deep probing depths.

Figure 22-33 The same patient as shown in Figure 22-32 with a full-thickness mucoperiosteal flap reflected to expose the elastic ligature and angular intrabony defects around the central incisors.

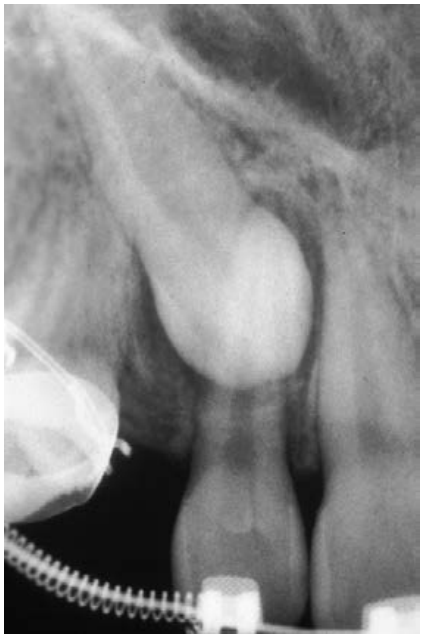

Figure 22-34 Radiograph of an impacted maxillary canine that required surgical exposure and orthodontic assistance to erupt.

Extraction of Impacted Third Molars

Numerous clinical studies have reported that the extraction of impacted third molars often results in the creation of vertical defects distal to the second molars.[7] This iatrogenic effect is unrelated to flap design[28] and appears to occur more often when third molars are extracted in individuals older than 25 years.[7,80,99] Other

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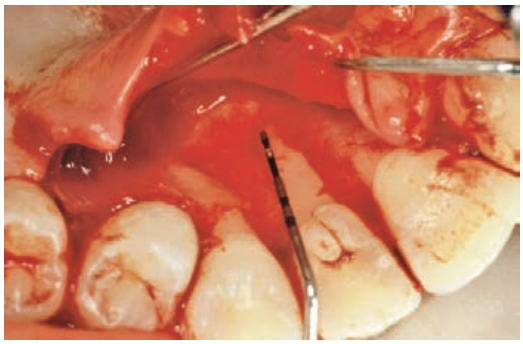

Figure 22-35 Same patient shown in Figure 22-34 with palatal flap reflected to reveal bony dehiscence on the maxillary lateral incisor after orthodontic therapy.

Figure 22-36 Panoramic radiograph illustrating a mesial impacted lower left third molar with a widened follicle and no apparent bone on the distal interproximal surface of the second molar. Alternatively, the lower right third molar is vertically impacted and exhibits interproximal bone distal to the second molar and mesial to the third molar.

factors that appear to play a role in the development of lesions on the distal surface of second molars, particularly in those older than 25 years, include the presence of visible plaque, bleeding on probing, root resorption in the contact area between second and third molars, presence of a pathologically widened follicle, inclination of the third molar, and close proximity of the third molar to the second molar (Figure 22-36).[80] Other potential iatrogenic adverse consequences of removal of third molars include permanent paresthesia (numbness of the lip, tongue, and cheek), which occurs at a frequency of approximately 1 in 100,000 among wisdom teeth removed in the United States.[45]

Habits and Self-Inflicted Injuries

Patients might not be aware of their self-inflicted injurious habits that may be important to the initiation and progression of their periodontal disease. Mechanical forms of trauma can stem from improper use of a toothbrush, wedging of toothpicks between the teeth, application of fingernail pressure against the gingiva, pizza burns, and others[18] (Figure 22-37). Sources of chemical irritation include topical applications of caustic medications such as aspirin or cocaine, allergic reactions to toothpaste and chewing gum, use of chewing tobacco, and concentrated mouthrinses.[146] Accidental and iatrogenic gingival injuries may be caused by a variety of chemical, physical, and thermal sources, yet they are generally self-limiting. Iatrogenic injuries are often acute, whereas factitious injuries tend to be more chronic in nature.[127]

Trauma Associated with Oral Jewelry. The use of piercing jewelry in the lip or tongue has become more common

Figure 22-37 Gingival recession on a maxillary canine caused by self-inflicted trauma from the patient's fingernail.

Figure 22-38 Tongue pierced with oral jewelry.

Figure 22-39 Probing depth of 8 mm with 10 mm clinical attachment loss on lingual surface of lower central incisor adjacent to oral jewelry in pierced tongue. Central incisor was found to be vital. *(Courtesy Dr. Leonidas Batas, Minneapolis.)*

recently among teenagers and young adults (Figure 22-38). Whittle and Lamden[170] surveyed 62 dentists and found that 97% had seen patients with either lip- or tongue-piercing jewelry within the previous 12 months. The incidence of lingual recession with pocket formation (Figure 22-39) and radiographic evidence of bone loss (Figure 22-40) was 50% among subjects with a mean age of 22 years who wore lingual "barbells" for 2 or more years.[34] Chipped lower anterior teeth were noted in 47% of the patients who wore tongue-piercing jewelry for 4 years or more. Patients need to be informed of the risks of wearing oral jewelry and cautioned against such practices.

Toothbrush Trauma. Abrasions of the gingiva, as well as alterations in tooth structure, may result from aggressive brushing

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Figure 22-40 Radiograph of lower incisor shown in Figure 22-39 depicting bone loss associated with tongue pierced by oral jewelry.

Figure 22-41 Overzealous use of a toothbrush resulted in denudation of the gingival epithelial surface and exposure of the underlying connective tissue as a painful ulcer.

in a horizontal or rotary fashion. The deleterious effect of excessively forceful brushing is accentuated when highly abrasive dentifrices are used. The gingival changes attributable to toothbrush trauma may be acute or chronic. The acute changes vary in their appearance and duration from scuffing of the epithelial surface to denudation of the underlying connective tissue with the formation of a painful gingival ulcer (Figure 22-41). Diffuse erythema and denudation of the attached gingiva throughout the mouth may be a striking result of overzealous brushing. Signs of acute gingival abrasion are frequently noted when the patient first uses a new brush. Puncture lesions may be produced when heavy pressure is applied to firm bristles that are aligned perpendicular to the surface of the gingiva. A forcibly embedded toothbrush bristle may be retained in the gingiva and cause an acute gingival abscess.

Chronic toothbrush trauma results in gingival recession with denudation of the root surface. Interproximal attachment loss is generally a consequence of bacteria-induced periodontitis, whereas buccal and lingual attachment loss is frequently the result of toothbrush abrasion.[153] Improper use of dental floss may result in lacerations of the interdental papilla.

Figure 22-42 Chemical burn caused by aspirin that depicts sloughing gingival tissue and resultant recession.

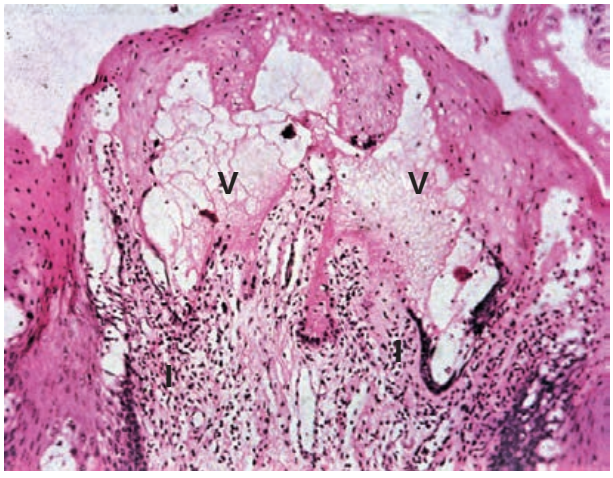

Figure 22-43 Biopsy of aspirin-induced chemical burn. Note intraepithelial vesicles *(V)* and an inflammatory infiltrate *(I)* within the underlying connective tissue.

Chemical Irritation. Acute gingival inflammation may be caused by chemical irritation resulting from either sensitivity or nonspecific tissue injury. In allergic inflammatory states, the gingival changes range from simple erythema to painful vesicle formation and ulceration. Severe reactions to ordinarily innocuous mouthwashes, dentifrices, or denture materials are often explainable on this basis.

Acute inflammation with ulceration may be produced by the nonspecific injurious effect of chemicals on the gingival tissues. The indiscriminate use of strong mouthwashes, topical application of corrosive drugs such as aspirin (Figure 22-42) or cocaine, and accidental contact with drugs such as phenol or silver nitrate are common examples of chemicals that cause irritation of the gingiva. A histologic view of an aspirin-induced chemical burn shows vacuoles with serous exudates and an inflammatory infiltrate in the connective tissue (Figure 22-43).

Smokeless Tobacco

Snuff and chewing tobacco constitute the two main forms of smokeless tobacco. Snuff is a fine cut form of tobacco available as loosely packed or in small sachets. Chewing tobacco is a more coarse-cut available in the form of loose leaves, a solid block, or

plug and as a twist of dried leaves. Chewing tobacco is typically placed in the mandibular buccal vestibule for several hours, during which time saliva and dilute tobacco is periodically expectorated.[167] The nicotine uptake of smokeless tobacco is similar to smoking cigarettes in that consumption of a 34-g container of smokeless tobacco is approximately equal to 1.5 packs of cigarettes.[55] Many professional baseball players use chewing tobacco. Perceived benefits of chewing tobacco are those derived from nicotine, including improved mental alertness and diminished reaction time, muscle relaxation, and reduced anxiety and appetite.[56,132] A 1990 survey of 1109 professional baseball players in the United States reported 39% of the players used smokeless tobacco with 46% of the users exhibiting leukoplakia within the gingiva and/or mucosa (Figure 22-44).[40] Histologic features of oral leukoplakia associated with smokeless tobacco include (1) chevronlike pattern of hyperkeratosis with focal areas of inflammation and (2) hyperplasia in the basal cell layer (Figure 22-45). Increased incidence of gingival recession, cervical root abrasion, and root caries have been reported with smokeless tobacco (Figure 22-46).[113,133,134,160] The incidence of gingival recession among adolescents who use smokeless tobacco has been reported as 42% compared to 17% among nonusers.[31,100,101,103] The NHANES III epidemiologic study investigated the adverse effect of smokeless tobacco on the periodontium and found double the incidence of severe periodontitis (odds ratio 2.1; 95% CI 1.2 to 3.7) among 12,932 adults who used smokeless tobacco but never smoked cigarettes.[41] However, Bergstrom et al found similar incidences of severe periodontitis among both smokeless tobacco users and nonusers.[12] It can be concluded that the use of smokeless tobacco is associated with at least localized gingival recession, clinical attachment loss, leukoplakia, and possibly enhanced susceptibility to severe periodontitis.

Radiation Therapy

Radiation therapy has cytotoxic effects on both normal cells and malignant cells. A typical total dose of radiation for head and neck tumors is in the range of 5000 to 8000 cGy (cGy = 1 rad) or equivalent to 50 to 80 Sieverts (Sv).[119] The total dose of radiation is generally given in partial incremental doses referred to as *fractionation*. Fractionation helps minimize the adverse effects of the radiation while maximizing the death rate for the tumor cells.[42] Fractionated doses are typically administered in the range of 100 to 1000 cGy, or 1 to 10 Sv, per week.

Radiation treatment induces an obliterative endarteritis that results in soft tissue ischemia and fibrosis while irradiated bone

Figure 22-44 Oral leukoplakia in vestibule associated with use of smokeless tobacco.

Figure 22-46 Oral leukoplakia, recession, and clinical attachment loss associated with use of smokeless tobacco.

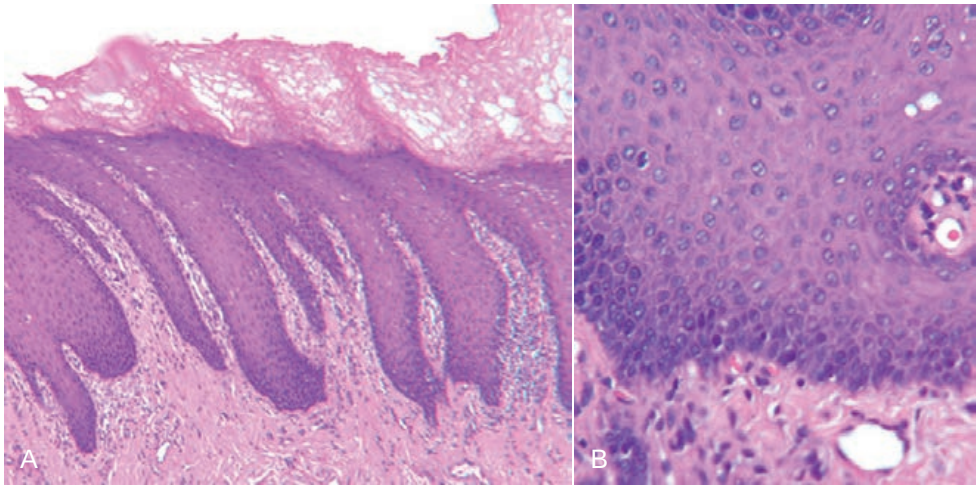

Figure 22-45 Histologic features of oral leukoplakia associated with smokeless tobacco. **A,** Chevronlike pattern of hyperkeratosis with focal areas of inflammation, **B,** Hyperplasia in basal cell layer.

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becomes hypovascular and hypoxic.[97] Adverse affects of head and neck radiation therapy include dermatitis and mucositis of the irradiated area, as well as muscle fibrosis and trismus, which may restrict access to the oral cavity.[135] The mucositis typically develops 5 to 7 days after radiation therapy is initiated. The severity of the mucositis can be reduced by asking the patient to avoid secondary sources of irritation, such as smoking, alcohol, and spicy foods, to the mucous membrane. Use of a chlorhexidine digluconate mouthrinse may help reduce the mucositis.[125] However, most chlorhexidine mouthrinses currently available in the U.S. have a high alcohol content that may act as an astringent, which dehydrates the mucosa, thereby intensifying the pain. Saliva production is permanently impaired when salivary glands that are located within the portal of radiation receive ≥6000 cGy or 60 Sv.[98] Xerostomia results in greater plaque accumulation and a reduced buffering capacity by saliva. The use of effective oral hygiene, professional dental prophylaxis cleanings, fluoride applications, and frequent dental examinations are essential to control caries and periodontal disease. The use of customized trays appears to be a more effective method of fluoride application as compared with the toothbrush.[76]

Periodontal attachment loss and tooth loss has been reported to be greater in cancer patients who were treated with high-dose unilateral radiation as compared with the nonradiated control side of the dentition.[39] Patients diagnosed with oral cancer that require radiation therapy should ideally be assessed for dental needs (mucositis, xerostomia, faulty restorations, periapical lesion, coronal and cervical decay, and periodontal status) before initiating radiation treatment.[59] Treatment and prevention of trismus, oral fungal infections, odontogenic infections, osteoradionecrosis, decay, and periodontal disease is critical to minimize oral morbidity for these patients. Dental and periodontal infections have the potential to be a severe risk for a patient who has been treated with head and neck radiation. The risk of osteoradionecrosis for oncology patients can be minimized by evaluating their oral status, providing dental care, and allowing time for tissue repair before the commencement of radiotherapy.[78]

Oral conditions that increased the risk of osteoradionecrosis for patients about to undergo radiation therapy for oral malignancies include >5 mm periodontal probing depths, >40% dental plaque score, and >60% alveolar bone loss.[74] Nonrestorable teeth and/or teeth with significant periodontal problems should be extracted before radiation therapy to reduce the risk of postradiotherapy osteoradionecrosis.[78] (See Chapter 37 for more detail on the periodontal management of medically compromised patients.)

The risk of osteoradionecrosis must be evaluated before performing atraumatic extractions or limited periodontal surgical procedures in previously irradiated sites. Therefore the dentist may choose to consult with the patient's oncologist before initiating therapy. A recent randomized multicenter clinical trial questions the merit of using hyperbaric oxygen (HBO) therapy to treat osteoradionecrosis, since at 1-year after treatment only 19% of test subjects responded to HBO versus 32% of placebo subjects.[5] Administration of the combination of pentoxifylline with vitamin E as antioxidants currently shows the greatest promise for revascularization and treatment of osteoradionecrosis sites.[33]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

Calculus plays a significant role in periodontal inflammation primarily because it provides an ideal surface for bacterial plaque accumulation. One of the universal goals for treatment of gingivitis and periodontitis is the complete removal of all calculus, particularly subgingival calculus. The presence of residual subgingival calculus after treatment predisposes the patient to ongoing periodontal destruction. The removal of calculus embedded into cementum with root planing is one of the most difficult procedures in periodontal therapy and requires special skills that clinicians are only able to develop with repeated procedures over time. Exposing subgingival calculus with surgical approaches increases the possibility of calculus removed compared to nonsurgical root planing. Therefore the cornerstone of all periodontal flap techniques is commitment to root planing, even though the patient may have previously had nonsurgical or initial therapy.

Other predisposing factors for periodontitis, such as restorative inadequacies and poorly designed removable partial dentures, are significant primarily because they increase the accumulation of bacterial dental plaque. Loss of even one tooth can lead to a plethora of positional changes of adjacent and opposing teeth with an increasing risk of periodontal breakdown caused by open contacts, plunger cusps, and occlusal instability. Thus it is good clinical procedure to replace missing teeth with implants or fixed bridges to maintain periodontal health of the remaining dentition.

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CHAPTER 23

Periodontal Microbiology

Wim Teughels, Marc Quirynen, and Nick Jakubovics

CHAPTER OUTLINE

THE ORAL CAVITY FROM A MICROBE'S PERSPECTIVE
BACTERIA AND THEIR BIOFILM MODE OF LIVING
- Structure of a Mature Dental Plaque Biofilm
- Accumulation of a Dental Plaque Biofilm
- Factors Affecting Supragingival Dental Plaque Formation
- De Novo Subgingival Plaque Formation

CHARACTERISTICS OF BIOFILM BACTERIA (LIFE IN "SLIME CITY")
- Metabolism of Dental Plaque Bacteria
- Communication Between Biofilm Bacteria
- Interactions Between Dental Plaque Bacteria
- Biofilms and Antimicrobial Resistance

BACTERIAL TRANSMISSION AND TRANSLOCATION
NONBACTERIAL INHABITANTS OF THE ORAL CAVITY
- Viruses
- Yeasts
- Protozoa
- Archaea

MICROBIOLOGIC SPECIFICITY OF PERIODONTAL DISEASES
- Nonspecific Plaque Hypothesis
- Specific Plaque Hypothesis
- Ecologic Plaque Hypothesis
- Complicating Factors
- Criteria for Identification of Periodontopathogens

THE TRANSITION FROM HEALTH TO DISEASE
- Host Susceptibility
- Pathogenic Bacteria
- Beneficial Species
- Periodontal Health
- Gingivitis
- Chronic Periodontitis
- Localized Aggressive Periodontitis
- Aggressive Periodontitis
- Necrotizing Periodontal Diseases
- Abscesses of the Periodontium
- Periimplantitis

VIRULENCE FACTORS OF PERIODONTOPATHOGENS
- Adhesive Surface Proteins and Fibrils
- Factors that Promote Tissue Destruction
- Strategies for Evading Host Immunity

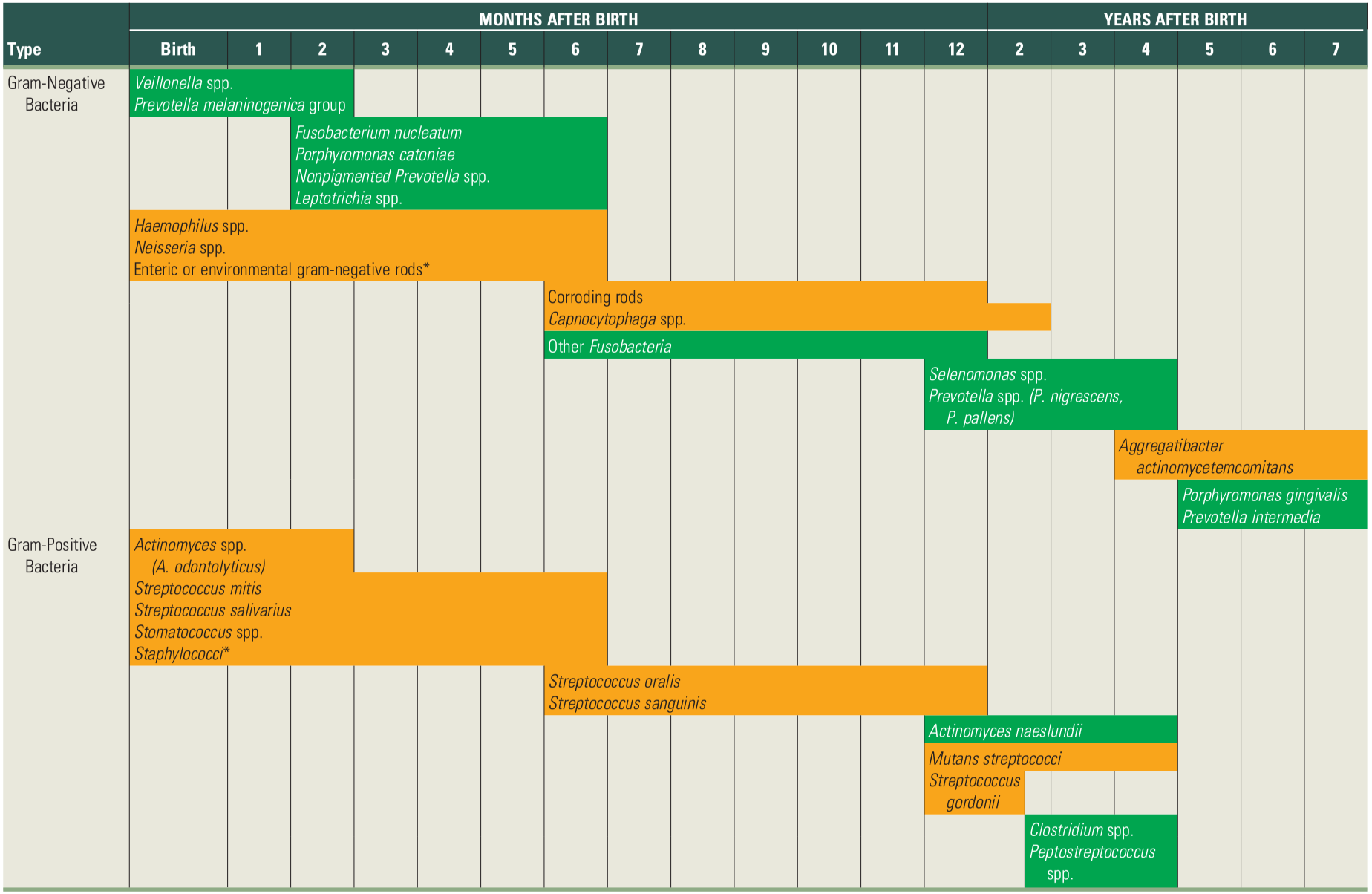
FUTURE ADVANCES IN PERIODONTAL MICROBIOLOGY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some text figures and tables may be out of numeric order in this printed chapter.

The human fetus inside the uterus is sterile but as soon as it passes through the birth canal, it acquires vaginal and fecal microorganisms.[105] Within 2 weeks, a nearly mature *microbiota* is established in the gut of the newborn baby. After weaning (>2 years), the entire human microbiota is formed and comprises a very complex collection of hundreds of different types of bacteria, totalling approximately 10^{14} microbial cells.[248] From this moment on, our body contains *10 times more bacteria* than human cells. It has been estimated that for a normal, healthy human being, the bacterial population comprises 2 kg of the total body weight. This is fascinating if one realizes that the average human brain weighs only about 1.4 kg.

The colonization of the oral cavity also starts close to the time of birth (Table 23-1). Within hours after birth, the sterile oral cavity will be colonized by low numbers of mainly facultative and aerobic bacteria.[368] From the second day, anaerobic bacteria can be detected in the infant's edentulous mouth.[89,319] The number of oral bacteria increases gradually as a result of exposure to external environmental microbial sources.[179,271,319] *Streptococcus salivarius* and *Streptococcus mitis* (Figure 23-1, *A*) have been identified as the first and most dominant oral microbes to colonize the oral cavity of newborn infants.[177,178,197] *Veillonella* spp. (Figure 23-1, *B*), *Neisseria* spp., *Actinomyces* spp. (Figure 23-1, *C* and *D*) and *Staphylococcus* spp. are also among the first colonizers of the oral cavity. After

TABLE 23-1 Colonization of the Oral Cavity



Adapted from Kononen E: *Oral Dis* 5:278, 1999; Kononen E: *Ann Med* 32:107, 2000.

Amenable periods for the establishment of most frequent bacterial species or groups (prevalence >25%) in infants' mouths. Anaerobic bacteria are indicated in green, and aerobic or facultative bacteria are indicated in orange. Bacteria indicated with an asterisk (*) decrease in prevalence with age.

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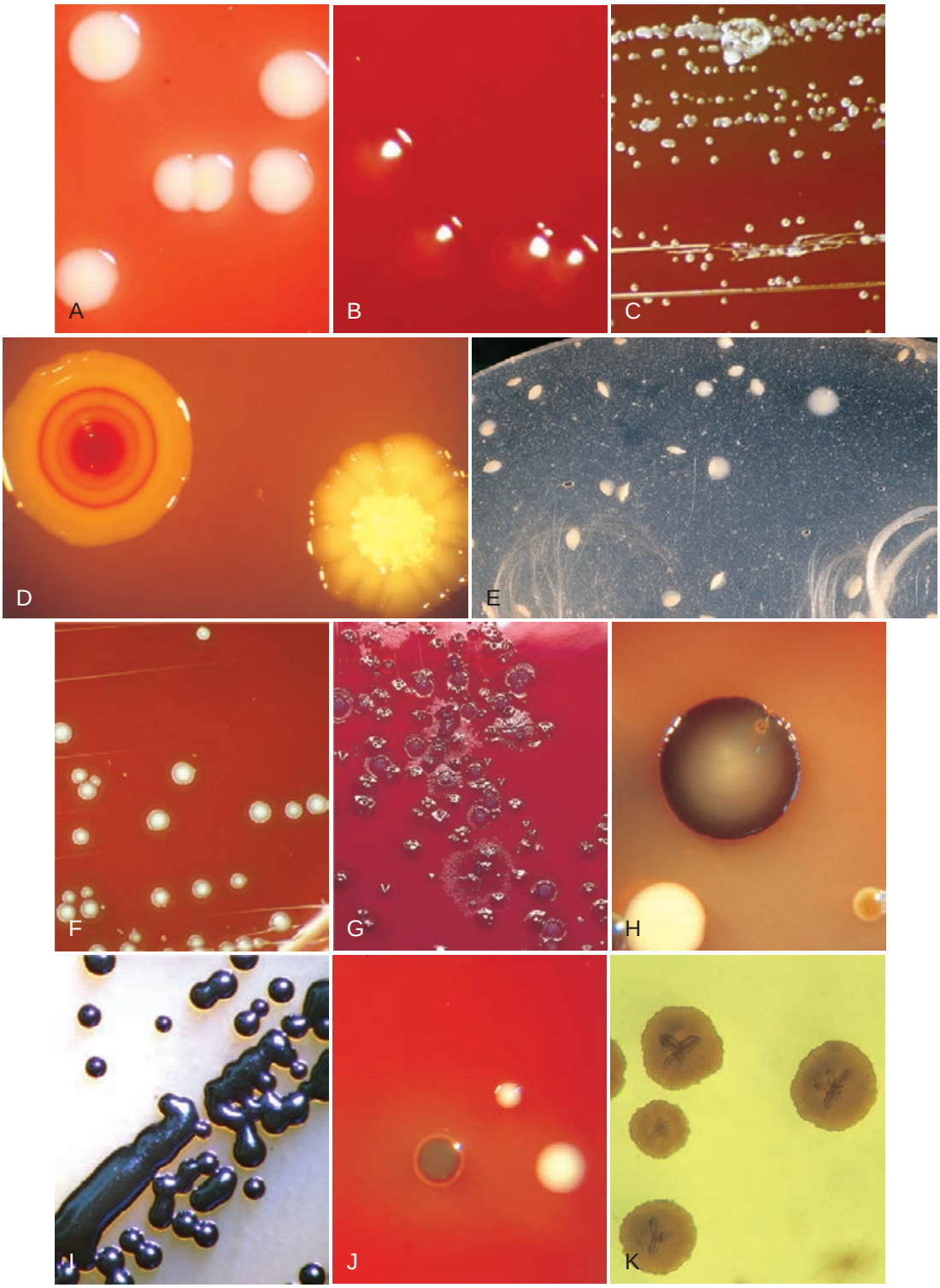

Figure 23-1 Various periodontal and cariogenic species grown on agar plates. A, *Streptococcus mitis* are gram-positive, fast-growing, facultatively anaerobic bacteria that are easy to culture on a blood-agar plate. A clear halo surrounding the colonies appears through hemolytic activity. **B,** *Veillonella parvula* are anaerobic gram-negative small cocci. They form small transparent colonies (<1.0 mm) after 48 hours of incubation. **C,** *Actinomyces viscosus* are microaerophilic to anaerobic gram positive rods with possible branches (pseudomycelium). They form slimy white spherical colonies in 48 hours. **D,** Typical colony morphology of *Streptococcus sanguinis (right)* and *Actinomyces odontolyticus (left)*. **E,** *Lactobacillus* spp. will typically grow on Rogosa agar as a sesame seed. **F,** *Streptococcus gordonii* are facultative anaerobic gram-positive cocci. On a blood plate, colonies of 1 to 3 mm are formed within 48 hours. These bacteria are α-hemolytic, resulting in the formation of a clear halo surrounding the colony. **G,** This selective agar plate containing crystal violet and erythromycin (CVE-agar plate) will allow *Fusobacterium nucleatum* to grow as a round, flat rhizoid, opaque purple colony. **H,** Detailed picture of *Porphyromonas gingivalis* (green-brown) and *Prevotella intermedia* (black) on a classic nonspecific blood-agar plate. **I,** *Prevotella nigrescens* forms like *Prevotella intermedia*, a black pigmented colony on a blood agar plate. It is a strictly anaerobic bacterium whose growth is limited to an oxygen-free environment. **J,** Detailed picture of *Parvimonas micra* (small white colony) next to *Porphyromonas gingivalis* (green-brown colony) on a classic non-specific blood-agar plate. **K,** Detailed picture of *Aggregatibacter actinomycetemcomitans* grown on a selective agar plate containing tryptic soy, horse serum, bacitracin and vancomycin (TSBV-agar plate).

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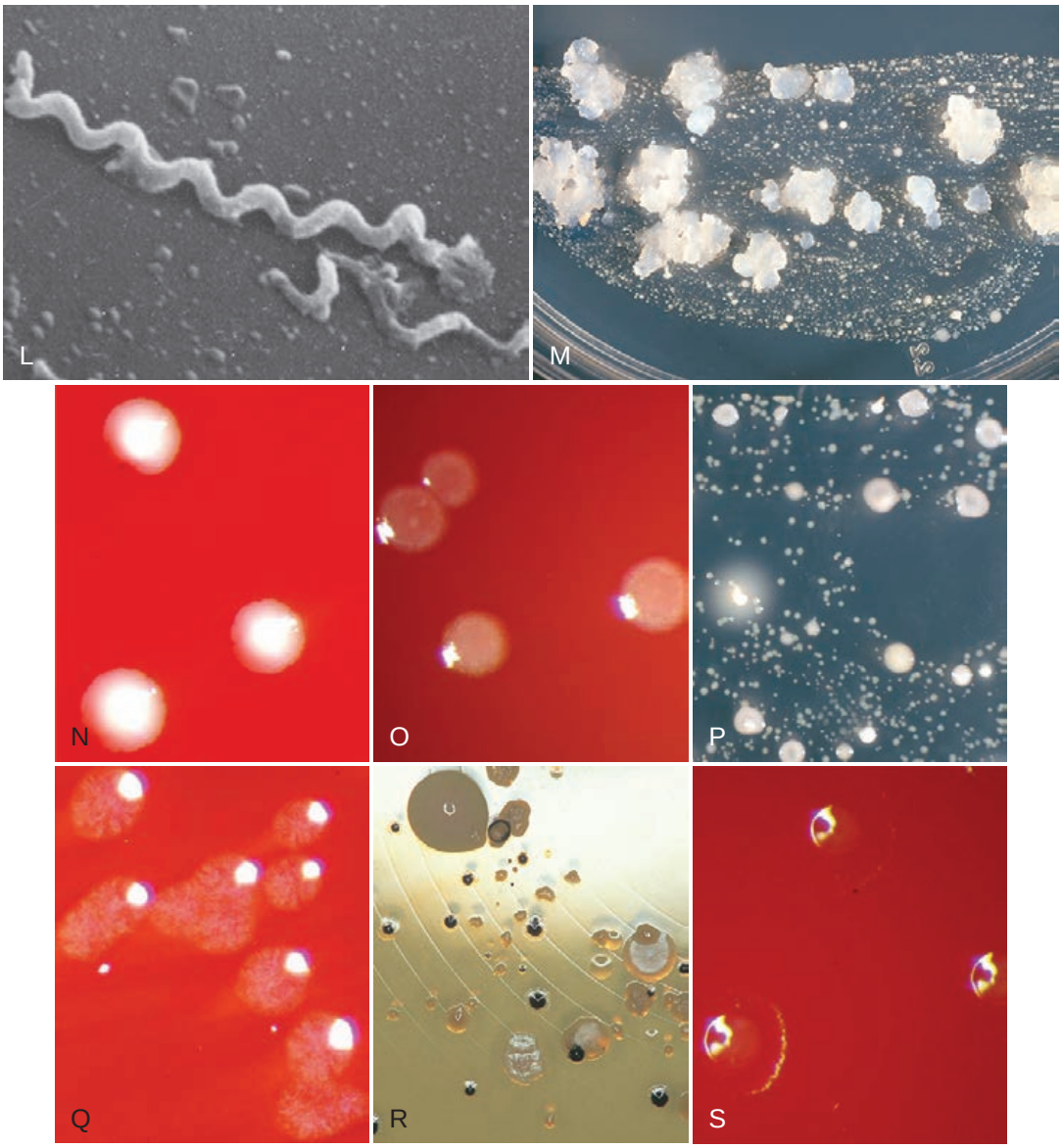

Figure 23-1, cont'd **L,** It is extremely difficult to culture *Treponema denticola* (spirochete) on an agar plate and therefore not possible to identify this bacteria with classic culture. Phase-contrast microscope, the dark-field microscope, or the electron microscope are often used to visualize this bacterium. Identification and quantification is only possible through DNA analysis. **M,** On a selective agar plate containing trypticase yeast-extract, cystine, sucrose, and bacitracin (TYCSB agar plate), *Streptococcus mutans* will grow as a sugar cube. **N,** *Eubacterium nodatum* colony morphology strongly depends on its substrate. Its growth is very slow, and it is an obligate anaerobic gram-positive rod. **O,** *Tannerella forsythia* are fastidious bacteria and are therefore difficult to culture. This organism grows on a blood-agar plate as a smooth white colony with a faded edge. The bacteria are strictly anaerobic. **P,** Typical colony morphology of *Streptococcus sobrinus* on TYCSB agar (colony with white halo). **Q,** Capnocytophaga are slowly growing bacteria that require an elevated CO_2 concentration for their growth. They are facultative anaerobic rods. **R,** *Campylobacter rectus* grows on a Hammond plate as small, smooth opaque, round colonies with a black color. **S,** *Eikenella corrodens* has a variable colony morphology and shows different biochemical and serologic reactions. Because of the difficult determination with classic culture, identification and quantification through DNA techniques is very suitable for this organism. *E. corrodens* cells are facultative anaerobic gram-negative rods. (Images A, B, C, F, I, L, N, O, Q, and S courtesy ADD Clinident, Malden, The Netherlands).

tooth eruption, a more complex oral microbiota is established. The species colonizing the teeth after eruption include *S. sanguinis* (Figure 23-1, *D*), *Lactobacillus* spp. (Figure 23-1, *E*), and *S. oralis*. Oral streptococci, including *S. oralis, S. anginosus, Mutans streptococci,* and *S. gordonii* (Figure 23-1, *F*), are commonly reported to be present after the first year of life.[49,53,271,319] In addition, anaerobes, including *Fusobacterium* spp. (Figure 23-1, *G*) and *Prevotella* spp. (Figure 23-1, *H* and *I*), can also be detected in young children.[49,179] In later childhood, the bacterial diversity and numbers in the oral cavity increase as more teeth erupt and provide more areas for the adherence and retention of bacteria.[32] Because of the paucity of longitudinal studies, relatively little is known about the first time of colonization by key microbes found in the oral cavity of children and adults.[197] It is estimated that *more than 500 different species* are capable of colonizing the adult mouth and that any individual typically harbors 150 or more different species.[250,363] When one thinks about bacteria, one almost immediately associates them with different pathologies. However, most oral bacteria are harmless commensals under normal circumstances. This means that this microbiota lives in harmony with its host, but under specific conditions (increased mass and/or pathogenicity, suppression of commensal or beneficial bacteria and/or reduced host response), disease

can occur. The importance of the commensal microbiota is clearly illustrated by the development of yeast infections when the normal oral microbiota is reduced, for example, after a longer period of systemic antibiotic usage.[410] Additionally, it has been shown that aggressive periodontitis is associated with a loss of colonization of *S. sanguinis*.[371]

It is obvious that the periodontal microbiota is extremely complex. Since it affects the host, the oral environment and periodontal treatment and since it is affected by the host, the oral environment and periodontal treatment, a profound knowledge of periodontal microbiology is necessary.

THE ORAL CAVITY FROM A MICROBE'S PERSPECTIVE

In general, with the exception of those microorganisms that are present in feces and in secretory fluids, all bacteria maintain themselves within their host by adhering to a surface. This principle also applies to the oral cavity. From an ecologic viewpoint, the oral cavity, which communicates with the pharynx, should be considered as an "open growth system" with an uninterrupted ingestion and removal of microorganisms and their nutrients. A dynamic equilibrium exists between the adhesion forces of microorganisms and a variety of removal forces originating from (1) swallowing, mastication, or blowing the nose; (2) tongue and oral hygiene implements; (3) the wash-out effect of the salivary, nasal, and crevicular fluid outflow; and (4) active motion of the cilia (nasal and sinus walls). Most organisms can only survive in the oropharynx when they adhere to either the soft tissues or the hard surfaces (teeth, dentures, and implants).

The ability of a bacterium to adhere to its host is crucial for the induction of infectious diseases, such as gingivitis or periodontitis.[310] Oral bacteria and especially pathogenic bacteria, such as *Porphyromonas gingivalis* (Figure 23-1, *H* and *J*) and *Aggregatibacter actinomycetemcomitans* (Figure 23-1, *K*), have a large battery of virulence factors, one of which is the ability to adhere to hard intraoral surfaces (teeth, restorative and prosthetic materials, and perimucosal implants) and/or to the oral mucosae, which have a significant variety of epithelial characteristics (Figure 23-2).[56,58,103,357]

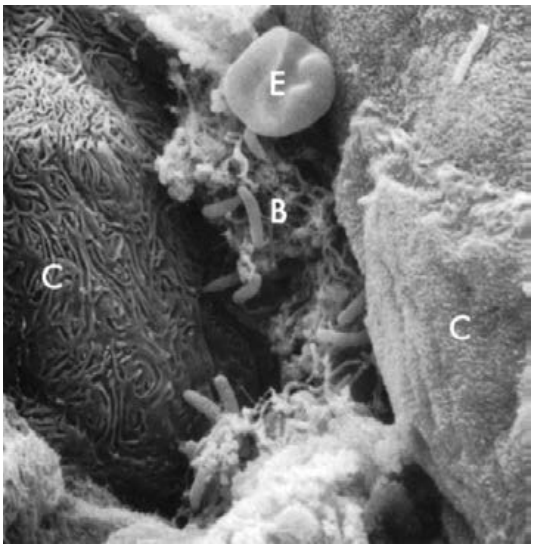

Figure 23-2 Scanning electron micrograph of epithelial intercellular spaces containing bacterial plaque *(B)* enmeshed in a fibrinlike material. *C,* Epithelial cells; *E,* erythrocyte. The cells to the left show signs of necrosis. (×4000.)

Several different types of surfaces are available for bacterial adhesion: soft tissues, such as mucosae, skin, and cornea, and hard tissues, such as teeth and nails. On the basis of physical and morphologic criteria, the oral cavity can be divided into *six major ecosystems* (also called *niches*), each with the following distinct ecologic determinants:

- The intraoral, supragingival, hard surfaces (teeth, implants, restorations, and prostheses)
- Subgingival regions adjacent to a hard surface, including the periodontal/periimplant pocket (with its crevicular fluid, the root cementum or implant surface, and the pocket epithelium)
- The buccal, palatal epithelium, and the epithelium of the floor of the mouth
- The dorsum of the tongue
- The tonsils
- The saliva

Table 23-2 summarizes several publications on the detection frequency of periodontopathogens in these different niches. Most species (with the exception of spirochetes (Figure 23-1, *L*) are able to colonize all of them. Some periodontopathogens (*Fusobacterium nucleatum* (Figure 23-1, *G*) and *Prevotella intermedia* (Figure 23-1, *H*) are involved in the etiology of tonsillitis and most periodontopathogens are able to colonize the maxillary sinus.[36,407]

The soft tissue surfaces are not passive bystanders in the process of bacterial adhesion and colonization.[146] They employ a variety of mechanisms to prevent adhesion of pathogenic organisms. One of the most important mechanisms is shedding. The high turnover rate of the intraoral epithelial cells, especially of the gingiva, prevents the permanent accumulation of large masses of microorganisms on these surfaces. In essence, this is a natural cleansing mechanism. However, bacteria can adhere to host cells and form a commensal relationship, beneficial for both parties.

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Also, in periodontal pockets, studies have shown high numbers of bacteria attached to pocket epithelial cells in vivo. In areas of gingival inflammation, there is an increased number of adhering bacteria.[79,393] These adhering bacteria can also infiltrate the pocket wall in relatively large numbers and reach the underlying stroma (Figure 23-3).[96,231,327] In general, there is a positive correlation between the adhesion rate of pathogenic bacteria to different epithelia and the susceptibility of that patient to certain infections.[262]

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There are some indications that the same may be true for periodontal infections. Isogai and co-workers reported a significantly lower adherence rate of *P. gingivalis* and *Prevotella intermedia* strains to gingival epithelial cells in rats, which were resistant to gingivitis when compared to susceptible rats.[153] An in vitro study on cultured human pocket epithelial cells (Figure 23-4) showed a similar tendency when patients resistant to periodontitis were compared to patients with severe periodontal breakdown.[299]

TABLE 23-2 Intraoral Habitats (Periodontal Pocket, Buccal Mucosa, Tongue, Saliva, Tonsils, and Supragingival Plaque) for Periodontopathogenic and Cariogenic Species

Authors	Infection	Age (n)	Species	# Positive Patients	DIFFERENT INTRAORAL NICHES					
					Pocket	Mucosa	Tongue	Saliva	Tonsils	Supra
Asikainen et al, 1991	Periodontitis	Adult	*A.a.*	–	100%*	–	56%	72%	–	–
Petit et al, 1994	–	Child (45)	*A.a.*	5	4†	3	1	3	2	–
			P.g.	1	0	1	0	1	1	–
			P.i.	34	23	18	25	31	22	–
			Spi	13	6	0	7	3	1	–
Petit et al, 1994	Periodontitis	Adult (24)	*A.a.*	13	13†	11	11	11	5	–
			P.g.	18	18	14	7	10	11	–
			P.i.	24	24	22	23	23	23	–
			Spi	22	22	0	2	5	1	–
von Troil-Lindén et al, 1995	Periodontitis	Adult (10)	*A.a.*	–	6‡	–	–	6	–	–
			P.g.	–	7	–	–	4	–	–
			P.i.	–	10	–	–	9	–	–
Danser et al, 1994	Perio/E	Adult (8)	*A.a.*	2	2/–§	2/0	2/0	2/0	2/0	1/0
			P.g.	6	6/–	2/0	4/0	4/0	3/0	2/0
			P.i.	8	8/–	4/1	6/3	6/4	5/3	6/1
			Prev.	8	3/–	4/5	7/6	7/5	4/6	5/4
Danser et al, 1996	Perio/R	Adult (15)	*A.a.*	11	9/4¶	10/9	–	5/2	–	2/0
			P.g.	10	6/5	9/7	–	4/1	–	6/0
			P.i.	15	11/1	15/15	–	14/13	–	11/1
Gibbons and van Houte, 1975	–¶	–¶	*S.m.*	–	?	<1¶	<1	<1	–	0-50
			L.		?	<0.1	< 0.1	<1	–	0-1

P.g., P. gingivalis, P.i., P. intermedia, A.a., A. actinomycetemcomitans, Spi, spirochetes, *Prev., Prevotella species (e.g., P. melaninogenica, P. denticola, P. loeschii, P. veroralis) S.m., S. mutans, L., Lactobacillus spp.*

*Percentage of "specific" positive sites in positive patients.

†Number of "specific" positive sites in positive patients.

‡Number of "specific" positive sites in patients with advanced periodontitis.

§Number of "specific" positive sites in positive patients before/after full mouth extraction (E) (e.g., 2 patients were positive for *Aa* before extraction, and no patients were positive for *Aa* after full mouth extraction).

¶Number of "specific" positive sites in positive patients before/after periodontal therapy including surgery (R).(e.g., 9 patients were positive for *Aa* before periodontal therapy compared to only 4 patients after periodontal therapy).

¶Percentage of total flora cultivable in anaerobically incubated agar in nonspecific patients as estimated from several studies.

In contrast to soft tissues, teeth and nails are the only naturally occurring nonshedding surfaces. Artificial nonshedding surfaces of medical importance are prosthetic devices such as catheters, artificial joints, dental implants, and heart valves. From a microbiologic viewpoint, teeth and implants are unique for two reasons: (1) they provide a hard nonshedding surface that allows the development of extensive structured bacterial deposits and (2) they form a unique ectodermal interruption. There is a special seal of epithelium (junctional epithelium) and connective tissue between the external environment and the internal parts of the body. The accumulation and metabolism of bacteria on these hard surfaces is considered the primary cause of caries, gingivitis, periodontitis, periimplantitis, and sometimes, bad breath.

In the periodontal pocket, different strategies contribute to bacterial survival such as swimming in the crevicular fluid, adhesion to the pocket epithelium, and when dentine is encountered, the colonization of the dentine tubules.[290] The crevicular fluid with its constant outflow does not favor the maintenance of unattached bacteria in the periodontal pocket.

It has been suggested that teeth are the primary habitat for periodontopathogens. Indeed, soon after full-mouth tooth extraction in patients with severe periodontitis, key pathogens, such as *A. actinomycetemcomitans* and *P. gingivalis,* disappeared from the oral cavity as determined by bacterial culturing techniques.[71] *Prevotella intermedia* and other black-pigmented *Prevotella* spp. could, however, remain but at lower detection frequencies and numbers (see Table 23-2).

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However, recent studies using molecular tools to detect and quantify oral bacteria seem to indicate that *A. actinomycetemcomitans* and *P. gingivalis* are not entirely eradicated after full-mouth extraction. They may remain colonizers of the oral cavity, but when teeth are lost, their relative numbers decrease.[304]

Cariogenic species, on the contrary, were thought to be relatively restricted to solid surfaces (see Table 23-2). For that reason *S. mutans* (Figure 23-1, *M*) was often called an *obligate periphyte.*[380] In some studies, this species was only detected from the time that the deciduous teeth erupted in the oral cavity.[50] In a longitudinal observation of adults with severe dental caries, the cariogenic species fell below detection level after full-mouth extraction but reappeared a few days after denture insertion.[51] Based on these

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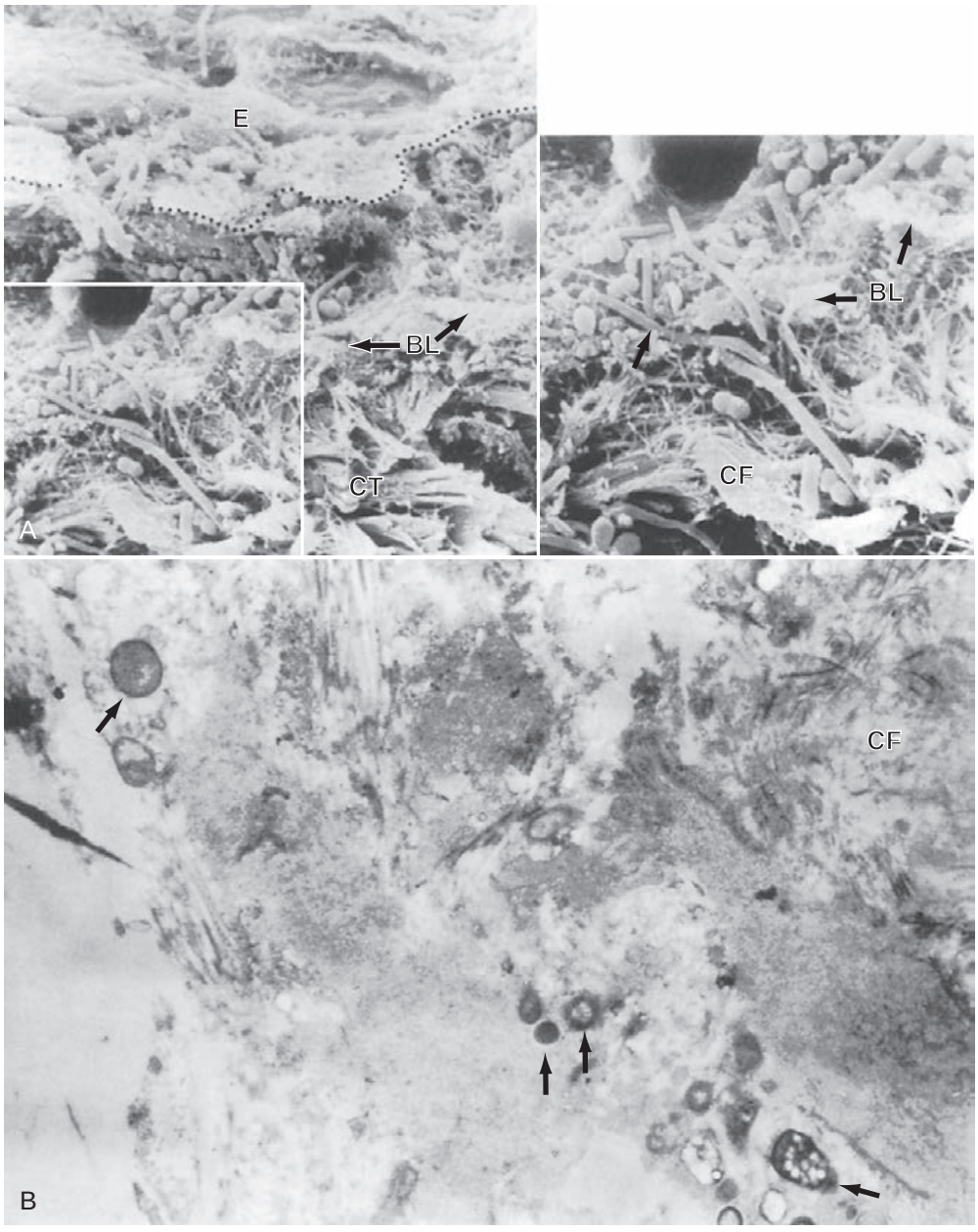

Figure 23-3 Bacterial penetration into the pocket wall in advanced periodontitis. **A,** The penetration through the pocket epithelium *(E)* and basement lamina *(BL)* into the connective tissue *(CT)* *(arrows)*. *CF,* Collagen fibers. **B,** Connective tissue–associated bacteria in advanced periodontitis. (**A** courtesy Dr. R. Saglie; **A** and **B** from Nissengard RJ, Newman MG: *Oral microbiology and immunology,* ed 2, Philadephia, 1994, Saunders.)

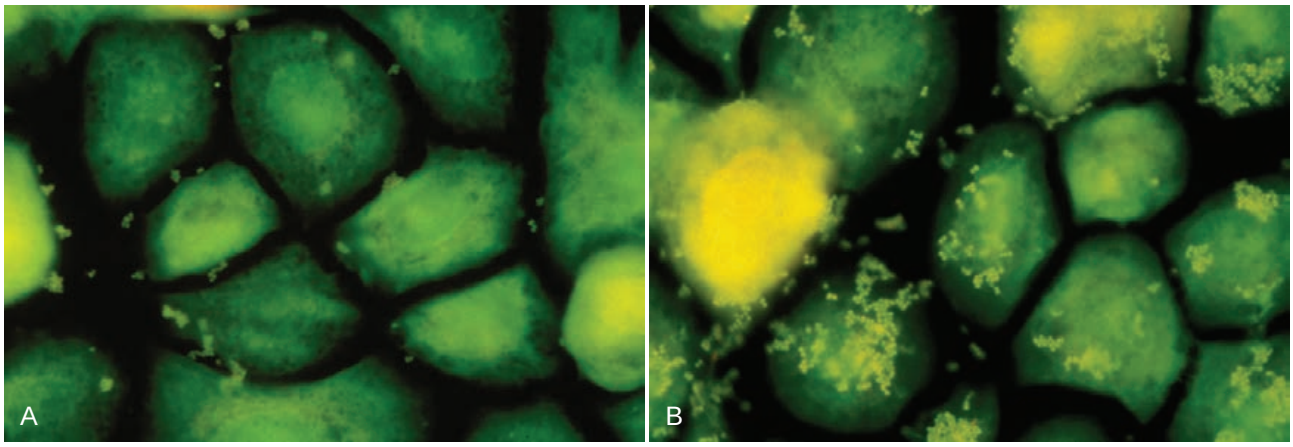

Figure 23-4 Microscopic confirmation of significant difference in adhesion capacity of *P. gingivalis (small green dots*) to epithelial cells from resistant patient *(left*) versus a patient suffering from severe periodontitis *(right*).

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Figure 23-5 Vertical section through a 4-day human plaque sample. An intraoral device designed for in vivo generation of plaque biofilms on enamel was used. Confocal microscopy enabled visualization of the section of plaque without the dehydration steps used in conventional histologic preparations. Notice the open fluid filled channels *(white arrows)* that traverse from the plaque surface through the bacterial mass (*M*; grey-white areas) to the enamel surface. An area in which the bacterial mass appears to attach to the enamel surface *(A)* is indicated. Scale bar = 25 µm. (From Wood SR, Kirkham J, Marsh PD, et al: *J Dent Res* 79:21, 2000.)

reports and on their own observations, Caufield and Gibbons concluded that most of the *S. mutans* cells in the saliva or on the tongue are derived from the biofilm present on the teeth and that the mucosae could not act as a reservoir for the infection of teeth by those organisms.[54] They suggested that there is a "window of infectivity" for the acquisition of *S. mutans* at a mean age of 26 months (range 9 to 44 months).[53] This observation has been supported by a few clinical studies, which showed that initial colonization of *S. mutans* varied between 7 to 36 months, which is a time period coinciding with eruption of the primary teeth.[6,49,99] On the other hand, longitudinal studies by Wan and co-workers[411,412] and Law and Seow[196] showed that there was increasing *S. mutans* colonization with increasing ages of the children, without any discrete window of infectivity. There is now clinical evidence that *S. mutans* can be detected in mouths of predentate children before eruption of the first tooth.[241,411,413]

BACTERIA AND THEIR BIOFILM MODE OF LIVING

To many people, the word "microbiologist" conjures images of someone in a laboratory coat swilling a flask of bacterial cells that are growing happily as pure cultures in nutrient-rich broth. In nature, bacteria rarely find life so easy. The major struggle faced by bacteria lies in obtaining sufficient nutrients to support growth. Competition is rife, and most microbial communities contain many different species, sometimes a hundred or more, sharing the same site. Nutrients tend to concentrate at interfaces, and consequently the densest bacterial populations are located at interfaces of various kinds. In aquatic ecosystems, the major interfaces are the solid-liquid boundaries, such as the surfaces of submerged rocks or soil particles, and the air-water interface that is present in open systems, such as oceans or lakes. Bacteria grow well in the laboratory at the solid-gas interface on the surface of an agar plate. However, desiccation rapidly kills most bacterial cells, and microbial growth at solid-gas interfaces is therefore generally restricted to areas where moisture is available. The human body provides several interfaces that support microbial populations, of which the most important in healthy individuals are the skin, gut, mouth, and female urogenital tract. As mentioned earlier, the oral cavity is somewhat unique in this regard since it provides hard, nonshedding surfaces (teeth) that are accessible for microbial colonization.

The importance of surfaces for microbial growth was recognized as early as the 1920s, when a number of workers independently noted that bacteria growing on glass slides submerged in soil were different from those that could be cultured in broth.[195] However, it was not until around 50 years later that sessile microbial populations were considered to be sufficiently different from free-living microorganisms to merit their own name, and the term "biofilms" was coined (Figure 23-5 and Figure 23-6, *B* and *C*). Biofilms are composed of microbial cells encased within a matrix of extracellular polymeric substances (EPS) such as polysaccharides, proteins, and nucleic acids.

For more information on biofilm bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

Biofilms are heterogenous: variations in biofilm structure exist within individual biofilms and between different types of biofilms. However, a number of structural features that are common to many biofilms have been noted. For example, biofilms frequently contain microcolonies of bacterial cells. Water channels are commonly found in biofilms, and these can form a primitive circulatory system, removing waste products and bringing fresh nutrients to the deeper layers of the film. Surface structures, such as fronds, can dissipate the energy of fluid flowing over the biofilm. In mixed-species biofilms, there is often heterogeneity in the distribution of different species. Steep chemical gradients exist, such as oxygen or pH, and these produce distinct microenvironments within the biofilm.

Microbial populations on the surfaces of teeth (dental plaque) are excellent examples of biofilm communities (Figure 23-7). The architecture of a dental plaque biofilm has many features in common with other biofilms. It is heterogeneous in structure, with clear evidence of open fluid-filled channels running through the plaque mass.[64,65,428] (see Figure 23-5). Nutrients make contact with the sessile (attached) microcolonies by diffusion from the water channels to the microcolony, rather than from the matrix. The bacteria exist and proliferate within the intercellular matrix through which the channels run. The matrix confers a specialized environment, which distinguishes bacteria that exist within the biofilm from those that are free-floating, the so-called planktonic state in solutions such as saliva or crevicular fluid. The biofilm matrix functions as a barrier. Substances produced by bacteria within the biofilm are retained and concentrated, which fosters metabolic interactions among the different bacteria.

The intercellular matrix consists of organic and inorganic materials derived from saliva, gingival crevicular fluid (GCF), and bacterial products.

Organic constituents of the matrix include polysaccharides, proteins, glycoproteins, lipid material, and (perhaps) DNA. Albumin, probably originating from crevicular fluid, has been identified as a component of the plaque matrix. The lipid material consists of debris from the membranes of disrupted bacterial and host cells,

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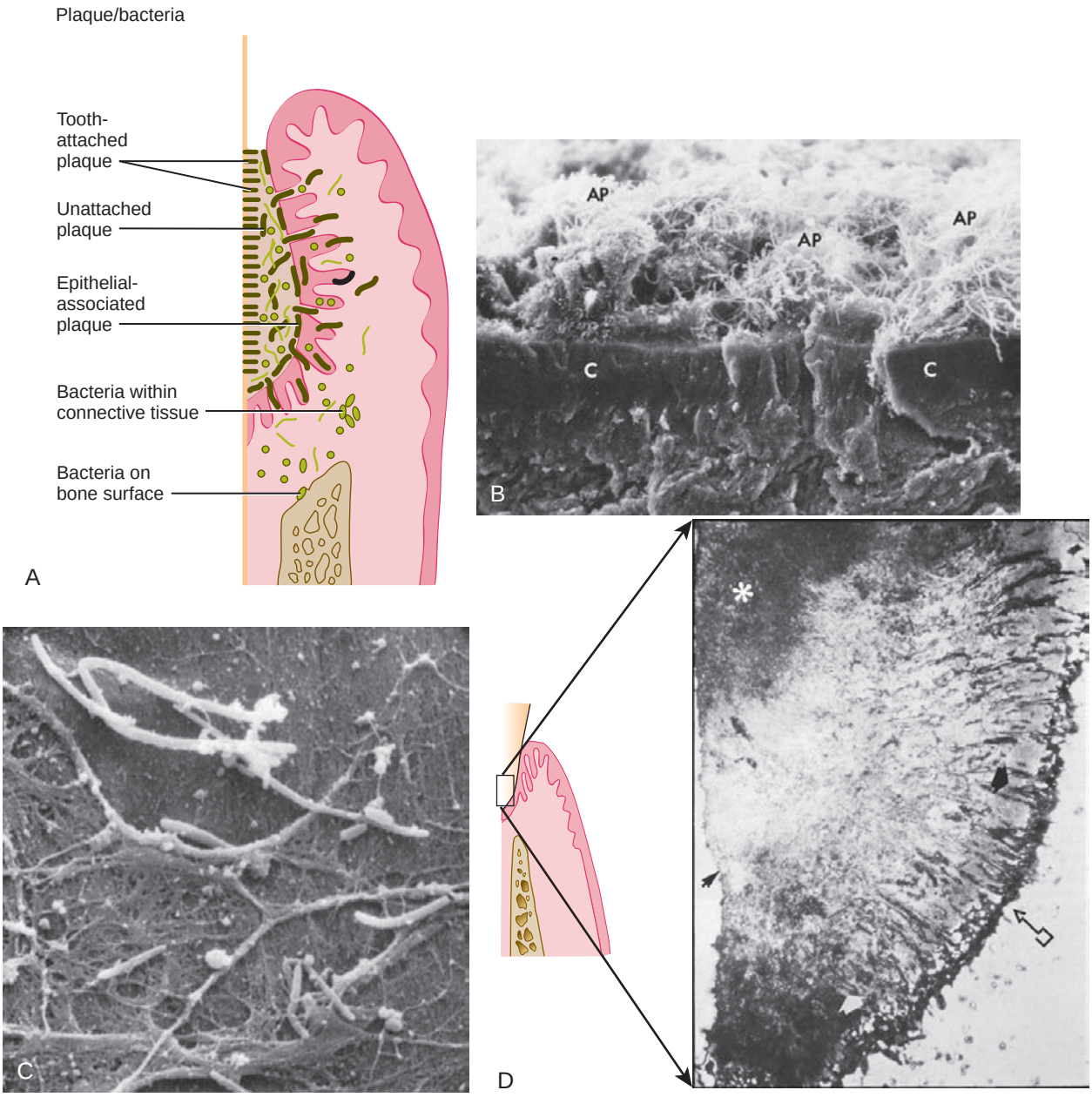

Figure 23-6 **A,** Diagram depicting the plaque-bacteria association with tooth surface and periodontal tissues. **B,** Scanning electron photomicrograph of cross-section of cementum *(C)* with attached subgingival plaque *(AP)*. Area shown is within a periodontal pocket. **C,** Scanning electron micrograph of cocci and filaments associated with surface of pocket epithelium in a case of marginal gingivitis. (Magnification ×3000.) **D,** *Left,* Diagrammatic representation of the histologic structure of subgingival plaque. *Right,* Histologic section of subgingival plaque. *Arrow with box,* Sulcular epithelium. *White arrow,* Predominantly gram-negative unattached zone. *Black arrow,* Tooth surface. *Asterisk,* Predominantly gram-positive attached zone. (**B** courtesy Dr. J. Sottosanti, La Jolla, CA.)

Figure 23-7 Clinical picture of 10-day-old supragingival plaque. The first signs of gingival inflammation *(arrows)* are becoming visible.

and possibly food debris. *Glycoproteins* from saliva are an important component of the pellicle that initially coats a clean tooth surface, but they also become incorporated into the developing plaque biofilm. Polysaccharides produced by bacteria also contribute to the organic portion of the matrix. They play a major role in maintaining the integrity of the biofilm.

The *inorganic components* of plaque are predominantly calcium and phosphorus, with trace amounts of other minerals such as sodium, potassium, and fluoride. The source of inorganic constituents of supragingival plaque is primarily saliva. As the mineral content increases, the plaque mass becomes calcified to form calculus (Figure 23-8). Calculus is frequently found in areas of the dentition adjacent to salivary ducts (e.g., the lingual surface of

the mandibular incisors and canines and the buccal surface of the maxillary first molars), reflecting the high concentration of minerals available from saliva in those regions. The inorganic components of subgingival plaque are derived from crevicular fluid (a serum transudate). Calcification of subgingival plaque also results in calculus formation (Figure 23-9). Subgingival calculus is typically dark green or dark brown, probably reflecting the presence of blood products associated with subgingival hemorrhage.

The importance of these biofilms for oral diseases, such as dental caries and periodontitis, together with the relative ease with which tooth surface biofilms can be accessed, has led to dental plaque becoming one of the most highly studied biofilm systems. It is hoped that by understanding the mechanisms involved in the accumulation of dental plaque and the transition from health to disease, it will be possible to improve our control over these processes and to further restrict plaque-associated oral diseases.

Figure 23-8 Supragingival calculus is depicted on the buccal surface of maxillary molars adjacent to the orifice for the parotid duct.

Figure 23-9 Dark-pigmented deposits of subgingival calculus are shown on the distal root of an extracted lower molar.

Structure of a Mature Dental Plaque Biofilm

Dental plaque (see Figure 23-7) is defined clinically as a structured, resilient yellow-grayish substance that adheres tenaciously to the intraoral hard surfaces, including removable and fixed restorations.[29] The tough extracellular matrix makes it impossible to remove plaque by rinsing or the use of sprays. Plaque can thus be differentiated from other deposits that may be found on the tooth surface such as materia alba and calculus. *Materia alba* refers to soft accumulations of bacteria, food matter, and tissue cells that lack the organized structure of dental plaque and are easily displaced with a water spray. *Calculus* is a hard deposit that forms by mineralization of dental plaque and is generally covered by a layer of unmineralized plaque (Table 23-3).

Dental plaque is composed primarily of microorganisms. One gram of plaque (wet weight) contains approximately 10^{11} bacteria.[339,360] The number of bacteria in supragingival plaque on a single tooth surface can exceed 10^9 cells. In a periodontal pocket, counts can range from 10^3 bacteria in a healthy crevice to $>10^8$ bacteria in a deep pocket. Using highly sensitive molecular techniques for microbial identification, it has been estimated that more than 500 distinct microbial phylotypes can be present as natural inhabitants of dental plaque.[1] In fact, this number may be much greater.

For more information on this topic, please visit the Expert CONSULT companion website at www.expertconsult.com.

Any individual may harbor 150 or more different species. Nonbacterial microorganisms that are found in plaque include archaea, yeasts, protozoa, and viruses.[62,203]

Dental plaque is broadly classified as supragingival or subgingival based on its position on the tooth surface toward the gingival margin.

- *Supragingival plaque* is found at or above the latter; when in direct contact with the gingival margin it is referred to as *marginal plaque.*
- *Subgingival plaque* is found below the gingival margin, between the tooth and the gingival pocket epithelium.

Supragingival plaque typically demonstrates a stratified organization of a multilayered accumulation of bacterial morphotypes (Figure 23-10). Gram-positive cocci and short rods predominate at the tooth surface, whereas gram-negative rods and filaments, as well as spirochetes, predominate in the outer surface of the mature plaque mass.

In general, the subgingival microbiota differs in composition from the supragingival plaque, primarily because of the local availability of blood products and a low reduction-oxidation (redox) potential, which characterizes the anaerobic environment.

TABLE 23-3 Differences Between Tooth Deposits

Materia Alba	Dental Plaque	Calculus
• White cheeselike accumulation • A soft accumulation of salivary proteins, some bacteria, many desquamated epithelial cells, and occasional disintegrating food debris • Lacks an organized structure and is therefore not as complex as dental plaque • Easily displaced with a water spray	• Resilient clear to yellow-grayish substance • Primarily composed of bacteria in a matrix of salivary glycoproteins and extracellular polysaccharides • Considered to be a biofilm • Impossible to remove by rinsing or the use of sprays	• Hard deposit that forms by mineralization of dental plaque • Generally covered by a layer of unmineralized dental plaque

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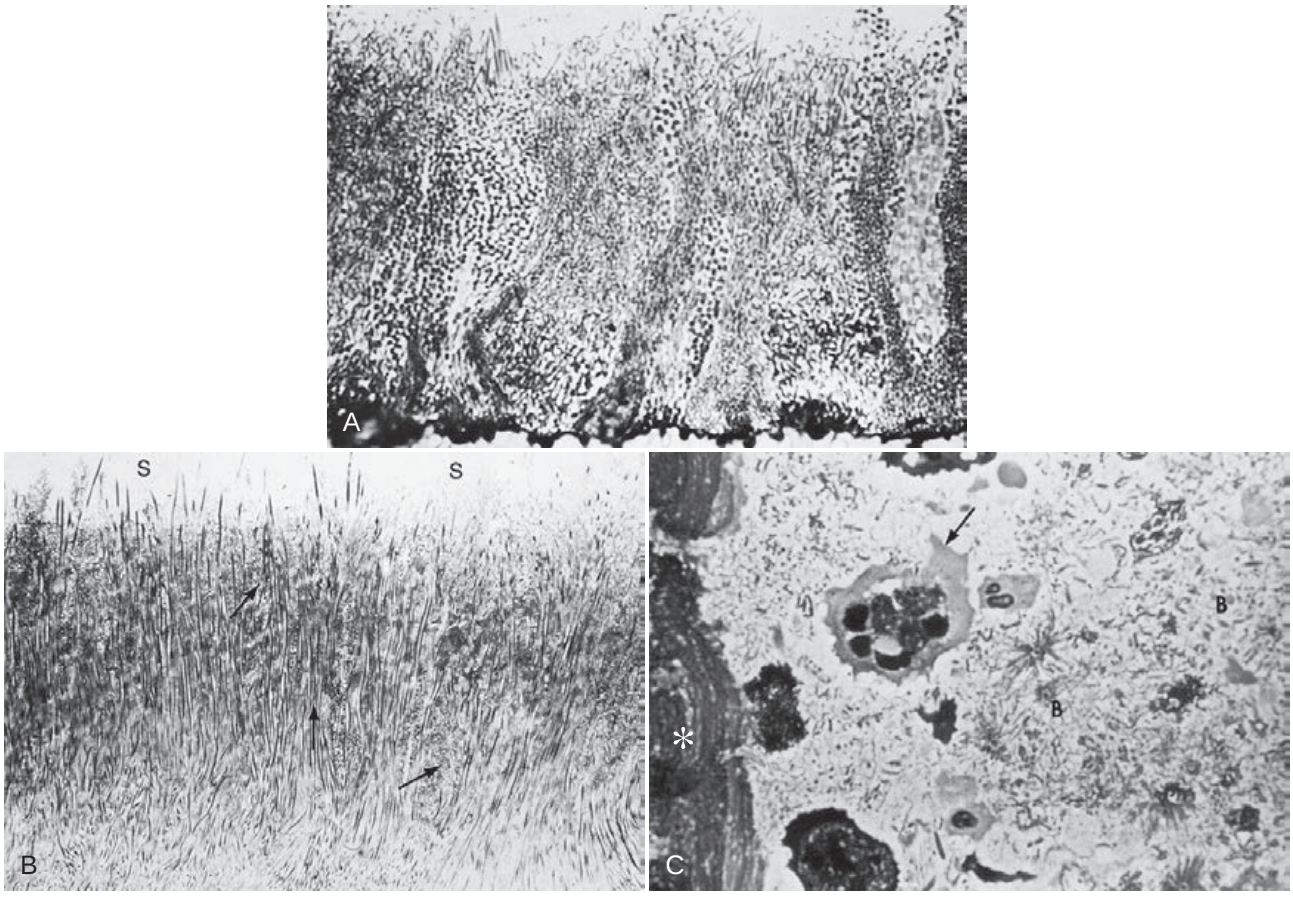

Figure 23-10 **A,** 1-day-old plaque. Microcolonies of plaque bacteria extend perpendicularly away from tooth surfaces. **B,** Developed supragingival plaque showing overall filamentous nature and microcolonies *(arrows)* extending perpendicularly away from tooth surface. Saliva-plaque interface shown *(S)*. **C,** Histologic section of plaque showing nonbacterial components such as white blood cells *(arrow)* and epithelial cells *(asterisk)*, interspersed among bacteria *(B)*. (Courtesy Dr. Max Listgarten, Philadelphia, PA.)

For more information on this topic and to view Figures 23-11 to 23-13, please visit the Expert CONSULT companion website at www.expertconsult.com.

The environmental parameters of the subgingival region differ from those of the supragingival region. The gingival crevice or pocket is bathed by the flow of crevicular fluid, which contains many substances which bacteria may use as nutrients. Host inflammatory cells and mediators are likely to have considerable influence on the establishment and growth of bacteria in the subgingival region. Both morphologic and microbiologic studies of subgingival plaque reveal distinctions between the tooth-associated and soft tissue-associated regions of subgingival plaque (see Figure 23-6, *A* to *C*).[209,253]

The tooth-associated cervical plaque, adhering to the root cementum, does not markedly differ from that observed in gingivitis. At this location, filamentous microorganisms dominate, but cocci and rods also occur. This plaque is dominated by gram-positive rods and cocci, including *S. mitis*, *S. sanguinis*, *Actinomyces oris* (a new species containing strains that were formerly classified as *A. naeslundii*), *A. naeslundii*, and *Eubacterium* spp. (see Figure 23-1, *N*). However, in the deeper parts of the pocket, the filamentous organisms become fewer in numbers, and in the apical portion they seem to be virtually absent. Instead, the microbiota is dominated by smaller organisms without a particular orientation.[209] The apical border of the plaque mass is separated from the junctional epithelium by a layer of host leukocytes, and the bacterial population of this apical tooth-associated region shows an increased concentration of gram-negative rods (see Figure 23-6, *D*).

The layers of microorganisms facing the soft tissue lack a definite intermicrobial matrix and contain primarily gram-negative rods and cocci, as well as large numbers of filaments, flagellated rods, and spirochetes. Studies on plaque associated with crevicular epithelial cells indicate a predominance of species such as *S. oralis*, *S. intermedius*, *Parvimonas micra* (formerly *Micromonas micra* and *Peptostreptococcus micros*), *P. gingivalis*, *Prevotella intermedia*, *Tannerella forsythia* (see Figure 23-10), and *F. nucleatum*.[76,79] Host tissue cells (e.g., white blood cells and epithelial cells) may also be found in this region (see Figure 23-10, *C*). Bacteria are also found within the host tissues, such as in the soft tissues (see Figure 23-3), and within epithelial cells (Figure 23-14), as well as in the dentinal tubules (Figure 23-15).[326,327]

The composition of the subgingival plaque depends on the pocket depth. The apical part is more dominated by spirochetes, cocci and rods, whereas in the coronal part more filaments are observed.

The site specificity of plaque is significantly associated with diseases of the periodontium. Marginal plaque, for example, is of prime importance in the initiation and development of gingivitis. Supragingival plaque and tooth-associated subgingival plaque are critical in calculus formation and root caries, whereas tissue-associated subgingival plaque is important in the tissue destruction that characterizes different forms of periodontitis. Biofilms also

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Figure 23-14 **A** to **C** show images of z-section No. 39 from a stack of 74 0.2-µm z-sections (magnification ×600; the scale bar in **A** also applies to **B** and **C**). Buccal epithelial cells (BEC) in this field were double-labeled with the *EUB338* universal probe **(A)** and the *A. actinomycetemcomitans*-specific probe **(B).** The cell in the center of **A** contained a large mass of brightly fluorescent intracellular bacteria *(red arrow)*. Other cells in the field contained smaller bacterial masses (not marked). **B** shows that a portion of the large mass labeled with the universal probe also hybridized with the *A. actinomycetemcomitans*-specific probe *(green arrow)*. Images from **A** and **B** were superimposed **(C),** confirming that bacteria labeled with both probes *(yellow arrow)* were adjacent to other bacteria labeled only with the universal probe *(red arrow)*. **D** presents a three-dimensional reconstruction of the same field. Bacteria recognized only by the universal probe are shown in solid red, whereas colocalization of the *A. actinomycetemcomitans* and universal probes is depicted by a green wireframe over a red interior. Reconstructed BEC surfaces are presented in blue. The red and green colors are muted when bacterial masses are intracellular, and brighter when bacteria appear to project out of the surface. The angle of view was rotated along the z-axis, and the image was zoomed. The large mass that appeared to have a lobular structure in z-section No. 39 was seen to be a cohesive unit containing *A. actinomycetemcomitans* in direct proximity to other species *(red and green arrows)*. (From Rudney JD, Chen R, Sedgewick GJ: *J Dent Res* 84:59-63, 2005.)

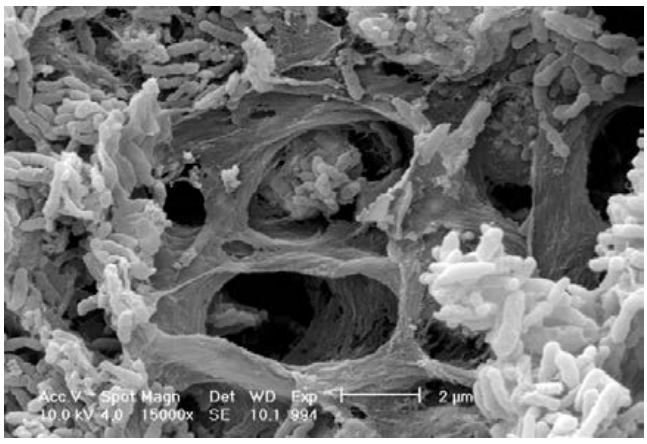

Figure 23-15 Scanning electron micrograph of bacteria within dentinal tubules.

form on artificial surfaces exposed to the oral environment such as prostheses and implants. A large series of papers compared the microbiota in pockets around teeth with those around implants of partially edentulous patients. The similarities were striking.*

Accumulation of a Dental Plaque Biofilm

The process of plaque formation can be divided into several phases: (1) the formation of the pellicle on the tooth surface, (2) initial adhesion/attachment of bacteria, and (3) colonization/plaque maturation.

*References 13, 149, 150, 167, 169, 198, 202, 235, 246, 267, 297, and 333.

Formation of the Pellicle. All surfaces in the oral cavity, including hard and soft tissues, are coated with a layer of organic material known as the *acquired pellicle*. The pellicle on tooth surfaces consists of more than 180 peptides, proteins, and glycoproteins, including keratins, mucins, proline-rich proteins, phosphoproteins (e.g., statherin), histidine-rich proteins, and other molecules that can function as adhesion sites (receptors) for bacteria.[346,347,432] Salivary pellicle can be detected on clean enamel surfaces within 1 minute after introduction into the mouths of volunteers.[134] By 2 hours, the pellicle is essentially in equilibrium between adsorption and detachment, although further pellicle maturation can be observed for several hours.

For more information on formation of the pellicle, please visit the Expert CONSULT companion website at www.expertconsult.com.

Consequently, bacteria that adhere to tooth surfaces do not contact the enamel directly but interact with the acquired enamel pellicle. However, the pellicle is not merely a passive adhesion matrix. Many proteins retain enzymatic activity when incorporated into the pellicle, and some of these, such as peroxidases, lysozyme, and α-amylase, may affect the physiology and metabolism of adhering bacterial cells.[129,131,132,133]

For more information on formation of the pellicle, please visit the Expert CONSULT companion website at www.expertconsult.com.

Initial Adhesion/Attachment of Bacteria. Toothbrushing removes most but not all bacteria from the exposed

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surfaces of teeth.[394] However, recolonization begins immediately, and bacteria can be detected within 3 minutes of introducing sterile enamel into the mouth.[130]

The initial steps of transport and interaction with the surface are essentially nonspecific (i.e., they are the same for all bacteria). The proteins and carbohydrates that are exposed on the bacterial cell surface become important once the bacteria are in loose contact with the acquired enamel pellicle. It is the specific interactions between microbial cell surface "adhesin" molecules and receptors in the salivary pellicle that determine whether a bacterial cell will remain associated with the surface. Only a relatively small proportion of oral bacteria possess adhesins that interact with receptors in the host pellicle, and these organisms are generally the most abundant bacteria in biofilms on tooth enamel shortly after cleaning. Over the first 4 to 8 hours, 60% to 80% of bacteria present are members of the genus *Streptococcus*.[77,258] Other bacteria commonly present at this time include species that cannot survive without oxygen (obligate aerobes), such as *Haemophilus* spp. and *Neisseria* spp., as well as organisms that can grow in the presence or absence of oxygen (facultative anaerobes) including *Actinomyces* spp. and *Veillonella* spp.[1,74] These species are considered the "primary colonizers" of tooth surfaces. The primary colonizers provide new binding sites for adhesion by other oral bacteria. The metabolic activity of the primary colonizers modifies the local microenvironment in ways that can influence the ability of other bacteria to survive in the dental plaque biofilm. For example, by removing oxygen, the primary colonizers provide conditions of low oxygen tension that permit the survival and growth of obligate anaerobes.

For more information on the initial adhesion/attachment of bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

Colonization and Plaque Maturation. The primary colonizing bacteria (Table 23-4) adhered to the tooth surface provide new receptors for attachment by other bacteria, in a process known as "coadhesion."[175] Together with growth of adherent microorganisms, coadhesion leads to the development of microcolonies (Figure 23-16) and eventually to a mature biofilm.

TABLE 23-4 Overview of Primary and Secondary Colonizers in Dental Plaque

Primary colonizers	*Streptococcus gordonii*
	Streptococcus intermedius
	Streptococcus mitis
	Streptococcus oralis
	Streptococcus sanguinis
	Actinomyces gerencseria
	Actinomyces israelii
	Actinomyces naeslundii
	Actinomyces oris
	Aggregatibacter actinomycetemcomitans serotype a
	Capnocytophaga gingivalis
	Capnocytophaga ochracea
	Capnocytophaga sputigena
	Eikenella corrodens
	Actinomyces odontolyticus
	Veillonella parvula
Secondary colonizers	*Campylobacter gracilis*
	Campylobacter rectus
	Campylobacter showae
	Eubacterium nodatum
	Aggregatibacter actinomycetemcomitans serotype b
	Fusobacterium nucleatum ssp *nucleatum*
	Fusobacterium nucleatum ssp *vincentii*
	Fusobacterium nucleatum ssp *polymorphum*
	Fusobacterium periodonticum
	Parvimonas micra
	Prevotella intermedia
	Prevotella loescheii
	Prevotella nigrescens
	Streptococcus constellatus
	Tannerella forsythia
	Porphyromonas gingivalis
	Treponema denticola

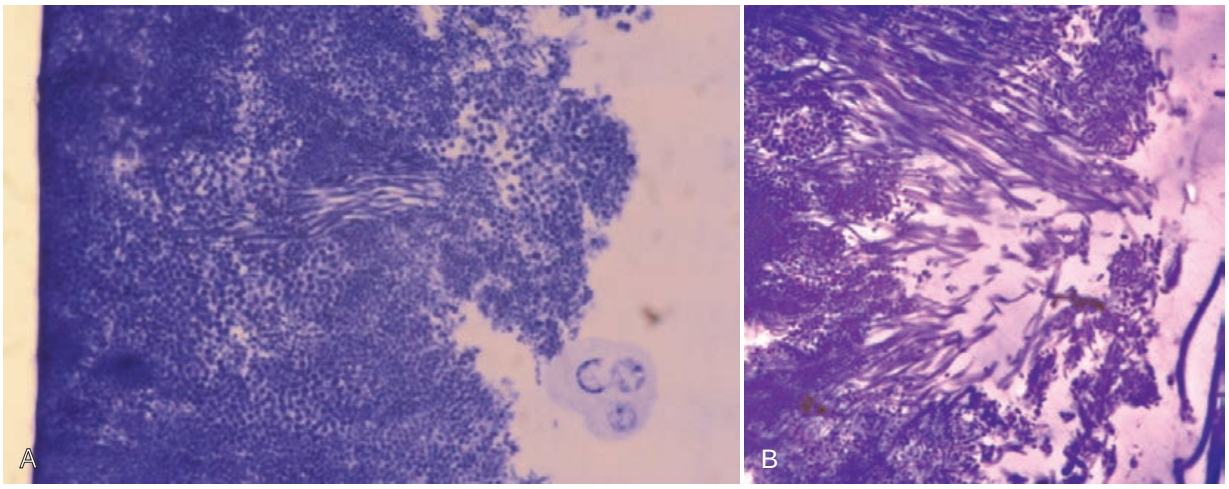

Figure 23-16 When a single microorganism adheres to the tooth surface, it can start to multiply and slowly forms a microcolony of daughter cells. These views were taken after plaque formation on a plastic strip (e.g., as shown in Figure 23-26), glued to a tooth surface.

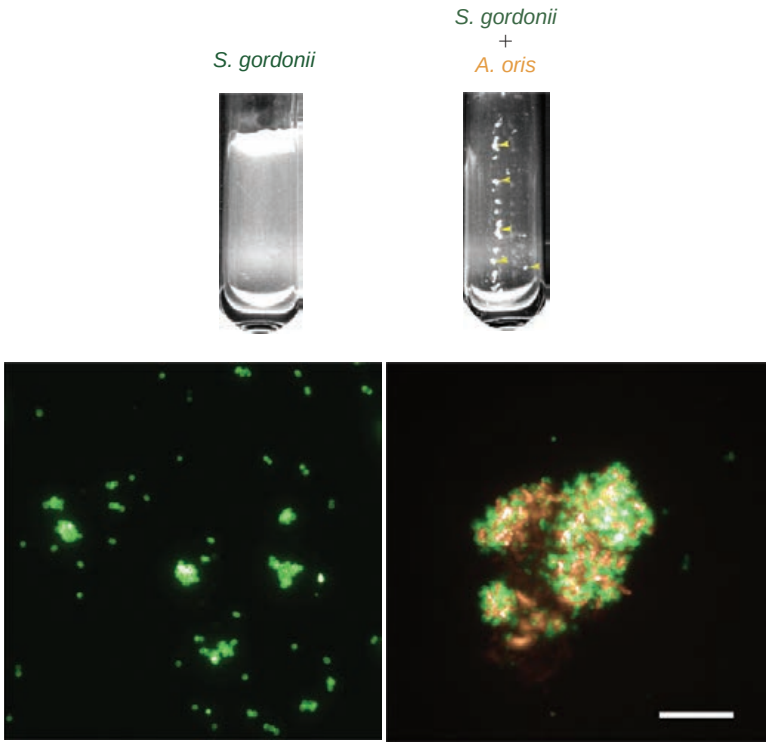

Figure 23-17 Coaggregation between *Streptococcus gordonii* DL1 and *Actinomyces oris* MG1 in vitro. A monoculture of *S. gordonii* appears uniformly turbid. Microscopically, cells labeled with specific anti-DL1 antibodies *(green)* are in small chains or clumps. Following addition of *A. oris*, cells clump together to form macroscopic coaggregates *(yellow arrows)*. Under the microscope, *S. gordonii (green)* are evenly distributed throughout the coaggregates with *A. oris (orange)*. Bar = 20 µm. (Image reproduced in part from Jakubovics NS, Gill SR, Iobst SE, et al: *J Bacteriol* 190:3646, 2008.)

For more information on colonization and plaque maturation, please visit the Expert CONSULT companion website at www.expertconsult.com.

In the laboratory, interactions between genetically distinct cells in suspension result in clumps, or "coaggregates," that can easily be seen with the naked eye (Figure 23-17).

For more information on colonization and plaque maturation, please visit the Expert CONSULT companion website at www.expertconsult.com.

Different species, or even different strains of a single species, have distinct sets of coaggregation partners (see Figure 23-18 online). Fusobacteria coaggregate with all other human oral bacteria while *Veillonella* spp., *Capnocytophaga* spp. (see Figure 23-1, *Q*) and *Prevotella* spp. bind to streptococci and/or actinomyces.[174,176,421] Each newly accreted cell becomes itself a new surface and therefore may act as a coaggregation bridge to the next potentially accreting cell type that passes by.

For more information on colonization and plaque maturation, please visit the Expert CONSULT companion website at www.expertconsult.com.

Well-characterized interactions of secondary colonizers (see Table 23-4) with early colonizers include the coaggregation of *F. nucleatum* with *S. sanguinis*, *Prevotella loescheii* with *A. oris*, and *Capnocytophaga ochracea* with *A. oris*.[162,164,416,417,418] Streptococci show intrageneric coaggregation, allowing them to bind to the nascent monolayer of already bound streptococci.[148,172,259,349]

Secondary colonizers (see Table 23-4), such as *Prevotella intermedia*, *Prevotella loescheii*, *Capnocytophaga* spp., *F. nucleatum*, and *P. gingivalis* do not initially colonize clean tooth surfaces but adhere to bacteria already in the plaque mass.[174] The transition from early, supragingival dental plaque to mature plaque growing below the gingival margin involves a shift in the microbial population from primarily gram-positive organisms to high numbers of gram-negative bacteria. Therefore, in the later stages of plaque formation, coaggregation between different gram-negative species is likely to predominate. Examples of these types of interactions are the coaggregation of *F. nucleatum* with *P. gingivalis* or *Treponema denticola* (see Figure 23-1, *L*).[166,171,176]

For more information on colonization and plaque maturation, please visit the Expert CONSULT companion website at www.expertconsult.com.

Factors Affecting Supragingival Dental Plaque Formation

Clinically, early undisturbed plaque formation on teeth follows an exponential growth curve when measured planimetrically.[298] During the first 24 hours starting from a clean tooth surface, plaque growth is negligible from a clinical viewpoint (<3% coverage of the vestibular tooth surface, which is an amount nearly undetectable clinically). This "lag time" is due to the fact that the microbial population must reach a certain size before it can be easily detected by the clinician. During the following 3 days, coverage progresses rapidly to the point where, after 4 days, on average 30% of the total coronal tooth area will be covered with plaque (see Video 23-1: Plaque Growth online).

For more information, and to view Figures 23-19 and 23-20, and Video 23-1, please visit the Expert CONSULT companion website at www.expertconsult.com.

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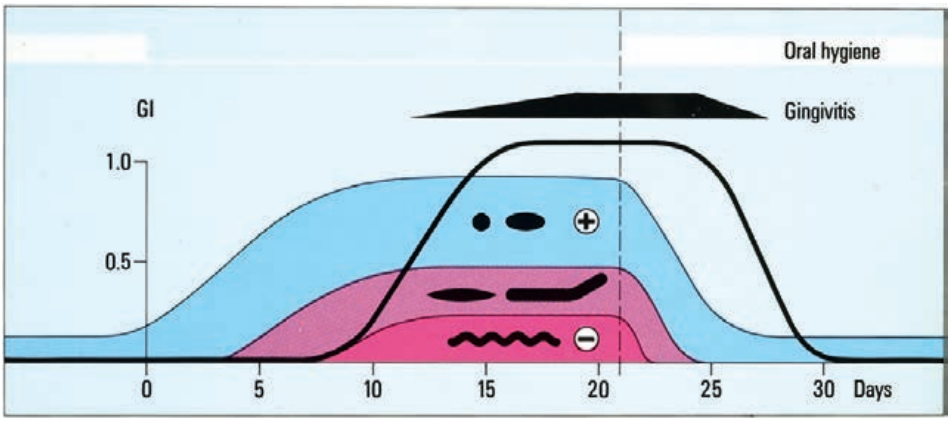

Figure 23-21 Artistic impression of the 1965 published classic experimental proof of the bacterial etiology of gingivitis using the experimental gingivitis model. In plaque-free subjects with clinically non-inflamed gingiva, plaque will slowly develop on the teeth when all mechanical plaque control is stopped. With time, the plaque composition changes. During the first few days, the plaque is mainly composed of gram positive (+) cocci and rods. Later on, the composition is shifted toward more gram-negative species, more rods, filaments, and finally gram-negative (–) spirochetes appear (see morphotypes in black). Within a few days, a mild gingivitis ensues (black line, gingival index according to Löe & Silness, GI=1). From the moment that proper plaque control is re-established (vertical line), the plaque composition returns to the initial situation and the symptoms of gingivitis disappear. *(From Loe H, Theilade E, Jensen SB: Experimental gingivitis in man,* J Periodontol *36:177, 1965.)*

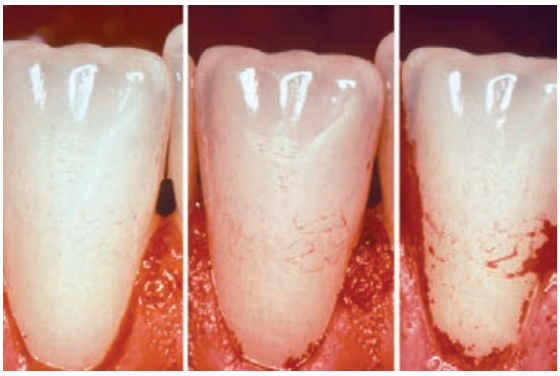

Figure 23-22 Clinical illustration of the typical topography of plaque growth. Initial growth starts along the gingival margins and from the interdental spaces (areas protected against shear forces), to further extend in a coronal direction. This pattern may fundamentally change, for example if the tooth surface contains irregularities (such as those evident in Figure 23-23).

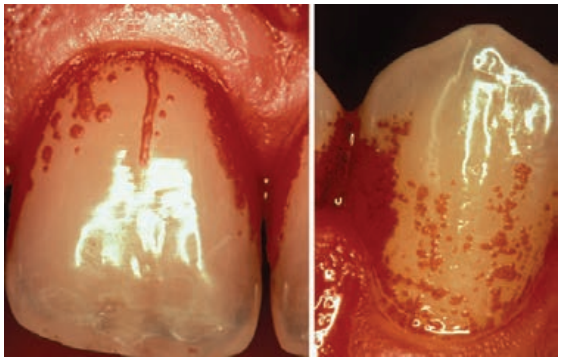

Figure 23-23 Important surface irregularities (crack on central upper incisor, several small pits on canine) are also responsible for the so-called individualized plaque growth pattern.

Several reports have shown that the microbial composition of the dental plaque will change with a shift toward a more anaerobic and a more gram-negative flora, including an influx of fusobacteria, filaments, spiral forms, and spirochetes (Figure 23-21). This was beautifully illustrated in experimental gingivitis studies.[375,387] In this ecologic shift within the biofilm, there is a transition from the early aerobic environment characterized by gram-positive facultative species to a highly oxygen-deprived environment in which gram-negative anaerobic microorganisms predominate. Bacterial growth in older plaque is much slower than in newly formed dental plaque, presumably because nutrients become limiting for much of the plaque biomass.[415]

Topography of Supragingival Plaque. Early plaque formation on teeth follows a typical topographic pattern (Figure 23-22), with initial growth along the gingival margin and from the interdental space (areas protected against shear forces). Later, a further extension in the coronal direction can be observed.[239,302] This pattern may fundamentally change when the tooth surface contains irregularities that offer a favorable growth path (Figure 23-23). Plaque formation can also start from grooves, cracks, perikymata, or pits.

For more information on topography of supragingival plaque, please visit the Expert CONSULT companion website at www.expertconsult.com.

By multiplication, the bacteria subsequently spread out from these starting up areas as a relatively even monolayer. Surface irregularities are also responsible for the so-called "individualized" plaque growth pattern (see Figure 23-23), which is reproduced in the absence of optimal oral hygiene.[239,240] This phenomenon illustrates the importance of surface roughness in plaque growth, which should lead to proper clinical treatment options.

Surface Microroughness. Rough intraoral surfaces (e.g., crown margins, implant abutments, and denture bases) accumulate and retain more plaque and calculus in terms of thickness, area, and colony-forming units.[289] Ample plaque also reveals an increased maturity/pathogenicity of its bacterial components, characterized by an increased proportion of motile organisms and spirochetes, and/or a denser packing of them (Figures 23-24 and 23-25).

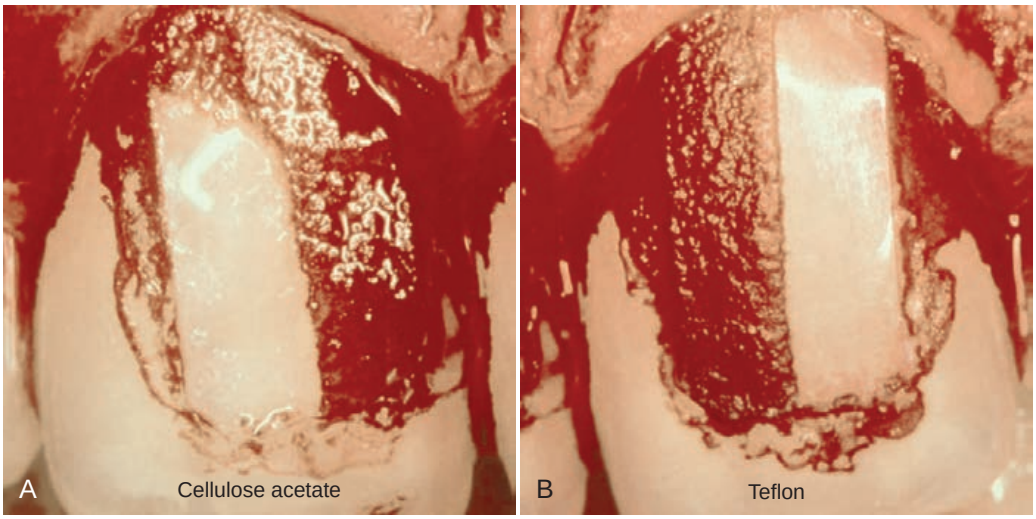

Figure 23-24 Photographs showing the clinical impact of surface roughness and surface free energy on de novo plaque formation. Two small strips were glued to the central upper incisors of a patient who refrained from oral hygiene for 3 days. Each strip was divided in two halves, a rough region (R_a 2.0 µm) located mesially, and a smooth region (R_a 0.1 µm) distally located. The left strip was cellulose acetate (medium surface free energy [sfe]: 58 $ergcm^{-2}$) and the right strip Teflon (low sfe: 20 $ergcm^{-2}$). Plaque was disclosed with 0.5% neutral red solution. The smooth regions show the decrease in biofilm formation because of the low sfe; the rough regions demonstrate the predominance of surface roughness (i.e., more plaque with no difference between the two surfaces), even with different sfe. (From Quirynen M, Listgarten MA: *Clin Oral Implants Res* 1:8, 1990.)

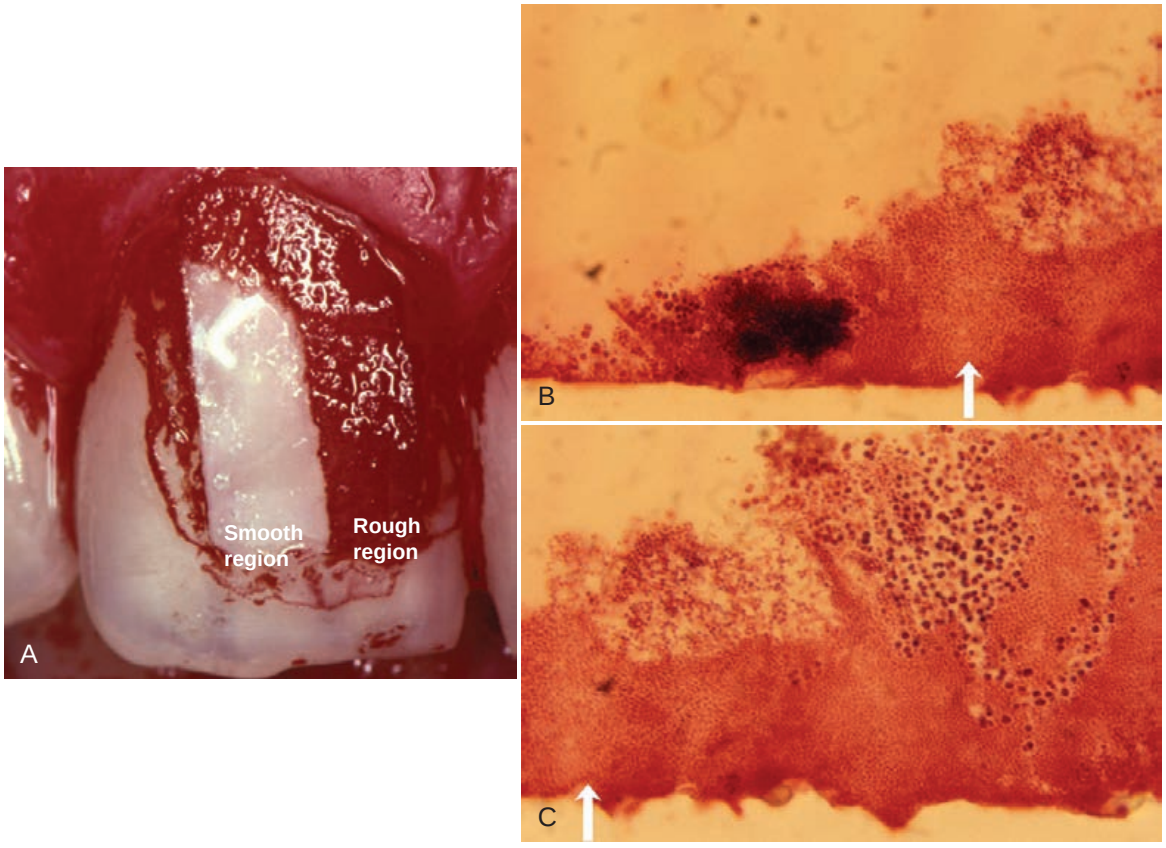

Figure 23-25 **A,** Small plastic strip, divided in half (a rough region, R_a 2.0 µm located mesially and a smooth region R_a 0.1 µm, distally located), had been glued to the central upper incisors of a patient who refrained from oral hygiene for 3 days. **B** and **C,** After removal, the strip had been cut in small slices for microscopic evaluation. It is obvious that the rough part **(C)** contains a thicker plaque layer than the smooth part **(B).** *Arrow* shows border between rough and smooth surface.

Smoothing an intraoral surface decreases the rate of plaque formation. Below a certain surface roughness (R_a < 0.2 µm), however, further smoothing does not result in an additional reduction in plaque formation.[27,291] There seems to be a threshold level for surface roughness (R_a around 0.2 µm), above which bacterial adhesion will be facilitated.[26] Although surface free energy and surface roughness are two factors influencing plaque growth, the latter predominates (see Figure 23-24 and 23-25).

Individual Variables Influencing Plaque Formation. The rate of plaque formation differs significantly between subjects, differences that might overrule surface characteristics. A distinction is often made between "heavy" (fast) and "light" (slow) plaque formers (see Video 23-2: Difference in Plaque Growth between Heavy and Light Plaque Former online).

To view Video 23-2, please visit the Expert CONSULT companion website at www.expertconsult.com.

For more information on variables influencing plaque formation, please visit the Expert CONSULT companion website at www.expertconsult.com.

A multiple regression analysis showed that the clinical wettability of the tooth surfaces, the saliva-induced aggregation of oral bacteria, and the relative salivary flow conditions around the sampled teeth explained 90% of the variation. Moreover, the saliva from light plaque formers reduced the colloidal stability of bacterial suspensions of, for example, *S. sanguinis*.[344]

For more information on variables influencing plaque formation, please visit the Expert CONSULT companion website at www.expertconsult.com.

Variation within the Dentition. Within a dental arch, large differences in plaque growth rate can be detected. In general, early plaque formation occurs faster: in the lower jaw (when compared to the upper jaw); in molar areas; on the buccal tooth surfaces, when compared to palatal sites (especially in the upper jaw); and in the interdental regions when compared to the buccal or lingual surfaces.[102,194,288]

Impact of Gingival Inflammation and Saliva. Several studies clearly indicate that early in vivo plaque formation is more rapid on tooth surfaces facing inflamed gingival margins than on those adjacent to healthy gingivae.[294,306,307] These studies suggest that the increase in crevicular fluid production enhances plaque formation. Probably, some substance(s) from this exudate (e.g., minerals, proteins, or carbohydrates) favor both the initial adhesion and/or the growth of the early colonizing bacteria. Additionally, it is known that during the night, plaque growth rate is reduced by some 50%.[302] This seems surprising, since one would expect that reduced plaque removal and the decreased salivary flow at night would enhance plaque growth. The fact that the supragingival plaque obtains its nutrients mainly from the saliva appears to be of greater significance than the antibacterial activity of saliva.[47]

The Impact of Patient's Age. Although older studies were contradictory, more recent papers clearly indicate that a subject's age does not influence de novo plaque formation. In a study by Fransson et al.[97] no differences could be detected in de novo plaque formation between a group of young (20 to 25 years of age) and older (65 to 80 years of age) subjects who abolished mechanical tooth cleaning measures for 21 days, neither in amount nor in composition.[97]

For more information on the impact of patient's age, please visit the Expert CONSULT companion website at www.expertconsult.com.

The developed plaque in the older patient group resulted, however, in a more severe gingival inflammation, which seems to indicate an increased susceptibility to gingivitis with aging.

Spontaneous Tooth Cleaning. Many clinicians still believe that plaque is removed spontaneously from the teeth such as during eating. However, based on the firm attachment between bacteria and surface, this seems unlikely. Even in the occlusal surfaces of the molars, plaque remains, even after chewing fibrous food (carrots, apples, or chips). The inefficiency of the spontaneous plaque removal is neatly illustrated by the clinical pictures in Figure 23-26, taken before and after dinner, starting from 4 days of undisturbed plaque formation. Only negligible differences in plaque extent could be observed.

De Novo Subgingival Plaque Formation

It is technically impossible to record the dynamics of subgingival plaque formation in an established dentition for the simple reason that one cannot sterilize a periodontal pocket. Some early studies, using culturing techniques, examined the changes within the subgingival microbiota during the first week after mechanical debridement and reported an only partial reduction around 3 logs (from

Figure 23-26 Lower premolars and molars from a dental student who refrained from oral hygiene for 100 hours to evaluate undisturbed plaque formation. **A** is before dinner, and **B** is after dinner, eating fibrous food. Nearly no reduction in plaque extension could be observed, illustrating the absence of spontaneous plaque removal.

10^8 bacterial cells to 10^5 cells), followed by a rapid regrowth towards nearly pretreatment levels (–0.5 log) within 7 days.[111,136,229] The fast recolonization was explained by several factors. A critical review of the effectiveness of subgingival debridement, for instance, revealed that a high proportion of treated tooth surfaces (5% to 80%) still harbored plaque and/or calculus after scaling. These remaining bacteria were considered the primary source for the subgingival recolonization.[276] Some pathogens penetrate the soft tissues or the dentinal tubules and eventually escape instrumentation (see Figure 23-15).[4,108,320] In a beagle dog study, Leknes et al studied the extent of subgingival colonization in 6 mm pockets with smooth or rough root surfaces.[201] They also observed that smooth surfaces harbored significantly less plaque and concluded that subgingival irregularities shelter submerged microorganisms. Moreover, biopsies of the soft tissues showed an elevated proportion of inflammatory cells in the junctional epithelium (and the underlying connective tissue) facing the rough surfaces.[200]

For more information on de novo subgingival plaque formation, please visit the Expert CONSULT companion website at www.expertconsult.com.

The introduction of oral implants, especially of the two-stage type, provides a new experimental set-up. When the transmucosal part of the implant (the abutment) is inserted on top of the osseointegrated endosseous part, a new "pristine" surface is created on which the intraoral translocation of bacteria can be investigated.[291] Recent papers demonstrated that a complex subgingival microbiota, including most periodontopathogens, is established within 1 week after abutment insertion. This is followed by a slow increase in the number of periodontopathogens.[101,303,304] Oral implants have also been used as a model to study the impact of surface roughness on subgingival plaque formation.* Smooth abutments (R_a < 0.2 µm) were found to harbor 25 times fewer bacteria than rough ones, with a slightly higher density of coccoid (i.e., nonpathogenic) cells. The subgingival microbiota was also largely dependent on the remaining presence of teeth and the degree of periodontitis in the remaining natural dentition (for review see Quirynen et al[293]). These observations highlight the importance of intraoral bacterial translocation for subgingival biofilms.

CHARACTERISTICS OF BIOFILM BACTERIA (LIFE IN "SLIME CITY")

Metabolism of Dental Plaque Bacteria

The majority of nutrients for dental plaque bacteria originate from saliva or GCF, although the host diet provides an occasional but nevertheless important food supply. The transition from gram-positive to gram-negative microorganisms observed in the structural development of dental plaque is paralleled by a physiologic transition in the developing plaque.

For more information on the metabolism of dental plaque bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

*References 27, 42, 118, 291, 301, and 316.

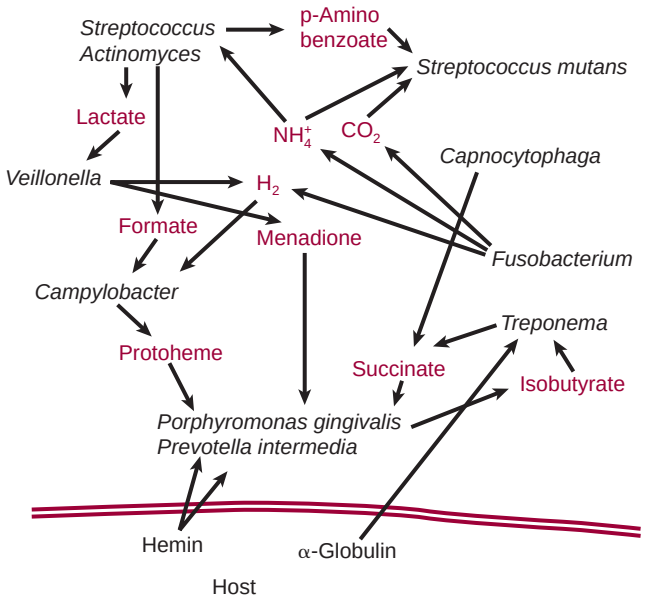

Figure 23-27 Schematic illustration of metabolic interactions among different bacterial species found in plaque, and between the host and plaque bacteria. These interactions are likely to be important to the survival of bacteria in the periodontal environment. (Based on Carlsson J: Microbiology of plaque associated periodontal disease. In Lindhe J, ed: *Textbook of clinical periodontology,* Munksgaard, 1983; Munksgaard International Publishers; Grenier D: Infect Immun 60:5298, 1992; Loesche WJ: *Periodontics* 6:245, 1968; Walden WC, Hentges DJ: *Appl Microbiol* 30:781, 1975.)

Laboratory studies have demonstrated many metabolic interactions among the different bacteria found in dental plaque (Figure 23-27).

For more information on the metabalism of dental plaque bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

The growth of *P. gingivalis* is enhanced by metabolic byproducts produced by other microorganisms, such as succinate from *C. ochracea* and protoheme from *Campylobacter rectus* (see Figure 23-1, *R*).[113,114,233] Overall, the total plaque population is more efficient than any one constituent organism at releasing energy from the available substrates.[422]

Metabolic interactions occur also between the host and plaque microorganisms.

For more information on the metabalism of dental plaque bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

Increases in steroid hormones are associated with significant increases in the proportions of *P. intermedia* found in subgingival plaque.[181] These nutritional interdependencies are probably critical to the growth and survival of microorganisms in dental plaque and may partly explain the evolution of highly specific structural interactions observed among bacteria in plaque.

Communication Between Biofilm Bacteria

Bacterial cells do not exist in isolation. In a biofilm, bacteria have the capacity to communicate with each other. One example of this

is quorum sensing, in which bacteria secrete a signaling molecule that accumulates in the local environment and triggers a response such as a change in the expression of specific genes once they reach a critical threshold concentration. The threshold concentration is reached only at a high-cell density, and therefore bacteria sense that the population has reached a critical mass, or quorum. There is some evidence that intercellular communication can occur after cell-cell contact and in this case, may not involve secreted signaling molecules.[155] Two types of signalling molecules have been detected from dental plaque bacteria: peptides released by gram-positive organisms during growth and a "universal" signal molecule autoinducer 2 (AI-2). Peptide signals are produced by oral streptococci and are recognized by cells of the same strain that produced them. Responses are induced only when a threshold concentration of the peptide is attained, and thus the peptides act as cell density, or quorum, sensors. Local concentrations of signalling molecules may be enhanced in biofilms if the signals become trapped in the biofilm matrix. The streptococcal peptides are known as *competence-stimulating peptides,* since the major response to these signals is the induction of competence, a physiologic state whereby cells are primed for DNA uptake and incorporation.

For more information on communication between biofilm bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

In contrast to the strain-specific competence-stimulating peptides, AI-2 is produced and detected by many different bacteria. Detection of AI-2 produces wide-ranging changes in gene expression, in some cases affecting up to one-third of the entire genome.[376]

For more information on communication between biofilm bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

Quorum sensing therefore appears to play diverse roles in, for example, modulating the expression of genes for antibiotic resistance, encouraging the growth of beneficial species to the biofilm, and discouraging the growth of competitors.

Interactions between Dental Plaque Bacteria

For more information on interactions between dental plaque bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

There is some evidence from laboratory studies that nonpathogenic organisms in subgingival dental plaque can modify the behavior of periodontal pathogens.

For more information on interactions between dental plaque bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

Using a proteomics approach to probe the phenotype of *P. gingivalis*, it has been shown that the expression of almost 500 *P. gingivalis* proteins is changed in model oral microbial communities containing *S. gordonii* and *F. nucleatum*.[186] At present, it is not clear how these changes affect the interaction between *P. gingivalis* and the host.

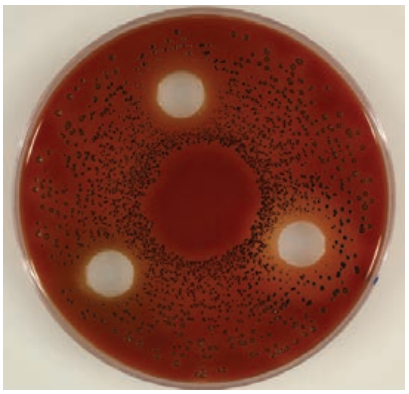

Figure 23-28 The growth of *Prevotella intermedia (black colonies)* is inhibited by the presence of *S. mitis, S. salivarius,* and *S. sanguinis (in the white holes)*. This is represented by a zone of no growth *(halo)* around the white holes. This is a typical example of growth inhibition induced by streptococci.

In multispecies biofilms in which many bacteria are juxtaposed to cells of different species, interactions between genetically distinct microorganisms can be mutually beneficial (see Figure 23-4, *A*). However, there are many examples of competitive interactions between different bacteria (Figure 23-28).

For more information on interactions between dental plaque bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

In fact, *S. oligofermentans* can convert lactic acid produced by *S. mutans* into H_2O_2, which then kills the *S. mutans* cells.[390]

For more information on interactions between dental plaque bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

Another example of competitive interaction exists between streptococci and periodontopathogens. *S. sanguinis*, *S. salivarius,* and *S. mitis* have been shown to inhibit hard and soft tissue colonization of *A. actinomycetemcomitans* (see Video 23-3: Colonization Inhibition online), *P. gingivalis,* and *Prevotella intermedia* in vitro.[351,383,397]

To view Video 23-3, please visit the Expert CONSULT companion website at www.expertconsult.com.

Surprisingly, when these bacteria were injected in freshly root-planed periodontal pockets in vivo, a significant inhibitory effect was observed on the recolonization of the periodontal pocket by periodontopathogens.[384] These observations highlight the importance of investigating nonpathogenic bacteria in periodontal biofilms, in addition to studying the (putative) pathogens, and might open doors for new treatment approaches.

Recent studies also show that these interactions also can influence the host. Although in the study by Teughels and co-workers, no clinical effect was expected, a reduction in bleeding on probing was noted in pockets that received the streptococci.[384] At that time, this was explained by the establishment of a more host-compatible

microbiota. However, an interaction with the host via the immune system seems an additional interesting hypothesis. Oral streptococci have been shown to modulate bacteria-host interactions involving both *F. nucleatum* and *A. actinomycetemcomitans*. Thus *S. cristatus* and other oral streptococci attenuate the ability of *F. nucleatum* to stimulate interleukin-8 (IL-8) production by host oral epithelial cells.[438] Sliepen and co-workers recently showed similar results for the streptococci that were used in the study of Teughels and co-workers.[352]

For more information on interactions between dental plaque bacteria, please visit the Expert CONSULT companion website at www.expertconsult.com.

Biofilms and Antimicrobial Resistance

Bacteria growing in microbial communities adherent to a surface do not "behave" the same way as bacteria growing suspended in a liquid environment (in a planktonic or unattached state). For example, the resistance of bacteria to antimicrobial agents is dramatically increased in the biofilm.[8,65,139,284] Almost without exception, organisms in a biofilm are 1000 to 1500 times more resistant to antibiotics than in their planktonic state. The mechanisms of this increased resistance differ from species to species, from antibiotic to antibiotic, and for biofilm growing in different habitats.

It is generally accepted that the resistance of bacteria to antibiotics is affected by their nutritional status, growth rate, temperature, pH, and prior exposure to subeffective concentrations of antimicrobial agents.[39,40,423] Variations in any of these parameters will thus lead to a varied response to antibiotics within a biofilm. An important mechanism of resistance appears to be the slower rate of growth of bacterial species in a biofilm, which makes them less susceptible to many but not all antibiotics.[18,37,65,431] The biofilm matrix, although not a significant physical barrier to the diffusion of antibiotics, does have certain properties that can retard antibiotic penetration.

For more information on biofilms and antimicrobial resistance, please visit the Expert CONSULT companion website at www.expertconsult.com.

In addition, extracellular enzymes such as β-lactamases, formaldehyde lyase, and formaldehyde dehydrogenase may become trapped and concentrated in the extracellular matrix, thus inactivating some antibiotics (especially positively charged hydrophilic antibiotics).

For more information on biofilms and antimicrobial resistance, please visit the Expert CONSULT companion website at www.expertconsult.com.

Recently, "super-resistant" bacteria were identified within a biofilm. These cells have multidrug resistance pumps that can extrude antimicrobial agents from the cell.[37] Since these pumps place the antibiotics outside the outer membrane, the process offers protection against antibiotics that target, for example, cell wall synthesis. The penetration and efficacy of antimicrobials against biofilm bacteria are critical issues for the treatment of periodontal infections.

Antibiotic resistance may be spread through a biofilm by intercellular exchange of DNA. The high density of bacterial cells in a biofilm facilitates the exchange of genetic information among cells of the same species and across species and even genera. Conjugation (the exchange of genes through a direct interbacteria connection formed by a sex pilus), transformation (movement of small pieces of DNA from the environment into the bacterial chromosome), plasmid transfer, and transposon transfer have all been shown to occur in biofilms.

BACTERIAL TRANSMISSION AND TRANSLOCATION

Transmission of pathogens, from one *locus* to another, is an important aspect of infectious diseases. In theory, such transmission may jeopardize the outcome of periodontal therapy but also transmission of pathogens from one person to another might be important in terms of disease transmission. The significance of such an intraoral or a vertical or horizontal transmission is, however, difficult to prove and/or to quantify.

One of the first questions in this regard is: "are oral bacteria transmissible between humans?" Molecular fingerprinting techniques clearly illustrated that periodontal pathogens are transmissible within members of a family[434] (Figure 23-29).

For more information on bacterial transimssion and translocation, please visit the Expert CONSULT companion website at www.expertconsult.com.

However, it was more often encountered that children carried a genotype identical to one of the parents than that spouses carried an identical genotype. This indicates that in terms of *A. actinomycetemcomitans* transmission, vertical transmission is more important than horizontal transmission. Similar observations were made for the transmission of cariogenic species from mother to child.[168] Vertical or horizontal transmission of *P. gingivalis* has rarely been observed.[401]

The existence of an "intraoral" translocation of bacteria (from one niche to another) has been investigated more thoroughly.

For more information on bacterial transimssion and translocation, please visit the Expert CONSULT companion website at www.expertconsult.com.

Christersson and co-workers demonstrated a translocation of *A. actinomycetemcomitans* via periodontal probes in localized juvenile periodontitis patients.[57] They were able to successfully colonize previously noninfected pockets with *A. actinomycetemcomitans* by a single course of probing with a probe previously inserted in an infected pocket of the same patient. Although the colonization was only temporary, the question remained whether the colonization could not have become permanent if the site had offered more suitable growth conditions (e.g., a deep bleeding pocket, as frequently encountered immediately after initial periodontal therapy). It is well established that colonization of an already established microbial niche by a new species is difficult since it is hampered by a variety of microbial interactions. Therefore intraoral translocation to and colonization of sterile surfaces might be different from translocation and colonization of already colonized surfaces. Within the oral cavity, such sterile surfaces are not naturally occurring. However, at the moment of abutment placement on a dental implant, a sterile subgingival pocket is created. A large series of papers compared the microbiota in pockets around teeth with that in periimplant pockets in partially edentulous patients and reported

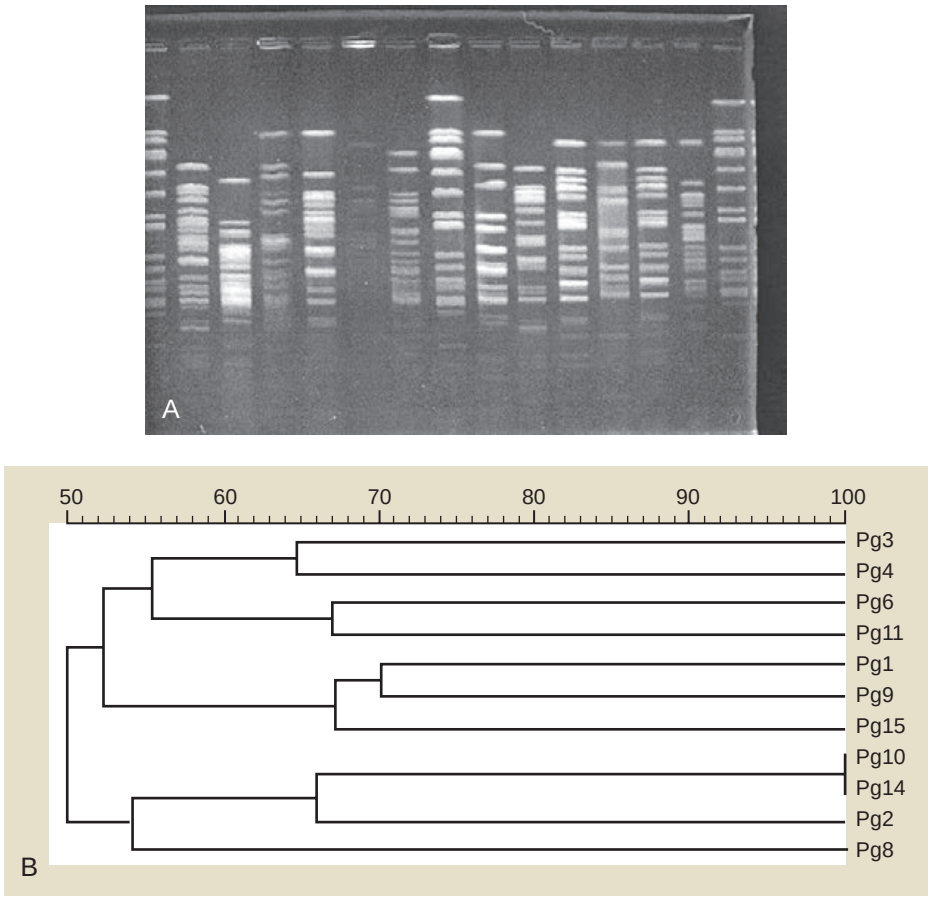

Figure 23-29 **A,** Band pattern of DNA isolated from multiple *Porphyromonas gingivalis* strains after pulsed-field gel electrophoresis. Lanes 1, 8, and 15 contain a reference strain DNA (*Staphylococcus aureus*) cut with *Sma*I (a restriction endonuclease) in lanes 2 to 7 and 9 to 14; 12 different *P. gingivalis* strains are compared on their genetic similarity. The genetic similarity is expressed in a dendrogram **(B).** Bacteria are considered genetically similar when their band pattern is similar for 80%.

a striking similarity.* Several recent studies have investigated the subgingival colonization of such "sterile" periimplant pockets in partially edentulous patients. It was shown that translocation to and colonization of these pockets is extremely fast. Periodontopathogens could already be detected in this niche 30 minutes after abutment placement.[101] The initial colonization of these pockets with periodontopathogens is completed within 1 week. After 2 weeks there are only minor additional changes in this newly developed microbiota.[101,303,305] These additional changes reflect primarily changes in the numbers of species. Based on the previously mentioned similarity between the microbial populations associated with teeth and implants, it has been suggested that at least in partially edentulous patients, teeth might act as a reservoir for the (re)colonization of the subgingival area around implants. This hypothesis was supported by Sumida and co-workers who detected similar intrasubject pulsed field gel electrophoresis patterns for *P. gingivalis* from teeth and implants (indicating that these locations contained precisely the same *P. gingivalis* strains), compared to large intersubject variations.[374]

For more information on bacterial transmission and translocation, please visit the Expert CONSULT companion website at www.expertconsult.com.

*References 13, 149, 150, 167, 169, 198, 202, 235, 246, 267, 297, 333.

NONBACTERIAL INHABITANTS OF THE ORAL CAVITY

Oral microbiology is often reduced to oral "bacteriology." However, the oral cavity comprises a much more diverse microbiota than merely a bacterial one. Viruses, fungi, archaea, and protozoa can be encountered in the oral cavity of humans. Many of these species can reside in the oral cavity, similar to oral bacteria, in harmony with the host as commensals, but they also can cause several oral diseases.

Viruses

Viral diseases of the oral mucosa and the perioral region are often encountered in dental practice. Viruses are important ulcerogenic and tumorigenic agents of the human mouth. The finding of an abundance of mammalian viruses in periodontitis lesions may suggest a role for viruses in more oral diseases than previously recognized.[341] A more in-depth review can be found in Slots.[356]

In contrast to most bacteria, viruses replicate only when present within eukaryotic (animals, plants, protists, and fungi) or prokaryotic (bacteria and archaea) cells, and not on their own. The extracellular virion particle ranges in size from 20 nm to 300 nm and consists of either DNA or ribonucleic acid (RNA) contained within a protective protein coat or capsid (Figure 23-30).

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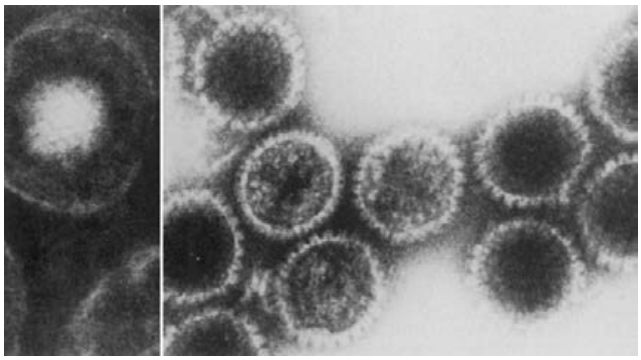

Figure 23-30 Electron micrographic picture of herpes simplex virus type 1, enveloped *(left)* and naked capsids *(right)*. (Magnification ×198,000.) (From Fields BN, Knipe DM: In Fields BN, eds: *Fields' virology,* New York, 1985, Raven Press.)

Some viruses have an additional envelope comprising a lipid bilayer derived from the outer cellular membrane, the internal nuclear membrane, or the endoplasmic reticulum membrane of the infected cell. Four major viral families are associated with the main viral oral diseases of adults, as follows:

1. The group of herpesviruses contains eight different members that all are enveloped double-stranded DNA viruses (Table 23-5). In the oral cavity, they are related to different ulcers, tumors, and other oral pathoses.
2. Human papillomaviruses are grouped within five genera and are nonenveloped double-stranded DNA viruses (Table 23-6). In the oral cavity, they are related to ulcers, tumors, and oral pathoses.
3. Picornaviruses are all nonenveloped, single-stranded RNA viruses (Table 23-7). In the oral cavity, they are related to ulcers and different oral pathoses.

TABLE 23-5 Herpesviruses that Can Be Recovered in the Oral Cavity

Virus	Characteristics	Disease Association	Oral Pathoses
Herpes simplex virus-1	Latency in sensory ganglia. Causes orolabial disease.	Herpetic gingivostomatitis, recurrent orolabial lesions, herpetic whitlow, keratoconjunctivitis, eczema herpeticum, pharyngitis, mononucleosis-like syndrome, encephalitis, neonatal infections.	Adult herpetic gingivostomatitis HIV/AIDS-related oral ulcers ? Recurrent oral aphthous stomatitis Behçet's syndrome Oral pemphigus vulgaris Erythema multiforme Dry socket after tooth extraction
Herpes simplex virus-2	Latency in sensory ganglia. Causes genital and newborn infections.	Genital infection, aseptic meningitis, sacral autonomic nervous system dysfunction.	—
Varicella-zoster virus	Latency in sensory ganglia. Only three major genotypes of the wild-type virus are known. More than 90% are infected before adolescence in an unvaccinated population.	Varicella (chickenpox), herpes zoster (shingles), central nervous system involvement, pneumonia, secondary bacterial infections and death. Available varicella vaccines (about 90% effectiveness) include a single-antigen vaccine and a combination vaccine against MMRV.	? Recurrent oral aphthous stomatitis Osteomyelitis Herpes zoster (Hunt syndrome)
EBV	Identified initially in 1964 from African Burkitt's lymphoma. Infects epithelial cells with a cytolytic infection and B lymphocytes with a latent infection.	Infectious mononucleosis, hairy leukoplakia of the tongue, Burkitt's lymphoma, B lymphoproliferative disease, Hodgkin's lymphoma, X-linked lymphoproliferative disease, nasal T-cell lymphoma, nasopharyngeal carcinoma, gastric carcinoma, parotid carcinoma, and leiomyosarcoma.	? Recurrent oral aphthous stomatitis Erythema multiforme Various epithelial type tumors such as lymphoepithelioma-like carcinoma, salivary gland lymphoepithelial carcinoma, Warthin's tumor (cystadenolymphoma) of the parotid gland, oral squamous cell carcinoma, tonsillar carcinoma, oral undifferentiated carcinomas, and oral hairy leukoplakia. Various lymphoid type tumors such as Hodgkin's lymphoma, T-cell/natural killer cell lymphoma, Burkitt's lymphoma, cyclosporine-related post-transplant lymphoproliferative disorder, oral post-transplant lymphoproliferative disorder/B-cell lymphoma, follicular lymphoid hyperplasia and plasmablastic lymphoma. Infectious mononucleosis

Continued

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TABLE 23-5 Herpesviruses that Can Be Recovered in the Oral Cavity—*cont'd*

Virus	Characteristics	Disease Association	Oral Pathoses
hCMV	Infects mainly T lymphocytes and macrophages. The gB protein in the virion envelope participates in the virus–cell interaction and is a major target of the immune response.	Preterm birth, preeclampsia, transplant rejection, immunosenescence, hemorrhagic retinal necrosis (HIV patients), encephalitis, infectious mononucleosis, atherosclerosis, gastrointestinal disease, pneumonia, and encephalitis.	HIV/AIDS-related oral ulcers ? Recurrent oral aphthous stomatitis Behçet's syndrome Oral pemphigus vulgaris Erythema multiforme Cyclosporine-steroid associated lymphoproliferative disorder Benign infantile hemangioendothelioma Kaposi's sarcoma Occasionally infectious mononucleosis Sialadenitis Osteomyelitis
Human herpesvirus-6	Cell tropism for T lymphocytes and neural cells. Frequently shed in the saliva of healthy donors.	Roseola infantum (sixth disease), meningitis, encephalitis, possibly multiple sclerosis.	Uvulopalatoglossal junctional ulcers Oral squamous cell carcinoma Oral leukoplakia Oral lichen planus
Human herpesvirus-7	Latency in macrophages and T lymphocytes. Frequently shed in the saliva of healthy donors.	Exanthema subitum, macular-papular rashes, transplant-recipient pathogen.	
Human herpesvirus-8	Six genetic subtypes with marked clustering to geographic areas. B lymphocytes and monocytes serve as reservoirs.	Kaposi's sarcoma, multicentric Castleman disease, primary effusion lymphoma, mononucleosis-like illness, aplastic anemia. (Unlike EBV, herpesvirus-8 is not involved in epithelial tumors.)	? Recurrent oral aphthous stomatitis Kaposi's sarcoma

Adapted from Slots J: *Periodontol 2000* 49:60, 2009.
EBV, Epstein-Barr virus; *hCMV,* human cytomegalovirus; *HIV,* human immunodeficiency virus; *AIDS,* acquired immunodeficiency syndrome; *MMRV,* measles, mumps, rubella, and varicella.

TABLE 23-6 Papillomaviruses that Can Be Recovered in the Oral Cavity

Virus	Characteristics	Disease association	Oral Pathoses
Papillomavirus	Epithelial cell proliferation with specificity principally in the anogenital area, urethra, skin, larynx, tracheobronchial, and oral mucosa.	Genital and cutaneous warts, cervical and anogenital cancers, condylomata acuminate (sexually transmitted disease), recurrent respiratory papillomatosis.	Unspecified oral ulcers ? Recurrent oral aphthous stomatitis Focal epithelial hyperplasia Oral squamous cell carcinoma/ verrucous carcinoma Oral leukoplakia Oral lichen planus

Adapted from Slots J: *Periodontol 2000* 49:60, 2009.

4. Retroviruses are divided into seven genera of which two are human pathogens. All retroviruses are enveloped single-stranded RNA viruses (Table 23-8). In the oral cavity, they are related to different tumors and oral pathoses.

For more information on viruses, please visit the Expert CONSULT companion website at www.expertconsult.com.

Clinical Manifestations of Oral Viral Diseases

Oral Ulcers. Ulcers/erosions are relatively common in the oropharyngeal mucosa.[337] Although oral ulcers can have a variety of etiologies, a viral cause of oral ulcers has been established for primary and recurrent herpetic gingivostomatitis (primarily herpes simplex virus-1 but also herpes simplex virus-2), varicella/herpes zoster outbreak (varicella-zoster virus), herpangina (Coxsackievirus), and hand, foot, and mouth disease (enteroviruses). Viruses may also play a role in some cases of recurrent aphthous stomatitis (varicella-zoster virus, CMV, other herpesviruses, adenoviruses, or measles virus) and in systemic diseases with an oral ulcerogenic component such as erythema multiforme (herpes simplex virus), Behçet's syndrome (herpes simplex virus and CMV), pemphigus vulgaris (herpes simples virus and CMV), and systemic lupus erythematosus (EBV).

Oral Tumors. Viruses can be connected to a single or a limited number of tumor types (e.g., hepatitis B virus) or to multiple tumor

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TABLE 23-7 Picornaviruses that Can Be Recovered in the Oral Cavity

Virus	Characteristics	Disease Association	Oral Pathoses
Coxsackievirus	Coxsackievirus A16 is closely related to Enterovirus-71, and both belong to a discrete subgroup of type A enteroviruses that are prominently associated with hand, foot and mouth disease.	Uncomplicated hand, foot, and mouth disease (Coxsackie virus A serotypes 10 and 16); herpangina (mostly Coxsackie virus A); myocarditis; infectious type 1 diabetes (Coxsackie virus B); atherosclerosis.	Herpangina Hand, foot, and mouth disease
Echovirus	Some echovirus replication occurs in the nasopharynx.	Meningitis; pericarditis; myocarditis; herpangina; Guillain-Barré syndrome.	Herpangina
Enterovirus	Enterovirus-71 was first isolated in 1969 from a child with encephalitis. Can cause large epidemics of acute disease. Mutates readily.	Hand, foot, and mouth disease (enterovirus-71); herpangina (enterovirus-71); poliomyelitis-like illness; meningoencephalitis (enterovirus-71); acute pulmonary edema (enterovirus-71); hemorrhagic conjunctivitis.	Herpangina Hand, foot, and mouth disease

Adapted from Slots J: *Periodontol 2000* 49:60, 2009.

TABLE 23-8 Retroviruses that Can Be Recovered in the Oral Cavity

Virus	Characteristics	Disease Association	Oral Pathoses
HIV-1	Global infection; infects cells containing CD4 receptor such as T-helper lymphocytes and cells of the macrophage lineage.	Rank order of AIDS-defining pathoses is as follows: Pneumocystis pneumonia (43%) Esophageal candidiasis (15%) Wasting (11%) Kaposi's sarcoma (11%) Disseminated *Mycobacterium avium* infection (5%) Mycobacterium tuberculosis (5%), cytomegalovirus disease (4%) HIV-associated dementia (4%) Recurrent bacterial pneumonia (3%) Toxoplasmosis (3%)	Oral hairy leukoplakia Plasmablastic lymphoma Kaposi's sarcoma Xerostomia (Sjögren's syndrome) Sialadenitis Osteomyelitis
HIV-2	Infection occurring mainly in West Central Africa (Guinea-Bissau).	HIV-2 is associated with similar types of diseases as HIV-1 but is generally less virulent.	Oral hairy leukoplakia Plasmablastic lymphoma Kaposi's sarcoma Xerostomia (Sjögren's syndrome) Sialadenitis Osteomyelitis

Adapted from Slots J: *Periodontol 2000* 49:60, 2009.
HIV, Human immunodeficiency virus; *AIDS,* acquired immunodeficiency syndrome.

types (e.g., EBV), a difference that probably reflects the extent of tissue tropism(s) of the viruses. Viruses may cause cell transformation and proliferation by directly expressing oncogenic genes in infected cells, or by acting as a necessary or a noncompulsory cofactor in the development of malignancy. However, even though most individuals harbor oncogenic viruses in the oral cavity, cancer occurring as a result of infection with such viruses is relatively rare. Risk factors apart from the viral infection are obviously important for cancer development, including family history, age, tobacco smoking, and alcohol consumption. The most important oncoviruses of the human mouth are EBV, herpesvirus-8, and papillomaviruses, and the most common virally related malignancies in the oral cavity are epithelial neoplasms, lymphomas, and Kaposi's sarcoma.[330]

Other Oral Pathologies. In addition to oral ulcers and tumors, viruses have also been implicated in infectious mononucleosis (primary EBV infection and occasionally a primary CMV infection), Sjögren syndrome (hepatitis C virus, human T-lymphotropic virus type I, HIV, and herpesviruses), osteomyelitis (herpes zoster virus and CMV), osteonecrosis (herpes zoster), oral leukoplakia (papillomaviruses, mainly the types 16 and 18), and oral lichen planus (papillomaviruses and hepatitis C virus).

Periodontitis. Bacteria are recognized as indispensable for the development of periodontitis and the current hypotheses on pathogenic mechanisms of periodontitis correctly emphasize the importance of assessing bacterial and host factors collectively. However, bacterial-host interactions alone appear insufficient to explain the

clinical characteristics of the disease.[355] The localized distribution and intermittent exacerbations of periodontal breakdown and other issues remain a riddle. In response, the involvement of herpesviruses in the etiology of periodontitis has been suggested based on their increased presence in inflamed gingival tissue, crevicular fluid, and subgingival plaque in periodontally diseased sites.[355] However, it remains unclear whether herpesvirus activation occurs spontaneously or as a result of concurrent infection, stress, or other factors reducing the host immune defence.[46] It is interesting to note that several risk factors for periodontitis also have the potential to reactivate herpesviruses.[66]

The hypothesis that herpesviruses are involved in the etiology of periodontitis is therefore controversial.[46] Yet, there is no doubt that herpesviruses are present in the periodontal pocket. Next to herpesviruses, many other viruses have been detected in periodontal pockets. Papillomaviruses, HIV, human T-lymphotropic virus type I, hepatitis B and C viruses, and Torquetenovirus all can inhabit periodontitis lesions.[356] Additionally, the inflamed periodontium may constitute the major oral reservoir for EBV, CMV, papillomaviruses, and hepatitis C virus.[356]

Yeasts

Many yeast species have been isolated from the oral cavity. The majority of isolates are *Candida*, and the most prevalent species is *C. albicans* (Figure 23-31). Together with *C. albicans*, some of the most common opportunistic fungal pathogens in humans are *C. tropicalis*, *C. glabrata*, *C. krusei*, *C parapsilosis*, *C. guilliermondii*, and *C. dubliniensis*.

For more information on yeasts, please visit the Expert CONSULT companion website at www.expertconsult.com.

Yeasts, such as *Rhodotorula glutinis* and *Saccharomyces cerevisiae*, are found in the oral cavity rarely and are not known to cause oral infections. *Cryptococcus neoformans* is occasionally isolated from the mouth, but usually from patients with pulmonary cryptococcosis.[370]

C. albicans is the species most frequently encountered in infected tissues, including oral mucosal layers.

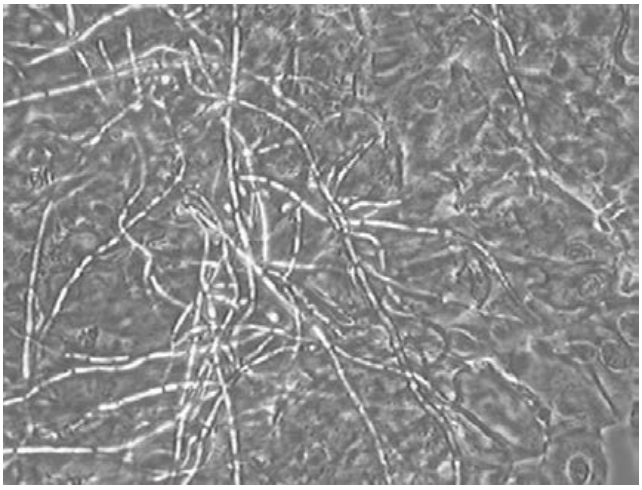

Figure 23-31 Sample of the palate of a patient with denture stomatitis with clear candidal mycelia visualized under a phase-contrast microscope.

For more information on yeasts, please visit the Expert CONSULT companion website at www.expertconsult.com.

C. albicans infections are increasing in prevalence, especially in denture wearers and aging people, and may lead to invasive disease, which has a high mortality rate.

For more information on yeasts, please visit the Expert CONSULT companion website at www.expertconsult.com.

A number of systemic mycotic diseases, once considered exotic, are now manifesting intraorally with increasing frequency because of the high prevalence of immunocompromised individuals in the community[227,329] (Table 23-9).

Protozoa

The mouth is the entry port for many parasites adapted to the human host. Only a few parasites affect the oral cavity, but an increasing body of literature claims that oral protozoa are more common than previously appreciated.[25] Depending on the type of infection, the parasitic infectious agents can be divided into two categories: those that induce local infections and those that induce systemic infections with indirect effects on the oral cavity. The former group comprises saprophytes (such as *Entamoeba gingivalis* and *Trichomonas tenax* (Figure 23-32; see Video 23-4: Phase Contrast online) that have the potential to turn into opportunistic pathogens, or free-living amoebae that occasionally become invasive but seldom present clinically.

To view Video 23-4, please visit the Expert CONSULT companion website at www.expertconsult.com.

The finding of these organisms in the mouth is associated with poor oral hygiene and they may be quite common in people who have a low standard of living.

TABLE 23-9 Etiologic Agents for Different Types of Oral Yeast Infections

Oral Mycoses	Etiologic Agent
Candidiasis	*Candida albicans, C. glabrata, C. tropicalis, C. dubliniensis, C. krusei,* etc
Aspergillosis	*Aspergillus fumigatus*
Blastomycosis	*Blastomyces dermatitidis*
Coccidioidomycosis	*Coccidioides immitis*
Cryptococcosis	*Cryptococcus neoformans*
Fusariosis	*Fusarium moniliforme*
Geotrichosis	*Geotrichum candidum*
Histoplasmosis	*Histoplasma capsulatum*
Mucormycosis	Order *Mucorales*
Paracoccidiomycosis	*Paracoccidioides brasiliensis*
Penicilliosis	*Penicillium marneffei*
Sporotrichosis	*Sporothrix schenckii*

Samaranayake LP, Keung LW, Jin L: Oral mucosal fungal infections, *Periodontol 2000* 49:39, 2009.

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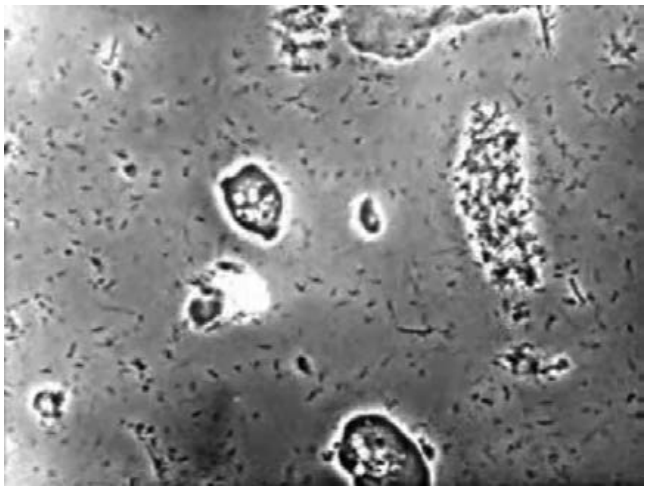

Figure 23-32 Trichomonas in dental plaque visualized under a phase-contrast microscope.

For more information on protozoa, please visit the Expert CONSULT companion website at www.expertconsult.com.

E. gingivalis is possibly less commonly found in the oral cavity than *T. tenax*. However, there are indications that *E. gingivalis* produces a particular, progressive periodontal disease in immunocompromised patients who often present with a necrotic gingival infection.

For more information on protozoa, please visit the Expert CONSULT companion website at www.expertconsult.com.

Among systemic parasitic infections, only the protozoan flagellate *Leishmania* can produce clinical symptoms affecting the oral cavity.[25] This effect is indirect and caused by disfigurations produced by infections with the mucocutaneous form of the infection, resulting in granulomatous growth involving the mouth and nose. While the distribution of the local, oral parasites is worldwide, leishmaniasis only exists in certain areas.

Archaea

Archaea are single-celled organisms that are as distinct from the bacteria as they are from eukaryotes. The role of archaea in oral diseases is only beginning to be explored. Methanogenic archaea produce methane from hydrogen gas (H_2)/carbon dioxide (CO_2) and sometimes from formate, acetate, methanol, or methylamine. These organisms have been isolated from patients with periodontal disease by enriching cultures with H_2 and CO_2.[24] Interestingly, in this study, archaea were not isolated from periodontally healthy subjects. Using molecular (culture-independent) methods, archaea were identified in 37 out of 48 samples from periodontal disease sites. Subsequently, a clear correlation between the presence of archaeal DNA and periodontal disease was established.[203] Despite these findings, virtually nothing is known about the role of archaea in periodontitis. Indeed, it is not clear whether these organisms contribute to disease or simply take advantage of the disease state to increase host colonization. Archaea have also been detected in endodontic infections, and again their contribution to disease is unknown.[405,406]

MICROBIOLOGIC SPECIFICITY OF PERIODONTAL DISEASES

Nonspecific Plaque Hypothesis

In the mid1900s, periodontal diseases were believed to result from an accumulation of plaque over time, eventually in conjunction with a diminished host response and increased host susceptibility with age.

For more information on nonspecific plaque hypothesis, please visit the Expert CONSULT companion website at www.expertconsult.com.

The nonspecific plaque hypothesis maintains that periodontal disease results from the "elaboration of noxious products by the entire plaque flora."[218] According to this thinking, when only small amounts of plaque are present, the noxious products are neutralized by the host. Similarly, large amounts of plaque would produce large amounts of noxious products, which would essentially overwhelm the host's defenses.

Several observations contradicted these conclusions. First, some individuals with considerable amounts of plaque and calculus, as well as gingivitis, never developed destructive periodontitis. Furthermore, individuals who did present with periodontitis demonstrated considerable site specificity in the pattern of disease. Some sites were unaffected, whereas advanced disease was found in adjacent sites. In the presence of a uniform host response, these findings were inconsistent with the concept that all plaque was equally pathogenic. Recognition of the differences in plaque at sites of different clinical status (i.e., disease versus health) led to a renewed search for specific pathogens in periodontal diseases and a conceptual transition from the nonspecific to the specific plaque hypothesis.[216,354,360]

Inherent in the nonspecific plaque hypothesis is the concept that control of periodontal disease depends on control of the amount of plaque accumulation. The current standard treatment of periodontitis by debridement (nonsurgical or surgical) and oral hygiene measures still focuses on the removal of plaque and its products and is founded on the nonspecific plaque hypothesis. Thus, although the nonspecific plaque hypothesis has been discarded in favor of the specific plaque hypothesis or the ecologic plaque hypothesis, much clinical treatment is still based on the nonspecific plaque hypothesis.

Specific Plaque Hypothesis

The specific plaque hypothesis states that only certain plaque is pathogenic, and its pathogenicity depends on the presence of or increase in specific microorganisms.[218] This concept predicts that plaque harboring specific bacterial pathogens results in a periodontal disease because these organisms produce substances that mediate the destruction of host tissues. The association of specific bacterial species with disease came about in the early 1960s, when microscopic examination of plaque revealed that different bacterial morphotypes were found in healthy *versus* periodontally diseased sites (see Video 23-4: Phase Contrast online). At about the same time, major advances were made in techniques used to isolate and identify periodontal microorganisms. These included improvements in procedures to sample subgingival plaque, handling of samples to prevent killing the bacteria, and media used to grow the bacteria in the laboratory.[362] The result was a tremendous increase in the ability to isolate periodontal microorganisms and considerable

refinement in bacterial taxonomy.[185] Acceptance of the specific plaque hypothesis was spurred by the recognition of *A. actinomycetemcomitans* as a pathogen in localized aggressive periodontitis.[254,354] These advances led to a series of association studies focused on identifying specific periodontal pathogens by examining the microbiota associated with states of health and disease in cross-sectional and longitudinal studies.

The introduction of molecular methods for bacterial identification has greatly increased the power of association studies, since we are no longer constrained to analyzing those bacteria that can be cultured (<50% of the total oral microflora). For example, specific members of the phylum *Synergistetes* that have never been cultured in isolation show a clear association with periodontal disease.[404] Molecular identification techniques such as DNA checkerboard hybridization can identify and enumerate many different organisms simultaneously and are thus well-suited to high-throughput studies. (see Figure 23-20 online) The association of Socranksy's "red complex" bacteria, *P. gingivalis*, *T. forsythia,* and *T. denticola* with periodontal disease was based on the analysis of 40 different bacteria in >13,000 plaque samples.[364] Nevertheless, disease association studies do not reveal whether the presence of specific bacteria *causes* or *correlates with* the presence of disease. In addition, these studies have shown that periodontal disease can occur even in the absence of defined "pathogens," such as red complex bacteria, and conversely that "pathogens" may be present in the absence of disease.

Ecologic Plaque Hypothesis

In the 1990s, Marsh and co-workers developed the "ecologic plaque hypothesis" as an attempt to unify the existing theories on the role of dental plaque in oral disease (Figure 23-33). According to the ecologic plaque hypothesis, both the total amount of dental plaque and the specific microbial composition of plaque may contribute to the transition from health to disease. The health-associated dental plaque microflora is considered to be relatively stable over time and in a state of dynamic equilibrium or "microbial homeostasis." The host controls subgingival plaque to some extent by a tempered immune response and low levels of GCF flow. Perturbations to the host response may be brought about by the excessive accumulation of (nonspecific) dental plaque, or by plaque-independent host factors (e.g., the onset of an immune disorder, changes in hormonal balance such as in pregnancy), or environmental factors (e.g., smoking, diet). Changes in the host status, such as inflammation, tissue degradation, and/or high GCF flow, may lead to a shift in the microbial population in plaque, culminating in periodontal disease. The ecologic plaque hypothesis is entirely consistent with observations that disease-associated organisms are minor components of the oral microflora in health; these organisms are kept in check by interspecies competition during microbial homeostasis. Disease is caused by the overgrowth of specific elements of dental plaque when the local microenvironment changes, but it is not necessarily the same species in each case. An important consideration of the ecologic plaque hypothesis is that therapeutic intervention can be useful on a number of different levels. Eliminating the disease-inducing stimulus, whether it is microbial, host, or environmental, will help to restore microbial homeostasis. Targeting specific microorganisms may be less effective since the conditions for disease will remain. One pathogen will simply be replaced by another.

Complicating Factors

The identification of bacterial pathogens in periodontal diseases has been difficult because of a number of factors.[362] The periodontal microbiota is a complex community of microorganisms, many of which are still difficult or impossible to cultivate or identify. The chronic nature of periodontal disease has complicated the search for bacterial pathogens. It was previously thought that periodontal diseases progressed at a slow but steady rate.[214] However, epidemiologic studies established that the disease progresses at different rates, with alternating episodes of rapid tissue destruction and periods of remission (Figure 23-34).[365] At this moment, by means of multilevel modelling, the linear and burst theories of periodontitis progression are considered to be a manifestation of the same phenomenon: some sites improve while others progress, in a cyclic manner.[107] Identification of the microorganisms found during the different phases of the disease progression is technically challenging. The interpretation of microbiologic data is further complicated by the clinical classification of the disease status, an area that has undergone a number of recent revisions.[15,396] Previous and perhaps current classifications involve the grouping of potentially different disease states because of difficulties in accurately distinguishing them clinically. It is important to recognize that groupings such as this may obscure microbiologic associations.

Currently, periodontitis is considered a mixed infection. This has significant implications for both the diagnosis and treatment of periodontitis. In the diagnosis, some evaluate the presence of up to 40 species, but it still is unclear whether some combinations of species are more pathogenic than others. The treatment is directed to the eradication/reduction of the number of all key periodontopathogens. Since several species might be involved, the appropriate use of antimicrobials (especially antibiotics) is extremely difficult to define because not all expected periodontopathogens are equally susceptible to the same antibiotic.

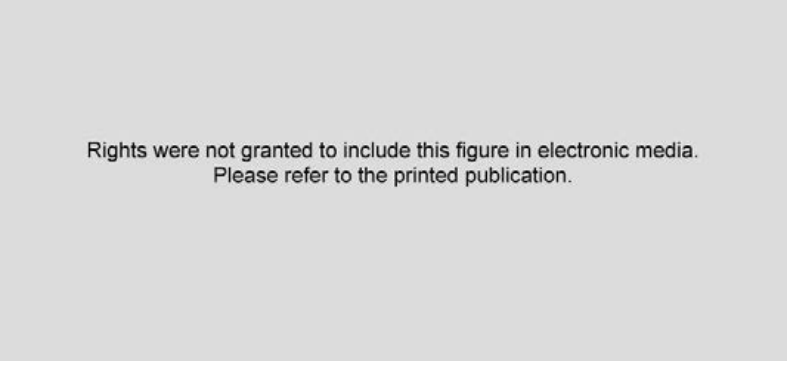

Figure 23-33 Ecologic plaque hypothesis in relation to periodontal diseases: gingivitis and periodontitis. Accumulation of plaque causes inflammation of adjacent tissues (gingivitis) and other environmental changes that favor the growth of gram-negative anaerobes and proteolytic species, including periodontopathogens. The increased proportions of such species results in destruction of periodontal tissues (i.e., periodontitis). *Eh,* Redox-potential; *GCF,* gingival crevicular fluid; *gram +ve,* gram-positive; *gram –ve,* gram-negative. (Adapted from Marsh PD: *Adv Dent Res* 8:263, 1994.)

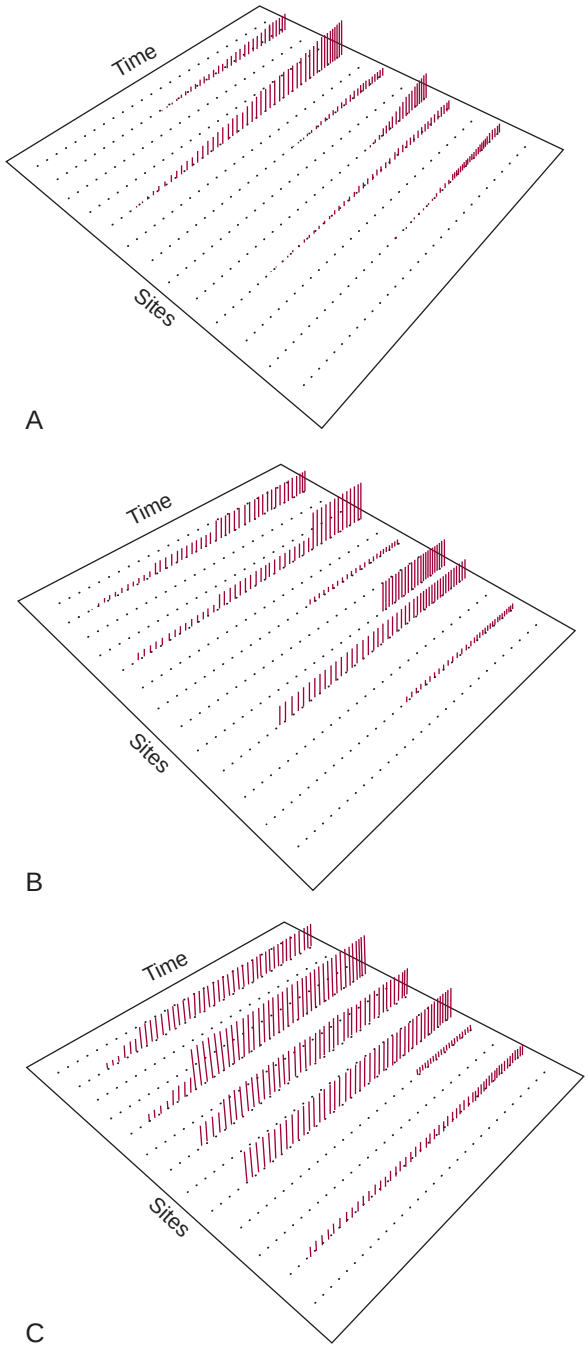

Figure 23-34 Diagrammatic representation of possible modes of progression of chronic destructive periodontal diseases. Sites on the x-axis are plotted against time on the y-axis, and activity is shown on the z-axis. **A,** Some sites show progressive loss of attachment over time, whereas others show no destruction. The time of onset and the extent of destruction vary among sites. **B,** Random burst model. Activity occurs at random at any site. Some sites show no activity, whereas others show one or several bursts of activity. The cumulative extent of destruction varies among sites. **C,** Asynchronous multiple burst model. Several sites show bursts of activity over a finite period, followed by prolonged periods of inactivity. Occasional bursts may occur infrequently at certain sites at later periods. Other sites show no periodontal disease activity at any time. The difference from the model shown in **B** is that in **C,** the majority of destructive disease activity takes place within a few years of the individual's life. (Courtesy Drs. S. Socransky, A. Haffajee, M. Goodson, and J. Lindhe, Boston, and Göteborg, Sweden.)

Recent microbiologic diagnostic tests indicated that the presence of periodontopathogens alone is not sufficient for the development of periodontitis. Because of the high sensitivity of these tests, several pathogens have been detected in periodontitis-free patients. Thus merely the presence of pathogens is not sufficient for disease and the amount of the pathogens plays a key role in relation to disease.[321] These observations have major clinical implications. First, the specificity of microbial detection is dramatically reduced (specificity = presence of pathogen means periodontitis). In other words, even though a microbiologic analysis is positive, the patient can be without disease.[222] Second, the understanding of the etiology becomes more complicated because the threshold level for periodontopathogens between health and disease is unknown and obviously subject dependent. Third, for several species, large intrastrain variations in genetic information have been detected (different genotypes) so that, in fact, information on the genotype is needed before the pathogenicity of the strain can be estimated.[55,72,115,272] The quality of the host response also plays an essential role, but this cannot yet be correctly estimated. Finally, one can question whether periodontopathogens are indigenous species or exogenous. Indeed, the newer techniques have reported high detection frequencies of all pathogens in healthy subjects.[222,332] This has significant impact on the treatment strategies. For indigenous species the endpoint of a therapy is the reduction of the species, whereas for exogenous species the endpoint is eradication and prevention of reinfection.

Currently, it is impossible to alter the susceptibility of the host, so periodontal therapy must be focused on the reduction/elimination of periodontopathogens in combination with the reestablishment, often by surgical pocket elimination, of a more suitable environment (less anaerobic) for a more beneficial microbiota. Several studies indicate that the presence of the previously mentioned periodontopathogens (persisting or reestablished after treatment) is associated with a negative clinical outcome of periodontal treatment.[120,311,312] Because several species might be involved, the use of antimicrobials (especially antibiotics) is extremely difficult since not all expected periodontopathogens are susceptible to the same antibiotic and susceptibility depends on the geographic region.[300,402]

It is obvious that several key questions still remain unanswered. Some researchers question whether the presence of specific microorganisms in the periodontal pocket is the cause or a consequence of the disease.[366] Since most periodontopathogens are fastidious strict anaerobes, they may contribute little to the initiation of disease in shallow gingival pockets but are found only in deep periodontal pockets, their preferred habitat.

Criteria for Identification of Periodontopathogens

In the 1870s, Robert Koch developed the classic criteria by which a microorganism can be judged to be a causative agent in human infections. These criteria, known as *Koch's postulates,* stipulate that the causative agent must do the following:

1. Be routinely isolated from diseased individuals.
2. Be grown in pure culture in the laboratory.
3. Produce a similar disease when inoculated into susceptible laboratory animals.
4. Be recovered from lesions in a diseased laboratory animal.

Difficulties exist in the application of these criteria to polymicrobial diseases, and the applicability of Koch's postulates has been increasingly challenged in recent years. In the case of periodontitis, three primary problems are (1) the inability to culture all the organisms that have been associated with disease (e.g., many of the oral

spirochetes), (2) the difficulties inherent in defining and culturing sites of active disease, and (3) the lack of a good animal model system for the study of periodontitis.[362] In fact, if the ecologic plaque hypothesis proves correct, it must be inherently impossible to fulfill Koch's postulates, since no single organism or group of organisms is responsible for all cases of disease.

Sigmund Socransky, a researcher at the Forsyth Dental Center in Boston, proposed criteria by which periodontal microorganisms may be judged to be potential pathogens.[362] According to these criteria, a potential pathogen must do the following:

1. Be associated with disease, as evident by increases in the number of organisms at diseased sites
2. Be eliminated or decreased in sites that demonstrate clinical resolution of disease with treatment
3. Induce a host response, in the form of an alteration in the host cellular or humoral immune response
4. Be capable of causing disease in experimental animal models
5. Produce demonstrable virulence factors responsible for enabling the microorganism to cause destruction of the periodontal tissues

Data supporting the role of *A. actinomycetemcomitans* and *P. gingivalis* as periodontal pathogens, based on these criteria, have been presented earlier. The association and elimination criteria are discussed in the preceding sections. The latter three criteria focus on the host parasite interaction, which is discussed in Chapter 25.

THE TRANSITION FROM HEALTH TO DISEASE

The oral microbial ecology is dynamic. The presence and numbers of particular microorganisms, even as a part of a community colonizing a niche in a human being, are controlled by the type and quantity of nutrients present (nutritional determinant), their ability to tolerate the physicochemical factors operating there (physicochemical determinants), and their ability to withstand any antimicrobial compounds (biological determinants) or mechanical removal forces (mechanical determinants).[424] Although these determinants are well-defined from a theoretic point of view, they overlap each other, especially in more complex environments (i.e., multispecies environments). Bacteria are living species not passive bystanders. They will interact with their environment and vice versa. Therefore the microbial ecology will change its composition or the composition of the microbial ecology will be changed when transitioning from a healthy status to a diseased status or vice versa. A change in the composition of a bacterial community as the result of external, nonmicrobial factors is termed *allogenic succession*. Smoking is a good example of such interaction. In *autogenic succession* (i.e., a change in the composition of a microbial community arising from microbial activities), interbacterial and viral-bacterial interactions are involved.

Early microscopy studies clearly demonstrated that subgingivally the number and proportions of different bacterial morphotypes differ between healthy and diseased sites (Figures 23-35 and 23-36; see Video 23-4: Phase Contrast online).[210,354,360] The total number of bacteria, determined by microscopic counts per gram of plaque, was twice as high in periodontally diseased sites than in healthy sites.[360] Because considerably more plaque is found at diseased sites, this suggests that in general the total bacterial load in diseased sites is greater than that at healthy sites. Fewer coccal cells and more motile rods and spirochetes are found in diseased sites than in healthy sites.[210] Although this morphologic criterion can be of diagnostic value, one should remember that nearly all key periodontal pathogens except *Campylobacter rectus* and spirochetes are immobile rods, which adds to the confusion regarding the bacterial etiology of periodontal diseases. By culturing it appears that the microbiota in periodontally healthy sites consist predominantly of gram-positive facultative rods and cocci (approximately 75%).[354] The recovery of this group of microorganisms is decreased proportionally in gingivitis (44%) and periodontitis (10% to 13%). These decreases are accompanied by increases in the proportions of gram-negative rods, from 13% in health to 40% in gingivitis and 74% in advanced periodontitis.

The current concept concerning the etiology of periodontal diseases considers 3 groups of factors that determine whether active periodontal destruction will occur in a subject: a susceptible host, the presence of pathogenic species, and the absence/small proportion of so-called beneficial bacteria.[12,359,362,369,427] The clinical manifestations of periodontal destruction thus result from a complex interplay between these etiologic agents. In general, small amounts of bacterial plaque can be controlled by the body's defense mechanisms without destruction, but when the balance between bacterial load and host response is disturbed, periodontal destruction might occur. The latter can be the case when the subject is very susceptible to periodontal infections, and/or when he or she is infected by a large amount of bacteria and/or by a very pathogenic microbiota.

Host Susceptibility

The susceptibility of the host is determined in part by genetic factors but can be influenced by environmental/behavioral factors such as smoking, stress, and viral infections. Genetic factors are known to play a role, especially in patients with so-called early onset periodontitis, now referred to as *aggressive periodontitis*.[237] Reports on the familial nature of chronic forms of periodontitis are less conclusive. Aggregation of aggressive periodontitis within

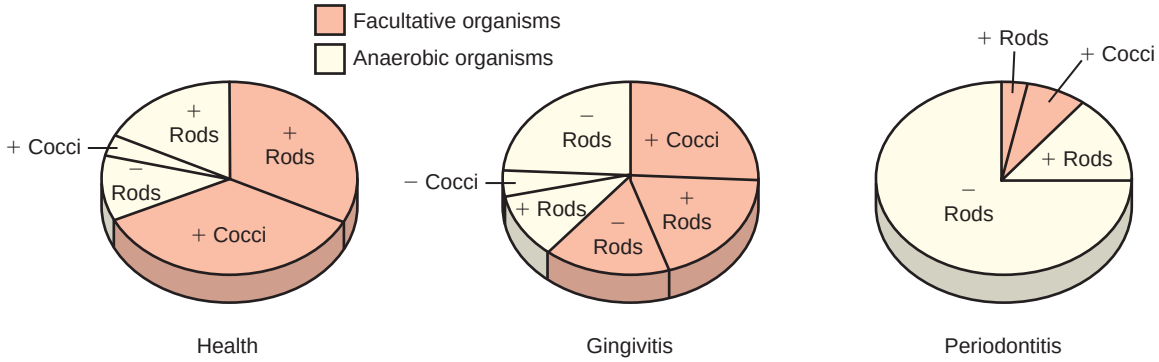

Figure 23-35 Pie charts based on culturing studies, representing the relative proportion of different morphotypes in subgingival samples in cases of periodontal health, gingivitis, and periodontitis. A clear distinction is made between facultative species and obligate anaerobic species. Spirochetes are not included.

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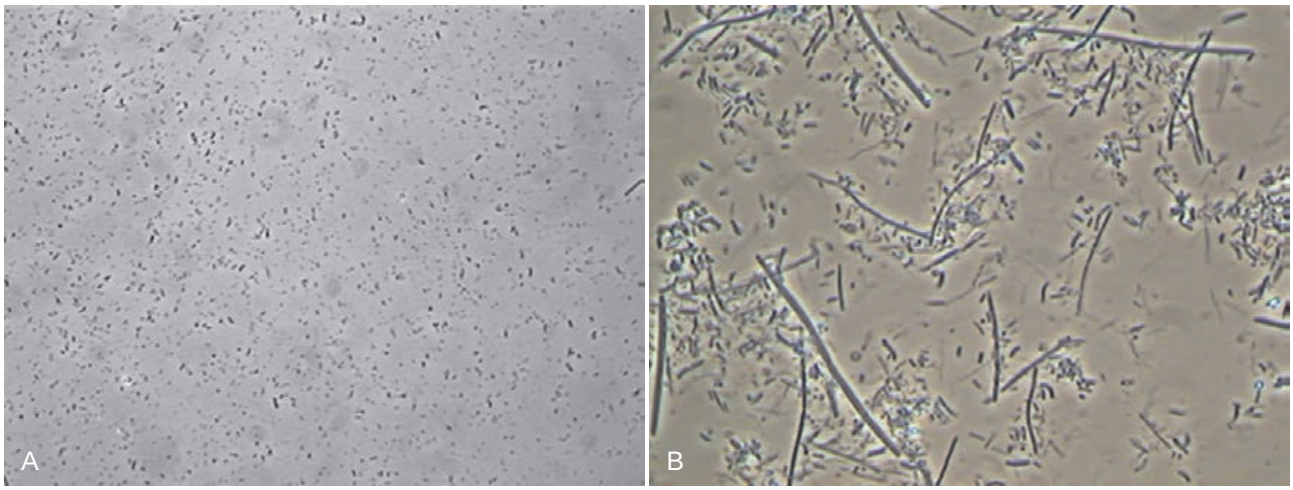

Figure 23-36 Phase-contrast micrograph of a plaque sample from a healthy **(A)** and a periodontitis patient **(B).** Note the large rods and spirochetes in the periodontitis plaque sample.

families is consistent with a genetic predisposition, although common environmental factors cannot be excluded.

For more information on host susceptibility, please visit the Expert CONSULT companion website at www.expertconsult.com.

Smoking is considered a behavioral or eventually an environmental factor. It dramatically increases host susceptibility to periodontal breakdown.

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Nevertheless, the exact nature of the relationship between periodontitis and smoking is unclear.[21,159]

For more information on host susceptibility, please visit the Expert CONSULT companion website at www.expertconsult.com.

On the other hand, there are conflicting reports regarding the effects of smoking on the oral microbiota. Some are in favor of an increased prevalence of *P. gingivalis*, *A. actinomycetemcomitans*, *T. forsythia, E. coli*, and *C. albicans* in smokers, while others report no difference.* Recently it has been shown that smoking cessation can alter the subgingival biofilm and suggest a mechanism for improved periodontal health associated with smoking cessation.[100] There is some evidence that epithelial cells of smokers are more prone to colonization by respiratory pathogens. Depending on the bacterial strain, the relative increase in the number of adhering pathogens in smokers versus nonsmokers ranged from 40% up to 150%.[91,325] Interestingly, cigarette smoke has recently been shown to modify the expression of *P. gingivalis* genes directly, including several virulence-associated genes.[20] After exposure to cigarette smoke extract, *P. gingivalis* cells induce a lower proinflammatory response than unexposed *P. gingivalis* cells.

Recently, viral infections have been added to the list of factors that modify host susceptibility. HIV and herpesviruses are often associated with periodontal infections.

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It is believed that virus-associated periodontal destruction originates from virus-induced changes in the host response to the local subgingival microbiota.[355]

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In relation to periodontopathogen adhesion to virally infected cells, Teughels and co-workers showed recently a 100% increase in *A. actinomycetemcomitans* colonization when epithelial cells were infected with hCMV.[385]

Increasing evidence suggests that emotional stress and/or negative life events can play an important role in the development and progression of periodontitis and may also modify the response to periodontal treatment.

For more information on host susceptibility, please visit the Expert CONSULT companion website at www.expertconsult.com.

Emotional and psychologic load can influence the immune system and will also alter oral health behaviors.[68] Both factors increase the susceptibility for periodontitis.

Racial factors also have been implicated in the differences in prevalence of early-onset periodontitis and in the frequency of detection of putative periodontopathogens.[22,215]

*References 28, 123, 285, 372, 392, 400, and 435.

Pathogenic Bacteria

The second essential factor for disease initiation and progression is the presence of one or more pathogens, of the appropriate clonal type and in sufficient numbers. Despite the difficulties inherent in characterizing the microbiology of periodontal diseases, a small group of pathogens is recognized because of their close association with disease. There are obvious data to support the designation of *A. actinomycetemcomitans, T. forsythia,* and *P. gingivalis* as key pathogens, since they are strongly associated with the periodontal disease status, the disease progression, and unsuccessful therapy. For the following list of bacteria, moderate evidence for etiology has been reported, at least if their concentration passes a certain threshold level: *P. intermedia, Prevotella nigrescens* (see Figure 23-1, *I*), *C. rectus, Parvimonas micra* (see Figure 23-1, *J*), *F. nucleatum, Eubacterium nodatum* (see Figure 23-1, *N*), and various spirochetes.[12,359,362,369,427] The significance for the role of these key pathogens is largely based on epidemiologic data, the ability of these microorganisms to produce disease when inoculated in animals, and/or their capacity to produce virulence factors. However, the mere presence of putative periodontopathogens in the gingival crevice is not in itself sufficient to initiate or cause periodontal inflammation. An elevation in the relative proportion/number of these pathogens, to reach a critical mass, seems more crucial to mount an effective tissue-damaging process. Indeed, even in health, periodontopathogens are or can be present in the gingival crevice, albeit in low numbers, as members of the normal resident flora.[222] Table 23-10 gives a general overview of the detection frequency for most key pathogens in different forms of periodontal infections. It is immediately obvious that there is no black-and-white situation; most pathogens might (but not necessarily) have to be present for specific forms of periodontitis. This also illustrates that it is most of the time difficult to use the microbial composition to differentiate between different forms of periodontal infections. Table 23-11 further highlights the complexity of the microbiology of periodontitis. Most periodontopathogens can also be detected in healthy subjects with frequencies ranging from 10% to 85%. It is obvious that this automatically reduces the specificity of microbiologic testing in periodontology.

Beneficial Species

The role of "beneficial" species of the host is less obvious in the development of periodontal diseases.[362] Often, bacteria and host cells establish a commensal relationship, beneficial for both.

For more information on beneficial species, please visit the Expert CONSULT companion website at www.expertconsult.com.

The role of beneficial species of the host is also less obvious in the progression of periodontitis.[317,362] These bacteria can affect the pathogenic species in different ways and thus modify the disease process, as follows: (1) by passively occupying a niche that may otherwise be colonized by pathogens, (2) by actively limiting a pathogen's ability to adhere to appropriate tissue surfaces, (3) by adversely affecting the vitality or growth of a pathogen, (4) by affecting the ability of a pathogen to produce virulence factors, or (5) by degrading virulence factors produced by the pathogen. The often-used textbook example of such a beneficial action is the effect of *S. sanguinis*, formerly known as *S. sanguis*, on *A. actinomycetemcomitans*.[391] *S. sanguinis* produces H_2O_2, which

TABLE 23-10 Prevalence of Microbial Species Associated with Various Clinical Forms of Periodontitis

	FORMS OF PERIODONTITIS		
Species	**Chronic Periodontitis**	**Localized Aggressive Periodontitis**	**Aggressive Periodontitis**
Aggregatibacter actinomycetemcomitans	28.2%	81.8%	40.9%
Porphyromonas gingivalis	53.8%	13.3%	79.6%
Prevotella intermedia/nigrescens	50.2%	53.4%	71.4%
Tannerella forsythia	50.6%	0	50.8%
Campylobacter rectus	40.3%	12.5%	47.8%

Adapted from Haffajee AD, Socransky SS: *Periodontol 2000* 5:78, 1994; and Mombelli A, Casagni F, Madianos PN: *J Clin Periodontol* 29 Suppl 3:10, 2002.
0, Occasionally isolated.

TABLE 23-11 Prevalence of Key Pathogens in Healthy Subjects and Patients with Periodontitis

	PREVALENCE		SIGNIFICANCE OF DIFFERENCE	
Species	**Health**	**Periodontitis**	**P-Value**	**Odds Ratio***
Aggregatibacter actinomycetemcomitans	12.8%	31.0%	0.002	3.1
Porphyromonas gingivalis	10.6%	59.5%	<0.001	12.3
Prevotella intermedia/nigrescens	69.1%	87.9%	0.001	3.3
Tannerella forsythia	47.9%	90.5%	<0.001	10.4
Fusobacterium nucleatum	85.1%	95.7%	0.014	3.9
Parvimonas micra	67%	94%	<0.001	7.7
Campylobacter rectus	13.8%	20.7%	NS	1.6

Adapted from Van Winkelhoff, Loos BG, van der Reijden WA, et al: *J Clin Periodontol* 29:1023, 2002.
*The ratio of the probability of developing the disease to the probability of nonoccurrence, thus 3 means 3 times more chance the disease will develop if that species is present.
NS, Not significant.

either directly or by host enzyme amplification can kill *A. actinomycetemcomitans*.[144] Although these so-called beneficial bacteria might be of importance for maintaining a healthy subgingival ecosystem, evidence is limited.[317] Despite a rapidly increasing knowledge on periodontopathogen-host interactions, the role of beneficial microbiota in this crosstalk remains obscure. In periodontal microbiology, a bacterial strain is considered beneficial when its prevalence is high in periodontal health and low in diseased situations. It is shown for other fields in microbiology that direct and indirect microbial interactions can suppress the emergence of pathogens.[35] The importance of the latter is evidenced within the oral cavity by the development of yeast infections when the normal oral flora is reduced, for instance after a period of systemic antibiotic usage.[410]

Since it is impossible so far to alter the susceptibility of the host, periodontal therapy is necessarily focused on the reduction/elimination of periodontopathogens in combination with the reestablishment, often by surgical pocket elimination, of a more suitable environment (e.g., less anaerobic) for a more beneficial microbiota. Several studies indeed indicated that the presence of the previously mentioned periodontopathogens (persisting or reestablished after treatment) is associated with a negative clinical outcome of periodontal treatment.[67,121,311,312,364]

Taking all of this together, it is clear that the periodontal microbiota will shift when going from a periodontally healthy situation to a periodontally diseased situation (Figure 23-37).

Comparing the microbiota between health, gingivitis, and periodontitis, the following microbial shifts can be identified as health progresses to periodontitis:

- From gram-positive to gram-negative
- From cocci to rods (and at a later stage to spirochetes)
- From nonmotile to motile organisms
- From facultative anaerobes to obligate anaerobes
- From fermenting to proteolytic species

Periodontal Health

The recovery of microorganisms from periodontally healthy sites is lower when compared with that from diseased sites (Table 23-12). The bacteria associated with periodontal health are primarily gram-positive facultative species and members of the genera *Streptococcus* and *Actinomyces* (e.g., *S. sanguinis, S. mitis, A. oris*, *A. israelii, A. gerencseriae, A. viscosus* (see Figure 23-1, *C*), and *A. naeslundii*). Small proportions of gram-negative species are also found, most frequently *P. intermedia, F. nucleatum, F. nucleatum* ssp. polymorphum, *F. periodonticum, Capnocytophaga* spp. *(C. gingivalis, C. ochracea, and C. sputigena*), *Neisseria* spp., and *Veillonella* spp. Microscopical analyses indicate that a few spirochetes and motile rods also may be found. Based on checkerboard DNA-DNA hybridization data, *Eubacterium saburreum, Propionibacterium acnes, S. anginosus, S. gordonii,* and *S. oralis* can also be considered as health-associated bacteria.[92,382,429,430]

Certain bacterial species have been proposed to be protective or beneficial to the host, including *S. sanguinis, Veillonella parvula* (see Figure 23-1, *B*), and *C. ochracea.* They are typically found in high numbers at periodontal sites that do not demonstrate attachment loss (inactive sites) but in low numbers at sites where active periodontal destruction occurs.[81,362] These species probably function in preventing the colonization or proliferation of pathogenic microorganisms. Clinical studies have shown that sites with high levels of *C. ochracea* and *S. sanguinis* are associated with a greater gain in attachment after therapy, further supporting this concept.[362] A better understanding of plaque ecology and the interactions between bacteria and their products in plaque will undoubtedly reveal many other examples.

Gingivitis

The development of gingivitis has been extensively studied in a model system referred to as *experimental gingivitis* and initially described by Harald Löe and co-workers.[216,387] Periodontal health is first established in human subjects by cleaning and rigorous oral hygiene measures, followed by abstinence from oral hygiene for 21 days. After 8 hours without oral hygiene, bacteria may be found at concentrations of 10^3 to 10^4 per square millimeter of tooth surface and will increase in number by a factor of 100 to 1000 in the next 24-hour period.[367] After 36 hours, the plaque becomes clinically visible (see Video 23-1 online). The initial microbiota of experimental gingivitis (see Figure 23-21) consists of gram-positive rods, gram-positive cocci, and gram-negative cocci. The transition to

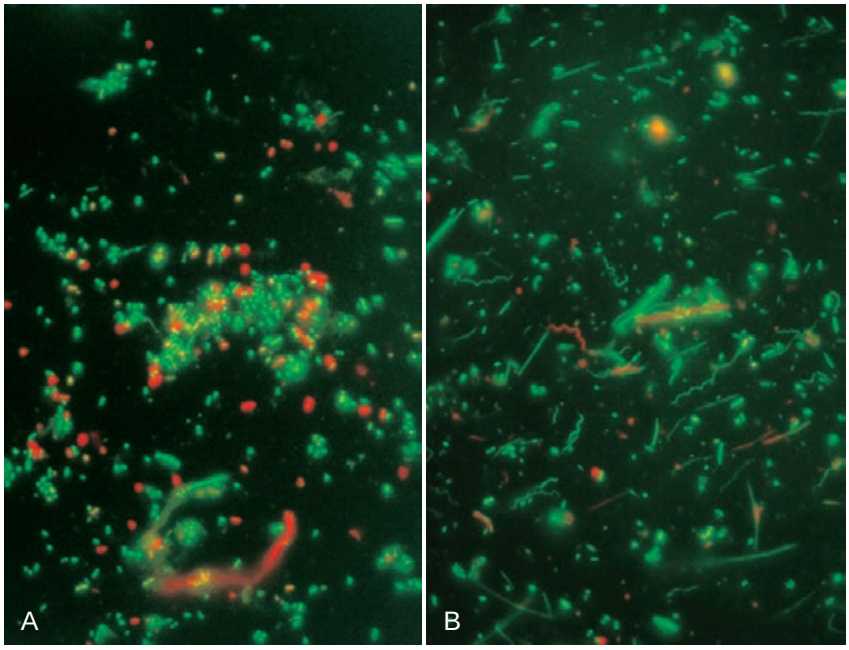

Figure 23-37 Vitality stain of subgingival plaque. **A,** Plaque derived from a healthy patient and primarily consists of cocci. **B,** Plaque derived from a periodontitis patient. Note the important morphologic difference with **A.** Green bacteria are alive; red bacteria are dead.

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TABLE 23-12 Summary of Bacterial Species Significantly Associated with Different Clinical Conditions (Based on Culture, Polymerase Chain Reaction (PCR), Checkerboard Data)

Health	Gingivitis	Chronic Periodontitis	Localized Aggressive Periodontitis	Aggressive Periodontitis	Necrotizing Periodontal Diseases	Abscesses of the Periodontium	Periimplantitis
Actinomyces gerencseriae *Actinomyces israelii* *Actinomyces naeslundii 1* *Actinomyces oris* *Actinomyces viscosus* *Capnocytophaga gingivalis* *Capnocytophaga ochracea* *Capnocytophaga* spp. *Capnocytophaga sputigena* *Eubacterium saburreum* *Fusobacterium periodonticum* *Fusobacterium polymorphum* *Neisseria* spp. *Propionibacterium acnes* *Streptococcus anginosus* *Streptococcus gordonii* *Streptococcus mitis* *Streptococcus oralis* *Streptococcus sanguinis* *Veillonella parvula*	*Actinomyces naeslundii 1* *Actinomyces viscosus* *Capnocytophaga* spp. *Streptococcus anginosus* *Streptococcus mitis* *Streptococcus oralis* *Streptococcus sanguinis* *Veillonella parvula* *Aggregatibacter actinomycetemcomitans* serotype a *Campylobacter concicus* *Campylobacter rectus* *Eikenella corrodens* *Eubacterium nodatum* *Fusobacterium nucleatum* *Haemophilus* spp. *Parvimonas micra* *Prevotella intermedia* spirochetes *Streptococcus intermedius* *Treponema* spp.	*Aggregatibacter actinomycetemcomitans* (serotype b) *Campylobacter rectus* *Eikenella corrodens* *Eubacterium nodatum* *Fusobacterium nucleatum* *Parvimonas micra* *Prevotella intermedia* Spirochetes *Treponema* spp. *Enterobacteriaceae* spp. *Leptotrichia buccalis* *Porphyromonas gingivalis* *Selenomonas noxia* *Tannerella forsythia* *Treponema denticola* *Prevotella nigrescens*	*Capnocytophaga* spp. *Aggregatibacter actinomycetemcomitans* high (Ltx clone) *Campylobacter rectus* *Eikenella corrodens* *Eubacterium nodatum* *Fusobacterium nucleatum* *Prevotella intermedia* Spirochetes *Treponema* spp. *Leptotrichia buccalis* *Porphyromonas gingivalis* *Tannerella forsythia* *Treponema denticola*	*Aggregatibacter actinomycetemcomitans* (serotype b) *Eubacterium nodatum* *Prevotella intermedia* *Spirochetes* *Treponema* spp. *Porphyromonas gingivalis* *Tannerella forsythia* *Treponema denticola* *Campylobacter gracialis* *Campylobacter showae* *Prevotella nigrescens*	*Fusobacterium nucleatum* *Prevotella intermedia* Spirochetes *Treponema* spp.	*Fusobacterium nucleatum* *Parvimonas micra* *Prevotella intermedia* Spirochetes *Porphyromonas gingivalis*	*Capnocytophaga* spp *Aggregatibacter actinomycetemcomitans* *Campylobacter rectus* *Fusobacterium nucleatum* *Parvimonas micra* *Prevotella intermedia* Spirochetes *Treponema* spp *Enterobacteriaceae* spp. *Porphyromonas gingivalis* *Tannerella forsythia* *Treponema denticola* *Pseudomonas aeruginosa* *Staphylococcus aureus* *Staphylococcus epidermidis*

Ltx, Leukotoxin.

gingivitis is evident by inflammatory changes and is accompanied first by the appearance of gram-negative rods and filaments, then by spirochetal and motile microorganisms.[387]

The subgingival microbiota of dental plaque-induced gingivitis (chronic gingivitis) differs from that of both health and chronic periodontitis (see Table 23-12). It consists of roughly equal proportions of gram-positive (56%) and gram-negative (44%) species, as well as facultative (59%) and anaerobic (41%) microorganisms.[354] It should be noted that the majority of predominant species in chronic periodontitis are already present in the gingivitis state, however, mostly in small numbers.

Predominant gram-positive species include *Streptococcus* spp. *(S. sanguinis, S. mitis, S. intermedius, S. oralis, and S. anginosus*), *Actinomyces* spp. *(A. viscosus, A. naeslundii*), *Eubacterium nodatum,* and *Parvimonas micra*. The gram-negative microorganisms are predominantly *Capnocytophaga* spp., *Fusobacterium* spp., *Prevotella* spp., *Campylobacter gracilis, Campylobacter concisus, V. parvula, Haemophilus* spp., and *E. corrodens.* Both groups are commonly associated with gingivitis. However, the following gram-negative species are commonly found in periodontitis but are also associated with gingivitis but in fewer numbers: *P. gingivalis, T. forsythia, P. intermedia, Campylobacter rectus, Treponema* spp., and *A. actinomycetemcomitans* serotype a.[244,250,354]

Pregnancy-associated gingivitis is an acute inflammation of the gingival tissues associated with pregnancy. This condition is accompanied by increases in steroid hormones in crevicular fluid and dramatic increases in the levels of *P. intermedia* and *C. rectus*, which use the steroids as growth factors.[181,433]

Studies of gingivitis support the conclusion that disease development is associated with selected alterations in the microbial composition of dental plaque and is not simply the result of an accumulation of plaque. Gingivitis was generally believed to precede the development of chronic periodontitis; however, many individuals demonstrate long-standing gingivitis that never advances to destruction of the periodontal attachment.[38,213]

Chronic Periodontitis

Numerous forms of periodontal disease are found in adult populations, characterized by different rates of progression (see Figure 23-34) and different responses to therapy.[15] Studies in which untreated populations were examined over long time intervals indicate disease progression at mean rates ranging from 0.05 to 0.3 mm of attachment loss per year (i.e., the gradual model).[38] When populations are examined over short time intervals, individual sites demonstrated short phases of attachment destruction interposed by periods of no disease activity (i.e., the burst model).[112] At this moment, by means of multilevel modelling, the linear and burst theories of periodontitis progression are considered to be a manifestation of the same phenomenon: some sites improve while others progress, in a cyclical manner.[107]

Microbiologic examinations of chronic periodontitis have been carried out in both cross-sectional and longitudinal studies; the latter have been conducted with and without treatment. These studies support the concept that chronic periodontitis is associated with specific bacterial agents. Microscopic examination of plaque from sites with chronic periodontitis has consistently revealed elevated proportions of spirochetes (see Video 23-4 online).[210,220] Cultivation of plaque microorganisms from sites of chronic periodontitis reveals high percentages of anaerobic (90%) gram-negative (75%) bacterial species[353,354] (see Figure 23-27 and Table 23-12).

In chronic periodontitis, the bacteria most often detected at high levels include *P. gingivalis, T. forsythia, P. intermedia, P. nigrescens, C. rectus, Eikenella corrodens* (see Figure 23-1, *S*), *F. nucleatum, A. actinomycetemcomitans* (often serotype b), *P. micra, E. nodatum, Leptotrichia buccalis, Treponema (T. denticola*), *Selenomonas* spp. *(S. noxia*), and *Enteric* spp.* When periodontally active sites (i.e., with recent attachment loss) were examined in comparison with inactive sites (i.e., with no recent attachment loss), *C. rectus, P. gingivalis, P. intermedia, F. nucleatum,* and *T. forsythia* were found to be elevated in the active sites.[80] Furthermore, detectable levels of *P. gingivalis, P. intermedia, T. forsythia, C. rectus,* and *A. actinomycetemcomitans* are associated with disease progression and their elimination via therapy is associated with an improved clinical response.[59,80,120,358,419] Both *P. gingivalis* and *A. actinomycetemcomitans* are known to invade host tissue cells, which may be significant in aggressive forms of periodontitis.[52,56,327]

Recent studies have documented an association between chronic periodontitis and viral microorganisms of the herpesviruses group, most notably EBV type 1 (EBV-1) and hCMV.[62] Further, the presence of subgingival EBV-1 and hCMV are associated with high levels of putative bacterial pathogens, including *P. gingivalis, T. forsythia, P. intermedia,* and *T. denticola.* These data support the hypothesis that viral infection may contribute to periodontal pathogenesis, but the potential role of viral agents remains to be determined.

Localized Aggressive Periodontitis

Several forms of periodontitis are characterized by rapid and severe attachment loss occurring in individuals during or before puberty. Localized aggressive periodontitis, previously referred to as *localized juvenile periodontitis* (LJP), develops around the time of puberty, is observed in females more often than in males, and typically affects the permanent first molars and incisors (Figure 23-38). This condition is almost uniformly seen in individuals who demonstrate some systemic defect in immune regulation, and often affected individuals demonstrate defective neutrophil function. Without treatment, the local form often extends to a more generalized form with severe attachment loss around many teeth. The first symptoms of LJP are already detectable in the deciduous dentition, especially via periodontal destruction around the canines and second molars.[48]

The microbiota associated with localized aggressive periodontitis is predominantly composed of gram-negative, capnophilic, and anaerobic rods[254,255,354] (see Table 23-12). Microbiologic studies indicate that almost all LJP sites harbor *A. actinomycetemcomitans,* which may comprise as much as 90% of the total cultivable microbiota.[182,247] Other organisms found in significant levels include *P. gingivalis, E. corrodens, C. rectus, F. nucleatum, B. capillus, Eubacterium brachy,* and *Capnocytophaga* spp. and spirochetes.[182,238,247,249] Recent checkerboard DNA-DNA hybridization studies have also shown high proportions of *E. nodatum, P. intermedia, Treponema* spp. *(T. denticola*), *L. buccalis,* and *T. forsythia.*[92,429] Herpesviruses, including EBV-1 and hCMV, also have been associated with localized aggressive periodontitis.[62,238,388]

A. actinomycetemcomitans is generally accepted as the primary etiologic agent in most but not all cases of localized aggressive periodontitis.[180,362] A high leukotoxic clone is uniquely associated with aggressive forms of periodontitis. However, the fact that not all aggressive periodontitis patients show this feature points to the possibility that several other factors may provoke this disease.[243] Studies of therapy indicate that mechanical debridement in combination with systemic antibiotic treatment are necessary to control

*References 183, 189, 217, 250, 354, 361, 362, 379, 382, 429, and 430.

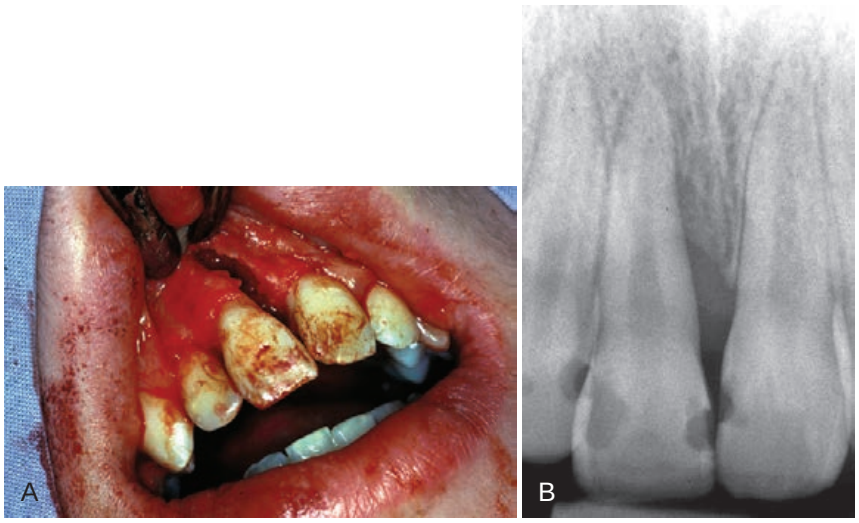

Figure 23-38 Clinical picture **(A)** and intraoral radiograph **(B)** of the dramatic bone destruction in adolescent with localized aggressive periodontitis.

Figure 23-39 Clinical picture of lower front teeth with necrotizing gingivitis.

the levels of *A. actinomycetemcomitans* in this disease.[182,313,314] The failure of mechanical therapy alone may relate to the ability of this organism to invade host tissues.[52,56,327]

Aggressive Periodontitis

Aggressive periodontitis, also known as *generalized aggressive periodontitis, early-onset periodontitis,* or *rapidly progressive periodontitis,* is a severe form of periodontitis that occurs at a relatively young age (between 20 to 40 years). It is characterized by a severe gingivitis, a large number of deepened pockets, and a high tendency for bleeding on probing. In relation to the patient's age, there is a lot of bone destruction. It is sometimes considered as a generalized form of localized aggressive periodontitis. From a microbiologic point of view, it has a lot of similarity toward localized aggressive periodontitis (see Table 23-12). However, aggressive periodontitis is clearly more dominated by *P. gingivalis, P. intermedia, T. forsythia,* and *Treponema* spp.*(T. denticola*) and less by *A. actinomycetemcomitans*. Recent checkerboard DNA-DNA hybridization studies indicate also high proportions of *E. nodatum, Campylobacter gracilis, Campylobacter showae,* and *P. nigrescens*.[92,429]

Necrotizing Periodontal Diseases

Necrotizing periodontal diseases (Figure 23-39) present as an acute inflammation of the gingival and periodontal tissues characterized by necrosis of the marginal gingival tissue and interdental papillae. Clinically, these conditions often are associated with stress or HIV infection. They may be accompanied by malodor, pain, and possibly systemic symptoms, including lymphadenopathy, fever, and malaise. Microbiologic studies indicate that high levels of *P. intermedia* and especially of spirochetes and *F. nucleatum* are found in necrotizing ulcerative gingivitis lesions. Spirochetes are found to penetrate necrotic tissue and apparently unaffected connective tissue.[211,212]

Abscesses of the Periodontium

Periodontal abscesses (Figure 23-40) are acute lesions that may result in very rapid destruction of the periodontal tissues. They often occur in patients with untreated periodontitis but also may be found in patients during maintenance or after scaling and root planing of deep pockets. Periodontal abscesses also may occur in the absence of periodontitis; for example, associated with impaction of a foreign object (e.g., a popcorn kernel or dental floss) or with endodontic problems.[142] Typical clinical symptoms of periodontal abscesses include pain, swelling, suppuration, bleeding on probing, and mobility of the involved tooth. Signs of systemic involvement may be present, including cervical lymphadenopathy and an elevated white blood cell count.[141] Investigations reveal that bacteria recognized as periodontal pathogens are commonly found in significant numbers in periodontal abscesses. These microorganisms include *F. nucleatum, P. intermedia, P. gingivalis, P. micra,* and *T. forsythia.*[124,141,254]

Periimplantitis

The term periimplantitis refers to an "inflammatory process" affecting the tissues around an already osseointegrated implant resulting in loss of supporting bone.[15] This inflammatory process has been associated (e.g., in animal studies, cross-sectional and longitudinal observations in man) with a microbiota comparable to that of periodontitis (high proportion of anaerobic gram-negative rods, motile organisms, and spirochetes), but this association does not necessarily prove a causal relationship (for review see Quirynen et al).[295] Healthy periimplant pockets are characterized by high proportions of coccoid cells, a low ratio of anaerobic/aerobic species, a low number of gram-negative anaerobic species, and low detection frequencies for periodontopathogens.[3,30,198,263] Implants with periimplantitis reveal a complex microbiota encompassing conventional periodontal pathogens (see Table 23-12). Species, such as *A. actinomycetemcomitans*, *P. gingivalis*, *T. forsythia*, *P. micra*, *C. rectus*, *F. nucleatum, P. intermedia, T. denticola,* and *Capnocytophaga,* are often isolated from failing sites but can also be detected around healthy periimplant sites.[343] Other species, such as *Pseudomonas aeruginosa*, *Enterobacteriaceae* spp, *C. albicans,* or staphylococci are also frequently detected around implants.[7] These organisms are

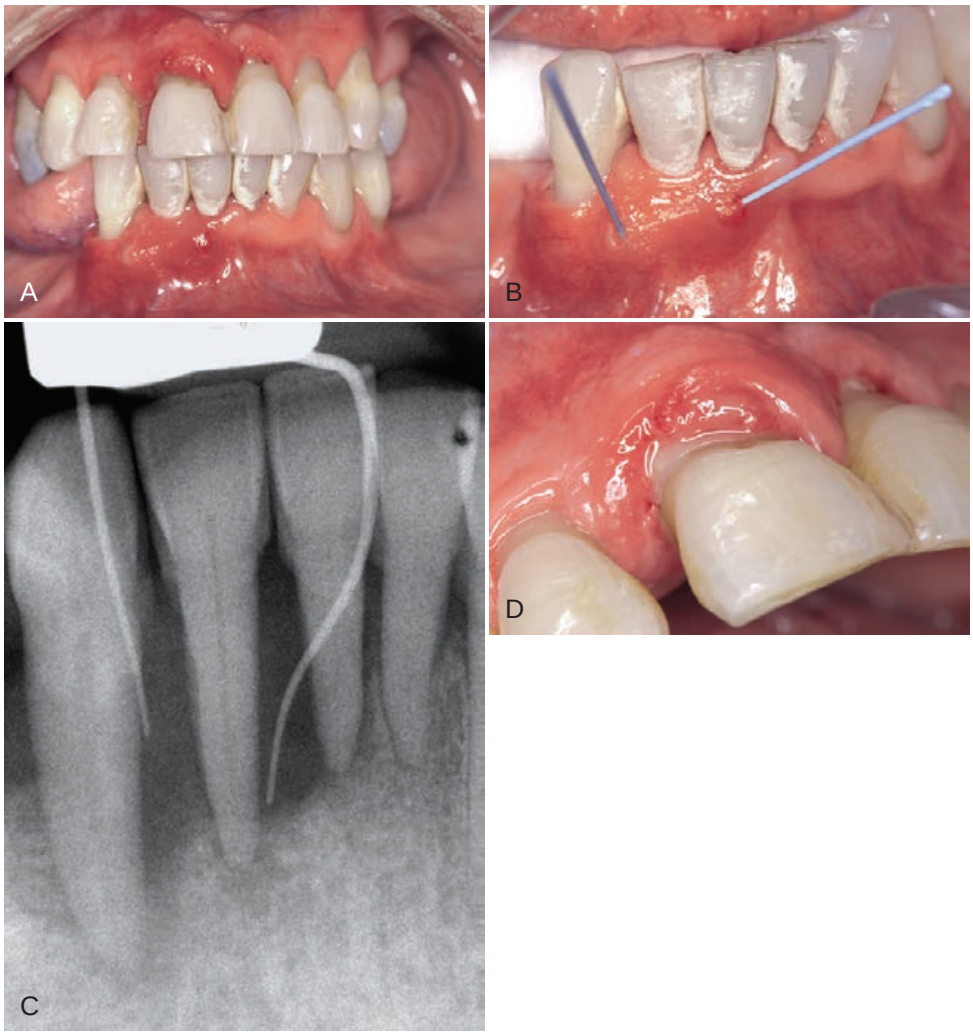

Figure 23-40 Clinical pictures (**A, B,** and **D**) of a patient with several periodontal abscesses. The intraoral radiograph **(C)** shows the severity of the periodontal destruction. Gutta-percha points (**B** and **C**) are inserted in the fistulae to show their course.

uncommon in the subgingival area but have been associated with refractory periodontitis.[359] High proportions of *Staphylococcus aureus* and *S. epidermidis* on oral implants have been reported.[308] The relative resistance of these organisms to commonly utilized antibiotics suggests that their presence might represent an opportunistic colonization secondary to systemic antibiotic therapy.[358]

For more information on periimplantitis, please visit the Expert CONSULT companion website at www.expertconsult.com.

VIRULENCE FACTORS OF PERIODONTOPATHOGENS

It is clear that some organisms, such as *P. gingivalis, A. actinomycetemcomitans,* spirochetes, and *P. intermedia,* are strongly associated with a number of periodontal diseases. However, periodontal disease never occurs in the absence of a complex microbiota and it is often difficult, if not impossible, to determine precisely how different organisms contribute to an individual case of disease. In fact, the contributions of specific bacteria to disease may be unimportant according to the ecological plaque hypothesis. Targeting one or more "pathogens" will not necessarily cure disease, since other organisms with similar activities might take their place. It may make sense therefore to focus on the specific molecules that contribute to disease (virulence factors), rather than on the microorganisms that produce them. In fact, it is often difficult to separate the virulence determinants from the organisms that produce them. For example, adhesins are produced by commensal organisms, as well as by pathogens, yet only those adhesins that promote attachment of a pathogenic organism could be considered virulence determinants. With this in mind, some of the known or putative virulence factors for periodontal disease are described below. It is important to note the following:

1. Only a proportion of periodontal bacteria have ever been isolated, and there are almost certainly many other virulence factors that are currently unknown.
2. Most of our understanding of virulence factors has come from studies on a very limited number of bacterial species and strains. It is far from clear that the molecules that have been studied in the greatest detail are truly representative of their classes.

Virulence factors of periodontal micro-organisms can be subdivided into (1) factors that promote colonization (adhesins), (2) toxins and enzymes that degrade host tissues, and (3) mechanisms that protect pathogenic bacteria from the host.

Adhesive Surface Proteins and Fibrils

To colonize the periodontal pocket, bacteria must adhere to cells or tissues in the region such as teeth, the existing microbial biofilm, or the pocket epithelium. Bacterial cell surface structures provide the points of contact. Often, these structures extend some distance from the cell surface. Fimbriae, or pili, are polymeric fibrils composed of repeating subunits that can extend several microns from the cell membrane. Pili were once thought to be unique to gram-negative bacteria but have now been identified in several gram-positive organisms, including streptococci and actinomyces.[230] Strains of *P. gingivalis* produce two types of fimbriae, known as the *major* and *minor fimbriae*.[126]

For more information on adhesive surface proteins and fibrils, please visit the Expert CONSULT companion website at www.expertconsult.com.

Factors that Promote Tissue Destruction

Many bacterial proteins that interact with host cells are recognized by the immune system and trigger immune responses. Fimbriae of *P. gingivalis* and *A. actinomycetemcomitans* are highly antigenic. Inflammation is a major contributor to tissue destruction in periodontal disease and is considered separately in Chapter 21. However, a number of bacterial products directly promote tissue destruction in addition to modulating host immunity, and the most notable of these are the extracellular proteolytic enzymes.

Bacterial proteolytic activity in dental plaque, and particularly trypsin-like protease activity, is closely correlated with clinical markers of periodontal disease.[260]

For more information on factors that promote tissue destruction, please visit the Expert CONSULT companion website at www.expertconsult.com.

However, the bulk of the host tissue degrading activity is restricted to a small number of these enzymes. In the case of *P. gingivalis*, three enzymes known as *gingipains* are responsible for at least 85% of the total host protein degradation activity.[281]

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Gingipains are multifunctional proteins that play important roles in adhesion, tissue degradation, and evasion of host responses.

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Proteases are produced by *A. actinomycetemcomitans* but appear to be less important for the virulence of this organism than the leukotoxin (LtxA).

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Most strains of *A. actinomycetemcomitans* produce low levels of LtxA. However, some strains are regarded as hyperleukotoxic, since they express high levels of the transcript from the *ltxA* gene, encoding LtxA. Hyperleukotoxic strains include the JP2 clone of *A. actinomycetemcomitans*, which is uniquely associated with localized aggressive periodontitis.[138,243] Two distinct mechanisms that give rise to high levels of *ltxA* expression have been described.[335] The JP2 clone contains a deletion in a regulatory gene, designated *orfA*, upstream of the *ltxCABD* gene locus. Hyperleukotoxic *A. actinomycetemcomitans* strains isolated in Japan do not contain deletions in *orfA* but instead harbor a mobile genetic element (transposon), IS*1301*, upstream of the *orfA* gene. The sequence of IS*1301* contains elements that direct enhanced transcription of the *orfA-ltxCABD* operon. From a diagnostic point of view, it is important to consider that simply detecting *A. actinomycetemcomitans* and/or LtxA in the periodontal biofilm is by no means indicative of disease. However, detection of a hyperleukotoxic strain of *A. actinomycetemcomitans* may be significant.

Strategies for Evading Host Immunity

Pathogenic bacteria have many and varied strategies for evading or subverting the host immune system, including (1) the production of an extracellular capsule, (2) proteolytic degradation of host innate and/or acquired immunity components, (3) modulation of host responses by binding serum components on the bacterial cell surface, and (4) invasion of gingival epithelial cells. A detailed description of bacteria-host interactions is given in Chapter 25. Selected examples of factors that mediate these processes are given next.

Strains of *P. gingivalis* produce polysaccharide capsules (Figure 23-41) that surround the outer membrane. Six different antigenic capsule types have been described based on differences in the polysaccharide K antigen.[190] In a mouse model, capsular strains of *P. gingivalis* produced a spreading type of infection, whereas acapsular strains tended to form more localized abscesses.[192] The majority of *P. gingivalis* isolates from periodontitis patients are encapsulated.[191] It is hypothesized that the capsule protects cells from the host immune system. However, protection against the complement system appears to be mediated primarily by a branched phosphomannan polysaccharide that is independent of the K antigen.[350]

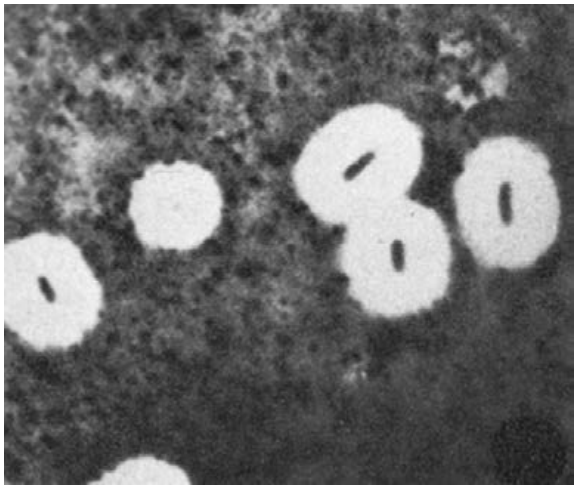

Figure 23-41 Glycocalyx or polysaccharide capsule of *Porphyromonas gingivalis* visualized with a East-Indian ink staining (clear halo).

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Several periodontopathogens are resistant to complement-mediated phagocytosis, and it is thought that proteolysis of complement components contributes to resistance to some extent. *P. gingivalis* gingipains have been shown to degrade complement components C3, C4, C5, and factor B.[280] More recently, a cysteine protease of *P. intermedia* termed *interpain A* has also been shown to degrade C3.[282] In vitro, interpain A acts synergistically with gingipains to decrease deposition of complement C3b.[282] These data fit with the hypothesis that periodontal disease processes are mediated by polymicrobial consortia rather than by individual periodontopathogens.

A novel mechanism for complement evasion has been identified in *A. actinomycetemcomitans*. This organism produces a 100 kDa protein on the outer membrane (Omp100 or ApiA), which mediates adhesion to and invasion of host cells.[17,204] Mutants lacking ApiA were sensitized to killing by human serum: wild-type cells were almost completely resistant to 30% or 50% normal human serum, whereas 90% of ApiA mutant cells were killed. ApiA was shown to bind factor H, an inhibitor of the complement cascade. Interestingly, H_2O_2 produced by *S. gordonii* induces the expression of ApiA and enhances the serum resistance of *A. actinomycetemcomitans*.[309] It has been proposed that H_2O_2 derived from oral streptococci may stimulate the immune response and that early detection of H_2O_2 by *A. actinomycetemcomitans* may provide an ecologic advantage under these conditions.[309] Alternatively, the ability of *A. actinomycetemcomitans* to detect streptococcal H_2O_2 may be a fortuitous consequence of a system that has evolved primarily to detect and respond to the neutrophil oxidative burst.

Periodontal pathogens are notoriously difficult to eradicate completely. Despite aggressive antibacterial attacks from the host defenses and from clinical treatments, it is often impossible to prevent reinfection. One potential reservoir for periodontopathogens is inside gingival epithelial cells. *P. gingivalis* and *A. actinomycetemcomitans*, for example, can invade epithelial cells in vitro.[193,236] In vivo, *P. gingivalis*, *T. forsythia*, *P. intermedia*, *T. denticola,* and *A. actinomycetemcomitans* can be detected within gingival epithelial cells of periodontitis patients before and after periodontal therapy.[160] Adhesion to host cells is critical for invasion. However, it has been suggested that there may be factors that specifically control the ability of bacteria to invade cells. In *A. actinomycetemcomitans*, a screen for invasion-associated genes identified two loci, encoding ApiA (see previous paragraph) and ApiBC that are related to known bacterial invasion proteins (invasins).[204] The ability of bacteria to invade cells may be influenced by their interactions with other organisms in the gingival sulcus. Thus coinfection of an epithelial cell line with *P. gingivalis* and a coaggregating strain of *F. nucleatum* resulted in increased invasion by *P. gingivalis* compared with a monoculture control.[328] Coaggregation with *F. nucleatum* is mediated by the *P. gingivalis* capsular polysaccharide and lipopolysaccharide, and therefore these molecules may indirectly contribute to invasion.[318]

SCIENCE TRANSFER

Subgingival plaque is an example of a complex biofilm composed of up to 150 bacterial species present at any one site with sharing of nutrients and deoxyribonucleic acid (DNA) and significant interdependencies between species for metabolites and adhesion. Biofilms give bacteria up to 1500 times more resistance to antimicrobial agents than that seen when bacteria exists in a liquid environment. There are significantly more bacteria present in diseased pockets (10^8 bacteria) compared to healthy gingival crevices (10^3 bacteria). One tooth surface may have as many as 10^9 bacteria in its supragingival biofilms.

After a tooth surface is cleaned during a prophylaxis treatment, a dental pellicle consisting of polysaccharides and glycoproteins from saliva appears on the surface within 1 minute. This is quickly followed within 3 minutes by the first bacterial components of the biofilm. These first bacteria include gram-positive cocci, which can initiate caries. It takes at least 4 to 7 days before the gram-negative anaerobic proteolytic bacteria related to periodontal disease occur. Thus caries can begin immediately, particularly if exposure occurs to sucrose from food. Gingival inflammation has a longer lag period.

There are specific groups of bacteria related to periodontal breakdown. These include the red complex of *Porphyromonas gingivalis, Tannerella forsythia,* and *Treponema denticola.* The broader group of putative periodontal bacteria can be seen in healthy sites but are only present in larger numbers in periodontal pockets. Viruses, such as human cytomegalovirus, may accentuate the destructive capacity of bacterial plaque.

The most effective method for changing the subgingival biofilm in patients with periodontal disease is the mechanical debridement using curettes and ultrasonic instruments. If this is followed by appropriate periodontal therapy that reduces pocket depths, then the patient is able to restrict the formation of a subgingival pathogenic biofilm with effective oral hygiene techniques.

At present, only a proportion of anaerobic gram-negative bacteria can be cultivated from periodontal pockets and many potential pathogenic species cannot be identified. This restricts the value of diagnostic cultural tests, and in the future, more sophisticated bacterial species identification procedures involving DNA will be needed before a clear picture of the role of periodontal microbiology in human periodontal disease can be substantiated.

FUTURE ADVANCES IN PERIODONTAL MICROBIOLOGY

Scientific progress at the end of the twentieth century, particularly in the field of molecular biology, has led to significant advances in our understanding of periodontal microbiology. DNA-based methodologies for the identification and detection of specific bacteria and viruses offer remarkable time and cost advantages, compared to culturing techniques. A dramatic increase in the number of samples that can be examined and the number of microorganisms enumerated is now possible. Perhaps even more relevant is the present ability to detect microorganisms that cannot be cultivated so far, which has underscored the limitations of our knowledge of this complex ecologic niche. Greater awareness of the role of the host response in periodontal disease will further improve the understanding of the severity and therapy of periodontal infections. Finally, the recognition of the beneficial activity of several groups of commensal species might open new strategies for treating periodontal disease, for example, using probiotics or microbial replacement therapy.[386]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 24

Genetic Factors and Periodontal Disease

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CHAPTER OUTLINE

GENETIC AND GENOMIC METHODS OF THE 21st CENTURY
Patterns in Populations and Pedigrees
Searching for Answers in the DNA
INHERITED VARIATION AND RISK OF PERIODONTITIS
Periodontitis in Genetic Syndromes and Other Diseases
Nonsyndromic Aggressive and Chronic Periodontitis
Challenges and Opportunities for the Future
INHERITED VARIATION AND TREATMENT OF PERIODONTITIS
Pharmacogenomics and Individualized Dentistry

It is now widely accepted that differences among individuals at risk for developing most diseases have a substantial inherited component. Factors in the environment, such as diet, smoking, preventive care, or exposure to pathogens, interact with each person's genetic predisposition to determine his or her health outcomes. This complex combination of variables determines if and when a disease affects the person, how fast and how severely symptoms of the disease progress, and how the person responds to different treatments in terms of both side effects and success of alternative therapies. Sometimes, as in diseases such as cystic fibrosis or muscular dystrophy, the genetic component of risk predominates, and differences in environment play only a minor role. In other diseases, factors in the environment are most important, and variation inherited in the person's deoxyribonucleic acid (DNA) has only an infrequent or minor influence on disease susceptibility or progression. Examples of the latter include infectious diseases, such as human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) or cancers, such as mesothelioma (strongly associated with asbestos exposure). The majority of human diseases fall half-way between these two extremes; genes and environment both play very important roles.

Most cases of periodontitis appear to fit this complex genes and environment model. With the exception of a handful of very rare syndromes caused by mutations in single genes, evidence indicates that inherited variation in DNA has a role roughly equal to that of the environment in determining who remains periodontally healthy versus who is affected by this disease. Beyond this broad generalization, however, and despite hundreds of studies of genetic polymorphisms reported to date, our understanding as to which specific genes are important remains extremely limited. We know even less about the likely (though unproven) role that individual genetic differences may have in determining how patients respond to alternative periodontal treatments.

In this chapter, the challenges and barriers that have thus far limited progress in advancing our knowledge of the complex genetics of periodontitis will be reviewed. This requires a clear understanding of not only the architecture of the human genome and the complexity of genetic susceptibility but also the critical roles that statistical power and sample size play in the process of discovery. These latter issues are important for all areas of research, but especially for situations in which a large number of variables need to be evaluated. In genetic studies, over 23,000 genes need to be considered as potential hypotheses or "candidates" for being disease risk factors. Depending on the gene's size and the frequency of genetic recombination in the gene's chromosomal region, scientists need to evaluate between a handful or up to several hundred inherited DNA variants in each gene as potential "biomarkers" of disease risk. Each one of these variants essentially amounts to a test of the hypothesis as to whether the variant is associated with disease risk. Furthermore, many complex diseases have been found to be very strongly associated with DNA variation in parts of the human genome where no genes are known to exist, but where the genetic material may have important functional effects nonetheless. Therefore, to fully evaluate the entire human genome, the total number of hypotheses that need to be tested is truly enormous—roughly involving the equivalent of one million independent statistical tests.

Only in the past few years have laboratory and computational tools been developed that make this scale of work technically possible, and thus far only one recent study has reported results of a whole genome analysis for periodontitis.

Opportunities for advanced study designs and next-generation genomic technologies to improve understanding of the inherited basis of periodontitis make it likely (although by no means certain) that genetic variation will become an important variable to be routinely considered by practicing dentists within the next 5 to 10 years. If and when this occurs, dentists will need to learn how to access and interpret the massive amounts of information coded in the >3 billion bases of DNA in the human genome and to incorporate this into improved evidence-based practices for periodontal disease prevention, diagnosis, and treatment. Alternatively, new interdisciplinary teams will need to be established with dentists as key members working closely with experts in bioinformatics, genomics, and genetic counseling so that patients will be able to reap the potentially significant benefits of these scientific advances. However the process evolves, it will surely lead to revolutionary changes in both dental education and dental practice in the not-so-distant future.[8,23]

GENETIC AND GENOMIC METHODS OF THE 21st CENTURY

Most health care professionals are aware of many major advances in genetics accomplished during the past 2 decades since the Human Genome Project officially began in 1990.[41] Headlines and announcements of "breakthroughs" have appeared regularly in print and television media. Too often, many of these stories seem to promise rapid and significant improvements in health care that are totally unrealistic.[11] More cautious assessments are usually not deemed as newsworthy by the media nor is cautious understatement always in the interest of private companies and granting agencies involved in basic research and clinical trials. Consequently, the public has often received an overly optimistic picture of what the future of medical care based on genomic medicine really holds for them. Despite this all-too-common overstatement about rapid translation to clinical practice, advances in technical capabilities for genomic data acquisition and the accumulation of knowledge in the field of genetics have been truly enormous. The eventual impact of this explosion of biologic knowledge on all areas of human health, including those concerning the field of dentistry, over the longer term is certain to be very substantial.

Unfortunately, for reasons explored further in the next section, genomic advances have thus far contributed very little to advancing our understanding of the molecular-pathologic causes of periodontitis or pointed to ways for improving treatment through "individualized" approaches based on patients' inherited genetic variation. Only recently has the possibility of making major progress been realized based on newly available genomic tools and approaches of Genome Wide Association Studies (GWAS, pronounced "gee-wahs") and "next generation" DNA sequencing techniques. For these research strategies to be successful, however, they must be combined with further improvement in research definitions of periodontal disease and much larger sample sizes than have been used in most previous genetic studies of this condition. To help improve understanding, some of the commonly used terms in genetics are explained in Table 24-1.

TABLE 24-1 Glossary of Terms Relevant to the Genetics of Periodontal Disease

Allele	One of several possible alternative forms of a gene caused by small or large differences in the DNA sequence within or near the gene. These differences arise by mutation, and some may affect the function of the gene product (i.e., a protein) or its abundance in different kinds of cells.
Autosome	A chromosome that is not a sex chromosome.
Autosomal dominant	DNA variation in a gene located on an autosome that has a dominant effect over other forms of variation at this location within the gene. When the dominant DNA sequence is present in combination with some other sequence, the gene's function is entirely or nearly entirely determined by the dominant sequence while the alternative sequence that occurs on the person's other chromosome is essentially silent.
Autosomal recessive	DNA variation in a gene located on an autosome that has an effect on the gene's function only when the person has inherited two copies, one from the mother and the other from the father. For example, if an individual has two copies of an abnormal gene that is autosomal recessive, they will be subject to the effects of that gene.
Chromosome	A nuclear structure that contains genetic information. Humans have 46 chromosomes, arranged in 23 pairs. There are 22 pairs of autosomes and a pair of sex chromosomes (either XX or XY).
Concordance	The probability that a pair of individuals (e.g., twins) both have a certain characteristic (e.g., periodontal disease), given that one of the pair has the characteristic. Presented as a number from 0 to 1, or as a percentage.
Dizygotic twins	Twins that have resulted from the fertilization of two separate eggs. They are no more similar to each other (from a genetic perspective) than are nontwin siblings. Nonidentical twins.
Epigenetics	Term used to describe the changes in phenotype or gene expression that result from mechanisms other than changes in the underlying DNA sequences (i.e., changes in which the gene is expressed rather than a change in the DNA sequence itself). Nongenetic factors cause the organism's genes to be expressed differently.
Exon	Protein coding regions of DNA.
Frameshift mutation	A mutation that results from insertion or deletion of one or more nucleotides into a gene, causing the coding regions to be read in the wrong frame and usually causing the protein produced to be defective in function.
Gene	The basic unit of heredity that occupies a specific position (locus) on a chromosome and has specific effect(s) on the phenotype of the organism. A piece of DNA that is transcribed into a molecule of RNA and then translated into a protein.
Gene expression	The process by which the information in a gene is utilized via transcription and translation, leading to production of protein. Differences in gene expression can affect the phenotype of the organism including risk of disease.

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TABLE 24-1 Glossary of Terms Relevant to the Genetics of Periodontal Disease—*cont'd*

Genetic code	In RNA and DNA, the consecutive nucleotide triplets (codons) that specify the sequence of amino acids for protein synthesis (translation).
Genome	The entire hereditary information of an organism. Refers to all the genes and other nongene portions of DNA carried by an individual cell.
Genotype	The genetic makeup of an organism or cell, distinct from its expressed features or phenotype.
Haplotype	A contraction of the term *haploid genotype*. Refers to a combination of alleles at multiple loci, which are usually transmitted together on the same region of a chromosome.
Heredity	The passing of traits to offspring from parents/ancestors. In biology, the study of heredity is referred to as *genetics*. As a result of heredity, variation among individuals allows species to evolve by natural selection in response to changes in their environment or by random change over long periods of time.
Homozygous	The presence of identical alleles at a specific position in a gene.
Heterozygous	The presence of two different alleles at a specific position in a gene.
Intron	A DNA region within a gene that is not translated into protein. These intervening (noncoding) portions of DNA or RNA are removed during RNA processing.
Isoform	Any of several different forms of the same protein. May be produced from related genes, or may arise from the same gene by alternative splicing. Many isoforms are caused by SNPs.
Ligand	A molecule that binds to another molecule, usually binding to a cellular receptor molecule.
Linkage	A term to describe the tendency for certain genes to be transmitted from parent to child together because they are located close to each other on the same chromosome.
Linkage disequilibrium	The occurrence of specific alleles at different locations in the DNA that are relatively close to each other (linked) more often than would be expected by chance alone (disequilibrium).
Locus	The physical location a gene occupies within a chromosome. (Plural: loci).
Monozygotic twins	Twins with identical genetic makeup (i.e., identical twins) as a result of fertilization of a single egg that then splits into two embryos.
Mutation	Changes in the DNA sequence of the genome can result from errors that occur during DNA replication or meiosis and can be caused by radiation, viruses, mutagenic chemicals. Most mutations have very little or no measurable effect on the gene's function; some are harmful and a rare few may be advantageous.
Nucleotide	Molecules that when linked together make up the structural units of RNA and DNA. They are composed of a phosphate group, the bases adenine, cytosine, guanine, and thymine, and a pentose sugar. In RNA the thymine base is replaced by uracil.
Penetrance	The proportion of individuals who have a particular allele/genotype who express an associated trait (phenotype). Genotypes with a high penetrance result in a larger number of individuals in the population with the associated phenotype compared to genotypes with a low penetrance.
Phenotype	The observable characteristics displayed by an organism (e.g., morphology, development, gender, eye color, physiologic properties, or behavior). Phenotype results from expression of the organism's genes, as well as the influence of environmental factors, and interactions between the two.
Polymorphism	Polymorphism exists when two or more different phenotypes exist within different individuals of the same population. In the context of genetics, it refers to a region of the genome that varies between individual members of the population in such proportions that the rarest of them cannot be maintained just by recurrent mutation. Polymorphism may be actively maintained in populations by natural selection and also by random drift.
Sequencing	Determining in the laboratory the linear arrangement of nucleotides (in RNA or DNA) or amino acids (in proteins).
Single nucleotide polymorphism (SNP)	A polymorphism in a gene caused by a change in a single nucleotide in the DNA sequence. A large number of protein isoforms result from SNPs. SNPs occur frequently, approximately every 100-1000 base pairs as a result of deletions, insertions, and substitutions. There are estimated to be >10 million SNPs in the human genome. Many SNPs that occur in genes have no effect on the encoded protein, but some SNPs do influence the function of the protein that the gene produces. An SNP initially arises as a very rare mutation but is considered to be an SNP if it occurs in at least 1% of the population.
Signal transduction	A cascade of intracellular events after binding of an extracellular signal (e.g., a hormone or a cytokine) to a receptor on the cell surface. The intracellular cascade can result in changes in gene expression in the nucleus and hence an altered phenotype of the cell (e.g., as a result of different protein production).
Splicing	The removal of introns from transcribed RNA. The process of removal can vary, and some exons are skipped or excluded from splicing. This causes production of "splice variants" or "alternatively spliced" protein isoforms, resulting in the formation of different proteins from the same initial RNA.
Transcription	RNA synthesis. The process of creating an RNA copy of an equivalent section of DNA is the first step of gene expression, and occurs in the nucleus. The RNA copy that is produced is called *messenger RNA* (mRNA).
Translation	The first stage of protein synthesis. mRNA produced in transcription is decoded to produce an amino acid chain that will later fold into an active protein. Translation occurs in the cytoplasm: ribosomes bind to the mRNA and then facilitate decoding by binding of transfer RNAs (tRNAs) that have complementary anticodon sequences to that of the mRNA. The tRNAs carry specific amino acids that are joined together to form a polypeptide as the mRNA passes through the ribosome.

DNA, Deoxyribonucleic acid; *RNA*, ribonucleic acid.

Patterns in Populations and Pedigrees

With all the attention focused on silicon arrays, laser scanners, and the other "glamorous" gadgets of advanced genomic technologies, it is important to consider how much needs to be learned about the genetic basis of a disease before stepping into a DNA laboratory. In fact, a strong foundation of knowledge about a disease's frequency in different populations and occurrence among closely and distantly related family members (pedigrees) is absolutely essential. Without this foundation, endless gigabytes of DNA sequence data will not enable scientist-clinicians to develop a solid understanding of the causes of a disease. The human genome has not evolved in a test tube nor inside a supercomputer but has existed for millennia in natural populations, transmitted from generation to generation—from parents to child. Only by carefully studying the genetics of a disease in populations and pedigrees can we hope to begin to unearth the complex interactions between genes and the environment that underlie individual differences in disease susceptibility. This field of research is known as *genetic epidemiology*.

Genetic epidemiologists often first look at whether a disease occurs more often in some human populations than others. These comparisons can include both populations in different geographic areas, as well as racial or ethnic groups living in the same region. Does the disease have more severe symptoms, or more rapid rates of progression, or an earlier age of onset in some populations? Such findings suggest (but do not prove) that genetic differences that are important for the disease may exist among the populations.

Before the massive human migrations in recent centuries, most human populations existed in semiisolation from other populations around the globe. As a consequence of natural selection (differential survival and/or reproduction), populations sometimes adapted genetically to their local environment. The most famous example of adaptation is the sickle cell hemoglobin variant that protects against the infectious disease malaria. This variant is common in populations living in areas where this mosquito-borne parasite has long been endemic because it provides strong protection against the severe symptoms of this disease. The variant persists at high frequency in these populations even though persons who inherit two copies of the mutation (one from mother and one from father) are severely affected by the disease sickle cell anemia. A balance between the benefit of malaria resistance in persons who inherit only one copy of the variant versus the disadvantage of sickle cell disease keeps the variant at relatively high frequencies in populations where malaria is present. Another example of population differentiation by natural selection is the ability to digest the milk sugar lactose as an adult that evolved in Europeans in conjunction with the domestication of dairy cattle over 8000 years ago. In addition to differentiation driven by natural selection as in these examples, a random process called *genetic drift* also causes populations with little or no migration between them to differentiate genetically over time, so one cannot assume that every population difference observed has a functional biological basis.

Regrettably, comparison of periodontitis in different populations across the globe is extremely challenging because of the lack of calibrated examiners and standardized disease definitions.[6] One of the most dramatic population differences in which data quality is not an issue is the observation that both localized and generalized forms of early-onset aggressive periodontitis occur about 10 times more frequently in African Americans compared to Caucasians.[28] Human racial and ethnic groups often differ dramatically in frequency of mutations at genes that have major effects on disease risk. For example, cystic fibrosis is caused exclusively by recessive mutations in the CFTR gene and varies in frequency from 1 in 3000 Caucasians to 1 in 15,000 African Americans in the United States (US), whereas only 1 in 350,000 Japanese are affected.[44] It is possible that the tenfold higher prevalence of early-onset aggressive periodontitis in African Americans is caused by elevated frequency of high-risk gene variants in this population. However, additional evidence is needed before drawing such a conclusion. Although comparative studies of different populations may provide clues as to possible genetic mechanisms underlying a disease, the environments of the populations may also be dissimilar in important ways. It is possible that variation in diet or in exposure to pathogenic oral bacteria or to some unknown and unmeasured environmental factors could entirely explain the observed difference in frequency of aggressive periodontitis between population groups. Until solid data confirm a genetic basis for population differences, we need to wait before drawing firm conclusions.

Comparison of disease occurrence or severity in identical (monozygotic) versus nonidentical (dizygotic) twins is a very powerful method for distinguishing between effects caused by variation in genes versus factors in the environment. This requires us to make what is usually a reasonable assumption that the environments of pairs of identical twins are no more or less similar than the environments shared by pairs of nonidentical twins. If variation among individuals in disease susceptibility or severity is caused entirely by factors in the environment, then we expect pairs of identical twins to be no more similar to each other than pairs of nonidentical twins. All twins (whether identical or nonidentical) are expected to be more similar to their co-twin on average than to unrelated members of their local population because they were raised in the same family environment (with similar diet, microbial exposures, etc). However, if genetic variation plays an important role in determining the trait, then genetically identical twin pairs will be more similar to each other than nonidentical twin pairs. This is because identical twins share 100% of the same genes, whereas nonidentical twin pairs share only 50% of their parents' genes on average. Genetic epidemiologists calculate a measure based on these correlations called *heritability* that estimates the portion of all variation in the trait that is attributable to inherited genetic variation. Traits whose variation is determined entirely by differences in environmental exposures have heritabilities of 0.0, whereas traits with variation attributable solely to inherited genetic differences without any environmental influence have heritabilities of 1.0. Heritabilities are sometimes reported as a percentage ranging from 0% to 100%.

Most human diseases and nondisease traits fall in the middle of this range with heritability ranging between 0.25 and 0.75. For example, type II diabetes was estimated to have a heritability of 0.26 and abnormal glucose tolerance a heritability of 0.61 in one study.[45] Note that for it to be feasible to use the twin method with adequate statistical power, the disease has to be fairly common so that the researcher can recruit enough twin pairs in which at least one of the twins is affected by the disease. For periodontal diseases, not surprisingly, only chronic periodontitis occurs frequently enough to have been studied using the twin design. Two twin studies of modest size (110 and 117 pairs) have been reported, and these estimate heritability of measures of chronic periodontitis ranging between 40% to 80%, thus clearly implicating genetic variation in disease risk.[38,39] Interestingly, a study of bacteria associated with periodontitis found no difference between identical versus nonidentical twins.[40] This suggests (at least for these twins, most of whom did not have severe periodontitis) that inherited variation in risk is not mediated by genes that influence the presence of specific bacteria in subgingival plaque.

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Another method used by genetic epidemiologists to understand and distinguish different mechanisms of transmission of diseases through families is called *segregation analysis.* This is relatively straightforward for traits in which mutation in a single gene causes the disease to develop with nearly 100% certainty in carriers, whereas persons who do not inherit the mutation are at little or no risk. For example, carriers of a single copy of the Huntington's disease gene mutation or carriers of two copies of a cystic fibrosis gene mutation always develop these diseases if they reach the ages at which symptoms of these conditions normally emerge. By tracking the transmission of these diseases in families, it is obvious, for example, that Huntington's disease is a dominant single gene disorder because it is transmitted with 50% probability to offspring of affected individuals and thus it is often found occurring across many generations of large pedigrees. By contrast, parents of cystic fibrosis cases are very rarely affected themselves and 25% of the siblings are affected by cystic fibrosis when the disease is present in a nuclear family. This pattern of transmission is expected if a disease is recessive (i.e., it requires inheritance of a mutated gene copy from both parents, who themselves have one normal and one mutated copy and so are not affected). For most common "complex" diseases, however, having a high-risk gene does not automatically lead to development of the disease (a phenomenon called *reduced penetrance*). Furthermore, several genes or even dozens of different genes may influence disease susceptibility (known as the *oligogenic inheritance* and *genetic heterogeneity*). Environmental exposures are also important modifiers of disease risk. Such highly complex combinations of multiple genetic and environmental risk factors make the challenge of deciphering genetic mechanisms by merely observing transmission patterns in families using the segregation analysis approach unfeasible. The limitations of this approach were humorously illustrated a number of years ago in an analysis that facetiously presented evidence of a recessive gene controlling the trait of attending medical school.[34] "Risk" for this outcome among first-degree relatives of a doctor was elevated 61 times above that of the general population. More recently, a robust quantitative analysis of the family histories of characters in the Harry Potter series suggested a dominant gene controls inheritance of magic abilities.[49] Because the etiology of periodontitis is likely to be highly complex, segregation analyses of this disease that have been reported in the literature should be viewed with considerable skepticism. Unfortunately, the simplifying assumptions required for this method make the results unreliable and potentially misleading. For highly complex diseases, such as most cases of periodontitis, assays at the DNA level need to be combined with careful evaluations of clinical measures among related individuals to derive robust conclusions about a disease's genetic architecture. Some of the key features of the different techniques for studying the genetics of periodontal disease are explained in Table 24-2.

Searching for Answers in the DNA

In theory, a *genetic marker* can be any type of biomolecule or assay that allows us to "read" inherited differences among individuals in their DNA sequences. Blood groups, protein isozymes, and human leukocyte antigens (HLAs) were among the first-developed markers, but even simple traits controlled by single genes, such as eye color, can also serve this purpose. Genomic methods have made these methods obsolete, as researchers can now determine a person's inherited variation directly at the DNA level for much lower cost and with greater speed and accuracy. So-called "next generation" DNA sequencing methods are projected to enable researchers and clinicians to obtain nearly the entire 3 billion DNA base human genetic blueprint for less than $1000 a few years from now.[15,60] At present, most genetic studies use a combination of whole genome arrays that can evaluate up to 1 million variable DNA sites in a single assay combined with lower-throughput methods used for fine mapping of chromosome regions of special interest.[48] These regions are said to contain *candidate genes* of high priority for further investigation either because of the genes' known biological functions or because results of previous genome-wide surveys indicate strong statistical chances that disease susceptibility genes are located in certain regions of one or more chromosomes. Types of variation include single nucleotide polymorphisms (SNPs, pronounced "snips") in which one DNA base is substituted for another, small insertions and deletions ("in/dels") of 1 or more DNA bases, and larger structural changes in the DNA such as inversions (in which a piece of DNA of hundreds or thousands of bases in size is cut out and sewn back into the chromosome in the opposite orientation) and copy number changes (in which a given segment of DNA is either missing or occurs in more than the usual two copies inherited as one copy from each parent).

Equipped with these powerful tools for rapidly measuring DNA variation, the next question is what kinds of study designs are most powerful for identifying the dozen or more variants that influence risk of disease from among the millions of DNA differences that exist between any two individuals in a typical population. One method that has been highly successful in finding molecular defects for simple genetic diseases (caused by mutation in a single gene), such as cystic fibrosis and Huntington's disease, is *linkage analysis.*[1,7] This gene-mapping strategy requires families with one or more members affected by the disease to be recruited and clinically and molecularly evaluated for a relatively small number of genetic markers. Depending on the type of marker used, as few as 500 markers or up to 10,000 markers distributed evenly across the genome are needed. Investigators usually try to recruit families with two or more close relatives, such as sibling pairs, who are affected by the disease, as well as parents and other siblings who may be unaffected. Under the *null hypothesis* that a region of the chromosome does *not* contain genetic variation that influences disease risk, siblings are expected to share identical genetic material inherited from their parents 50% of the time on average. However, if the region of the chromosome being evaluated contains a gene that has a substantial effect on disease risk (e.g., increases risk by tenfold or more), then pairs of siblings who are both affected by the disease will share the chromosome region containing the disease gene substantially more often than 50% of the time, and the null hypothesis of 50% sharing will be statistically rejected if the study has an adequately large enough sample of such families. This simple example illustrates how linkage analysis is performed. In practice, both small and large extended families are studied, including simultaneous evaluation of the sharing of genetic material among both affected and unaffected relatives. Sophisticated mathematical algorithms and computer programs are used to carry out the huge number of calculations required for data analyses.

After achieving many successes using linkage analysis for "simple" diseases caused by mutation of a single gene, this method was extended to complex diseases caused by combinations of multiple susceptibility genes and environmental risk factors. Unfortunately, these conditions proved to be beyond the reach of linkage analysis in most instances. Numerous studies conducted during the 1990s either failed to find any genes or initially positive findings failed to replicate. Linkage studies of very large numbers of carefully diagnosed families for complex diseases, such as orofacial clefting in which twin studies had firmly established heritability of 70%, identified at most a tiny fraction of this genetic variation. Mathematical analyses have subsequently shown that the linkage

TABLE 24-2 Techniques for Studying the Genetics of Periodontal Disease

Candidate gene approach	A gene mapping approach that tests whether one allele of a gene occurs more often in patients with the disease than in subjects without the disease. These methods are also referred to as *association analyses* and aim to identify which genes are associated with the disease. Candidate genes are chosen on the basis of their known or presumed function (i.e., they have some plausible role in the disease process such as producing a protein that is important in the disease pathogenesis). Conceptually, this makes sense but requires some knowledge of the candidate gene to look for it!
Case-control studies	Studies in which the genetic makeup is compared in cases (who have the disease in question) and controls (who do not). The populations need to be carefully matched, otherwise apparent observed differences between cases and controls could arise because of ethnic or geographic variation, for example.
Twin studies	Comparisons of traits, including diseases in monozygotic, dizygotic, or usually both types of twins aimed at determining whether variation in the trait among members of a population is caused by genetic variation in inherited DNA sequences, environmental exposures in the subjects' previous lives, or some combination of both of these processes. Twin studies often measure the *concordance rates* of twins with a particular trait or disease of interest. Monozygotic (identical) twins are very nearly identical in their DNA, whereas dizygotic (nonidentical) twins share on average half of their DNA as identical sequences inherited from their parents. If a disease has high heritability, identical twins will be more likely to either be both affected or both unaffected (concordant). However, this assumption is complicated in many diseases. A genetic mutation may not have complete *penetrance,* environmental conditions may contribute to the development of the disease (e.g., one twin may smoke and the other may not). Furthermore, many diseases are polygenic (i.e., caused by alterations in multiple genes).
Familial aggregation and relative risk	Many diseases run in families, and the degree of clustering within the family can be estimated by comparing the number of disease cases in relatives of patients to the risk of disease in the general population. Difficulties with this approach relate to the fact that in addition to having many genes in common, family members also share many aspects of a common environment (e.g., diet, nutrition, smoking, infectious organisms, and shared socioeconomic factors).
Segregation analyses	Statistical analyses of the patterns of transmission of a disease in families in an attempt to determine the relative likelihood that the disease is caused by a single gene with dominant or recessive inheritance, by multiple genes, or entirely by variation in exposure to risk factors. The observed proportions of offspring who have the trait or disease being evaluated (i.e., the phenotype) are compared with the proportions that are expected in the general population.
Linkage analysis	A technique used to map a gene responsible for a trait to a specific location on a chromosome. These studies are based on the fact that genes that are located close to each other on the chromosome tend to be inherited together as a unit. As such, these genes are said to be linked. Because linkage analysis requires use of initially very expensive DNA markers, this was originally only considered justified after finding strong evidence of a genetic basis for a trait using segregation analyses and/or family aggregation studies. A difficulty with linkage analyses is that many diseases are not caused by a single gene of "major" effect but rather by multiple genes of "minor" effect. In the latter situation, multiple genes each contribute a small amount to the phenotype/disease/trait, and the linkage study approach has little power for detection while association analysis methods may still be quite powerful.
Genome wide analyses	A Genome Wide Association Study (GWAS) investigates genetic variation across the entire genome simultaneously, with the aim of identifying genetic associations related to a trait or disease of interest. The completion of the Human Genome Project in 2003 and the development of microarray technologies capable of assaying >0.5M SNPs have made GWAS possible. This method has the potential to identify the genetic contributions to common diseases. An important advantage of this approach is, because the entire genome is analyzed, the technique permits the genetics of a disease to be investigated in a nonhypothesis-driven way. It is not necessary to correctly guess which candidate genes are most interesting to evaluate. A GWAS requires that well-characterized cases and controls be identified. A disadvantage of GWAS is that large clinical sample sizes are required to reduce the likelihood of differences between cases and controls being observed simply by chance as a result of the hundreds of thousands of multiple statistical tests required to search the entire human genome.

analysis gene-mapping strategy has extremely low statistical power for complex diseases in which each individual susceptibility gene has a relatively small effect on risk (e.g., twofold or less) and in which there is extensive heterogeneity among different families that have different combinations of susceptibility genes and environmental exposures.[50] Consequently, it is not surprising that linkage analysis has only been successfully applied to syndromic forms of periodontitis (summarized later).

Disappointment over this setback to human disease mapping caused by the initially unrecognized limitations of linkage analysis was short-lived. An alternative approach called *association analysis* was also available, although this had been relegated to studies of HLA and a few other markers of special interest during the prime of linkage approaches.[1,7] Mathematical analyses indicated that if we make some reasonable assumptions about the nature of genetic factors in human disease, this method could provide adequate statistical power for finding genes of small-to-modest effect on risk while requiring only moderately large sample sizes that would be feasible to recruit.[50] There was, however, one major catch—to search the entire genome using the GWAS method, the number of genetic

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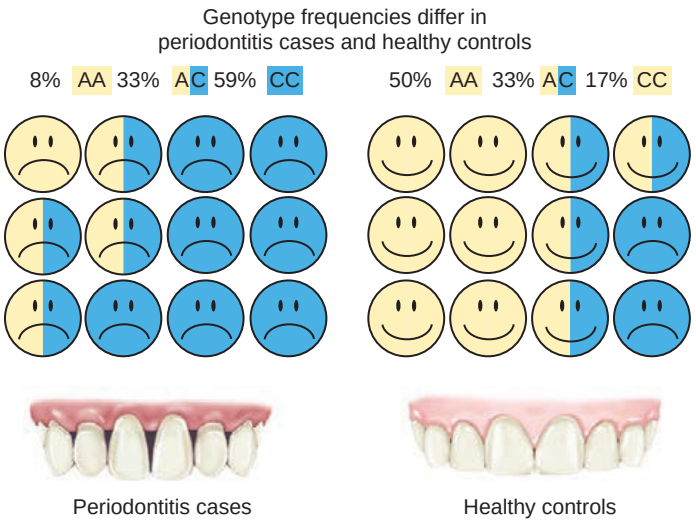

Figure 24-1 Case-control design for a genetic association study. Periodontitis cases shown on the left have frequencies for a hypothetical genetic marker of 8% AA, 33% AC, and 59% CC, which are substantially different from genotype frequencies found in healthy periodontal controls of 50% AA, 33% AC, and 17% CC. In this example, the CC genotype is associated with increased risk because it occurs at much higher frequency in the disease cases, whereas the AA allele is protective because this variant is much more common in the healthy controls.

markers needed was several orders of magnitude greater (500,000 to one million assays needed per subject). Fortunately, advances in molecular assay technologies converged at this time period and several methods of array-based genotyping provided such a capability at an acceptable cost.[48]

How association studies are used to find disease susceptibility genes is illustrated for the case-control design in Figure 24-1. Association analyses are sometimes referred to as *case-control* studies, although this is only one of several sampling methods that can be used (including studies of families). Genotype frequencies of an inherited DNA variant for a group of periodontitis cases are statistically compared to the frequencies of the variant in a matched group of periodontally healthy control subjects. If the genotype frequencies differ so greatly that the results are very unlikely to occur by chance, then we conclude that the genotype that is more common in the cases compared to the controls is "associated" with increased disease risk. In Figure 24-1, 59% of the cases have the CC genotype (having inherited a C allele from both of their parents), while only 17% of the healthy controls inherited the CC genotype. Therefore this DNA variant could be used to predict periodontitis risk (but not until after the finding was validated in additional, independent studies). Conversely, we can also say that the AA genotype is "protective" against disease because it occurs much more often in the healthy controls (50%) compared to periodontitis cases (8%). Ideally, the cases and healthy controls are matched as closely as possible for race and ethnicity, smoking behavior, age, gender, etc, so that differences in genotype frequency are likely to be caused by real biologic effects on disease development or progression rather than as artifacts of some kind. For example, it is well known that races and ethnic groups sometimes differ dramatically in genotype frequencies because of their historic isolation in different geographic regions. Consider, for example, a study that had mostly Swedish cases and mostly Italian controls. We know that there are thousands of DNA variants that differ substantially among these populations because of their geographic isolation in human history. Few, if any, of these variants have anything to do with differences in disease risk but could falsely appear to be associated because of the failure to carefully match ethnicity in cases and controls. In practice, this is usually not a problem provided that investigators take reasonable precautions in how cases and controls are selected. It is also now routine practice to use several statistical methods to check for mismatching and then adjust for this in data analyses if it occurs.

The good news is now clearly in: association studies have been a boon for discovery of inherited genetic variation important for a wide range of complex diseases, including diabetes, cardiovascular disease, metabolic disorders, obesity, and mental illnesses. A recent review cites 24 genes identified with unquestionable statistical confidence for type 2 diabetes alone and the list continues to grow.[57] Most of these genetic polymorphisms with elevated risk are very common in the population (from 5% up to >50%). Although each variant only increases risk slightly (twofold or less), because the risk alleles are so common, they can account for a nontrivial proportion of the occurrence of disease in the population (a measure epidemiologists called *attributable risk*).

An especially attractive aspect of the GWAS approach is that because the entire human genome is searched, we no longer have to depend on prior hypotheses about the disease's molecular pathology. In most GWAS studies, about half of the statistically definitive findings point to genes that experts in the field had no suspicion whatsoever were involved in the disease's etiology. This allows researchers to open up entirely new pathways for investigation that may lead to insights about the disease's biologic mechanism and suggest novel molecular strategies for pharmaceutical or other therapeutic interventions. In more than a few cases, robust GWAS findings implicate regions of the human genome in which no genes appear to be present, highlighting the limitations of our knowledge even today about basic genome functions.

Although great progress has been made in understanding the etiology of many complex human diseases using GWAS methods, the approach has nevertheless usually failed to account for most of the heritability known to exist for these conditions.[12,33] A recent study found that well-established nongenetic diabetes risk factors, such as gender, smoking, family history, body mass index, and blood lipids and glucose, were better predictors of risk than a combination of the top 20 genetic markers for this disease.[58] To improve gene-based risk estimates, the missing heritability needs to be found and high hopes are now being placed on the emergence of next generation DNA-sequencing tools. In theory, these may enable researchers to identify the less common (1% to 5%) genetic variants that are predicted to have individual gene effects of greater magnitude on disease risk (>twofold but <tenfold) that cannot readily be found using either GWAS or linkage analysis methods.

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INHERITED VARIATION AND RISK OF PERIODONTITIS

Because of the appropriate focus on the role of bacterial infections in the disease pathogenesis of periodontitis, inherited human genetic variation is often referred to as *host defense* or (somewhat more broadly) *host response.* These terms, however, cover only a small portion of the range of gene functions that may be important for periodontitis risk. Many additional biologic processes not directly related to defenses against or responses to infection by microbial pathogens are also likely to play important roles in determining an individual's susceptibility to this disease.

Periodontitis in Genetic Syndromes and Other Diseases

A number of extremely rare conditions consistently include periodontitis among the array of clinical manifestations that define a syndrome. Many genetic syndromes involve mutations of single genes or larger chromosomal regions. However, a number of syndromes, such as fetal alcohol syndrome, are purely environmental in origin. Some of the syndromes that include periodontitis are caused by mutations in specific genes. For example, mutations in the cathepsin C gene have been shown to cause both Papillon-Lefèvre (Figure 24-2) and Haim-Munk syndromes, some forms of nonsyndromic prepubertal periodontitis, and may also be associated with risk of aggressive periodontitis.[43] Periodontitis frequently occurs in some subtypes of Ehlers-Danlos syndrome, Kindler syndrome, Down syndrome (trisomy 21), leukocyte adhesion deficiencies (Figure 24-3), hypophosphatasia, two types of neutropenia, and aplasia of lacrimal and salivary glands. A large tri-racial extended family showed evidence of a single gene causing both early-onset aggressive periodontitis and dentinogenesis imperfecta. The gene has been mapped using linkage to a chromosomal region that contains a dentin matrix protein gene.[32] Many of these conditions are so rare that few periodontists see even a single case in a lifetime of practice. However, dentists should be aware that these single gene conditions exist and be prepared to extend clinical evaluations to close relatives and seek the assistance of or referral to appropriately trained genetics counselors or specialists if a patient's medical history or presentation of multiple symptoms raises the possibility that he or she may be affected. Clinicians can obtain updated information on these conditions by accessing the publicly available Online Mendelian Inheritance in Man database and typing in "periodontitis OR periodontal disease" as query terms.[2] Further research is necessary to determine whether inherited variation in the genes that cause these rare syndromes might also influence risk of nonsyndromic forms of aggressive or chronic periodontitis.

Nonsyndromic Aggressive and Chronic Periodontitis

In this section, evidence for association of inherited genetic variation with aggressive and chronic periodontitis will be considered for cases that present without the co-occurrence of anomalies or disorders of other parts of the body or of the subject's behavior. Such cases are appropriately classified as *nonsyndromic periodontitis.* This terminology is similar to the way that other human diseases, such as orofacial clefting, have long been recognized as occurring in syndromic and nonsyndromic forms. Elevated risk of periodontitis associated with metabolic conditions, such as diabetes (addressed elsewhere in this book), is more appropriately considered a "co-morbidity" rather than being designated a syndrome.

Figure 24-2 Oral **(A),** radiographic **(B),** and dermatologic (**C** and **D**) findings in Papillon-Lefèvre syndrome. Advanced periodontal disease usually affects the primary and secondary dentition shortly after teeth erupt, and many patients become edentulous while in their teens. Palmar-plantar keratodermas affect the palms of the hands and the plantar surfaces of the feet **(D)** but can also affect other sites, in this case, the elbow **(C).** *(Courtesy Dr. Robert J. Gorlin, Minneapolis.)*

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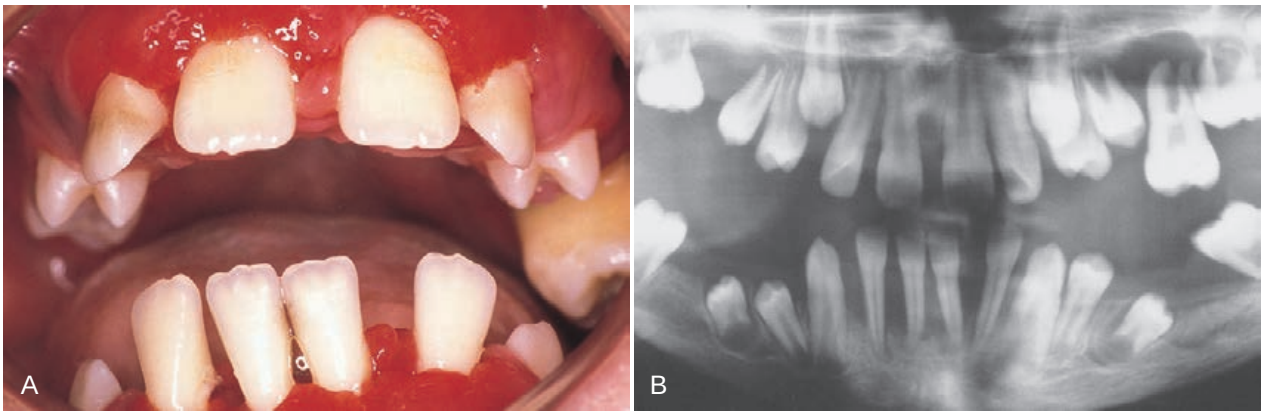

Figure 24-3 Oral **(A)** and radiographic **(B)** appearance of a patient with leukocyte adhesion deficiency (LAD). The child was deficient in CD18 (i.e., LAD type I), which results in absent or severely reduced levels of the β-2 integrin molecule. The patient suffered from recurrent infections of the middle ear, tongue, and perirectal areas, as well as the periodontium. (From Majorana *A, Notarangelo LD, Savoldi E et al:* Oral Surg Oral Med Oral Pathol Oral Radiol Endod 87:691, 1999.)

> "A finding of no statistical significance in a well designed, conceptually sound, adequately powered study testing an important hypothesis is likely to provide more useful information than significance in a study that does not meet these criteria."[5]

As reviewed above, twin studies have shown that chronic periodontitis has very substantial heritability and we know that aggressive periodontitis aggregates strongly in families. Since aggressive periodontitis occurs so rarely, it is not feasible to perform a twin study to confirm the heritability of this condition. Neither segregation analyses nor gene-mapping linkage studies are capable of providing reliable information about the genetic etiology of a highly complex disease such as periodontitis. However, large numbers of susceptibility genes have been identified for complex disorders, such as diabetes and cardiovascular disease, using association analysis. It seems reasonable to expect that similar successes could be achieved for periodontitis using this approach. In fact, during the past decade, several hundred papers have reported associations of nonsyndromic aggressive and/or chronic periodontitis with polymorphisms in a number of candidate genes. Certain classes of genes, such as cytokines, that have long been a focus of attention by immunologists and cell biologists studying pathogenic mechanisms associated with periodontitis have received the most attention. Early reports of relatively weak associations with variation in interleukin-1 (IL-1) genes led to a large number of attempts to replicate and extend these findings. Unfortunately, with very few exceptions, association studies of periodontitis have been very inadequately powered to detect genetic variation having modest effects on disease risk or progression (i.e., much too small sample sizes). In addition, inconsistency in the methods used to classify subjects as periodontal cases versus controls or to quantitatively measure disease severity and extent greatly limit our ability to draw sound conclusions by comparing results reported in different studies.

The reason why association studies of periodontitis have largely failed will be challenging to fully address. The first issue is straightforward, simply a matter of numbers. It is noteworthy that the successes achieved for many complex diseases, such as diabetes, using the GWAS mapping approach were based on sample sizes involving thousands of cases and controls, with multiple replications by *independent* teams of investigators. Statistical theory shows

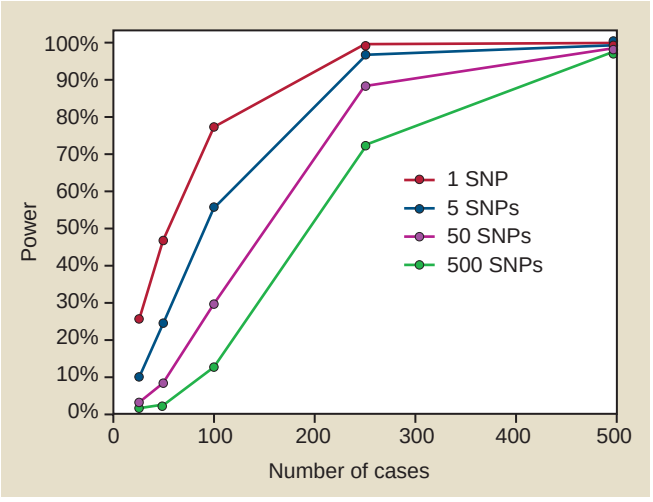

Figure 24-4 Statistical power estimates are shown for a situation in which we hypothesize that a dominantly transmitted disease susceptibility gene that increases risk by twofold and has an allele frequency in the population of 25% is being mapped by a case-control association study. The lines illustrate the loss of statistical power caused by the requirement to adjust for multiple comparisons when a research study involves the evaluation of not just a single genetic marker but as many as 5, 50, or 500 independent genetic polymorphisms. While only 100 cases (and 100 controls) may provide sufficient power if only a single marker is being tested, 250 cases and 250 controls are needed if 5 or 50 SNPs are assayed; a minimum of 500 cases and 500 controls are necessary if a study investigates 500 independent genetic markers.

that to detect genes of modest effect these large sample sizes are absolutely essential. Using a statistical power calculator developed for case-control studies,[47] sample sizes required for 80% power are shown in Figure 24-4 for a study that involves only a single genetic marker, as well as for studies that evaluate 5, 50, or 500 independent genetic markers in which the effects of multiple comparisons need to be accommodated. In this example, we assume that the risk gene acts in a dominant manner, with the high risk allele occurring at frequency of 25% in the population, and that this allele causes risk among carriers to increase by twofold (a greater effect on risk than observed for many susceptibility alleles found in GWAS studies of

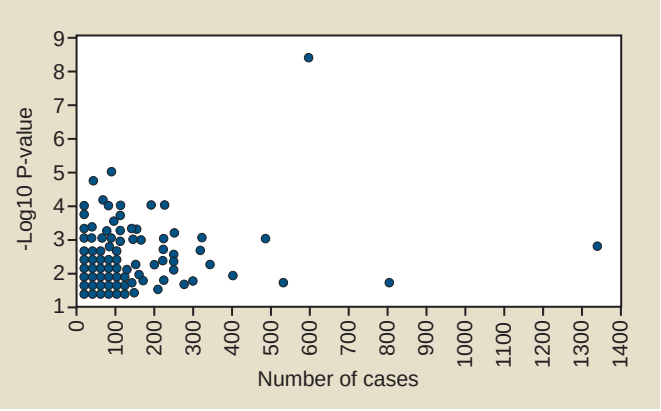

Figure 24-5 Number of cases and the P-values reported for 307 gene association tests for aggressive or chronic periodontitis reported in the literature. Only findings with P-values ≤0.05 are included, and when multiple tests were performed, the strongest association (i.e., the smallest P-value) is the one used for this presentation.

other complex diseases). Many periodontitis association studies reported in the literature involved multiple markers in each publication, and often the same research team reports positive findings for other genes in subsequent papers. Furthermore, because of the difficulties of publishing negative findings (i.e., when no association is found), many research teams working in this area may assay 50 or more genetic markers over the course of their work over several years. Results shown in Figure 24-4 demonstrate that to obtain 80% power a study of 50 markers would require over 200 cases and 200 controls. Even if a research team assays only 5 SNPs, their study would still require 100 cases and 100 controls in order to achieve adequate power.

Our review of the literature identified 307 periodontitis gene association tests with a P-value less than or equal to 0.05 for at least one statistical test performed on the data. These results are summarized in Figure 24-5 in which the x-axis indicates the number of cases included in the study and the P-value for the strongest finding is plotted on the y-axis. When more than one statistical test was reported, only the one with the smallest P-value is shown in this figure, thus in some cases inflating the actual statistical significance, as investigators rarely adjust their findings for these multiple tests when these are reported in a publication. This analysis shows clearly that most association findings have been drastically underpowered if periodontitis is assumed to be a complex disease. The majority (66%) of these association reports for chronic and aggressive periodontitis are based on samples of 100 cases or less, and 41% are based on less than 60 cases. As shown in Figure 24-4, studies of such small sample size have little power to detect a susceptibility gene that increases risk by twofold. Given the added concern about publication bias (i.e., that positive findings are more likely to be accepted for publication), we can have little confidence that even the more statistically significant findings are valid and likely to be independently replicated if based on such very inadequate samples sizes.

Aside from important lessons about how *not* to carry out association studies of a complex disease, there are some tentative conclusions that can be drawn from results reported to date. Results of our review and manual curation of 298 publications reporting association findings for periodontitis are presented in Table 24-3 online.

To view a comprehensive table on independent gene association reports for aggressive periodontitis and/or chronic periodontitis, please visit the companion Expert CONSULT website at www.expertconsult.com.

Key findings are condensed in Table 24-4. This summary includes a total of 41 genes plus a combination of the IL-1A and IL-1B genes analyzed jointly. Only genes that had at least two findings classified as "weak" or one finding of association with a "moderate" or "strong" level of support are included in this summary. The technical criteria for these designations are based on a combination of each study's sample size (assuming larger studies are less likely to produce false positives) and the study's P-value (statistical significance or type I error) as described in the Table 24-4 footnotes and in online supplementary documentation.

Genes in Table 24-4 have usually been evaluated in multiple, independent studies for association with either aggressive or chronic periodontitis or both. Occasionally, these subtypes of the disease have been pooled together, so it is not possible to determine from the publication whether the association involves primarily one subtype or both. With the exception of only the one GWAS study that has been published for this disease, all association tests reported to date involve evaluation of candidate genes that were selected based on known or postulated mechanisms of disease pathogenesis.

For example, when bacteria challenge gingival tissue, CD14 binds lipopolysaccharide and TLR4 plays a key role in pathogen recognition and activation of innate immunity.[26] Lactotransferrin (LTF) plays an antimicrobial role as the first line of host defense and can also neutralize endotoxin and inhibit the induction of nuclear factor-κB (NF-κB) in monocytes in response to LPS.[24,59] Myeloperoxidase (MPO) is an oxidative enzyme expressed in polymorphonuclear leukocytes. It is involved in the defense against periodontal bacteria and is also able to mediate inflammatory tissue destruction in periodontal disease.[35] Glutathione S-transferase mu 1 (GSTM1)[10] and *N*-acetyltransferase 2 (NAT2)[25] genes are responsible for detoxification of a wide range of chemicals, including tobacco carcinogens. HLA complex genes[56] play a central role in the immune system by presenting extracellular peptides that are important for either self-recognition or initiating immune responses to foreign pathogens. The Fcγ receptor genes (FcγRs) encode receptors for the Fc portion of immunoglobulin G and are involved in the removal of antigen-antibody complexes from the circulation, as well as other antibody-dependent responses.[30] Formyl peptide receptor (FPR1) is a G-protein coupled receptor of phagocytic cells interacting with bacterial peptides and mediates chemotaxis, degranulation, and superoxide production involved in inflammation.[17] Cytokines, such as interleukins (IL-1, IL-2, IL-4, IL-6, IL-10), tumor necrosis factor (TNF), and lymphotoxin alpha (LTA) play a number of important roles in the immunopathology of periodontal disease.[42] Prostaglandin-endoperoxide synthase (PTGS2), also known as *cyclooxygenase 2*, is the key enzyme in prostaglandin biosynthesis. It is regulated by specific stimulatory events, suggesting that it is responsible for the prostanoid biosynthesis involved in inflammation.[20] S100 calcium binding protein A8 (S100A8), the light subunit of calprotectin, is also associated with inflammatory diseases, including periodontitis.[27] Fibrinogen (FGB) is an acute-phase protein whose levels are elevated during inflammation and has been associated with cardiovascular disease risk.[51] Vitamin D receptor (VDR)[19] and estrogen receptor (ESR1)[61] are hormone receptors involved in skeletal muscle metabolism,

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TABLE 24-4 Number of Independent Gene Association Reports for Aggressive Periodontitis and/or Chronic Periodontitis*

Gene Symbol†	AGGRESSIVE PERIODONTITIS				CHRONIC PERIODONTITIS		
	None‡	Weak	Moderate	Strong	None	Weak	Moderate
CD14	3				7	5	
CDKN2BAS		1	1				
ESR1	1	1				2	
FCGR2A	8	1			12	3	
FCGR2B	2	1			2	1	
FCGR3A	7	1			7	3	
FCGR3B	3	8			11	2	2
FGB	1				1	2	
FPR1	4	4	1		2		
GLT6D1				1			
GSTM1			1			2	
HLA-A	3	11			2	5	
HLA-B	8	2			1	4	
HLA-C	8				1	2	
HLA-DQ	1	1			1	1	
HLA-DQB1	3	3			2		
HLA-DR	3	2			2	1	
HLA-DRB1	3	2			2	2	
HLA-DRB3/4/5	1	1			1	1	
IL10	10	2			10	4	2
IL1A	17	5			23	5	3
IL1A.IL1B	3	1			9	8	4
IL1B	16	9	1		29	11	2
IL1RN	10	2	1		10	3	1
IL2	2				2	2	
IL4	2	2			7	1	
IL6	4	3	1		8	11	
IL6R		1				1	
LTA					1	2	
LTF	1	3				1	
MMP1	2	1			5	3	
MMP9	1	1			1	3	
MPO						2	
NAT2						2	1
NOS3		1				1	
PTGS2			1			2	1
S100A8	1	2				1	
SERPINE1	1					2	1
TGFB1	1				4	3	
TLR4	6	1			12	1	1
TNF	13	1			17	6	
VDR	3	6			7	12	

*Only genes ≥2 reports of association are included.
†Full gene names and a summary of knowledge about the gene's function can be found by typing the gene symbol into the search box at: http://www.ncbi.nlm.nih.gov/gene.
‡Strength of association defined as: Weak (P-value <0.05 but ≥0.001 or if <50 cases in the study then the finding is considered weak regardless of P-value); Moderate (P-value <0.001 but ≥0.0001 or if <101 cases in the study regardless of P-value); Strong (P-value <0.0001 and >250 cases in the study).

including calcium absorption and bone loss. The matrix metalloproteinases (MMPs) are a group of endogenous proteinases that contribute to degradation of extracellular and basement membrane components.[18] CDKN2B antisense RNA (CDKN2BAS) is a nonprotein coding gene of unknown function that has also been reported to be associated with coronary heart disease.[52] The one GWAS reported for periodontitis (described in detail later) revealed strong association of a glycosyltransferase gene (GLT6D1) with aggressive periodontitis,[54] but there is no clear functional relationship of this gene with periodontal disease pathogenesis.

With few exceptions, there are just as many or more reports in Table 24-4 in which no significant association was found for the gene as there are positive findings. For example, HLA-B has nine negative reports and six weak positive findings. IL-1B has 45 negative reports, 20 weak associations, and 3 moderately supportive findings. The inconsistency among these findings requires a much deeper analysis to understand what may be going on. One possibility is that genetic variation at the candidate gene is *not* associated with periodontitis. When a large number of studies are performed using a multitude of alternative ways of classifying small numbers

of cases versus controls, when multiple alternative statistical analyses are run for the same small data sets, and when there may be a bias against publication of negative results, then a substantial portion of studies should report positive findings, even if no real association exists. Then again, heterogeneity among studies may be real. The negative studies may differ in terms of the racial or ethnic composition of the subjects, and the findings may be valid for some human populations but not others. Different genetic polymorphisms in the candidate gene may be evaluated in the different studies and only some of these may actually be associated with disease risk. Different clinical definitions or sources of information (e.g., clinical attachment loss versus bone loss measured from radiographs) may be used to define cases, or various quantitative measures might be employed, and these sources of variation might also influence the outcome of association tests. With data limited to mostly small studies, it is not currently possible to definitively determine which of these potential explanations applies to most of the findings shown in Table 24-4.

Figure 24-5 shows that a handful of periodontitis association studies have been reported that approach the sample size needed for complex diseases with over 400 cases.[14,29,36-37,52-53] This includes the GWAS in which over 322,825 SNP genetic markers were evaluated for association with risk of aggressive periodontitis.[54] In the GWAS, statistical testing was performed in sequence for 3 independent sets of samples with a total of 438 cases and 1320 controls. Interestingly, this relatively powerful analysis failed to yield significant support for "the usual suspect" candidate genes shown in Table 24-4, after adjusting for the large number of hypotheses tested in the analysis. Instead, several novel chromosomal regions and candidate genes were implicated, among the strongest being a glycosyltransferase gene (GLT6D1), one of several glycosyltransferases in the human genome. This had an unadjusted P-value of 0.000000006. However, the P-value was reduced by 3 orders of magnitude to 0.000006 after accounting for the effects of gender, smoking, and diabetes. The adjusted P-value indicates that we should expect to see a difference in genotype frequency of this magnitude by chance alone of about 1 out of 166,666 times under the null hypothesis of no association with disease risk. This may seem to be very strong evidence for rejection of the null hypothesis, but this adjusted finding is actually only a marginal statistical significance in the context of a GWAS. In fact, this sometimes occurs by chance alone in the absence of a valid association, since this study involved testing over 300,000 genetic markers. Because there is no any obvious functional connection of this gene with what we currently know about pathogenesis of periodontitis (the protein is thought to play a role in signalling in development), it might be tempting to further downplay the finding's significance. However, experience with GWAS studies of other complex diseases has shown that quite often valid gene associations are discovered in pathways that experts in the field did not previously realize were even related to the disease's biology. This makes such discoveries all the more valuable, assuming that they are definitively and independently replicated, as they offer the potential of revealing completely new avenues for further exploration at the cellular and molecular levels and potentially may also provide novel targets for therapeutic interventions. It will be very interesting and important to see whether the association with GLT6D1 and the other statistically interesting findings reported in this first periodontitis GWAS can be replicated.

Challenges and Opportunities for the Future

The classification of disease used for research studies is an especially difficult challenge to be faced if we are to benefit fully from the opportunities offered by the genomics revolution.[46] If we cannot agree which subjects in a study are affected by disease, by a particular subtype of disease, how severely they are affected, or how quantitative measures related to disease should be obtained and analyzed, then the chances of making progress will be very low, regardless of how advanced our molecular or bioinformatics technologies become. This problem is important for all kinds of research, not only genetics, and the issue of diagnosis is addressed elsewhere in this volume. However, genetics may have a unique role to contribute toward solving this dilemma. This may best be illustrated by an example from the early 1980s, in which medical geneticists and oncologists could not agree on classification of the disease neurofibromatosis (NF). "Splitters" argued for up to a dozen different etiologic subtypes, whereas "lumpers" suggested there might be only a single disease with a lot of variation among individuals as a result of differences in environmental exposures and variation in "genetic background" (a term that encompasses the cumulative influence of all other genes distributed throughout the genome in addition to the "major gene"). It had been discovered in research conducted in earlier decades that NF was transmitted in families as a single dominant gene, but the question remained as to how many different genes were involved (locus heterogeneity). Furthermore, even if only one gene was involved, it was possible that different mutations caused unique patterns of signs and symptoms (allelic heterogeneity). The controversy was largely resolved by the discovery of the NF1[31] and NF2[55] genes. After these genes were identified, individual patients could be distinguished by DNA testing, and the diagnostic classification controversies were resolved. In hindsight, it became clear that clustering of signs and symptoms fell into two major categories among different types of families and depended on which NF1 or NF2 mutation was involved and on what kind of gene mutation was involved. Although such gene-based clarity is unlikely for a complex multigenic condition such as periodontitis, the potential exists that subtypes of patients may eventually be classified more effectively by examining their DNA to see what kinds of susceptibility genes they inherited.

To move toward this desired outcome, however, we need to use the best strategies available today to classify research subjects into categories such as cases versus controls. Alternatively, we can attempt to use quantitative measures of bone loss or attachment loss to classify research subjects along a more continuous gradient ranging from persons with extremely healthy periodontium with little or no signs of disease to those with tooth loss and very high measures of pocket depth and attachment loss in remaining teeth. Family studies have numerous advantages in genetics, and for these designs, using a quantitative measures approach is especially attractive. When searching for gene associations in unrelated cases and controls, we can decide to select only subjects who are clearly affected as cases and compare these to subjects who clearly are very periodontally healthy assigned as controls. When studying families, however, one has to assign a disease status to all of the family members to fully utilize all of the information in the biologic unit. We may initially recruit the family based on an unambiguous case known as the *proband* (the subject who makes the family eligible for inclusion in the study), but the handling of parents, siblings, and other close relatives who may be neither clearly periodontally healthy nor clearly diseased is not easy to decide. For example, if we establish a threshold and require that two or more teeth have a minimum of 4 mm attachment loss for subjects to be classified as affected, then how we handle family members who are borderline becomes the challenge—siblings of the proband might have several teeth at 3 mm attachment loss in addition to one tooth with 6 mm loss. Such subjects are close to but not quite over the threshold.

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They are neither clearly healthy nor clearly diseased, and this makes them very problematic for categorical data analyses that require them to be classified as cases versus controls.

One aspect of the diagnosis challenge that is especially poorly understood is how and why different teeth are affected by periodontitis. Dentists have long recognized that incisors and first molars are more likely to be affected in early-onset disease, but it is not straightforward to incorporate this information into threshold disease definitions. One way to begin to address the problem of variation among teeth is to apply multivariate methods, such as principal components analysis, as was done in a study of aggressive periodontitis.[13] This method maximizes the proportion of the total variation in attachment loss among all 28 teeth explained by a limited number of master variables called *principal components*. In this study, three principal components explained 77.8% of the total variance. The results are displayed in Figure 24-6 using color intensities to help visualize the patterns of correlation among different tooth types. Mixtures of red, green, and blue are "painted" onto the teeth, with each color's intensity adjusted according to the tooth's relative "weight" calculated for each of the principal components. This quantitative analysis shows how attachment loss is correlated among different kinds of teeth. First molar teeth are consistently painted yellow color by their principal components scores and these teeth are very different from all of the other teeth in their patterns of attachment loss. There is a gradual change in the patterns of attachment loss for the other teeth extending out from the central incisors (magenta) to the lateral incisors (purple), cuspids (blue), premolars (blue green), and second molars (green). In addition to generally exploring patterns of periodontal disease dispersed in the "geography" of the mouth, because these are studies conducted in families we can also use genetic epidemiologic methods to validate and compare alternative measures of disease.

Specifically, we can calculate heritability for different quantitative variables such as simple mean attachment loss averaged across all teeth, or averaged for specific groups of teeth, as well as for more complex variables like these principal components. In the study of aggressive periodontitis illustrated in Figure 24-6, the principal component most heavily weighted on first molars had heritability of 30%, which was actually slightly higher than the 26% heritability estimated for a simple mean of attachment loss in first molars. By using heritability and other genetic measures such as association with specific inherited polymorphisms, it may be possible to refine diagnostic and disease classification systems by aligning these according to subgroups of subjects who share homogeneous etiologies.

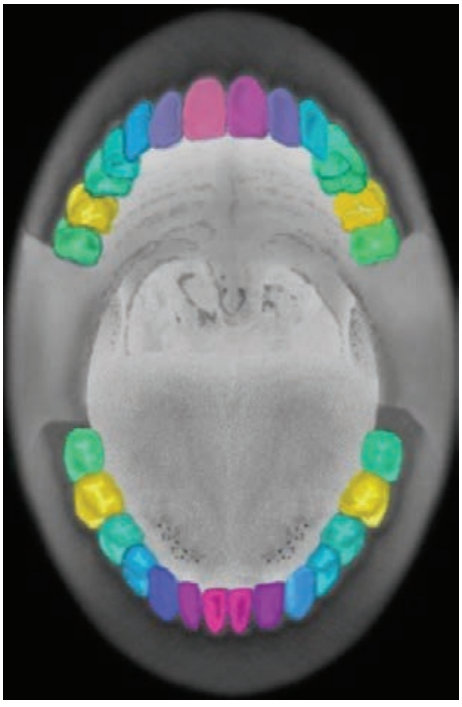

Figure 24-6 Illustration of the similarity and differences among different kinds of teeth in the frequency and severity of attachment loss observed in early onset aggressive periodontitis patients and their unaffected family members using the principal components analysis method.[13]

> Learning from the experience of other complex diseases, Sir Isaac Newton famously remarked in 1676: "What Descartes did was a good step. You have added much several ways … If I have seen a little further it is by standing on the shoulders of Giants."

Although there remains great enthusiasm for GWAS studies, as was noted, even the most successful GWAS results, such as those for type 2 diabetes, have failed to identify most of the genetic variation responsible for the disease. Discovery of over 30 gene associations for type 2 diabetes accounts for a small fraction of the disease's heritability.[12] To track down the missing genetic variation, a great deal of attention is now turning to whole genome DNA-sequencing methods that will be available at very low cost within 3 to 5 years. Sequencing methods also expand capabilities for measuring gene expression in various tissues, and these data, combined with the ever-increasing availability of proteomic assays, will challenge investigators with huge quantities of information. Aside from generating the many gigabytes of data involved in this technology, the greatest challenges of "systems biology" may lie in developing the bioinformatics tools to sift through and identify the key bits of information important for advancing our knowledge from among the vast rising sea of biologic data pouring from our laboratories.[4]

Strategies for improving our understanding of the genetics of periodontitis, as well as possibilities for translation of this knowledge to benefit patients in the clinic, are being mapped-out today by teams of geneticists, clinicians, and information scientists who are currently focused on major medical conditions. By continuing to learn from the experiences of these early genomic explorers, it is reasonable to hope that dental researchers will be able to avoid some of their mistakes and follow the quickest path to advances in knowledge about our diseases of interest.

INHERITED VARIATION AND TREATMENT OF PERIODONTITIS

Pharmacogenomics and Individualized Dentistry

If individuals differ in their susceptibility to disease and especially if underlying the superficial signs and symptoms of a disease there really are etiologically distinct subtypes of disease pathogenesis, then the traditional medical model of "one size fits all" will not be optimal. As shown in Figure 24-7, cases of disease (illustrated by circles with unhappy faces) may differ in their genotype for a gene that determines which treatment works best for each individual. Some cases inherited a G allele from both their mother and their father and have GG genotype, whereas others have GT or TT genotype. The gene might distinguish between different subtypes

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Some rare diseases are associated with a mutation of the gene that controls cathepsin C (a molecule expressed in control of various components of the immune response) that have aggressive periodontitis as part of their manifestation (e.g., Papillon-LeFevre syndrome). Also, there are various groups of genetic abnormalities that result in significant periodontal breakdown (e.g., neutropenia, Down syndrome, Ehlers-Danlos syndrome, and hypophosphatasia).

The human genome has an enormous number of genes that could partially influence the susceptibility of patients to periodontitis, but at present, there is no strong evidence to show any specific genetic pattern that can be absolutely tied to disease activity. The genes controlling interleukin-1 (IL-1) production have a weak connection with periodontal susceptibility in some populations. Genetic investigations using a new genetic identification tool named *Genome Wide Association Studies* (GWAS) have shown a statistical relationship between periodontitis and the gene GLT6D, which controls glycosyltransferase. The clinical mechanism for this association has not been elucidated.

The most important payoff for clinicians would be to identify those patients whose genetic encoding could influence response to periodontal therapy. Once again, at present, no connections have been confirmed, and the genes controlling IL-1 polymorphism do not apparently play a significant role in prognosis.

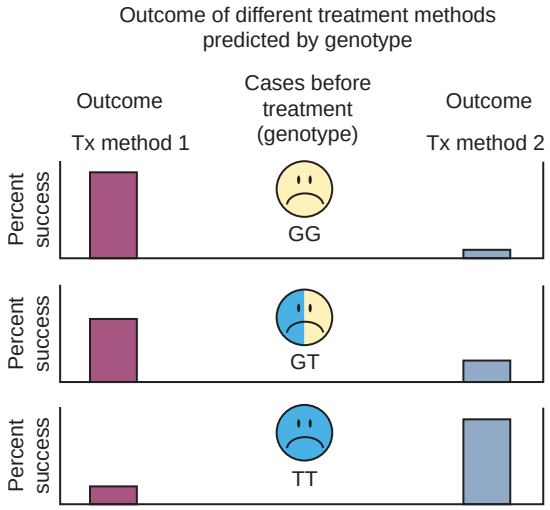

Figure 24-7 Inherited genetic variation determines treatment success. Individuals who inherited the GG or GT genotypes at a gene involved in a biologic pathway related to either the underlying disease susceptibility mechanisms or to the body's response to the therapy have very good chances for success from treatment method 1 *(Tx method 1),* but a poor prognosis with treatment method 2 *(Tx method 2),* while individuals with the TT genotype respond positively to treatment method 2 only.

of disease etiology or pathogenesis, or it might code for an enzyme, transporter, or receptor important for the metabolism of a therapeutic drug and have nothing directly to do with disease susceptibility. In the example illustrated in this figure, treatment method 1 *(Tx method 1)* has very high success when provided to subjects with the GG or GT genotypes but usually fails for cases with the TT genotype. TT genotypes experience much better outcomes when provided with treatment method 2 *(Tx method 2),* but this method often fails for subjects with the GT or GT genotypes. Sometimes, concern about severe side effects that limit use of a therapy rather than differences in efficacy and risk of side effects can also be genetically determined. If clinicians are not aware of the relationships between these genotypes and treatment success or side effects, it would appear that neither treatment is consistently effective. Clinicians would be forced to use a trial and error approach—first trying one method and then switching to the other if the first method fails. At best, this causes unnecessary expense and does not provide optimal quality of care. Furthermore, especially for deadly diseases such as cancer, precious time may be lost in halting the advance of the disease and by the time the clinician discovers an individual's best treatment, it may be too late. This strategy of individualized medicine is already being practiced for an increasing number of diseases.[9,16]

In dentistry, a test based on genetic variation at IL-1α and IL-1β cytokine and receptor antagonist genes to predict progression and severity of periodontitis has been commercialized and is still offered today, although it does not appear to be widely used. A number of reviews of the large number of studies on the IL-1 gene polymorphisms (see Table 24-3 [online] and Table 24-4) indicate that genetic variation at these loci may be associated with a modest effect on disease risk.[21,42] Huynh-Ba et al concluded in 2007 that "there is insufficient evidence to establish if a positive IL-1 genotype status contributes to progression of periodontitis and/or treatment outcomes."[21] Similar lack of supporting evidence for use of IL-1 genetic testing to predict implant success has also been reported.[3,22]

As genetic testing becomes widespread, especially through "direct to consumer" commercial models, increasing responsibility will fall on dentists to fully understand and counsel their patients on the implications of test results for their treatment options and their risk of developing various dental diseases in the future. Tests for diseases of the oral cavity may likely combine inherited polymorphisms with oral microbial profiles and might also include assays of gene expression or proteomic data measured in saliva or other oral tissues. Few dentists practicing today have been educated to prepare themselves for these future challenges. This major shortfall of knowledge urgently needs to be addressed either by continuing education of today's dental professionals about advances in knowledge of human genetics and the uses and potential misuses of genetic testing, by establishment of multidisciplinary teams of health practitioners in which dentists work closely with genetics counselors and medical geneticists, or by combinations of these approaches.

Acknowledgments

We thank Olga Korczeniewska for assistance in preparation of the figures and acknowledge support provided by NIH NIDCR grants 5R01DE016057 and 5R01DE018635 and the Foundation of the University of Medicine and Dentistry of New Jersey.

References and Suggested Reading can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 25

Molecular Biology of the Host-Microbe Interaction in Periodontal Diseases

Carlos Rossa Jr. and Keith L. Kirkwood

CHAPTER OUTLINE

INNATE IMMUNITY IN PERIODONTAL DISEASES
- Vigilance and Tolerance
- Cell Signalling Pathways and the Expression of Biologically-Active Mediators in the Innate Immune Response

ADAPTIVE IMMUNITY IN PERIODONTAL DISEASES
- Activation of Adaptive Immunity in Periodontal Diseases
- Host-Microbe Interactions in Adaptive Immunity (online only)

PATHOBIOLOGY OF PERIODONTAL DISEASE PROGRESSION
- Cytokines and Mediators of Inflammation
- Cell Signalling Events Modulating Inflammatory Mediator Expression

THERAPEUTIC STRATEGIES FOR DISRUPTING HOST-CELL SIGNALLING IN THE TREATMENT OF PERIODONTAL DISEASES

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Some text, figures, references, and tables may be out of numeric order in this printed chapter.

This chapter provides an overview of the molecular biology of the host-parasite relationship from a cellular signalling perspective specific to pathogen-associated lipopolysaccharide (LPS). The implications of periodontal microbiota-associated LPS and other microbiota-associated molecular patterns (MAMPs) on the induction of the innate immune response, toll-like receptor (TLR) signalling, and periodontal pathogenesis are reviewed. The second half of the chapter highlights the pathobiology of periodontal disease and the induction of disease by proinflammatory cytokines that generate host-tissue destruction and alveolar bone loss. The chapter concludes with ramifications for the development of therapeutic strategies that perturb innate immune signal transduction for the treatment of periodontitis.

For the past two decades, the host response to the bacterial challenge originating from the dental biofilm has been considered to play a major role in initiation and tissue destruction of periodontal diseases.[176] The significance of host-microbial interactions is reinforced by epidemiologic data indicating different susceptibilities to periodontal disease among individuals, in spite of the long-term presence of oral biofilm.[20,21,194] Other studies demonstrating increased susceptibility and greater severity of periodontal disease in individuals with impaired immune response caused by systemic conditions also indicate the significance of the host response to the bacterial challenge.[88,209] In the current paradigm of periodontal disease, periodontal pathogens are essential for disease initiation; however, the extent and severity of tissue destruction are largely dependent on the nature of the host-microbial interactions. These interactions are dynamic, since the microbial composition of the dental biofilm and host immune competency can vary widely between patients resulting in differences in host responses and subsequent alveolar bone loss. This concept has evolved in parallel with a more advanced appreciation of the immune response resulting in an increased emphasis focused on mechanisms of host-microbial interactions in periodontal disease pathobiology, as well as on the development of novel therapeutic strategies.

Periodontal diseases provide a unique situation to study microbial-host interactions. Over 500 different microbial species can be found in the oral biofilm;[237] however, only a few of those are associated with periodontal disease.[293,294] This implicates that the recognition of both nonpathogenic/commensal bacteria and pathogenic bacteria by the host requires vigilance and tolerance mechanisms to mount an appropriate response that can prevent dissemination of infection without inducing an exacerbated reaction that could result in damage to the host tissues. Direct recognition of microbes by the host is mediated by the recognition of MAMPs by pattern-recognition receptors (PRRs).[35,37]

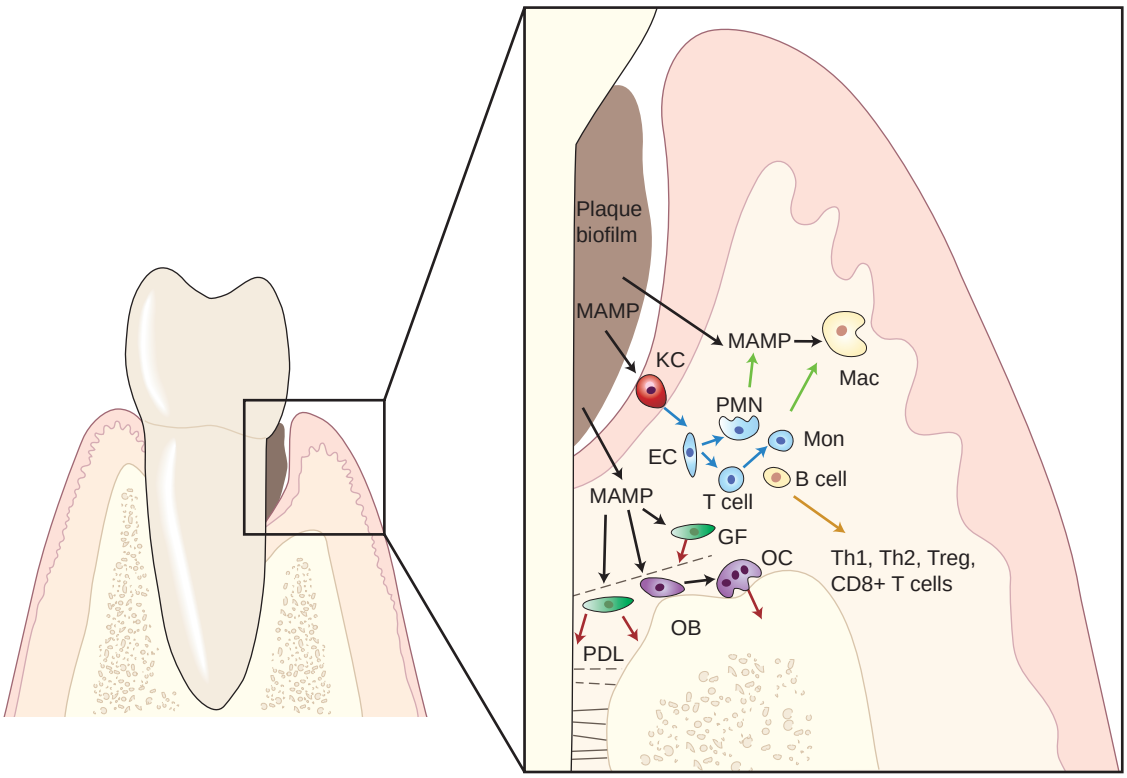

Figure 25-1 Microbial-associated molecular pattern (MAMP) from microorganisms in the dental biofilm activate inflammatory responses in periodontal tissues. Biologic mediators will either affect neighboring cells *(blue arrows)* by inducing the expression of other mediators (e.g., RANKL) or by triggering chemotaxis *(green arrows)*. Direct damage to periodontal tissues may also result after MAMPs stimulation *(red arrows)*, such as metalloproteinase secretion by gingival and periodontal ligament fibroblasts. *MAMP,* Microbial-associated molecular patterns; *RANKL,* receptor activator of nuclear factor (NF) -κB ligand; *KC,* keratinocyte; *Mac,* macrophage; *GF,* gingival fibroblast; *PDL,* periodontal ligament fibroblast; *OB,* osteoblast; *OC,* osteoclast; *EC,* endothelial cell; *PMN,* polymorphonuclear neutrophil; *Mon,* monocyte; *T cell,* T lymphocyte, *B cell,* B lymphocyte.

Host response to periodontal infection requires expression of a number of bioactive agents, including proinflammatory and anti-inflammatory cytokines, growth factors, and enzymes that are the result of the activation of multiple signalling pathways. This activation of intracellular signalling may initiate exclusively as an innate immune response associated with PRR-mediated sensing of MAMPs. However, since the oral cavity is one of the most readily colonized postnatal mucosal sites, the long-standing interactions between microbes (commensal and pathogenic) and host results in a "priming" of the immune system with the common presence of adaptive immune cells in the periodontal tissues.

The biologic mediators expressed as a result of PRR-signalling activation include costimulatory molecules involved in the induction of adaptive immunity.[37] This results in a cascade of events that will establish very complex cytokine and signalling networks. There is accumulating evidence for a direct role of "classic" innate immunity signalling through PRRs in adaptive immune cells that can modulate the function of these cells. Abundant evidence indicates a key role for the adaptive immune response—humoral and cellular aspects—in mediating the host response to microorganisms existing within the oral biofilm and also in the majority of tissue destruction associated with periodontal diseases.* Even though cells participating in the adaptive immune response are considered to be the primary source of cytokines leading to bone resorption,[168] additional data exist showing that periodontal bone loss occurs in the absence of B and T cells, suggesting a role for the innate immune response in periodontal disease initiation or progression.[22,25,26]

Innate immunity and inflammation are not synonymous; however, inflammation arises primarily in response to infection. More importantly, innate and adaptive immune responses are not mutually exclusive, and the bridge between these arbitrarily and didactically distinguished arms of the immune response has been shortened in recent years. However, considering that this chapter focuses on host-microbe interaction, we will emphasize the direct recognition of MAMPs by cells participating in the immune response and the molecular mechanisms activated downstream of this recognition (Figure 25-1).

INNATE IMMUNITY IN PERIODONTAL DISEASES

Vigilance and Tolerance

Even though adaptive and innate immune responses are traditionally distinguished, in vivo they are integral parts of the host defenses against infections (i.e., innate immunity is not inactivated once the adaptive response is initiated). Innate immunity is required for the

*References 24, 26, 84, 85, 124, and 169.

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activation of a more specific adaptive immune response, but it also plays an important role in managing host-microbial interactions since the innate immune system is rapidly activated (within minutes) and is responsible for the defense during the initial hours and days of the infection, whereas adaptive immunity requires at least 7 to 10 days before an adequate cellular or humoral response occurs. This role of innate immunity is especially important considering that our host cells are outnumbered 10:1 by the microbial cells living on skin and mucosal surfaces. This coexistence, however, is usually pacific and even beneficial for one or both cohabitants but requires both vigilance and tolerance mechanisms that ultimately determine if the host response is to clear the infection, or suppression of the host response is required to maintain the homeostatic equilibrium between host and microbes.

With regard to periodontal diseases, current paradigm indicates that some groups or complexes of bacteria are more strongly associated with the presence of periodontal diseases, but this association is not universally found, as there are individuals harboring the disease-associated complexes that are free of periodontal disease. Moreover, the microbial flora of the oral cavity can include more than 500 different species and there is no uniformity on the level of infection that is sufficient to produce disease. The complexity of the oral microbial flora is particularly intriguing given the limited number of receptors that are able to recognize microbial antigens. Tolerance mechanisms probably play a role in modulating the host responses to commensal/nonpathogenic bacteria. One of the primary challenges of the innate immune system is to discriminate among a large number of periodontal pathogens from the host with a limited number of cell surface receptors. This challenge is compounded because microbial pathogens have the ability to mutate as a mechanism to escape host recognition. The innate immune system has met this challenge through recognition of evolutionarily conserved structures on pathogens that are not present in higher eukaryotes (PRRs). These molecular motifs (MAMPs) have essential roles in the pathogen's ability to evade host defense and thus are not subject to high mutation rates. MAMPs are shared among various species of microbes but are not expressed by the host. Although many PRRs have been known for years, it was not clear how the innate immune system functioned until the discovery of TLRs, which have proved to be critical for recognition of microbes by the innate immune system and for bridging the innate and acquired immune responses. Interestingly, within the periodontal tissues, the expression of various TLRs appears to be increased in severe disease states.[29] Table 25-1 illustrates PRRs and the host cells that express them and their ligands (MAMPs) present in microorganisms.

However, there are other PRRs that can be secreted into the plasma as humoral proteins and others that are localized in the cytoplasm as intracellular sensors. Soluble PRRs include various proteins, including collectins, ficolins, and acute-phase pentraxins (e.g., C-reactive protein) that represent functional ancestors of antibodies. These soluble mannose-binding receptors can interact with structures from microbes and activate the complement system via mannan-binding lectin-associated serine kinase (MASP) pathway.[269] In fact, direct interaction between complement precursors and microbes can initiate activation through an unclear mechanism that culminates with opsonization and lysis of the microbes. Another example of soluble PRRs is the lectin-complement system,

TABLE 25-1 Pattern-Recognition Receptors (PRRs), Host Cells (Mouse or Human) that Express Them, and Their Ligands (MAMPs) Present in Microorganisms Relevant to Periodontal Diseases

PRR	Expressed in	MAMP	Periodontal Pathogen	References
TLR2	Monocytes, neutrophils, epithelial cells, fibroblasts, macrophages, T and B lymphocytes	Lipoproteins	*Tannerella forsythia*	130
		Atypical lipopolysaccharide (LPS)	*Porphyromonas gingivalis, Capnocytophaga ochracea*	46, 117, 138, 200, 282, 342
		Outer membrane proteins	Oral treponemes	16, 132
		Fimbriae	*P. gingivalis*	15, 118–122, 137
		Nonendotoxic glycoprotein	*Prevotella intermedia*	302
		Whole bacteria	*Treponema denticola*	267
		Cell-surface BspA protein	*T. forsythia*	232
TLR4	Monocytes, macrophages, epithelial cells, fibroblasts, neutrophils, T and B lymphocytes	LPS	*P. gingivalis, C. ochracea*	342
		Heat shock protein (HSP)-60 (GroEL)	*P. gingivalis*	322
		Atypical LPS	*P. gingivalis*	31
TLR9	Epithelial cells, dendritic cells, T and B lymphocytes	CpG-containing DNA	*Aggregatibacter actinomycetemcomitans, P. gingivalis, P. micros*	220
Nod1	Cytotoxic T cells, dendritic cells, epithelial cells fibroblasts, osteoblasts, macrophages	Meso-diaminopimelic acid (meso-DAP) containing peptidoglycan (PGN) fragments (mostly gram-negative bacteria)		90, 303, 320, 321
Nod2	Neutrophils, monocytes, dendritic cells, epithelial cells, fibroblasts, osteoblasts, macrophages	Muramyl dipeptide (MDP) found in PGN of both gram-positive and gram-negative bacteria		90, 303, 320, 321

which is involved in recognizing the microbe by the carbohydrate-binding lectin, leading to its subsequent opsonization and phagocytosis.[331]

Nucleotide-oligomerization domain (NOD) protein-like receptors represent cytoplasmic PRRs and are characterized by C-terminal leucine-rich repeats (LRR domain, similar to the TLRs), an N-terminal caspase-activating recruitment domain (CARD), and a nucleotide-binding domain (NBD). These were initially described as cytosolic TLRs, analogous to the R proteins present in plants.[70] NOD proteins are capable of recognizing different peptidoglycan molecules: Nod1 recognizes peptidoglycan containing meso-diaminopimelic acid (meso-DAP) fragments present in most gram-negative and some gram-positive bacteria,[106] whereas Nod2 recognizes muramyl dipeptide (MDP), which is found in peptidoglycan from both gram-negative and gram-positive bacteria.[207] More recent evidence indicates that NOD proteins, specifically Nod1 and Nod2, are involved in activation of inflammatory gene expression[90] and even LPS recognition independently of TLR.[152] The relevance of NOD proteins for the immune response is demonstrated by the association between genetic mutations on Nod1 and Nod2 and development of allergic conditions and Crohn's disease, respectively.[148,153] Another type of intracellular PRR is represented by a cytoplasmic protein family, the retinoic acid-inducible gene I (RIG-I)-like receptors (RLRs), which play a critical role in recognizing viral ribonucleic acid (RNA) and induction of type I interferon (IFN) expression. However, since the role of retroviruses in periodontal disease is not clear, RLRs will not be discussed here (for a review of RLRs, see Yoneyama and Fujita[340]).

All of these different PRRs represent the necessary armamentaria for recognition of MAMPs by the host and are expressed by a variety of cells that play a role in innate immunity. Importantly, our understanding of the roles of PRRs in inflammation and immune responses has been expanded, so it is now appreciated that these receptors not only recognize various MAMPs to activate innate immune response, but they can also bind to endogenous molecules derived from damaged tissue and have a role in inflammation and adaptive immune responses. The cells involved in innate immunity include macrophages and polymorphonuclear cells as professional phagocytes with the primary function of engulfing and destroying microbes; dendritic cells as professional antigen-presenting cells and activators of adaptive immunity; and natural killer (NK) cells, the innate cytotoxic lymphocytes that recognize and kill host cells that are altered (e.g., tumor cells) or infected with viruses. However, other cell types can also play important roles in innate immunity, since they are able to recognize MAMPs through their PRRs and respond by expressing biologically-active molecules, such as cytokines and matrix metalloproteases (MMPs), that will have an effect on homeostasis of the host tissues in the periodontal microenvironment. Resident, "nonprofessional" cells such as fibroblasts and osteoblasts are also capable of producing a variety of cytokines, such as interleukin-6 (IL-6), prostaglandin E_2 (PGE_2), MMPs, and receptor activator of NF-κB ligand (RANKL). Table 25-2 presents these biologic mediators elicited by different MAMPs in resident and nonresident cells involved in the pathogenesis of destructive periodontal disease. Because of the sheer proportion of fibroblasts in the periodontal tissues and also to the proximity and relevance of both fibroblasts and osteoblasts to non-mineralized and mineralized tissue turnover, respectively, these cells can play important roles in innate immunity during periodontal diseases. Epithelial cells represent the initial point of contact with MAMPs in the periodontium and play an important role in innate immunity not only because of their function as a physical barrier, but also because these cells are equipped with PRRs and respond to MAMPs by secreting various cytokines and chemokines, including IL-8 and antimicrobial peptides (e.g., beta defensins, cathelicidins).[69,156]

Despite the alleged nonspecificity of the innate immune response, it has long been known that there are important quantitative and qualitative differences in cytokine release on stimulation with gram-positive or gram-negative bacteria.

For more information on vigilance and tolerance mechanisms, please visit the companion Expert CONSULT website at www.expertconsult.com.

There are some important differences between the oral epithelium and the intestinal mucosa, especially the presence of specialized immune cells subjacent to the mucosal epithelium in the intestines (gut-associated lymphoid tissues [GALT] and Peyer's patches); however, the concept of a tolerance mechanism that renders the immune system hyporesponsive to commensal bacteria, while retaining the capacity to respond to pathogenic organisms has also been investigated in oral epithelial cells. A comparison of the responses of oral epithelial cells to nonperiodontopathic and periodontopathogenic bacteria demonstrated that, in general, commensal bacteria (such as *Streptococcus gordonii* or *Streptococcus sanguinis*) induce expression of antimicrobial peptides without inducing the chemoattractant cytokine IL-8, whereas periodontopathogenic bacteria from the "orange complex" (*Fusobacterium nucleatum* and *Prevotella intermedia*) induce strong expression of both antimicrobial peptides and IL-8. Interestingly, the bacteria that are more strongly associated with periodontitis ("red complex" organisms *Treponema denticola, Tannerella forsythia,* and *Porphyromonas gingivalis*) tended to suppress the innate immune response by inhibiting the expression of antimicrobial peptides, IL-8, or both simultaneously.[156,179,324] Tolerance mechanisms may involve the activation of different signalling pathways, since the activation of NF-κB (which is associated with the expression of many inflammatory genes) was not required for the expression of antimicrobial peptides by oral epithelial cells after stimulation with commensal bacteria, as opposed to the periodontopathogenic microorganisms.[56] There is still much to be explored in these tolerance mechanisms, but it is conceivable that commensal bacteria may be beneficial to the host as they induce expression of antimicrobial peptides, which will clear some of the nonpathogenic and pathogenic bacteria while allowing nonpathogenic periodontal bacteria that are resistant to antimicrobial peptides, such as *S. gordonii* and *S. mutans*,[156,251] to establish themselves in the oral biofilm. The weak induction of IL-8 may function to maintain immune vigilance without causing too much collateral damage to the host tissues. On the other hand, pathogenic bacteria (such as the orange complex microorganisms, *F. nucleatum* and *P. intermedia*) that induce both antimicrobial peptides and IL-8 expression may be limiting their survival in the biofilm. Interestingly, the inhibition of expression of antimicrobial peptides and IL-8 in epithelial cells by red complex bacteria (such as *P. gingivalis* and *T. denticola*) can represent host-evading strategies that allow them to survive and establish themselves in the oral biofilm.[44]

Activation of the innate immune system is critical for lymphocyte activation and other immune cells to help clear the infectious microorganisms. However, exuberant production of proinflammatory cytokines leads to severe pathology, including alveolar bone

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TABLE 25-2 Biological Mediators Elicited by Different MAMPs in Resident and Nonresident Cells Involved in the Pathogenesis of Destructive Periodontal Disease

Cell type	MAMPs	Biological Mediator	References
Epithelial cells	LPS, fimbriae, glycoprotein, whole bacteria, cell wall extracts, *P. gingivalis*, gingipains	IL-8, G-CSF, GM-CSF, Beta-defensin2, MMP-3, MMP-9, MMP-13, MIP-1α, IL-1β	56, 71, 116, 126, 146, 147, 223, 232, 271, 277, 299, 304, 329
Dendritic cells	Fimbriae, LPS, CpG DNA, DNA	IFN-α, IL-6, IL-8, IL-10, IL-12, TNF-α, GM-CSF,	155, 162, 163, 249, 296
Endothelial cells	LPS, heat shock proteins	IL-6, GM-CSF, ICAM-1	72, 95, 160, 198
Gingival fibroblasts	LPS, peptidoglycan, CpG DNA, gingipains, *T. forsythia* detaching factor	IL-1β, IL-6, IL-8, TNF-α, PGE_2, MCP-1, MMP-2	12, 38, 82, 133, 220, 223, 229, 231, 256, 307, 315, 329, 330
Periodontal ligament fibroblasts	LPS	IL-6, IL-8, MMP-13, RANKL	133, 264, 266, 334
Cementoblasts	LPS	OPN, OCN, RANKL, IL-6	215, 218
Macrophages	LPS, CpG DNA, leukotoxin	IL-1α, IL-1β, IL-6, IL-12, TNF-α, MMP-1, NO	170, 174, 206, 220, 229, 259, 309
Osteoblasts	LPS	IL-1β, IL-6, TNF-α, RANKL, PGE_2, NO, MMP-2, MMP-9	173, 242, 300, 347
Neutrophils	DNA, LPS	IL-8, chemotaxis, shedding of L-selectin, MIP-1α	271, 316, 317
Monocytes	LPS, CpG DNA, fimbriae	IFN-γ, IL-1α, IL-1β, IL-6, IL-8, IL-12, TNF-α, LIF, RANKL, PGE_2	27, 28, 39, 72, 80, 95, 119, 128, 157, 183, 196, 214, 255, 259
B lymphocytes	CpG DNA, heat stress proteins, cell sonicate extracts	IL-6, IL-10, IL-12, TNF-α, proliferation, antibody production	50, 228, 326, 344
T lymphocytes	LPS, CpG DNA, peptidoglycan	IFN-γ, IL-4, IL-10, IL-13, inhibition of apoptosis, decrease Th2 cytokines	103, 202, 203, 249, 308, 344

LPS, Lipopolysaccharide; *DNA,* deoxyribonucleic acid; *IL,* interleukin; *G-CSF,* granulocyte colony-stimulating factor; *GM-CSF,* granulocyte macrophage colony-stimulating factor; *IFN-γ,* interferon-γ; *MMPs,* matrix metalloproteinases; *PGE2,* prostaglandin E_2; *RANKL,* receptor activator of NF-κB ligand; *TNF-α,* tumor necrosis factor-alpha; *LIF,* leukemia-inhibiting factor; *MIP-1α,* macrophage inflammatory protein-1α; OPN, osteopontin; OCN, osteocalcin; NO, nitric oxide.

loss.[17,76,77,113,114] To help prevent deleterious effects of TLR activation, a number of signalling mechanisms have evolved. These mechanisms include downregulation of surface TLR receptor expression, transcriptional induction of negative regulators such as IL-1 receptor-associated kinase (IRAK-M), suppressor of cytokine signalling-1 (SOCS1), and SH2-containing inositol phosphatase (SHIP), and production of antiinflammatory cytokines, including IL-10 and transforming growth factor-beta (TGF-β).[191] In contrast to the release of proinflammatory cytokines and mediators, production of these negative immune regulators occurs over a much longer time frame, providing a vital role in controlling the extent of proinflammatory and antiinflammatory mediators in a proper temporal sequence.[191]

Cell Signalling Pathways and the Expression of Biologically-Active Mediators in the Innate Immune Response

Once MAMPs are recognized by the appropriate PRRs, a signal is initiated within the cell. This signal is transduced through the cytoplasm and nucleus more commonly by sequential posttranslational modification (e.g., phosphorylation) of a cascade of kinases that are constitutively expressed and ultimately will determine the cell response to the MAMPs detected (Figure 25-2).

TLRs are single-pass transmembrane proteins with N-terminal presenting LRRs that are responsible for the recognition of their ligands and with a C-terminal cytoplasmic domain that is very similar to the cytoplasmic region of the IL-1 receptor,[3] which is called the *Toll/IL-1 receptor domain* (TIR). Thus, subsequent to the recognition of a ligand by TLRs, the signal generated uses pathways similar to those utilized by the IL-1 receptor. TLR signalling was originally described in the context of the activation of the IFN regulatory factor IRF family of transcription factors and NF-κB, leading to the expression of IFN-γ and early-response inflammatory genes, respectively.

For more information on cell signalling pathways, and to view Figure 25-2, please visit the companion Expert CONSULT website at www.expertconsult.com.

ADAPTIVE IMMUNITY IN PERIODONTAL DISEASES

Activation of Adaptive Immunity in Periodontal Diseases

Besides its role in the immediate detection of infectious agents and toxic/degradation ("danger") products to elicit an inflammatory response and activate effector processes aimed at the elimination or confinement of microbial invaders, innate immunity also has the pivotal role of initiating and modulating adaptive immune responses. Thus adaptive immunity requires the innate "branch" of immunity

to be activated; however, in vivo, this didactic distinction does not exist and the immune response should be understood as a continuum, a constantly adjusting host reaction to the presence of microbes and their MAMPs. In the context of this chapter, the signalling and molecular aspects of the host-microbe interactions related to the adaptive immune response will be emphasized.

Innate immune mechanisms and signalling are not "turned off" once the adaptive immune response is activated but rather are "supplemented" by the latter in the defense against microbes. On the other hand, cells from adaptive immune response also express PRRs and respond to MAMPs, which further emphasizes the dynamic interaction between the two "arms" of the immune response.

In T lymphocytes, TLR agonists have been shown to modulate the expression of costimulatory receptors (such as CD28, CD69, and CD152) and increase their proliferation and IFN-γ production, suggesting that microbes and their MAMPs may also have a direct functional role in the regulation of adaptive immunity.[47,292] This is especially important to realize in the context of periodontal disease, which is a chronic inflammatory condition and as such has a dense lymphocytic infiltrate. Ever since the classic studies of the host response in periodontal disease, including the recognition of immunoglobulin-producing plasma cells in the gingival tissues of patients with periodontal disease,[43] the classic descriptions of initial, established, and advanced lesions[233] and the characterization of the inflammatory infiltrate in the dog model,[192] the nature of the inflammatory infiltrate and its association with the inflammatory response and ultimately the quiescence or progression of disease, has been debated.

For more information on activation of adaptive immunity in periodontal diseases, please visit the companion Expert CONSULT website at www.expertconsult.com.

Host-Microbe Interactions in Adaptive Immunity

For more information on host-microbe interactions in adaptive immunity, please visit the companion Expert CONSULT website at www.expertconsult.com.

PATHOBIOLOGY OF PERIODONTAL DISEASE PROGRESSION

Host response to periodontal infection requires expression of a number of bioactive agents, including proinflammatory and anti-inflammatory cytokines, growth factors, and enzymes that are the result of the activation of multiple signalling pathways. This activation of intracellular signalling may initiate exclusively as an innate immune response associated with PRR-mediated sensing of MAMPs. However, the biologic mediators expressed as a result of PRR-signalling include costimulatory molecules involved in the induction of adaptive immunity.[36] This results in a cascade of events that will establish very complex cytokine and signalling networks.

Innate immunity and inflammation are not synonymous; however, inflammation arises primarily in response to infection. A better understanding of direct host-microbe interactions is especially important to understand how inflammation is initiated in response to microorganisms in the dental biofilm. In this sense, PRR signalling is considered the most important interface between the host and the microbes,[36] and as discussed previously in this chapter, it can modulate both innate and adaptive aspects of the host response.

Cytokines and Mediators of Inflammation

Inflammation involves a number of biochemical and cellular alterations. An inappropriate inflammatory response is the cause of many common diseases, including periodontitis. The local inflammatory reaction, in response to bacteria in the dental biofilm, is characterized by an initial increase in blood flow, enhanced vascular permeability, and the influx of cells from the peripheral blood to the gingival crevice. These initial events are triggered by bioactive molecules (such as histamine, bradykinin, PGE_2, and nitric oxide) and produced by innate immune cells and resident, nonimmune cells present in the periodontal tissues. PMNs or neutrophils attracted to the area by other bioactive molecules (e.g., IL-8) migrate through the epithelial lining of the gingival sulcus to be the initial defense against invading plaque bacteria and their by-products.[195] These cells are nonspecific phagocytes responsible for an acute and rapid defense. Subsequently, there is an increase in the number of monocytes/macrophages, as well as an influx of T and B cells to the area.[97]

Once activated by cytokines, bioactive molecules, and MAMPs present in the area, infiltrating cells produce other inflammatory mediators that modulate the activity of other cells and affect homeostasis of nonmineralized and mineralized tissues in the periodontium[59,111] (Table 25-2 and Figure 25-3). Cytokines responsible for early responses to microbial aggression include IL-1α, IL-1β, IL-6, and TNF-α.[185] Other proinflammatory mediators include leukemia-inhibiting factor (LIF); IFN-γ; oncostatin M (OSM); ciliary neurotrophic factor (CNTF); TGF-β; GM-CSF; IL-11, IL-12, IL-17, IL-18, IL-8, and a variety of other chemokines that attract inflammatory cells.[49,226,275,276] The net effect of an inflammatory response is determined by the balance between proinflammatory and antiinflammatory cytokines such as IL-4, IL-10, IL-13, IL-16, IFN-α, TGF-β, IL-1Ra, GM-CSF, and soluble receptors for TNF or IL-6.[112,186]

Patients with periodontal inflammation have high concentrations of TNF-α, IL-1β, RANKL, and MMP-13* in the gingival crevicular fluid (GCF). Increased levels of IL-1β, IL-2, IL-6, IL-17, TNF-α, and IFN-γ in gingival tissues are also associated with destructive periodontal disease.[110,188]

However, in spite of an abundance of publications investigating cytokine profiles associated with periodontal disease, the definitive description of the cytokine response still remains elusive. Among the reasons for the variability of the reports on the cytokine profile are the difficulties in defining periodontal disease and especially in the identification of actively progressing disease sites (i.e., those with ongoing attachment loss and alveolar bone resorption). A gene-expression profiling (or transcriptome analysis) of changes in mRNA levels of over 38,000 genes with 54,000 DNA probes was performed in healthy and diseased periodontal tissues, and demonstrated that over 12,000 probes were differentially regulated in periodontal diseased tissues, including genes involved in inflammation, apoptosis and angiogenesis.[78] These data agree with the evidence demonstrating that the cytokine profile associated with periodontal disease in vivo varies and includes both Th1- and Th2-type cytokines. IL-1α, IL-1β, IL-8, and TNF-α mRNA were detected in macrophages present in inflamed gingival tissues,[201] whereas Th2 cytokine IL-4 and pleiotropic IL-6 protein were also

*References 86, 96, 110, 284, 291, and 298.

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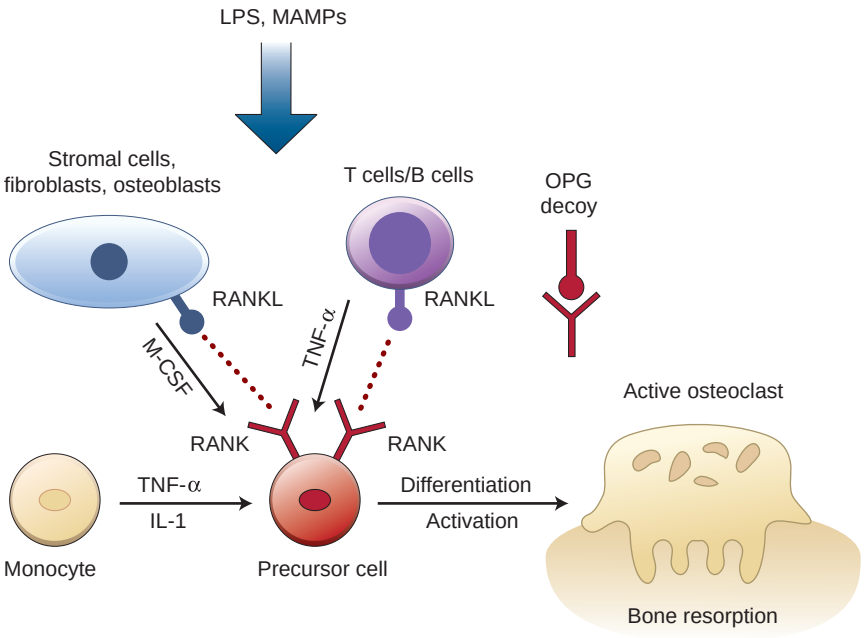

Figure 25-3 Stimuli and factors regulating osteoclastogenesis. Cytokines (interleukin-1 *[IL-1]*, tumor necrosis factor alpha *[TNF-α]*, macrophage-colony stimulating factor *[M-CSF]*) produced by resident cells (bone stromal cells, fibroblasts, osteoblasts) and immune cells (monocytes, T- and B-lymphocytes) are key regulators of this process. Osteoprotegerin *(OPG)* acts as a decoy receptor that prevents receptor activator of nuclear factor (NF)-κB ligand *(RANKL)* from binding to its receptor activator of NF-κB *(RANK)* on precursor cells and downregulate osteoclastogenesis. Bacterial lipopolysaccharide (LPS) and other microbial-associated molecular patterns *(MAMPs)* may directly or indirectly initiate expression of RANKL and osteoclastogenesis.

observed in diseased periodontal tissues.[339] A characteristic cytokine profile has been associated with each type of periodontal disease (i.e., inflammation of marginal soft tissues without active bone resorption [gingivitis] or with active bone resorption [periodontitis]). Thus expression of Th1-type cytokines has been associated with gingivitis, whereas Th2 cytokines were found in higher levels in periodontitis-affected tissues,[186,314] even though this distinction was not clear because both Th1 and Th2 cytokines were produced in gingivitis- and periodontitis-affected tissues; and the predominant profile may actually represent the current activity of tissue destruction.[101,286,335] The relevance of cytokines as biologic mediators of periodontal disease progression is demonstrated by studies showing that conventional periodontal therapy aimed at the reduction of the bacteria and associated metabolic by-products result in their decrease, as shown for IL-1β[4,61,313] and TNF-α.[154]

Once the immune and inflammatory processes are initiated and the complex cytokine network is established, various inflammatory molecules that play a direct role in the degradation of both nonmineralized and mineralized tissues of the periodontium are produced in response to MAMPs and/or to the host-derived cytokines.[59,111] One such family of inflammatory molecules is the MMPs, which are released from different cell types present in the lesion, including macrophages, leukocytes, and fibroblasts or other resident cells.[178] MMPs are a family of neutral proteases that are zinc/calcium-dependent with an essential role in extracellular matrix (ECM) turnover and degradation. Their activity is regulated at multiple levels (transcriptional, posttranscriptional, and post-translational) and also by endogenous inducible inhibitors, the tissue inhibitors of MMPs (TIMPs). Collectively, MMPs are capable of degrading virtually all components of ECM, and increased levels have been associated with periodontal disease in both humans[61,87,89,180] and animal models.[74,102] The same difficulties associated with the determination of disease activity apply to the determination of MMPs in periodontal disease, even though the trend seems to have a positive correlation (i.e., MMPs increase with inflammation and disease activity[108]) and their detection in saliva has been recently suggested as one possible host-response biomarker of periodontal disease.[254] Interestingly, the role of individual MMPs in periodontal disease progression may differ, as MMP-8 was recently shown to have a protective effect on alveolar bone loss induced by *P. gingivalis* infection in mice[182]; whereas MMP-13 expression was positively correlated with the severity of inflammation in ligature- and LPS-induced experimental periodontitis in rats[74] and also with severity of periodontal disease in humans.[291]

The relevance of MMPs to the pathogenesis of periodontal diseases is supported by the decrease in bone loss associated with their nonselective inhibition in animal models of periodontal disease[252,253] and especially by the improved clinical results observed after periodontal treatment associated with systemic inhibition of MMPs by a sub-antimicrobial dose of doxycycline.[99,107,247,248]

As periodontal disease progresses, the collagen fibers and connective tissue attachment to the tooth are destroyed, the junctional epithelial cells proliferate apically along the root surface, and these structural changes are reflected clinically as attachment loss and/or increased probing pocket depth. These changes represent not only an increased severity of inflammation but also an apical displacement of the inflammatory infiltrate, which thus is in close proximity to the alveolar bone. This affects homeostasis of bone tissue by triggering the resorptive process that represents the main characteristic of destructive periodontal disease. RANKL plays a pivotal role in bone resorption, since it is involved in osteoclast differentiation, activation, and survival.[42,171,310] Osteoprotegerin (OPG) is the endogenous inhibitor of RANKL and functions as its decoy receptor, preventing binding of RANKL to the specific membrane-bound receptor expressed in osteoclast precursor cells (RANK).[42,139,301] RANKL is produced as either a membrane-bound or secreted protein by fibroblasts, osteoblasts, chondrocytes, mesenchymal cells, and T- and B-lymphocytes, whereas OPG is secreted primarily by osteoblastic cells, bone marrow stromal cells, and fibroblasts (see Figure 25-3). The ratio between RANKL and OPG is the current paradigm for modulation of coupled bone turnover, and specifically in periodontal disease, this concept is supported by observations demonstrating that patients with advanced periodontitis present with higher levels of RANKL and lower levels of OPG than periodontally healthy patients.[65,213] Further support to the relevance of RANKL/OPG cytokine system for bone homeostasis comes from studies in which local delivery of OPG decreases alveolar bone resorption in experimental

periodontal disease models.[54,159] However, other biologically-active mediators present in the complex network of cytokines established in the periodontal disease microenvironment can play important roles in periodontal disease pathogenesis and alveolar bone resorption, as indicated by studies targeting inhibition of TNF-α.[57,79]

It is important to consider that there is significant heterogeneity of clinical presentation of periodontal disease in terms of its extent, severity, progression, and response to treatment. This heterogeneity is thought to reflect the high variability of the levels of inflammatory cytokines and bioactive molecules produced by different individuals, which cannot be completely explained by differences in the microbial population of the dental biofilm. Based on susceptibility analysis, individual differences in the host response to MAMPs and to host-derived cytokines that are the result of genetic variations may play an important role in modulating the pathogenesis of periodontal diseases.[341] This is supported by epidemiologic studies of the association between various genetic syndromes and periodontal diseases[222] and the familial trend for occurrence of some types of aggressive periodontal disease.[9,216] There are numerous studies correlating multiple variations in genetic information encoding genes and receptors involved in the immune response and inflammation with periodontal disease,[93,151] and these genetic variations are also heterogeneic according to race and ethnic aspects of human populations.[13,199,217,273,328] It is interesting to note that there is also evidence suggesting that genetic polymorphisms for the "effector" genes of MMPs[18,52,246] and RANKL/OPG[235,295] are associated with the severity and extent of periodontal disease. However, the strength of this association, especially for MMPs, is still controversial and may depend on specific types of MMPs, as other studies failed to demonstrate an association between polymorphisms for some MMPs and periodontal disease status.[75,115,140]

Another important aspect to consider is how the external environment affects the production of inflammatory mediators by the host. Thus, even though there is a "personalized genetic program" controlling how each individual responds to the microbial challenge, this is further modulated by transitory aspects of each individual's life that can affect production of cytokines and inflammatory mediators, including hormonal imbalances, stress, acquired diseases, medications, and smoking. These are all factors that come into play in determining the individual's response to the microbial challenge and ultimately, periodontal tissue destruction. Moreover, it has been recently suggested that genetic variations modulating host responses may represent a common trait for susceptibility to periodontitis and systemic diseases such as cardiovascular disease.[283]

Cell Signalling Events Modulating Inflammatory Mediator Expression

In the complex scenario of host-microbe interactions associated with periodontal disease, the signalling pathways originally shown to be relevant for recognition of stress, inflammatory, and infectious extracellular stimuli are of special interest. Production of cytokines and inflammatory mediators is usually a tightly-controlled process that is initiated by external stimuli or "signals" that are rapidly transduced through the cytoplasm and into the nucleus where gene expression starts with the transcription of DNA into pre-mRNA. From this start to the final assembly of the biologically-active protein, there are a great number of regulatory mechanisms that can affect gene expression and various signalling pathways can participate in many of these mechanisms, both at transcriptional and posttranscriptional levels. Thus cytokine production is a fast and transient process, initiated and controlled by even faster mechanisms represented by the signalling pathways. The fact that a given signalling pathway regulates the expression of various inflammatory mediators is especially important for therapeutic applications if one considers that targeting expression of a single cytokine may not be particularly effective as the result of compensation of its biologic role by other proinflammatory cytokines.

For more information on cell-signalling events modulating inflammatory mediator expression, please visit the companion Expert CONSULT website at www.expertconsult.com.

THERAPEUTIC STRATEGIES FOR DISRUPTING HOST-CELL SIGNALLING IN THE TREATMENT OF PERIODONTAL DISEASES

A variety of treatment strategies have been developed to target the host response to LPS-mediated tissue destruction. MMP inhibitors, such as low dose formulations of doxycycline, have been used in combination with scaling and root planing[48] or surgical therapy.[98] In addition, high-risk patient populations, such as those with diabetes, have benefited from systemic administration of MMP inhibitors.[51,221,270] Encouraging results have also been shown using soluble antagonists of TNF and IL-1 delivered locally to the periodontal tissues in nonhuman primates.[17] Other therapeutic strategies that are being explored are aimed at inhibiting signal transduction pathways involved in inflammation. Pharmacological inhibitors of NF-κB and p38 MAP kinase pathways are actively being developed to manage rheumatoid arthritis and inflammatory bone diseases[1,158,181] and have been applied in periodontal disease models with some benefits observed.[175,260] Using this novel strategy, inflammatory mediators, including proinflammatory cytokines (IL-1, TNF, IL-6), MMPs and others, would be inhibited at the level of cell signalling pathways required for transcription factor activation necessary for inflammatory gene expression or mRNA stability (Figure 25-2). Indeed, targeting RNA-binding proteins that mediate effects on inflammatory cytokines does have therapeutic value in small animal models of periodontal disease progression.[241] These therapies may provide the next wave of adjuvant chemotherapeutics that may be used to manage chronic periodontitis.

References and Suggested Reading can be found on the companion Expert CONSULT website at www.expertconsult.com.

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SCIENCE TRANSFER

A small number of the 500 plus bacterial species found in dental biofilms are related to periodontal destruction. These are gram-negative anaerobic bacteria that reside in the subgingival plaque.

The extent and severity of periodontitis is closely related to the host response to these putative pathologic bacteria. Initially, the host oral bacterial biofilm stimulates the establishment of an innate immune response based on the classical inflammatory cells. With initiation of periodontal disease, dendritic cells signal a more widespread immune reaction (adaptive immune response) that involves not only the B and T lymphocytes, polymorphonuclear leukocytes (PMNs), macrophages, and osteoclasts but also has contributions of signalling molecules and tissue destructive cytokines from resident epithelial cells, fibroblasts, and osteoblasts. The resulting molecular response is complex and interrelated and involves molecular signalling with genetic control of many of the proteins involved. Recent research has unraveled only part of these bioactive networks, and the more that is discovered, the more complicated and diversified are the mechanisms of periodontal destruction.

Most clinicians will have difficulty appreciating all of the signaling pathways and feedback mechanisms that are elucidated, but they should appreciate the importance of these adaptive immune responses in disease progression. New therapies based on regulating tissue destruction by inhibiting such molecules as matrix metalloproteinases (MMPs) with long-term low-dose doxycycline are already in widespread use as supportive therapy for conventional periodontal treatment. Newer approaches soon to be available include antagonists to cytokines and agents to inhibit signalling pathways associated with tissue destructive proteins. Because of the multitudinous molecular mechanisms of tissue destruction, it is unlikely that any one of these approaches will have a singular dramatic therapeutic affect, rather they will be only effectively used in combination with conventional antibacterial treatments such as initial therapy, antimicrobial agents, and periodontal surgery.

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CHAPTER 26

Smoking and Periodontal Disease

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CHAPTER OUTLINE

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THE SMOKING EPIDEMIC

Smoking is highly prevalent and can be considered an epidemic in both developed and developing nations. In the United States (US) in 1993, 25.4% of the population smoked, 27.0% of men and 24.0% of women.[1] By 2007, this had decreased to 20.8% of the total population, 23.9% of men and 18.0% of women (http://www.cdc.gov/tobacco). Among dentate individuals during the period 1988 to 1994, 27.9% of adults were current smokers and 23.3% were former smokers.[2] Smoking was higher in younger age groups (≤34 years) than in older age groups (≥55 years) and was more common in males (30.9%) compared to females (25.1%).[2] In 2009, roughly 43.4 million Americans were smokers with 443,000 deaths in the US attributable to smoking each year (http://www.cdc.gov/tobacco). In the European Union, approximately 29.0% of the population are smokers, ranging from 16% in Sweden to 48% in Greece, and smoking accounts for 650,000 deaths each year (or 1 in 7 of all deaths) (http://www.ash.org.uk). Furthermore, 79,000 nonsmoking Europeans die each year as a result of passive smoking. Worldwide, it is estimated that there are >1.3 billion smokers (around 1 billion men and 300 million women), with just over 80% living in low- and middle-income countries. Globally, smoking accounts for 1 in 5 deaths among men over 30 and 1 in 20 deaths among women over 30.

Smoking is harmful to almost every organ in the body and is associated with multiple diseases reducing the expectancy and quality of life. Diseases associated with smoking include lung cancer, heart disease, stroke, emphysema, bronchitis, and cancers of the oral cavity, bladder, kidney, stomach, liver, and cervix. Approximately half of long-term smokers will die early on account of smoking, and those who die before the age of 70 will lose on average around 20 years of life.[3] Most deaths from smoking are due to lung cancer, chronic obstructive pulmonary disease, and coronary heart disease.

Tobacco smoke contains thousands of noxious chemicals, and comprises a gaseous phase and a solid (particulate) phase. The gas phase contains carbon monoxide, ammonia, formaldehyde, hydrogen cyanide, and many other toxic and irritant compounds, including more than 60 known carcinogens such as benzo(a)pyrene and dimethylnitrosamine. The particulate phase includes nicotine, "tar" (itself made up of many toxic chemicals), benzene, and benzo(a)pyrene. Tar is inhaled with the smoke and in its condensate form, is the sticky brown substance that stains fingers and teeth yellow/brown. Nicotine, an alkaloid, is found within the tobacco leaf and evaporates when the cigarette is lighted. It is quickly absorbed in the lung and reaches the brain within 10 to 19 seconds. Nicotine is highly addictive and causes a rise in blood pressure, increased heart rate and respiratory rate, and peripheral vasoconstriction.

All dental patients must be asked about their smoking status. Current smoking status is the minimal information that must be recorded (e.g., patient is currently smoking *X* cigarettes per day), but the importance of cumulative exposure to cigarette smoke mandates that it is more appropriate to record pack-years of smoking (Box 26-1). Biochemical tests can also be used to assess smoking status, including exhaled carbon monoxide (CO) and measurement of cotinine (the major metabolite of nicotine) in serum, saliva, or urine. Cotinine is measured in preference to nicotine as the half-life of nicotine is short ($t_{½} \approx 1$ to 2 hours),[4] whereas that of cotinine is approximately 20 hours.[5] Plasma and saliva cotinine concentration in smokers is approximately 300 ng/ml and the urine concentration is approximately 1500 ng/ml.

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BOX 26-1 The Challenge of Assessing Smoking Status

Current Smokers

Ask about current smoking and past smoking. Many smokers are trying to quit and therefore simply asking how many cigarettes they are smoking today may not give an accurate assessment of their lifetime exposure (e.g., a patient currently smoking 5 cigarettes per day may have been smoking 40 cigarettes per day until yesterday when they decided to cut down). To get around this problem, pack-years should be calculated as follows:

- Pack-years = (number of packs smoked per day) × (number of years of smoking)

1 pack-year is the cumulative exposure that corresponds to smoking 1 pack of 20 cigarettes per day for 1 year. For example, a smoker who has smoked 20 cigarettes per day for 15 years has 15 pack-years of smoking.

Former Smokers

Ask the patient about past smoking. A patient with periodontitis may have a significant smoking history that has had an impact on their periodontal status, even though they may no longer smoke. Former smokers should always be congratulated on their achievement in quitting, but it is also very important to document the following:

- How much they used to smoke
- How many years they smoked
- When they quit

Is the patient's response accurate? Inaccurate or false reporting of smoking status is common (e.g., because the patient tells you what they think you want to hear or is embarrassed because they have not managed to cut down yet). Many patients report smoking 20 per day, because this is the number in a pack in most countries, so 20 may be a convenient response rather than an accurate response. Cultural factors may also influence responses.[94]

When is a smoker not a smoker?

- Smokers have smoked ≥100 cigarettes in their lifetime and currently smoke.
- Former smokers have smoked ≥100 cigarettes in their lifetime and do not currently smoke.
- Nonsmokers have not smoked ≥100 cigarettes in their lifetime and do not currently smoke.

It must be noted that many periodontal research studies have not used such definitions, however, and this can become particularly problematic in the context of what constitutes a former smoker. For example, from an exposure point of view, there is a big difference between someone who smoked 5 cigarettes per day for 10 years and quit 30 years ago compared to someone who smoked 40 cigarettes per day for 20 years and quit 6 months ago. It is always best in clinical practice to gather full information about the smoking history.

Nonsmokers usually have plasma and saliva concentrations <2 ng/ml (unless they are passive smokers).

Smoking is *the* major risk factor for periodontitis, affecting the prevalence, extent, and severity of disease. In addition, smoking adversely impacts the clinical outcome of nonsurgical and surgical therapy, as well as the long-term success of implant placement. With 41.9% of periodontitis cases in the US *attributable* to smoking,[2] it is essential to understand its impact on the initiation, progression, and management of the disease. This chapter discusses the effects of smoking on the prevalence, severity, etiology, and pathogenesis of periodontal disease, as well as the impact on treatment. The reader is referred to several excellent reviews on the topic for the detailed results of studies.[6–13]

TABLE 26-1 Effects of Smoking on Prevalence and Severity of Periodontal Disease

Periodontal Disease	Impact of Smoking
Gingivitis	↓ Gingival inflammation and bleeding on probing
Periodontitis	↑ Prevalence and severity of periodontal destruction ↑ Pocket depth, attachment loss, and bone loss ↑ Rate of periodontal destruction ↑ Prevalence of severe periodontitis ↑ Tooth loss ↑ Prevalence with increased number of cigarettes smoked per day ↓ Prevalence and severity with smoking cessation

↓, Decreased; ↑, increased.

EFFECTS OF SMOKING ON THE PREVALENCE AND SEVERITY OF PERIODONTAL DISEASES

Gingivitis

Controlled clinical studies have demonstrated that in human models of experimental gingivitis, the development of inflammation in response to plaque accumulation is reduced in smokers compared with nonsmokers (Table 26-1).[14,15] In addition, cross-sectional studies have consistently demonstrated that smokers present with less gingival inflammation than nonsmokers.[16–20] These data suggest that smokers have a decreased expression of clinical inflammation in the presence of plaque accumulation compared with nonsmokers. The microbiologic, immunologic, and physiologic factors that might account for this observation are discussed in detail later.

Periodontitis

Although gingival inflammation in smokers appears to be reduced in response to plaque accumulation compared with nonsmokers, an overwhelming body of data points to smoking as a major risk factor for increasing the prevalence and severity of periodontal destruction. Multiple cross-sectional and longitudinal studies have demonstrated that pocket depth, attachment loss, and alveolar bone loss are more prevalent and severe in patients who smoke compared with nonsmokers.[8,9,12,13] An assessment of the relationship between cigarette smoking and periodontitis was performed in more than 12,000 dentate individuals >18 years of age as part of the third National Health and Nutrition Examination Survey (NHANES III).[2] Periodontitis was defined as one or more sites with clinical attachment loss ≥4 mm and pocket depth ≥4 mm. Smoking status was defined using criteria established by the Centers for Disease Control and Prevention (CDC) (see Box 26-1). Of >12,000 individuals studied, 9.2% had periodontitis. This represented approximately 15 million cases of periodontitis in the US. On average, smokers were four times as likely to have periodontitis as persons who had never smoked after adjusting for age, gender, race/ethnicity, education, and income/poverty ratio. Former smokers were 1.7 times more likely to have periodontitis than persons who had never smoked. This study also demonstrated a dose-response relationship between cigarettes smoked per day and the odds of having periodontitis. In subjects smoking nine or fewer cigarettes

per day, the odds for having periodontitis were 2.8, whereas subjects smoking 31 or more cigarettes per day were almost six times more likely to have periodontitis. With former smokers, the odds of having periodontitis declined with the number of years since quitting. These data indicated that approximately 42% of periodontitis cases (6.4 million cases) in the US adult population were attributable to current smoking and approximately 11% (1.7 million cases) were attributable to former smoking. These data highlight the serious threat to dental public health posed by cigarette smoking and raise questions about the best methods for managing periodontitis in patients who smoke (Box 26-2).

These data are consistent with the findings of other cross-sectional studies performed in the United States and Europe. The odds ratio for periodontitis in current smokers has been estimated to range from as low as 1.5 to as high as 7.3, depending on the observed severity of periodontitis.[11] A metaanalysis of data from six such studies involving 2361 subjects indicated that current smokers were almost three times more likely to have severe periodontitis than nonsmokers.[10] The detrimental impact of long-term smoking on the periodontal and dentate status of older adults has been clearly demonstrated. Older adult smokers are approximately three times more likely to have severe periodontal disease,[21,22] and the number of years of tobacco use is a significant factor in tooth loss, coronal root caries, and periodontal disease.[23,24]

BOX 26-2 Should We Change How We Manage Periodontal Disease?

FACT 1: Smoking is *the* major risk factor for periodontal disease.

FACT 2: According to the literature, smoking may be responsible for more than half of periodontitis cases among adults in the United States.[2]

FACT 3: Depending on the study, approximately 10%-15% of adults in most populations examined have advanced chronic periodontitis.

Questions

- Would there be a benefit at the population level in periodontal health if all smokers in the population quit today?
- Would the dental profession be more successful in managing periodontitis in smokers if we focused on smoking cessation as *the* primary treatment strategy for managing their periodontitis?

These questions are intended to be controversial! It is clear that smoking has a huge deleterious impact on periodontal status. Smoking cessation MUST be a core part of periodontal treatment protocols in patients with periodontitis who smoke. The answer to the first question is almost certainly a resounding "YES" but probably will never be able to be tested. The answer to the second question is more difficult. Certainly, smokers with periodontitis must be educated about the harm they are causing to their periodontal tissues and must be encouraged and helped to quit. Treatment outcomes are improved in smokers who quit compared to those who continue to smoke.[92]

Two Final (Contentious!) Points

- If more than half of the cases of periodontitis are attributable to smoking, patients may be better served if the dental profession put more than half of its efforts in treating these patients into smoking cessation (as opposed to other forms of periodontal therapy).
- It is absurd for smokers in the West to worry about anything except stopping smoking.[95]

Smoking also has been shown to affect periodontal disease severity in younger individuals. Cigarette smoking is associated with increased severity of generalized aggressive periodontitis in young adults,[25] and those age 19 to 30 years who smoke are 3.8 times more likely to have periodontitis than nonsmokers.[26] Longitudinal studies have demonstrated that young individuals smoking more than 15 cigarettes per day showed the highest risk for tooth loss.[27] Also, smokers are more than six times as likely as nonsmokers to demonstrate continued attachment loss.[28] Over a 10-year period, bone loss has been reported to be twice as rapid in smokers as in nonsmokers[29] and proceeds more rapidly even in the presence of excellent plaque control.[30] Less information is available on the effects of cigar and pipe smoking, but it appears that effects similar to cigarette smoking are observed with these forms of tobacco use.[31–34] The prevalence of moderate and severe periodontitis and the percentage of teeth with ≥5 mm attachment loss was most severe in current cigarette smokers, but cigar and pipe smokers showed a severity of disease intermediate between the current cigarette smokers and nonsmokers.[31] Tooth loss is also increased in cigar and pipe smokers compared with nonsmokers.[34]

Interestingly, former smokers have less risk for periodontitis than current smokers but more risk than nonsmokers, and the risk for periodontitis decreases with the increasing number of years since quitting smoking.[2] This suggests that the negative effects of smoking on the host are reversible with smoking cessation and therefore that smoking cessation programs must be an integral component of periodontal education and therapy (Box 26-3). Several tobacco intervention approaches can be used in helping the patient deal with the nicotine withdrawal symptoms and psychologic factors associated with smoking cessation (Box 26-4).[35,36]

EFFECTS OF SMOKING ON THE ETIOLOGY AND PATHOGENESIS OF PERIODONTAL DISEASE

The increased prevalence and severity of periodontal destruction associated with smoking suggests that the host-bacterial interactions normally seen in chronic periodontitis are altered, resulting in more extensive periodontal breakdown (Table 26-3). This imbalance between bacterial challenge and host response may be caused by changes in the composition of the subgingival plaque, with increases in the numbers and virulence of pathogenic organisms, changes in the host response to the bacterial challenge, or a combination of both. This section discusses recent evidence on the effects of smoking on the microbiology, immune-inflammatory response, and physiology of periodontitis.

Microbiology

Studies have failed to demonstrate a difference in the rate of plaque accumulation of smokers compared with nonsmokers, suggesting that if an alteration in the microbial challenge in smokers exists, it results from a *qualitative* rather than quantitative alteration in the plaque.[17] Several studies have explored the possible changes in subgingival plaque caused by smoking, with conflicting and inconclusive results. In a study of 142 patients with chronic periodontitis, plaque samples from deep pockets (≥6 mm) showed no differences in the counts of *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, and *Prevotella intermedia*.[37] In a similar study of 615 patients using immunoassay, the prevalence of *A. actinomycetemcomitans*, *P. gingivalis*, *P. intermedia*, and *Eikenella corrodens* was not found to be significantly different between smokers and nonsmokers.[38] In contrast, other studies have shown differences in the

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BOX 26-3 Helping Your Patients to Quit Smoking

Smoking cessation is a public health priority for governments around the world. Excellent online resources are available to provide information about the harmful effects of smoking and to help people to quit, as follows:

- http://www.cdc.gov/tobacco
- http://www.ash.org.uk
- http://smokefree.nhs.uk
- http://www.tobacco-oralhealth.net

Smoking cessation must be an integral part of the management of dental patients who smoke, and it is the responsibility of all dental care professionals to address this issue with their patients. The dental team is well placed to provide this treatment, since they see patients on a regular basis over time as part of ongoing routine dental management. Furthermore, interventions to help patients quit smoking in dental practices are effective, with quit rates of 15%- 20% compared to around 5% in control groups.[96] The whole dental team should therefore be involved in smoking cessation, but this is not always undertaken. Why? Some of the barriers to providing smoking cessation counseling in the dental practice are shown in Table 26-2.

Various methods for helping patients to quit smoking in the dental environment have been described, and these are typically referred to as *Brief Intervention Programs*. One such program[8] is the 5 As, as follows:

ASK Ask the patient about their smoking status (see Box 26-1).

This should be part of the medical history.

ADVISE Advise smokers of the associations between oral disease and smoking.

Be informative, honest, and helpful but not judgmental.

The patient's response to this information will reveal their interest in quitting.

ASSESS Assess the patient's interest and readiness to attempt smoking cessation.

Patients may not yet be in an action phase to quit smoking, which is why it is always important to make these assessments every time you see the patient.

ASSIST Assist the patient in their quit attempt.

If you are trained, there are many techniques that can be used (see Box 26-4).

Alternatively, assist the patient in seeking the help they need.

ARRANGE Arrange follow-up or referral to professional smoking cessation services.

The most important aspect of this is to keep in regular contact particularly around the quit date and in the immediate period after.

A simplified version that is particularly useful for the dental team is the AAR program, as follows:

ASK Ask the patient about their smoking status.

ADVISE Advise smokers of the associations between oral disease and smoking.

REFER Refer the patient to a professional smoking cessation program.

BOX 26-4 Methods for Smoking Cessation

Set a Quit Date

This is the very first step and is highly important. The patient must set their own quit date on which they will stop smoking and will remove all tobacco products from their environment.

Will Power Alone

Success rate at 12 months is 3%. This is the least effective method of smoking cessation, with only 3% of smokers managing to quit after 12 months.

Self-Help Materials

Success rate at 12 months is 4%. This includes a variety of literature and online resources that patients can access. The dental team can be helpful in providing literature and guiding patients towards resources they can access.

Brief Intervention Program in Primary Care

Success rate at 12 months is 5%–10%.[36] Brief advice is very important and should always be given at every dental visit. Enhancing the role of specially trained dental hygienists and prevention auxiliaries in giving lifestyle advice is of major benefit. If the dental team gave even brief advice to most of their smoking patients and achieved a low cessation rate of 5% over time, a significant proportion of smokers would be assisted to quit each year.

Nicotine Replacement Therapy (NRT)

Success rate at 12 months is 10%–20%. NRT generally doubles the success rate of smoking cessation. For example, in primary care settings in which brief advice is given, 12-month success rates double from around 5% to around 10% if NRT is used. In an intensive setting such as a smokers clinic, success rates increase from around 10%-20%. NRT is not a magic cure but helps with craving and withdrawal when a person quits smoking, and although it does contain nicotine, it does not contain the toxic products, such as tar and carbon monoxide (CO), in cigarette smoke. NRT products include the following:

- Patches (available in different doses and worn for 16–24 hours per day).
- Lozenges and gum (available in different flavors, should be chewed slowly to allow the nicotine to be absorbed through the mouth).
- Nasal spray (delivers nicotine solution in a nasal spray).
- Inhalator (plastic mouthpiece with a supply of nicotine cartridges that fit on the end, nicotine is absorbed in the mouth by drawing on it like a cigarette).

Bupropion

Success rate at 12 months is 20%–30%. This medication is used as an antidepressant at higher doses but is effective for smoking cessation at lower doses. It is usually prescribed starting 1–2 weeks before the quit date, initially at 150 mg once per day for 6 days then 150 mg twice daily for 7–9 weeks. There are serious potential drug interactions so this medication should be prescribed by the patient's medical doctor.

Other Methods

Whatever works for the patient is good! In addition to combinations of the above, techniques for smoking cessation can include intensive counseling, motivational interviewing, cognitive behavioral therapy, hypnosis, and acupuncture.

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TABLE 26-2 Barriers Against and Stimuli for Provision of Smoking Cessation Advice by the Dental Team in Dental Practice

	Barriers	Stimuli
Professional characteristics	Perceived lack of efficacy	Self-efficacy
	Concerned about disturbing the patient-dentist relationship	Positive attitude regarding giving advice as their role
	Lack of knowledge about how to give the correct advice	
	Belief that counseling is unnecessary	Training
	A perception that advising patients is frustrating and has a low success rate	Self-belief
	The dental team member may be a smoker themselves	
	Lack of confidence in giving the advice	
Practice organization	Lack of time	Task delegation
	No organizational support in the practice to deliver advice	A practice focus to help smokers quit
Health care system	No reimbursement	Local availability of smoking cessation clinics
	No referral options for further help	

Adapted from Rosseel JP, Jacobs JE, Hilberink SR, et al: *Br Dent J* 206:E13. 2009.

TABLE 26-3 Effects of Smoking on the Etiology and Pathogenesis of Periodontal Disease

Etiologic Factor	Impact of smoking
Microbiology	No effect on rate of plaque accumulation. ↑ Colonization of shallow periodontal pockets by periodontal pathogens. ↑ Levels of periodontal pathogens in deep periodontal pockets.
Immune-inflammatory response	Altered neutrophil chemotaxis, phagocytosis, and oxidative burst. ↑ TNF-α and PGE_2 in GCF. ↑ Neutrophil collagenase and elastase in GCF. ↑ Production of PGE_2 by monocytes in response to LPS.
Physiology	↓ Gingival blood vessels with ↑ inflammation. ↓ GCF flow and bleeding on probing with ↑ inflammation. ↓ Subgingival temperature. ↑ Time needed to recover from local anesthesia.

↓, Decreased; ↑, increased; *TNF-α,* tumor necrosis factor alpha; *GCF,* gingival crevicular fluid; *PGE2,* prostaglandin E_2; *LPS,* lipopolysaccharide.

microbial composition of subgingival plaque between smokers and nonsmokers. In a study of 798 subjects with different smoking histories, it was found that smokers had significantly higher levels of *Tannerella forsythia (*previously called *Bacteroides forsythus*) and that smokers were 2.3 times more likely to harbor *T. forsythia* than nonsmokers and former smokers.[39] Of particular interest was the observation that smokers do not respond to mechanical therapy as well as nonsmokers; this is associated with increased levels of *T. forsythia*, *A. actinomycetemcomitans*, and *P. gingivalis* remaining in the pockets after therapy in the smoking group when compared with nonsmokers.[40–43]

Many discrepancies between the findings of microbiologic studies are a function of the methodology involved, including bacterial counts versus proportions or prevalence of bacteria, number of sites sampled and the pocket depths selected, the sampling technique, the disease status of the subject, and the methods of bacterial enumeration and data analysis. In an attempt to overcome some of these problems, a recent study sampled subgingival plaque from all teeth with the exception of third molars in 272 adult subjects, including 50 current smokers, 98 past smokers, and 124 nonsmokers.[44] Using checkerboard DNA-DNA hybridization technology to screen for 29 different subgingival species, it was found that members of the orange and red complexes, including *Eikenella nodatum*, *Fusobacterium nucleatum* ss *vincentii*, *P. intermedia*, *Peptostreptococcus micros*, *Prevotella nigrescens*, *T. forsythia*, *P. gingivalis*, and *Treponema denticola,* were significantly more prevalent in current smokers than in nonsmokers and former smokers. The increased prevalence of these periodontal pathogens was caused by an increased colonization of shallow sites (pocket depth ≤4 mm), with no differences among smokers, former smokers, and nonsmokers in pockets 4 mm or greater. In addition, these pathogenic bacteria were more prevalent in the maxilla than the mandible. These data suggest that smokers have a greater extent of colonization by periodontal pathogens than nonsmokers or former smokers and that this colonization may lead to an increased prevalence of periodontal breakdown.

Immune-Inflammatory Responses

The immune response of the host to plaque accumulation is essentially protective. In periodontal health and gingivitis, a balance exists between the bacterial challenge of plaque and the immune-inflammatory responses in the gingival tissues, with no resulting loss of periodontal support. In contrast, periodontitis is associated with an alteration in the host-bacterial balance that may be initiated by changes in the bacterial composition of subgingival plaque, changes in the host responses, or a combination of both.

Smoking exerts a major effect on the protective elements of the immune-inflammatory response, resulting in an increase in the extent and severity of periodontal destruction. The deleterious effects of smoking appear to result in part from a down-regulation of the immune response to bacterial challenge. The neutrophil is an important component of the host response to bacterial infection, and alterations in neutrophil number or function may result in localized and systemic infections. Critical functions of neutrophils include *chemotaxis* (directed locomotion from the bloodstream to the site of infection), *phagocytosis* (internalization of foreign particles such as bacteria), and *killing* using oxidative and nonoxidative mechanisms. Neutrophils obtained from the peripheral blood, oral

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cavity, or saliva of smokers or exposed in vitro to whole tobacco smoke or nicotine have been shown to demonstrate functional alterations in chemotaxis, phagocytosis, and the oxidative burst.[45,46] In vitro studies of the effects of tobacco products on neutrophils have shown detrimental effects on cell movement, as well as the oxidative burst.[47–51] In addition, levels of antibody to periodontal pathogens essential for phagocytosis and killing of bacteria, specifically immunoglobulin G2 (IgG2), have been reported to be reduced in smokers versus nonsmokers with periodontitis,[42,52–54] suggesting that smokers may have reduced protection against periodontal infection. In contrast, elevated levels of tumor necrosis factor alpha (TNF-α) have been demonstrated in the gingival crevicular fluid (GCF) of smokers,[55] as well as elevated levels of prostaglandin E_2 (PGE_2), neutrophil elastase, and matrix metalloproteinase-8 (MMP-8).[56] In vitro studies also have shown that exposure to nicotine increases the secretion of PGE_2 by monocytes in response to lipopolysaccharide (LPS).[57]

These data suggest that smoking may impair the response of neutrophils to plaque bacteria but may also increase the release of tissue-destructive enzymes. The exact changes in the immunologic mechanisms involved in the rapid tissue destruction seen in smokers are currently unclear. Further studies are needed to define the effects of tobacco use on the immune-inflammatory response and tissue destruction in periodontitis.

Physiology

Previous studies have shown that the clinical signs of inflammation are less pronounced in smokers than in nonsmokers.[15,18] This may result from alterations in the inflammatory response in smokers, as outlined previously, or from alterations in the vascular response of the gingival tissues. Although no significant differences in the vascular density of healthy gingiva have been observed between smokers and nonsmokers,[58] the response of the microcirculation to plaque accumulation appears to be altered in smokers compared with nonsmokers. With developing inflammation, increases in GCF flow, bleeding on probing,[14] and gingival blood vessels[59] are less in smokers than nonsmokers. In addition, the oxygen concentration in healthy gingival tissues appears to be less in smokers than nonsmokers, although this condition is reversed in the presence of moderate inflammation.[60] Subgingival temperatures are lower in smokers than nonsmokers,[61] and recovery from the vasoconstriction caused by local anaesthetic administration takes longer in smokers.[62,63] These cumulative data suggest that significant alterations are present in the gingival microvasculature of smokers compared with nonsmokers and that these changes lead to decreased blood flow and decreased clinical signs of inflammation. This explains the long observed phenomenon of a transient increase in gingival bleeding when a smoker quits.

EFFECTS OF SMOKING ON RESPONSE TO PERIODONTAL THERAPY

Nonsurgical Therapy

Numerous studies have indicated that current smokers do not respond as well to periodontal therapy as nonsmokers or former smokers (Table 26-4). The majority of clinical research supports the observation that pocket depth reduction is more effective in nonsmokers than in smokers after nonsurgical periodontal therapy, including oral hygiene instruction, scaling, and root planing.[40–43,64–66] In addition, gains in clinical attachment as a result of nonsurgical treatment are less pronounced in smokers than in nonsmokers. In a study of patients with previously untreated advanced periodontal disease, nonsurgical therapy resulted in significantly greater mean reductions in pocket depth and bleeding on probing in nonsmokers than in smokers when evaluated 6 months after completion of therapy.[43] Average pocket reductions of 2.5 mm for nonsmokers and 1.9 mm for smokers were observed in pockets that averaged 7 mm before treatment. In another study, the nonsurgical management of pockets ≥5 mm showed that smokers had less pocket depth reduction than nonsmokers after 3 months (1.29 mm versus 1.76 mm), as well as less gain in clinical attachment levels.[40] When a higher level of plaque control can be achieved as part of nonsurgical care, the differences in the resolution of 4 mm to 6 mm pockets between nonsmokers and smokers became clinically less significant.[66] It can be concluded that smokers respond less well to nonsurgical therapy than nonsmokers. With excellent plaque control, however, these differences may be minimized (but the emphasis is on truly excellent plaque control). When comparing current smokers with former smokers and nonsmokers, the former and nonsmoking subjects appear to respond equally well to nonsurgical care,[40] reinforcing the need for patients to be informed of the benefits of smoking cessation.

TABLE 26-4 Effects of Smoking on Response to Periodontal Therapy

Therapy	Effects of Smoking
Nonsurgical	↓ Clinical response to root surface debridement. ↓ Reduction in pocket depth. ↓ Gain in clinical attachment levels. ↓ Negative impact of smoking with ↑ level of plaque control.
Surgery and implants	↓ Pocket depth reduction and ↓ gain in clinical attachment levels after access flap surgery. ↑ Deterioration of furcations after surgery. ↓ Gain in clinical attachment levels, ↓ bone fill, ↑recession, and ↑ membrane exposure after GTR. ↓ Pocket depth reduction after bone graft procedures. ↑ Risk for implant failure and periimplantitis.
Maintenance care	↑ Pocket depth and attachment loss during maintenance therapy. ↑ Disease recurrence in smokers. ↑ Need for re-treatment in smokers. ↑ Tooth loss in smokers after surgical therapy.

↓, Decreased; ↑ increased; *GTR,* guided tissue regeneration.

Surgical Therapy and Implants

The less favorable response of the periodontal tissues to nonsurgical therapy that is observed in current smokers is also observed after surgical therapy. In a longitudinal comparative study of the effects of four different treatment modalities, including coronal scaling, root planing, modified Widman flap surgery, and osseous resection surgery, smokers ("heavy" defined as ≥20 cigarettes/day; "light" defined as ≤19 cigarettes/day) consistently showed less pocket

reduction and less gain in clinical attachment than nonsmokers or former smokers.[65] These differences were evident immediately after the completion of therapy and continued throughout 7 years of supportive periodontal therapy. During the 7 years, deterioration at furcation areas was greater in heavy and light smokers than in former smokers and nonsmokers. Smoking also has been shown to have a negative impact on the outcomes of guided tissue regeneration (GTR)[67,68] and the treatment of infrabony defects by bone grafts.[69] By 12 months after GTR therapy at deep infrabony defects, smokers demonstrated less than half the attachment gain as nonsmokers (2.1 mm versus 5.2 mm).[67] In a second study, 73 smokers also showed less attachment gain than nonsmokers (1.2 mm versus 3.2 mm), more gingival recession, and less bone infill of the defect. Similarly, after the use of bone grafts for the treatment of infrabony defects, smokers showed less reduction in pocket depths than nonsmokers.[69]

Open flap access surgery without regenerative or grafting procedures is the most common surgical procedure used for accessing the root and bone surfaces. By 6 months after this procedure, smokers showed significantly less reduction of deep pockets (≥7 mm) than nonsmokers (3.0 mm for smokers versus 4.0 mm for nonsmokers) and significantly less gain in clinical attachment (1.8 versus 2.8 mm), even though all of the patients received supportive periodontal therapy every month for 6 months.[70] Of clinical relevance was the observation that only 16% of deep pockets in smokers returned to 3 mm or less at 6 months after surgery, whereas 47% of deep pockets in nonsmokers were 3 mm or less after completion of therapy.

Three metaanalyses have investigated the influence of smoking on the short- and long-term outcomes of implant therapy and have identified that smoking increases the risk of implant failure.[71–73] These studies used various definitions to define implant failure, including implant loss, implant bone loss, mobility, pain, and periimplantitis. Overall, the risk for implant failure in smokers appears to be approximately double the risk for failure in nonsmokers, and the risks appear to be higher in maxillary implants and when implants are placed in poor quality bone. Smoking has also been shown to be a risk factor for periimplantitis, with a majority of studies showing a significant increase in periimplant bone loss compared with nonsmokers.[74] Collectively, these data indicate that implant failure is more common in smokers than nonsmokers, but since numerous factors can influence implant success (see Part 7), further controlled clinical trials are needed to address the role of smoking as an independent variable in implant failure. However, given the current evidence, patients should be informed and advised of the benefits of smoking cessation and the potential risks of smoking for implant failure.

Maintenance Therapy

The detrimental effect of smoking on treatment outcomes appears to be long-lasting and independent of the frequency of maintenance therapy. After four different modalities of therapy, including scaling, scaling and root planing, modified Widman flap surgery, and osseous surgery, maintenance therapy was performed by a hygienist every 3 months for 7 years.[65] Smokers consistently had deeper pockets than nonsmokers and less gain in attachment when evaluated each year for the 7-year period. Even with more intensive maintenance therapy given every month for 6 months after flap surgery,[70] smokers had deeper and more residual pockets than nonsmokers, even though no significant differences in plaque or bleeding on probing scores were found. These data suggest that the effects of smoking on the host response and the healing characteristics of the periodontal tissues may have a long-term effect on pocket resolution in smokers, possibly requiring more intensive management in the maintenance phase. Smokers also tend to experience more periodontal breakdown than nonsmokers after therapy.[75,76] In studies of patients who failed to respond to conventional therapy, including different combinations of oral hygiene instruction, scaling and root planing, surgery, and antibiotics, approximately 90% of these poorly responding patients were smokers.[75,77]

It is clear from these studies that smokers (1) may present with periodontal disease at an early age, (2) may be difficult to treat effectively with conventional therapeutic strategies, and (3) may continue to have progressive or recurrent periodontitis. For this reason, smoking cessation counseling must be a cornerstone of periodontal therapy in smokers.

EFFECTS OF SMOKING CESSATION ON PERIODONTAL TREATMENT OUTCOMES

The effect of smoking cessation on periodontal status has been studied in a large number of cross-sectional and cohort observational studies, in which the periodontal status of smokers, former smokers and nonsmokers is compared.* Similarly, periodontal treatment outcomes have been assessed in smokers, former smokers, and nonsmokers.[41,65,86–89] Collectively, these studies have demonstrated that smokers have significantly worse periodontal status (deeper probing depths, more attachment loss and bone loss) than either former smokers or nonsmokers and usually have poorer treatment outcomes. The periodontal status of former smokers is intermediate to that of current smokers or nonsmokers and appears to be usually closer to that of nonsmokers.

There are very few intervention studies on the effect of smoking cessation on periodontal treatment outcomes (i.e., studies in which smokers were helped to quit and the effect on periodontal status was then assessed). Two short-term studies have indicated that smoking has a negative impact on the gingival vasculature and that these changes are reversible on quitting smoking.[90,91] One intervention study has assessed the impact of smoking cessation on outcomes after nonsurgical periodontal treatment.[92] This study employed dental hygienists who were trained as smoking cessation advisors and achieved a 20% quit rate at 12 months in a population of smokers who also had periodontitis using a variety of strategies, including counseling, NRT, and bupropion. This confirms the effectiveness of dental health care professionals in providing smoking cessation counseling. All the patients in the study received nonsurgical therapy as treatment for their periodontitis, in addition to smoking cessation counseling. Those individuals who successfully quit smoking for the entire 12 months of the study had the best response to the periodontal treatment. The treatment responses in the nonquitters and the failed quitters (i.e., the "oscillators" who initially quit, but then resumed smoking) were significantly poorer than those seen in the quitters, and did not differ significantly from each other.

The benefit of smoking cessation on the periodontium is likely to be mediated through various pathways, such as a shift towards a less pathogenic microflora, recovery of the gingival microcirculation and improvements in aspects of the immune-inflammatory responses. In support of this, in the interventional study described previously,[92] plaque samples were collected as the study progressed.

*References 2, 23, 26, 29, 30, and 78–85.

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It became clear that subgingival microbial profiles differed significantly between smokers and quitters at 6 and 12 months following smoking cessation.[93] At 6 and 12 months posttreatment, the microbial community in smokers was similar to that observed at baseline (i.e., before periodontal treatment/smoking cessation counseling), whereas the quitters demonstrated significantly divergent profiles, and changes in bacterial levels contributed to this shift. These data suggest a critical role for smoking cessation in altering the subgingival biofilm.

To conclude, smoking is the major risk factor for periodontitis, and smoking cessation should be an integral part of periodontal therapy in patients who smoke and needs to be considered a priority for the management of periodontitis in smokers.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

Smoking results in a dramatic increased risk of periodontitis and increases the likelihood of poor responses to periodontal therapy. Nonsurgical therapy can be successful in smokers if excellent oral hygiene is achieved, but surgical treatments, including implant placement, all have decreased favorable outcomes in smokers. Smokers have less gingivitis and bleeding on probing, so it is more difficult to detect attachment loss and bone destruction; thus it is imperative that comprehensive pocket depth recording and full-mouth radiographs accompany their periodontal examination.

Dental clinicians have a unique and obligatory responsibility to inform smokers of their susceptibility to advanced periodontal destruction and poor response to surgical procedures. The demonstration of specific smoking-related periodontal problems is often the first salutary information that the patient accepts concerning the body's deleterious reaction to smoking. This can trigger a desire for quitting smoking and the dental team has a special role in initiating smoking cessation. Nicotine replacement therapy and behavior modification approaches generally have a 20% success rate. The addition of systemic medication, such as bupropion and varenicline, may add another 10% of quitters at 1 year, but because of the occasional serious side effects (seizures, arrhythmias, tachycardia, depression, and suicidality), it is suggested that these agents may be best used under the direction of a physician.

Smoking cessation should precede periodontal surgical intervention, and if patients continue to smoke, clinicians may consider limiting periodontal treatment to nonsurgical approaches accompanied by rigorous short interval (2 to 3 months) maintenance visits until smoking ceases.

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PART 5

Relationship Between Periodontal Disease and Systemic Health

Perry R. Klokkevold

A significant interrelationship between periodontal disease and systemic health is more evident today than ever before. Inflammation is the primary pathologic feature of periodontal disease, and bacterial plaque is the essential etiologic factor responsible for inducing the host inflammatory process. Systemic diseases and variations in the host response to the bacterial challenge lead to differences in the severity of periodontal disease from one individual to another. Whereas, increasing evidence supports the premise that chronic inflammatory periodontal disease has a significant effect on systemic health conditions such as coronary heart disease, stroke, or adverse pregnancy outcomes.

The relationship between periodontal disease and systemic health is a two-way road, with systemic host factors acting locally to reduce resistance to periodontal destruction and the local bacterial challenge generating an inflammatory response with the potential to induce adverse systemic outcomes. The chapters in Part 5 describe the influence of systemic disease and conditions on the periodontium and the role of inflammatory periodontal disease on systemic conditions.

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CHAPTER 27

Influence of Systemic Conditions on the Periodontium

Perry R. Klokkevold and Brian L. Mealey

CHAPTER OUTLINE

ENDOCRINE DISORDERS AND HORMONAL CHANGES
- Diabetes Mellitus
- Female Sex Hormones (online only)
- Hyperparathyroidism (online only)

HEMATOLOGIC DISORDERS AND IMMUNE DEFICIENCIES
- Leukocyte (Neutrophil) Disorders
- Leukemia
- Anemia (online only)
- Thrombocytopenia (online only)
- Antibody Deficiency Disorders (online only)

GENETIC DISORDERS
- Chédiak-Higashi Syndrome (online only)
- Lazy Leukocyte Syndrome (online only)
- Leukocyte Adhesion Deficiency (online only)
- Papillon-Lefèvre Syndrome (online only)
- Down Syndrome

STRESS AND PSYCHOSOMATIC DISORDERS
- Psychosocial Stress, Depression, and Coping (online only)
- Stress-Induced Immunosuppression (online only)
- Influence of Stress on Periodontal Therapy Outcomes
- Psychiatric Influence of Self-Inflicted Injury

NUTRITIONAL INFLUENCES
- Fat-Soluble Vitamin Deficiency (online only)
- Water-Soluble Vitamin Deficiency (online only)
- Protein Deficiency (online only)

MEDICATIONS
- Bisphosphonates
- Corticosteroids

OTHER SYSTEMIC CONDITIONS
- Osteoporosis
- Congenital Heart Disease (online only)
- Hypophosphatasia (online only)
- Metal Intoxication (online only)

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Some text, figures, references, and tables may be out of numeric order in this printed chapter.

Many systemic diseases, disorders, and conditions have been implicated as risk indicators or risk factors in periodontal disease. Clinical and basic science research over the past several decades has led to an improved understanding and appreciation for the complexity and pathogenesis of periodontal diseases.[183] Although there is clear evidence for a bacterial etiology and there are *specific* bacteria (periodontal pathogens) associated with destructive periodontal disease, the presence of these pathogens does not invariably cause disease. Their absence, on the other hand, appears to be consistent with periodontal health. The role of bacteria in disease etiology and pathogenesis is discussed in Chapters 23 and 25.

Perhaps the most significant advance in our understanding of the pathogenesis of periodontitis is that the host response varies between individuals and that an altered, deficient, or exaggerated host immune response to bacterial pathogens may lead to more severe forms of the disease. In other words, the individual host immune response to periodontal pathogens is very important and likely explains much of the differences in disease severity observed from one individual to another. Furthermore, systemic diseases, disorders, and conditions alter host tissues and physiology, which may impair the host's barrier function and immune defense against periodontal pathogens creating the opportunity for destructive periodontal disease.

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Recent evidence also suggests that periodontal infections can adversely affect systemic health with manifestations such as coronary heart disease, stroke, diabetes, preterm labor, low-birthweight delivery, and respiratory disease.[163] The role of periodontal infections on these systemic health conditions is discussed in Chapter 28.

The interrelationships between periodontal infections and host defense are complex. A number of environmental, physical, and psychosocial factors have the potential to alter periodontal tissues and the host immune response, resulting in more severe periodontal disease expression. It is important to recognize that the systemic diseases, disorders, or conditions themselves do not cause periodontitis, but they may predispose, accelerate, or otherwise increase its progression. This chapter discusses the influence of several notable systemic diseases, disorders, and conditions on the periodontium.

ENDOCRINE DISORDERS AND HORMONAL CHANGES

Endocrine diseases, such as diabetes and hormonal fluctuations, that are associated with puberty and pregnancy are well-known examples of systemic conditions that adversely affect the condition of the periodontium. Endocrine disturbances and hormone fluctuations affect the periodontal tissues directly, modify the tissue response to local factors, and produce anatomic changes in the gingiva that may favor plaque accumulation and disease progression. This section describes the evidence supporting the relationship among endocrine disorders, hormonal changes, and periodontal disease.

Diabetes Mellitus

Diabetes mellitus is an extremely important disease from a periodontal standpoint. It is a complex metabolic disorder characterized by chronic hyperglycemia. Diminished insulin production, impaired insulin action, or a combination of both result in the inability of glucose to be transported from the bloodstream into the tissues, which in turn results in high blood glucose levels and excretion of sugar in the urine. Lipid and protein metabolism is altered in diabetes as well. Uncontrolled diabetes (chronic hyperglycemia) is associated with several long-term complications, including microvascular diseases (retinopathy, nephropathy, or neuropathy), macrovascular diseases (cardiovascular, cerebrovascular), an increased susceptibility to infections, and poor wound healing. An estimated 23.6 million individuals (children and adults), or 7.8% of the United States (US) population, have diabetes.[192] Approximately 5.7 million of these individuals are unaware that they have the disease.

There are two major types of diabetes, type 1 and type 2, with several less common secondary types. *Type 1* diabetes mellitus, formerly insulin-dependent diabetes mellitus (IDDM), is caused by a cell-mediated autoimmune destruction of the insulin-producing beta cells of the islets of Langerhans in the pancreas, which results in insulin deficiency. Type 1 diabetes accounts for 5% to 10% of all cases of diabetes and most often occurs in children and young adults. This type of diabetes results from a lack of insulin production and is very unstable and difficult to control. It has a marked tendency toward ketosis and coma, is not preceded by obesity, and requires injected insulin to be controlled. Patients with type 1 diabetes mellitus present with the symptoms traditionally associated with diabetes, including polyphagia, polydipsia, polyuria, and predisposition to infections.

Type 2 diabetes mellitus, formerly non–insulin-dependent diabetes mellitus (NIDDM), is caused by peripheral resistance to insulin action, impaired insulin secretion, and increased glucose production in the liver. The insulin-producing beta cells in the pancreas are not destroyed by cell-mediated autoimmune reaction. It typically begins as insulin resistance leading to reduced pancreas production of insulin as the demand increases. Type 2 diabetes is the most common form of diabetes, accounting for 90% to 95% of all cases, and usually has an adult onset. Individuals often are not aware they have the disease until severe symptoms or complications occur. Type 2 diabetes generally occurs in obese individuals and can often be controlled by diet and oral hypoglycemic agents. Ketosis and coma are uncommon. Type 2 diabetes can present with the same symptoms as type 1 diabetes but typically in a less severe form.

An additional category of diabetes is *hyperglycemia* secondary to other diseases or conditions. A prime example of this type of hyperglycemia is gestational diabetes associated with pregnancy. *Gestational diabetes* develops in 2% to 5% of all pregnancies but disappears after delivery. Women who have had gestational diabetes are at increased risk of developing type 2 diabetes later in life. Other secondary types of diabetes are those associated with diseases that involve the pancreas and destruction of the insulin-producing cells. Endocrine diseases, such as acromegaly and Cushing's syndrome, tumors, pancreatectomy, and drugs or chemicals that cause altered insulin levels, are included in this group. Experimentally induced types of diabetes generally belong in this category rather than in type 1 or 2 diabetes mellitus.

Oral Manifestations. Numerous oral changes have been described in diabetic patients, including cheilosis, mucosal drying and cracking, burning mouth and tongue, diminished salivary flow, and alterations in the flora of the oral cavity, with greater predominance of *Candida albicans,* hemolytic streptococci, and staphylococci.[2,22,99,158] An increased rate of dental caries has also been observed in poorly controlled diabetes.[74,84] Importantly, however, these changes are not always present, are not specific, and are not pathognomonic for diabetes.[161] Furthermore, these changes are less likely to be observed in well-controlled diabetic patients. Individuals with controlled diabetes have a normal tissue response, a normally developed dentition, a normal defense against infections, and no increase in the incidence of caries.[233]

The influence of diabetes on the periodontium has been thoroughly investigated. Although it is difficult to make definitive conclusions about the specific effects of diabetes on periodontium, a variety of changes have been described, including a tendency toward enlarged gingiva, sessile or pedunculated gingival polyps, polypoid gingival proliferations, abscess formation, periodontitis, and loosened teeth[112] (Figure 27-1). Perhaps the most striking changes in uncontrolled diabetes are the reduction in defense mechanisms and the increased susceptibility to infections, leading to destructive periodontal disease. In fact, periodontal disease is considered to be the sixth complication of diabetes.[144] Periodontitis in type 1 diabetic patients appears to start after age 12 years.[47] The prevalence of periodontitis has been reported as 9.8% in 13- to 18-year-old patients, increasing to 39% in those 19 years and older.

The extensive literature on this subject and the overall impression of clinicians indicate that *periodontal disease in diabetic patients follows no consistent or distinct pattern.* Severe gingival inflammation, deep periodontal pockets, rapid bone loss, and frequent periodontal abscesses often occur in poorly controlled diabetic patients with

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Figure 27-1 Periodontal condition in patients with diabetes. **A,** Adult with diabetes (blood glucose level > 400 mg/dl). Note the gingival inflammation, spontaneous bleeding, and edema. **B,** Same patient as in **A.** Improved control of diabetes following 4 days of insulin therapy (blood glucose level <100 mg/dl). The clinical periodontal condition has improved without local therapy. **C,** Adult patient with uncontrolled diabetes. Note the enlarged, smooth, erythematous gingival margins and papilla in the anterior area. **D,** Same patient as in **C.** Lingual view of the right mandibular area. Note the inflamed and swollen tissues in the anterior and premolars area. **E,** Adult patient with diabetes. Suppurating abscess on the buccal surface of the maxillary premolars in a patient with uncontrolled diabetes.

poor oral hygiene[3] (Figures 27-2 and 27-3). Children with type 1 diabetes tend to have more destruction around the first molars and incisors than elsewhere, but this destruction becomes more generalized at older ages.[47] In juvenile diabetic patients, extensive periodontal destruction often occurs as a consequence of having more severe disease at a younger age.

Other investigators have reported that the rate of periodontal destruction appears to be similar for those with diabetes and those without diabetes, up to 30 years of age.[90,227] After age 30, diabetic patients have a greater degree of periodontal destruction, possibly related to more disease destruction over time. Patients showing overt diabetes over more than 10 years have greater loss of periodontal support than those with a diabetic history of less than 10 years.[90] This destruction may also be related to the diminished tissue integrity that continues to deteriorate over time (see later section for description of altered collagen metabolism).

Although some studies have not found a correlation between the diabetic state and the periodontal condition, the majority of well-controlled studies show a higher prevalence and severity of periodontal disease in individuals with diabetes than in nondiabetic persons with similar local factors.* Findings include a greater loss of attachment, increased bleeding on probing, and increased tooth mobility. A study of risk indicators for a group of 1426 patients, ages 25 to 74, revealed that individuals with diabetes are twice as likely to exhibit attachment loss as nondiabetic individuals.[106] The lack of consistency across studies is most likely related to the different degrees of diabetic involvement, variations in level of disease control and diversity of indices, and patient sampling from one study to another.

Recent studies suggest that uncontrolled or poorly controlled diabetes is associated with an increased susceptibility to and severity of infections, including periodontitis.[16,207] As with other systemic conditions associated with periodontitis, diabetes mellitus does not cause gingivitis or periodontitis, but evidence indicates that it alters the response of the periodontal tissues to local factors,

*References 14, 22, 38, 50, 109, 173, 175, 176, and 230.

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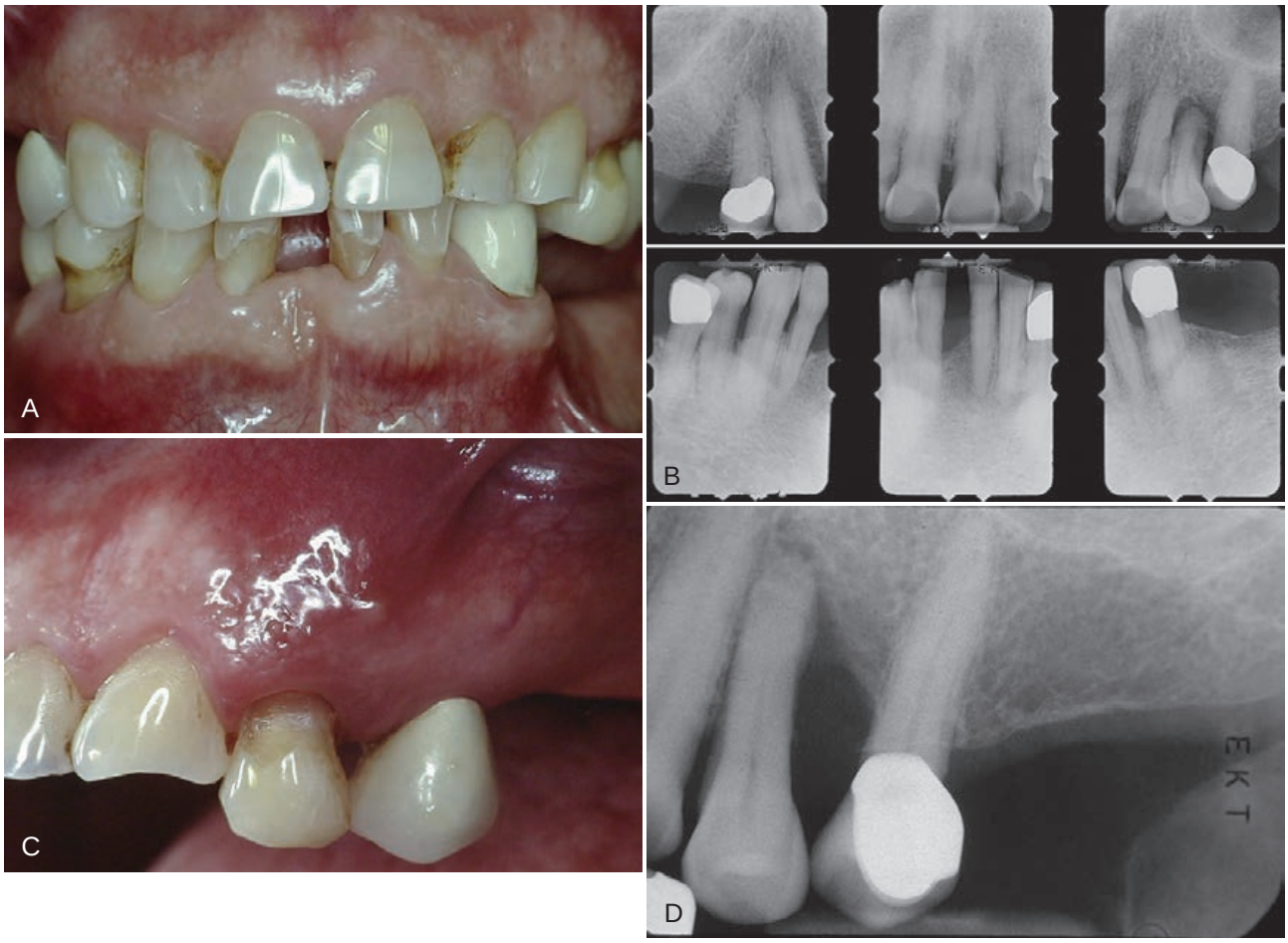

Figure 27-2 Sixty-year-old patient with long-term history of type 2 diabetes. **A,** Anterior retracted view of dental/periodontal condition. Note missing posterior teeth, supereruption of premolars, and mild generalized gingival inflammation. **B,** Periapical radiographs of remaining teeth. Note mild, generalized bone loss with localized areas of severe bone loss. Failure to replace posterior teeth adds to the occlusal burden of the remaining dentition. **C,** Clinical photograph of maxillary premolar area presenting with abscess. Notice diffuse erythema, inflammation surrounding abscess area. **D,** Periapical radiograph of maxillary premolar showing extensive bone loss associated with abscess.

hastening bone loss and delaying postsurgical healing. Frequent periodontal abscesses appear to be an important feature of periodontal disease in diabetic patients.

Approximately 40% of adult Pima Indians in Arizona have type 2 diabetes. A comparison of individuals with or without diabetes in this Native American tribe has shown a clear increase in prevalence of destructive periodontitis, as well as a 15% increase in edentulousness, in diabetic patients.[217] The risk of developing destructive periodontitis increases threefold in these individuals.[71]

Bacterial Pathogens. The glucose content of gingival fluid and blood is higher in individuals with diabetes than in those without diabetes, with similar plaque and gingival index scores.[77] The increased glucose in the gingival fluid and blood of diabetic patients could change the environment of the microflora, inducing qualitative changes in bacteria that could contribute to the severity of periodontal disease observed in those with poorly controlled diabetes.

Patients with type 1 diabetes mellitus and periodontitis have been reported to have a subgingival flora composed mainly of *Capnocytophaga*, anaerobic vibrios, and *Actinomyces* species. *Porphyromonas gingivalis, Prevotella intermedia,* and *Aggregatibacter actinomycetemcomitans*, which are common in periodontal lesions of individuals without diabetes, are present in low numbers in those with the disease.[108,159] Other studies, however, found scarce *Capnocytophaga* and abundant *A. actinomycetemcomitans* and black-pigmented *Bacteroides*, as well as *P. intermedia, P. melaninogenica,* and *Campylobacter rectus*.[158,210] Black-pigmented species, especially *P. gingivalis, P. intermedia,* and *C. rectus,* are prominent in severe periodontal lesions of Pima Indians with type 2 diabetes.[87,261] Although these results suggest an altered flora in the periodontal pockets of patients with diabetes, the exact role of these microorganisms has not been determined.

Polymorphonuclear Leukocyte Function. The increased susceptibility of diabetic patients to infection has been hypothesized as being caused by polymorphonuclear leukocyte (PMN) deficiencies resulting in impaired chemotaxis, defective phagocytosis, or impaired adherence.[162,230] In patients with poorly controlled diabetes, the function of PMNs and monocytes/macrophages is impaired.[116] As a result, the primary defense (PMNs) against periodontal pathogens is diminished, and bacterial proliferation is more likely. No alteration of immunoglobulin A (IgA), G (IgG), or M (IgM) has been found in diabetic patients.[201]

Altered Collagen Metabolism. Chronic hyperglycemia impairs collagen structure and function, which may directly impact the integrity of the periodontium. Decreased collagen synthesis,

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Figure 27-3 Abscess in 28-year-old patient with poorly controlled type 1 diabetes. **A,** Periodontal abscess in patient with poorly controlled diabetes. He presented with pain and abscess a few weeks after scaling and root planing of the area. **B,** Radiograph of mandibular right premolar area demonstrating severe localized destruction of bone in the area of periodontal abscess. **C,** Radiograph of mandibular right premolar area taken 2 months before presentation of abscess. Note presence of calculus and level of interproximal bone before abscess.

osteoporosis, as well as a reduction in alveolar bone height has been demonstrated in diabetic animals.[91,212] Chronic hyperglycemia adversely affects the synthesis, maturation, and maintenance of collagen and extracellular matrix. In the hyperglycemic state, numerous proteins and matrix molecules undergo a nonenzymatic glycosylation, resulting in *accumulated glycation end-products* (AGEs). The formation of AGEs occurs at normal glucose levels as well, but in hyperglycemic environments, AGE formation is excessive. Many types of molecules are affected, including proteins, lipids, and carbohydrates. Collagen is cross-linked by AGE formation, making it less soluble and less likely to be normally repaired or replaced. Cellular migration through cross-linked collagen is impeded, and perhaps more importantly, tissue integrity is impaired as a result of damaged collagen remaining in the tissues for longer periods (i.e., collagen is not renewed at a normal rate).[106] As a result, collagen in the tissues of patients with poorly controlled diabetes is older and more susceptible to pathogenic breakdown (i.e., less resistant to destruction by periodontal infections).

AGEs play a central role in the classic complications of diabetes[33] and may play a significant role in the progression of periodontal disease as well. Poor glycemic control, with the associated increase in AGEs, renders the periodontal tissues more susceptible to destruction.[211] The cumulative effects of altered cellular response to local factors, impaired tissue integrity, and altered collagen metabolism undoubtedly play a significant role in the susceptibility of diabetic patients to infections and destructive periodontal disease.

For more information on female sex hormones, to view Figures 27-4 to 27-7, and hyperparathyroidism, please visit the companion Expert CONSULT website at www.expertconsult.com.

HEMATOLOGIC DISORDERS AND IMMUNE DEFICIENCIES

All blood cells play an essential role in the maintenance of a healthy periodontium. White blood cells (WBCs) are involved in inflammatory reactions and are responsible for cellular defense against microorganisms as well as for proinflammatory cytokine release. Red blood cells (RBCs) are responsible for gas exchange and nutrient supply to the periodontal tissues and platelets and are necessary for normal hemostasis, as well as recruitment of cells during inflammation and wound healing. Consequently, disorders of any blood cells or blood-forming organs can have a profound effect on the periodontium.

Certain oral changes, such as hemorrhage, may suggest the existence of a blood dyscrasia. However, a specific diagnosis requires a complete physical examination and a thorough hematologic study. Comparable oral changes occur in more than one form of blood dyscrasia, and secondary inflammatory changes produce a wide range of variation in the oral signs.

Gingival and periodontal disturbances associated with blood dyscrasias must be viewed in terms of fundamental interrelationships between the oral tissues and the blood cells and blood-forming organs rather than in terms of a simple association of dramatic oral changes with hematologic disease. Hemorrhagic tendencies occur when the normal hemostatic mechanisms are disturbed. Abnormal bleeding from the gingiva or other areas of the oral mucosa that is difficult to control is an important clinical sign suggesting a hematologic disorder. Petechiae (Figure 27-8) and ecchymosis (Figure 27-9) observed most often in the soft palate area are signs of an underlying bleeding disorder. It is essential to diagnose the specific etiology to appropriately address any bleeding or immunological disorder.

Deficiencies in the host immune response may lead to severely destructive periodontal lesions. These deficiencies may be primary

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Figure 27-8 Petechiae evident on the soft palate of patient with underlying bleeding disorder (thrombocytopenia).

Figure 27-9 Ecchymosis evident on the lateral aspects of the soft palate and tonsillar pillars of patient with chemotherapy-induced thrombocytopenia.

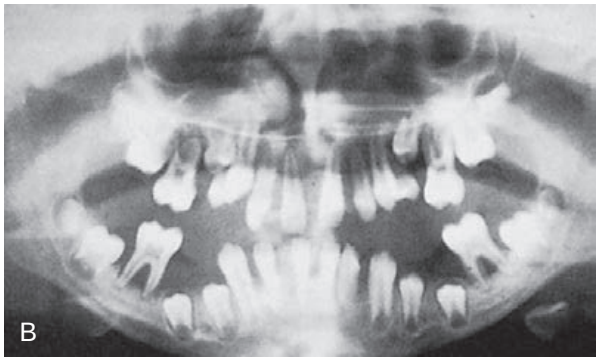

Figure 27-10 Aggressive periodontitis in 10-year-old male with cyclic neutropenia and agammaglobulinemia. **A,** Clinical presentation of periodontal condition. Note the severe swelling and inflammation of marginal and papillary gingiva. There is gross migration of teeth caused by loss of bone support. **B,** Panoramic radiograph demonstrating severe bone loss around all permanent teeth that have erupted into the oral cavity.

(inherited) or secondary (acquired), caused by immunosuppressive drug therapy, or pathologic destruction of the lymphoid system. Leukemia, Hodgkin's disease, lymphomas, and multiple myeloma may result in secondary immunodeficiency disorders. This section discusses common hematologic and certain non–HIV/AIDS-related immunodeficiency disorders (see Chapter 19 for a detailed discussion of the HIV-infected patient).

Leukocyte (Neutrophil) Disorders

Disorders that affect production or function of leukocytes may result in severe periodontal destruction. The PMN (neutrophil) in particular plays a critical role in bacterial infections because PMNs are the first line of defense (see Chapter 25). Quantitative deficiency of leukocytes (neutropenia, agranulocytosis) are typically associated with a more generalized periodontal destruction affecting all teeth.

Neutropenia. Neutropenia is a blood disorder that results in low levels of circulating neutrophils. it is a serious condition that may be caused by diseases, medications, chemicals, infections, idiopathic conditions, or hereditary disorders. It may be chronic or cyclic, severe, or benign. It affects as many as one in three patients receiving chemotherapy for cancer. An absolute neutrophil count (ANC) of 1000 to 1500 cells/µl is diagnostic for mild neutropenia. An ANC of 500 to 1000 cells per microliter is considered moderate neutropenia and an ANC less than 500 cells/µl is a severe neutropenia. Infections are sometimes difficult to manage and may be life threatening, particularly in severe neutropenia.

Agranulocytosis. Agranulocytosis is a more severe neutropenia involving not only the neutrophil but also basophils and eosinophils. It is defined as an ANC of less than 100 cells/µl. It is characterized by a reduction in the number of circulating granulocytes and results in severe infections, including ulcerative necrotizing lesions of the oral mucosa, skin, and gastrointestinal and genitourinary tracts. Less severe forms of the disease are called *neutropenia* or *granulocytopenia.*

Drug idiosyncrasy is the most common cause of agranulocytosis, but in some cases, its cause cannot be explained. Agranulocytosis has been reported after the administration of drugs such as aminopyrine, barbiturates and their derivatives, benzene ring derivatives, sulfonamides, gold salts, or arsenical agents.[134,149,166,194] It generally occurs as an acute disease. It may be chronic or periodic with recurring neutropenic cycles (e.g., cyclic neutropenia).[231]

The onset of disease is accompanied by fever, malaise, general weakness, and sore throat. Ulceration in the oral cavity, oropharynx, and throat is characteristic. The mucosa exhibits isolated necrotic patches that are black and gray and are sharply demarcated from the adjacent uninvolved areas.[126,153] The absence of a notable inflammatory reaction caused by lack of granulocytes is a striking feature. The gingival margin may or may not be involved. Gingival hemorrhage, necrosis, increased salivation, and fetid odor are accompanying clinical features. In cyclic neutropenia, the gingival changes recur with recurrent exacerbation of the disease.[49] The occurrence of generalized aggressive periodontitis has been described in patients with cyclic neutropenia[215] (Figure 27-10).

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Because infection is a common feature of agranulocytosis, differential diagnosis involves consideration of such conditions as necrotizing ulcerative gingivitis, noma, acute necrotizing inflammation of the tonsils, and diphtheria. Definitive diagnosis depends on the hematologic findings of pronounced leukopenia and almost complete absence of neutrophils.

Leukemia

Leukemia is an important disease to understand and appreciate because of the seriousness of the disease and the periodontal manifestations. The leukemias are malignant neoplasias of WBC precursors characterized by (1) diffuse replacement of the bone marrow with proliferating leukemic cells, (2) abnormal numbers and forms of immature WBCs in the circulating blood, and (3) widespread infiltrates in the liver, spleen, lymph nodes, and other body sites.[200]

According to the cell type involved, leukemias are classified as *lymphocytic* or *myelogenous*. A subgroup of the myelogenous leukemias is *monocytic* leukemia. The term *lymphocytic* indicates that the malignant change occurs in cells that normally form lymphocytes. The term *myelogenous* indicates that the malignant change occurs in cells that normally form RBCs, some types of WBCs and platelets. According to their evolution, leukemias can be *acute,* which is rapidly fatal, *subacute,* or *chronic.* In acute leukemia, the primitive "blast" cells released into the peripheral circulation are immature and nonfunctional, whereas in chronic leukemia the abnormal cells tend to be more mature with normal morphologic characteristics and function when released into the circulation.

All leukemias tend to displace normal components of the bone marrow elements with leukemic cells, resulting in reduced production of normal RBCs, WBCs, and platelets, which leads to anemia, *leukopenia* (reduction in number of *nonmalignant* WBCs) and thrombocytopenia. Anemia results in poor tissue oxygenation, making tissues more friable and susceptible to breakdown. A reduction of normal WBCs in the circulation leads to a poor cellular defense and an increased susceptibility to infections. *Thrombocytopenia* leads to bleeding tendency, which can occur in any tissue but in particular affects the oral cavity, especially the gingival sulcus (Figure 27-11). Some patients may have normal blood counts while leukemic cells reside primarily in the bone marrow. This type of disease is called *aleukemic leukemia*.[97]

Figure 27-11 Spontaneous bleeding from the gingival sulcus in patient with thrombocytopenia. Normal coagulation is evident by the appearance of the large clot that forms in the mouth. However, platelets are inadequate to establish hemostasis at the site of hemorrhage.

The Periodontium in Leukemic Patients. Oral and periodontal manifestations of leukemia may include leukemic infiltration, bleeding, oral ulcerations, and infections. The expression of these signs is more common in acute and subacute forms of leukemia than in chronic forms.

Leukemic Infiltration. Leukemic cells can infiltrate the gingiva and less frequently the alveolar bone. Gingival infiltration often results in *leukemic gingival enlargement* (see Chapter 9).

A study of 1076 adult patients with leukemia showed that 3.6% of the patients with teeth had leukemic gingival proliferative lesions, with the highest incidence in patients with acute monocytic leukemia (66.7%), followed by acute myelocytic-monocytic leukemia (18.7%), and acute myelocytic leukemia (3.7%).[63] It should be noted, however, that monocytic leukemia is an extremely rare form of the disease. Leukemic gingival enlargement is not found in edentulous patients or in patients with chronic leukemia, suggesting that it is the accumulation of immature leukemic blast cells in the gingiva adjacent to tooth surfaces with bacterial plaque. Leukemic gingival enlargement consists of a basic infiltration of the gingival corium by leukemic cells that increases the gingival thickness and creates gingival pockets in which bacterial plaque accumulates, initiating a secondary inflammatory lesion that contributes to the enlargement of the gingiva. It may be localized to the interdental papilla area (Figure 27-12) or expand to include the marginal gingiva and partially cover the crowns of the teeth (Figure 27-13, *C* and *D*). Clinically, the gingiva appears bluish red and cyanotic, with a rounding and tenseness of the gingival margin. The abnormal accumulation of leukemic cells in the dermal and subcutaneous connective tissue is called leukemia cutis and forms elevated and flat macules and papules[63,200] (Figure 27-13, *A* and *B*).

Microscopically, the gingiva exhibits a dense, diffuse infiltration of predominantly immature leukocytes in the attached and marginal gingiva. Occasionally, mitotic figures indicative of ectopic hematopoiesis may be seen. The normal connective tissue components of the gingiva are displaced by the leukemic cells (Figure 27-14). The nature of the cells depends on the type of leukemia. The cellular accumulation is denser in the entire reticular connective tissue layer. In almost all cases the papillary layer contains comparatively few leukocytes. The blood vessels are distended and contain predominantly leukemic cells, and the RBCs are reduced in number. The epithelium presents a variety of changes and may be thinned or hyperplastic. Common findings include degeneration associated with intercellular and intracellular edema and leukocytic infiltration with diminished surface keratinization.

The microscopic picture of the marginal gingiva differs from that of other gingival locations in that it usually exhibits a notable

Figure 27-12 Leukemic infiltration causing localized gingival swelling of the interdental papillae between the maxillary lateral and central incisor. Note the tense induration of the area.

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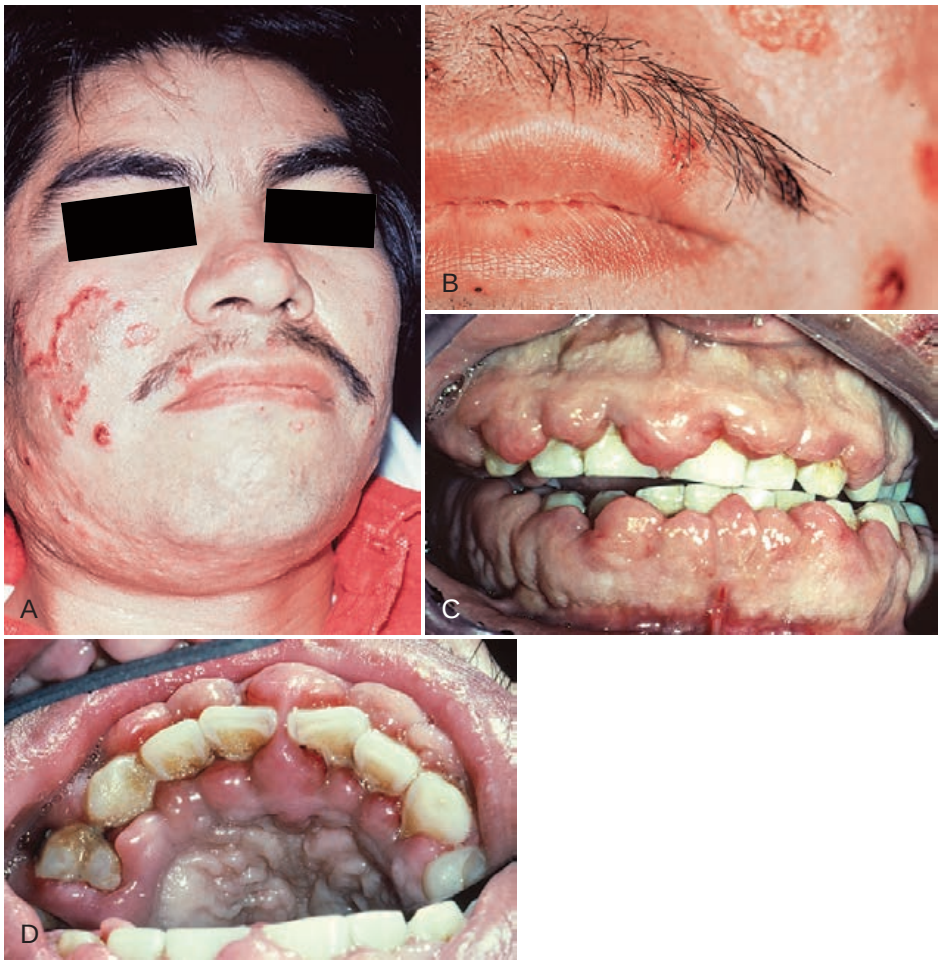

Figure 27-13 Adult male with acute myelocytic leukemia. **A,** View of patient's face. Note the elevated, flat macules and papules (leukemia cutis) on the right cheek. **B,** Close-up view of skin lesions. **C,** Intraoral view showing pronounced gingival enlargements of the entire gingival margin and interdental papilla areas of both arches. **D,** Occlusal view of maxillary anterior teeth. Note the marked enlargement in both the facial and the palatal aspects. (Courtesy Dr. Spencer Woolfe, Dublin, Ireland.)

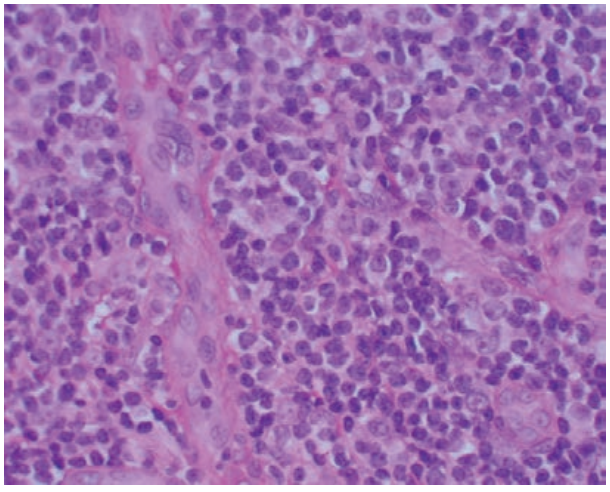

Figure 27-14 Human histologic appearance of leukemic infiltrate with dense, diffuse infiltration of predominantly immature leukocytes. The normal connective tissue components of the gingiva are displaced by the leukemic cells. The cellular accumulation is denser in the entire reticular connective tissue layer. (Courtesy of Dr. Russell Christensen, University of California, Los Angeles.)

inflammatory component, in addition to the leukemic cells. Scattered foci of plasma cells and lymphocytes with edema and degeneration are common findings. The inner aspect of the marginal gingiva is usually ulcerated, and marginal necrosis with pseudomembrane formation may also be seen.

The periodontal ligament and alveolar bone may also be involved in acute and subacute leukemia. The periodontal ligament may be infiltrated with mature and immature leukocytes. The marrow of the alveolar bone exhibits a variety of changes, such as localized areas of necrosis, thrombosis of the blood vessels, infiltration with mature and immature leukocytes, occasional RBCs, and replacement of the fatty marrow by fibrous tissue.

In leukemic mice the presence of infiltrate in marrow spaces and the periodontal ligament results in osteoporosis of the alveolar bone with destruction of the supporting bone and disappearance of the periodontal fibers[31,39] (Figure 27-15).

Bleeding. Gingival hemorrhage is a common finding in leukemic patients (see Figure 27-11), even in the absence of clinically detectable gingivitis. Bleeding gingiva can be an early sign of leukemia. It is caused by the thrombocytopenia resulting from replacement of the bone marrow cells by leukemic cells and from the inhibition of normal stem cell function by leukemic cells or their

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products.[200] This bleeding tendency can also manifest in the skin and throughout the oral mucosa, where petechiae are often found, with or without leukemic infiltrates. A more diffuse submucosal bleeding manifests as ecchymosis (see Figure 27-9). Oral bleeding has been reported as a presenting sign in 17.7% of patients with acute leukemia and in 4.4% of patients with chronic leukemia.[148] Bleeding may also be a side effect of the chemotherapeutic agents used to treat leukemia.

Oral Ulceration and Infection. In leukemia, the response to bacterial plaque or other local irritation is altered. The cellular component of the inflammatory exudate differs both quantitatively and qualitatively from that in nonleukemic individuals in that there is a pronounced infiltration of immature leukemic cells in addition to the usual inflammatory cells. As a result, the normal inflammatory response may be diminished.

Granulocytopenia (diminished WBC count) results from the displacement of normal bone marrow cells by leukemic cells, which increases the host susceptibility to opportunistic microorganisms and leads to ulcerations and infections. Discrete, punched-out ulcers penetrating deeply into the submucosa and covered by a firmly attached white slough can be found on the oral mucosa.[15] These lesions occur in sites of trauma such as the buccal mucosa in relation to the line of occlusion or on the palate. Patients with past history of herpesvirus infection may develop recurrent herpetic oral ulcers, often in multiple sites, and large atypical forms, especially after chemotherapy is instituted[102] (Figure 27-16).

A gingival (bacterial) infection in leukemic patients can be the result of an exogenous bacterial infection or an existing bacterial infection (e.g., gingival or periodontal disease). Acute gingivitis and lesions resembling necrotizing ulcerative gingivitis are more frequent and severe in terminal cases of acute leukemia[21] (Figures 27-17 and 27-18). The inflamed gingiva in patients with leukemia differs clinically from that in nonleukemic individuals. Gingiva is a peculiar bluish red, is spongelike and friable, and bleeds persistently on the slightest provocation or even spontaneously in leukemic patients. This greatly altered and degenerated tissue is extremely susceptible to bacterial infection, which can be so severe as to cause acute gingival necrosis with pseudomembrane formation (Figure 27-19) or bone exposure (Figure 27-20). These are secondary oral changes superimposed on the oral tissues altered by the blood dyscrasia. They produce associated disturbances that may be a source of considerable difficulty to the patient, such as systemic toxic effects, loss of appetite, nausea, blood loss from persistent gingival bleeding, and constant gnawing pain. Eliminating or

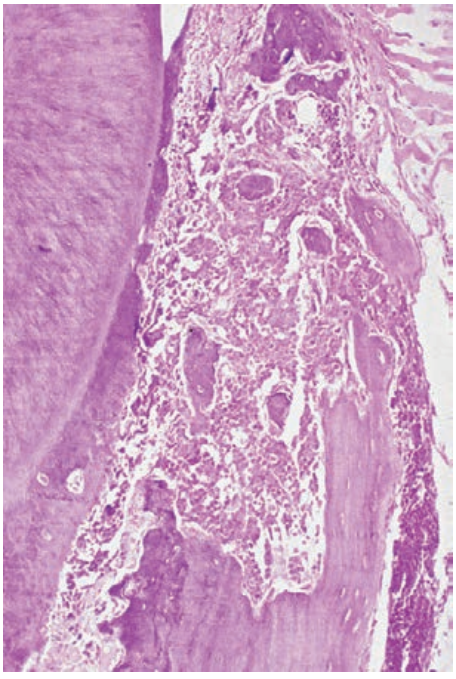

Figure 27-15 Leukemic infiltrate in alveolar bone in leukemic mouse. Note the leukemic infiltrate causing destruction of bone and loss of periodontal ligament.

Figure 27-16 Large ulcerations on the palate of patient with granulocytopenia secondary to leukemia. These atypical ulcerations are caused by herpesvirus opportunistic infection. Notice the smaller, discrete, round ulcerations that have coalesced into the larger lesion.

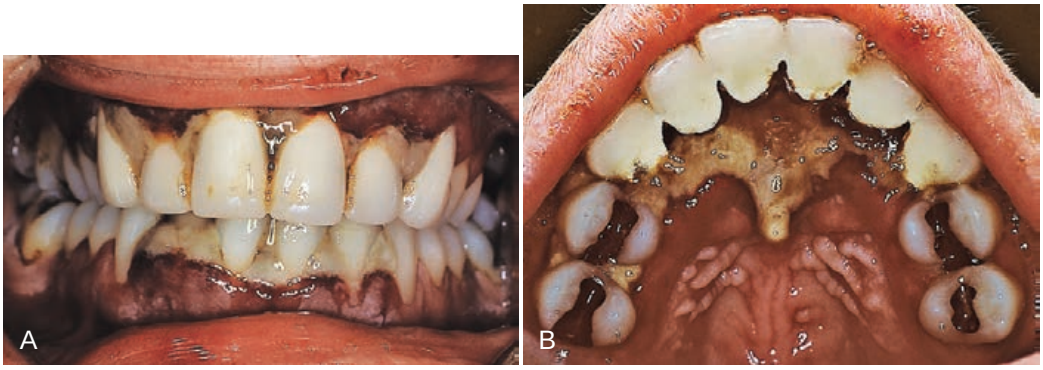

Figure 27-17 Adult female with acute myelocytic leukemia. **A,** Anterior view of patient with acute myelocytic leukemia. Interdental papillae are necrotic with a highly inflamed and swollen gingival tissue at the base of the lesions. **B,** Palatal view demonstrating extensive necrosis of interdental and palatal tissues behind the maxillary incisors.

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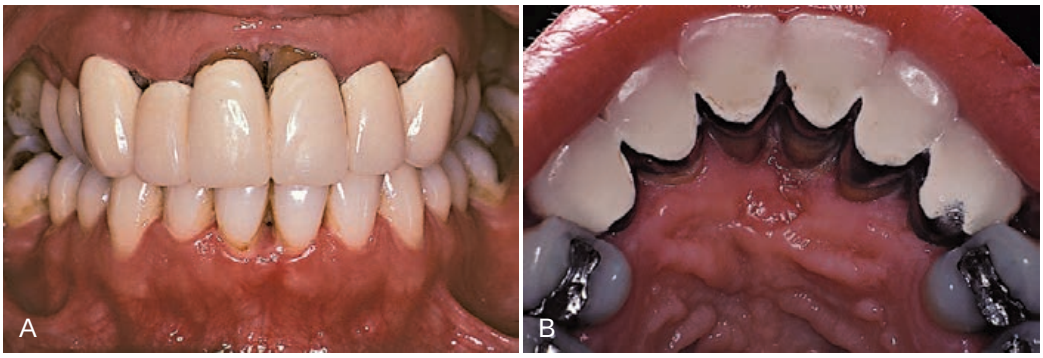

Figure 27-18 Same patient as in Figure 27-17 after chemotherapy that resulted in remission of leukemia. **A,** Anterior view reveals dramatic improvement in gingival health following remission of leukemia. Note the loss of interdental papillae as well as gingival recession in the anterior areas. **B,** Palatal view shows extensive loss of gingival tissue around maxillary incisors.

Figure 27-19 Opportunistic bacterial infection of gingiva in patient with hospitalized with leukemia. Gingival tissue is highly inflamed, bleeding, and necrotic with pseudomembrane formation.

Figure 27-20 Opportunistic bacterial infection in immunosuppressed patient caused complete destruction of gingiva, exposing underlying alveolar bone.

reducing local factors (e.g., bacterial plaque) can minimize severe oral changes in leukemia. In some patients with severe acute leukemia, symptoms may only be relieved by treatment that leads to remission of the disease.

In chronic leukemia, oral changes suggesting a hematologic disturbance are rare. The microscopic changes in chronic leukemia may consist of replacing the normal fatty marrow of the jaws with islands of mature lymphocytes or lymphocytic infiltration of the marginal gingiva without dramatic clinical manifestations.

The existence of leukemia is sometimes revealed by a gingival biopsy performed to clarify the nature of a troublesome gingival condition. In such cases the gingival findings must be corroborated by medical examination and hematologic study. In patients with diagnosed leukemia, gingival biopsy may indicate the extent to which leukemic infiltration is responsible for the altered clinical appearance of the gingiva. *Although such findings are of interest, their benefit to the patient is insufficient to warrant routine gingival biopsy studies in patients with leukemia.* Furthermore, it is important to note that the absence of leukemic involvement in a gingival biopsy specimen does not rule out the possibility of leukemia. A gingival biopsy in a patient with chronic leukemia may reveal typical gingival inflammation without any suggestion of a hematologic disturbance.

For more information on anemia and thrombocytopenia, and to view Figures 27-21 to 27-23, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on antibody deficiency disorders, please visit the companion Expert CONSULT website at www.expertconsult.com.

GENETIC DISORDERS

Many systemic conditions associated with or predisposing to periodontal destruction include genetic disorders that result in an inadequate number or function of circulating neutrophils. This underscores the importance of the neutrophil in the protection of the periodontium against infection. Severe periodontitis has been observed in individuals with primary neutrophil disorders such as neutropenia, agranulocytosis, Chédiak-Higashi syndrome, and lazy leukocyte syndrome. In addition, severe periodontitis has also been observed in individuals who exhibit secondary neutrophil impairment, as seen in Down syndrome, Papillon-Lefèvre syndrome, and inflammatory bowel disease. This section discusses conditions with primary and secondary leukocyte disorders.

For more information on genetic disorders, and to view Figures 27-24 and 27-25, please visit the companion Expert CONSULT website at www.expertconsult.com.

Down Syndrome

Down syndrome (mongolism, trisomy 21) is a congenital disease caused by a chromosomal abnormality and characterized by mental deficiency and growth retardation. The prevalence of periodontal

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Figure 27-26 Severe periodontal destruction in 14-year-old patient with Down syndrome. Note the extensive loss of periodontal support around the mandibular incisors as evidenced by severe gingival recession. Moderate-heavy bacterial plaque is associated with moderate gingival inflammation in the area.

disease in Down syndrome is high (occurring in almost 100% of patients younger than 30 years).[52,59] Although plaque, calculus, and local irritants (e.g., diastemata, crowding of teeth, high frenum attachments, and malocclusion) are present and oral hygiene is poor, the severity of periodontal destruction exceeds that explainable by local factors alone.[48,50,52,198,226]

Periodontal disease in Down syndrome is characterized by formation of deep periodontal pockets associated with substantial plaque accumulation and moderate gingivitis (Figure 27-26). These findings are usually generalized, although they tend to be more severe in the lower anterior region. Moderate recession is sometimes seen in this region as well. The disease progresses rapidly. The high prevalence and increased severity of periodontal destruction associated with Down syndrome is most likely explained by poor PMN chemotaxis, phagocytosis, and intracellular killing.*

STRESS AND PSYCHOSOMATIC DISORDERS

Psychologic conditions, particularly psychosocial stress, have been implicated as risk indicators for periodontal disease.[85] The most notable example is the documented relationship between stress (e.g., experienced by soldiers at war or by students during examinations) and acute necrotizing ulcerative gingivitis (NUG) (see Chapters 10 and 17). The presence of NUG in soldiers stressed by wartime conditions in the trenches led to one of the early diagnostic terms used to describe this condition, namely "trench mouth." Despite this well-known association between stress and NUG, *confirming* the connection between psychologic conditions and other forms of periodontal disease (e.g., chronic periodontitis) has been elusive. These relationships are difficult to elucidate because, as with many common diseases, the etiology and pathogenesis of periodontal disease is multifactorial and the role of individual risk factors is difficult to define.

Some studies have failed to recognize a relationship between psychological conditions and periodontal disease despite specific efforts to identify them. In a study of 80 patients (40 with aggressive periodontitis and 40 with chronic periodontitis), Monteiro da Silva et al failed to find a relationship between psychologic factors and periodontal disease.[172] They were able to identify depression and smoking as *marginally* significant in the aggressive periodontitis group. Their inability to find a relationship may be attributed to a lack of significant differences in psychologic characteristics between the two groups in the study. In an earlier study, the same group identified depression and loneliness as significant factors associated with aggressive periodontal disease in 50 patients compared with 50 periodontally healthy individuals and 50 individuals with chronic periodontitis.[171] Another challenge in defining a relationship between psychosocial status and periodontitis is the myriad of confounding factors and the difficulty in controlling for them.[65]

*References 12, 13, 53, 57, 103, 119, and 127.

For more information on psychosocial stress, depression, and coping, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on stress-induced immunosuppression, please visit the companion Expert CONSULT website at www.expertconsult.com.

Influence of Stress on Periodontal Therapy Outcomes

Psychologic conditions, such as stress and depression, may also influence the outcome of periodontal therapy. In a large-scale retrospective study of 1299 dental records from a health maintenance organization (HMO) database, 85 individuals with depression had posttherapy outcomes that were less favorable (below median) compared to those without depression.[70] More than half (697) were complete enough for a comprehensive evaluation, including both periodontal diagnosis and psychologic profiles. The authors concluded that depression might have a negative effect on periodontal treatment outcomes.

A recent study investigating the relationship between psychologic stress and wound repair in patients after routine surgery (inguinal hernia open incision repair) revealed that stress impairs the inflammatory response and matrix degradation.[30] Forty-seven adults were given a standardized questionnaire before surgery to assess their psychologic stress before surgery. Wound fluids were collected over the first 20 hours after surgery to measure inflammatory markers (interleukin-1 [IL-1], IL-6, and matrix metalloproteinase-9 [MMP-9]). Greater psychologic stress was significantly associated with lower levels of IL-1 and MMP-9, as well as significantly more painful, poorer, and slower recovery.

Another study compared the psychiatric characteristics of individuals with different outcomes to periodontal therapy.[10] Two groups were compared to evaluate the psychologic characteristics of 11 individuals who were responsive to periodontal treatment compared with 11 individuals who were not responsive to periodontal treatment. The responsive group had a more rigid personality, whereas the nonresponsive group had a more passive, dependent personality. Furthermore, the nonresponsive group reported more stressful life events in their past.

These studies suggest that both stressful life events and the individual's personality and coping skills are factors to consider in assessing the risk of periodontal disease destruction and the potential for successful periodontal therapy. If patients are identified with emotional or defensive coping skills, care should be taken to ensure that they receive information in a manner that does not elicit a "defensive" reaction.

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Figure 27-27 Severe gingival recession localized to the labial surface of all mandibular incisors. This finding was discovered under general anesthesia in an uncooperative, institutionalized adult with mental disorders. The patient was known to pace around the home with all four fingers inside his lower lip.

Psychiatric Influence of Self-Inflicted Injury

Psychosomatic disorders may result in harmful effects to the health of tissues in the oral cavity through the development of habits that are injurious to the periodontium. Neurotic habits, such as grinding or clenching the teeth, nibbling on foreign objects (e.g., pencils, pipes), nail biting, and excessive use of tobacco, are all potentially injurious to the teeth and the periodontium. Self-inflicted gingival injuries, such as gingival recession, have been described in both children and adults (Figure 27-27). However, these types of self-inflicted, factitious injuries do not appear to be common in psychiatric patients.[209]

NUTRITIONAL INFLUENCES

Some clinicians enthusiastically adhere to the theory in periodontal disease that assigns a key role to nutritional deficiencies and imbalances. Previous research did not support this view, but numerous problems in experimental design and data interpretation could be responsible for making these research findings inadequate.[5,208] The majority of opinions and research findings regarding the effects of nutrition on oral and periodontal tissues point to the following:

1. *There are no nutritional deficiencies that by themselves can cause gingivitis or periodontitis.* However, nutritional deficiencies can affect the condition of the periodontium and thereby may accentuate the deleterious effects of plaque-induced inflammation in susceptible individuals. Theoretically, one might presume that an individual with a nutritional deficiency is less able to defend against a bacterial challenge compared with a nutritionally competent individual.
2. *There are nutritional deficiencies that produce changes in the oral cavity.* These changes include alterations of tissues of the lips, oral mucosa, gingiva, and bone. These alterations are considered to be periodontal and oral manifestations of nutritional disease.

The role of nutrition in periodontal disease may be related to the effect of nutrition on inflammation. A recent review of the literature evaluating the effect of nutritional factors on inflammation demonstrated that subtle shifts in nutritional status are associated with the prevalence of periodontitis.[43] More specifically, they reported that the results of contemporary animal and human studies have demonstrated the role of specific micronutrients in the modulation of the host's inflammatory response by reducing inflammatory biomarkers, which may in turn be responsible for periodontal destruction. The evidence for the effect of nutrition on inflammation is significant. Data suggest that diets containing foods rich in antioxidants are beneficial, whereas foods containing high levels of refined carbohydrates are detrimental to the inflammatory process.[43]

For a review of existing knowledge on the effects of vitamin and protein deficiencies on changes in the periodontium, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on nutritional influences, please visit the companion Expert CONSULT website at www.expertconsult.com.

MEDICATIONS

Some medications prescribed to cure, manage, or prevent diseases may have an adverse effect on periodontal tissues, wound healing, or the host immune response. Bisphosphonates are a class of widely prescribed medications that have recently been associated with osteonecrosis of the jaw (ONJ). Corticosteroids have long been prescribed to suppress the immune system in the control and management of autoimmune disease, in cancer treatment, and as an antirejection medication in transplant patients. This section discusses the effect of bisphosphonates and corticosteroids on the periodontium.

Bisphosphonates

Bisphosphonate medications are primarily used to treat cancer (intravenous [IV] administration) and osteoporosis (oral administration). They act by inhibiting osteoclastic activity, which leads to less bone resorption, less bone remodeling, and less bone turnover.[206] The use of bisphosphonates in cancer treatment is aimed at preventing the often lethal imbalance of osteoclastic activity. In the treatment of osteoporosis, the goal is simply to harness osteoclastic activity to minimize or prevent bone loss and in many cases, to increase bone mass by creating an advantage for osteoblastic activity. The major difference in the use of bisphosphonates for cancer versus osteoporosis is the potency and route of administration of the specific bisphosphonate medication used. Potency is influenced by the chemical properties, as well as the binding and release pharmacokinetic properties, of these agents with bone. Specifically, the strength of binding and the ease of release of bisphosphonates with hydroxyapatite make them more or less potent.

Bisphosphonates were first synthesized in the 1950s as a substitute for pyrophosphate, a compound used in detergent. The ability of bisphosphonates to increase bone mass was discovered after animal studies in 1966, but the potential advantage of using bisphosphonates in humans with low bone mass was not appreciated until 1984.[244] The Food and Drug Administration (FDA) approved the use of alendronate for osteoporosis in 1995.

The chemical structure of bisphosphonate consists of two phosphate groups covalently bonded to a central carbon (Figure 27-28). In addition to the two phosphate groups, the central carbon also has two side chains, R1 and R2. Both the short R1 side chain and the long R2 side chain influence the chemical properties and pharmacokinetics. The long R2 side chain also influences the mode of action and determines the strength or potency of the medication.

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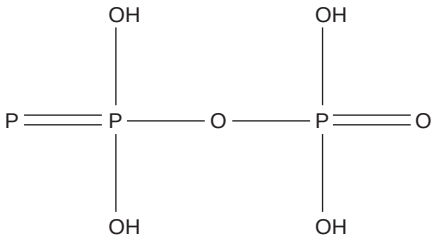
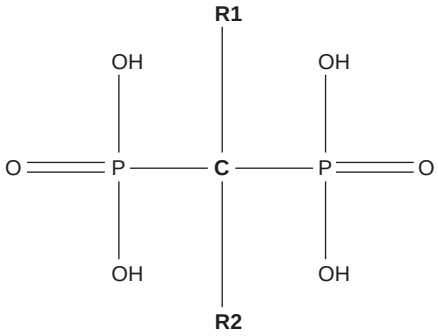
Bisphosphonates inhibit osteoclasts by two mechanisms that depend on whether the R2 side chain contains nitrogen. Nonaminobisphosphonates are metabolized by osteoclasts to form an adenosine triphosphate (ATP) analog that interferes with energy production and causes osteoclast apoptosis. Aminobisphosphonates (risedronate, zoledronate, ibandronate, and alendronate) are more potent and have multiple effects on osteoclasts, including (1) inactivation of ATP, (2) osteoclast cytoskeletal disruption, (3) impairment of osteoclast recruitment, and (4) induction of osteoblasts to produce osteoclast-inhibiting factor.[181] Table 27-1 lists some of the common bisphosphonate medications used for osteoporosis and cancer treatment that are currently available in the US.

Bisphosphonates have a high affinity for hydroxyapatite and are rapidly absorbed in bone, especially in areas of high activity, which may explain why bisphosphonate-induced osteonecrosis is only found in the jaws.[244] The bisphosphonate molecule gets incorporated into bone without being metabolized or modified. During osteoclastic resorption of bone, the trapped bisphosphonate is released and able to affect osteoclasts again. As a result, the half-life of bisphosphonates in the bone is estimated to be 10 years or more.

Pyrophosphate



Generic bisphosphonate



R1 – Enhances binding to hydroxapatite
-C- Enhances chemical stability
R2 – Determines antiresorptive potency

Figure 27-28 Chemical structure of bisphosphonate molecule. Two phosphate groups are covalently bonded with a central carbon. The carbon also has two side chains, R1 and R2.

Osteonecrosis of the jaw associated with bisphosphonates was first reported in 2003 by Marx in a report of 36 cases of patients with avascular necrosis of the jaws who were treated with IV bisphosphonate for malignant tumors.[155] Several case series reporting an association between bisphosphonates and osteonecrosis of the jaws were published in the months and years after this initial report.[157,204] Various terms have been used to describe this type of osteonecrosis of the jaw, including avascular necrosis, bisphosphonate-related or -associated ONJ (BRONJ), and bisphosphonate-induced ONJ (BIONJ).[244] Today, with the widespread recognition of BIONJ, it is important to remember that necrotic bone exposure of the jaw (ONJ) is a condition with multiple possible etiopathogenic factors, including systemic medications, radiation, infection, trauma, direct chemical toxicity, or other idiopathic mechanisms, and clinicians should carefully consider all factors before concluding a diagnosis of BIONJ.[8]

The condition of BIONJ has been defined as exposure/necrosis of portions of the jaw bone in patients exposed to bisphosphonates that has persisted more than 8 weeks with no past history of radiation therapy to the jaws.[203] The stage of osteonecrosis is used to categorize patients and make treatment decisions.[1,156,157] Stage 0 is defined as patients at risk who have been treated with IV or oral bisphosphonates but have no apparent exposed/necrotic bone. Stage 1 is exposed/necrotic bone in patients who are asymptomatic with no infection. Stage 2 is exposed/necrotic bone in patients with pain and clinical evidence of infection. Stage 3 is exposed/necrotic bone in patients with pain, infection, and one or more of the following: pathologic fracture, extraoral fistula, or osteolysis extending to the inferior border.

Clinically, BIONJ presents as exposed alveolar bone occurring spontaneously or after a dental procedure (Figures 27-29 and 27-30). The sites may be painful with surrounding soft tissue induration and inflammation. Infection with drainage may be present. Radiographically, lesions appear radiolucent with sclerosis of lamina dura, loss of lamina dura, or widening of periodontal ligament in areas where teeth are present. Histologically, bone appears necrotic with empty lacunae demonstrating a lack of living osteocytes. In advanced cases, pathologic fracture may be present through the area of exposed/necrotic bone.

The high potency of nitrogen-containing bisphosphonates, especially those administered via IV for cancer treatment (e.g.,

TABLE 27-1 Current Nonaminobisphosphonate and Aminobisphosphonate Medications and Common Therapeutic Uses

Generic Name	Commercial Name	Route of Administration	Therapeutic Use	N-Containing R2 Side Chain	Antiresorptive Potency
etidronate	Didronel	PO	Paget's disease	No	1
tiludronate	Skelid	PO	Paget's disease	No	10
risedronate	Actonel	PO	Osteoporosis, Paget's disease	Yes	5000
ibandronate	Boniva	PO	Osteoporosis	Yes	10,000
alendronate	Fosamax	PO	Osteoporosis, Paget's disease	Yes	1000
pamidronate	Aredia	IV	Paget's disease, Cancer	Yes	100
zoledronate	Zometa	IV	Cancer	Yes	10,000+

PO, By mouth; *IV,* intravenous.

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Figure 27-29 Clinical photograph of exposed bone on palatal surface of maxilla adjacent to molar root in 60-year-old female with bisphosphonate-induced osteonecrosis of bone (maxilla). The bone exposures were noted about 1 year during treatment with bisphosphonate (Aredia and Zometa). *(Courtesy of Drs. Eric S. Sung and Evelyn M. Chung, University of California, Los Angeles.)*

Figure 27-30 Clinical photograph of exposed bone on lingual surface of posterior mandible in 70-year-old male with bisphosphonate-induced osteonecrosis of bone (mandible). The bone exposure was noted after 3 years of bisphosphonate (Aredia and Zometa) for multiple myeloma. *(Courtesy of Drs. Eric S. Sung and Evelyn M. Chung, University of California, Los Angeles.)*

zoledronate), may explain the high incidence of BIONJ in these patients as compared to osteoporotic patients taking oral bisphosphonates. The incidence in patients treated for cancer has been reported to range from 2.5% to 5.4%.[248] Estimating the incidence in patients taking oral bisphosphonates for osteoporosis is more difficult due to the large number of patients taking this medication (prescriptions) and lack of good reporting or documentation for these patients. Estimates from different sources project the incidence to range from 0.007% to 0.04%.[244] Clearly, the incidence is low.

In addition to bisphosphonate therapy, other factors make individuals susceptible to BIONJ. Potential risk factors that may contribute to BIONJ include systemic corticosteroid therapy, smoking, alcohol, poor oral hygiene, chemotherapy, radiotherapy, diabetes, and hematologic disease.[28] The precipitating factors or events leading to BIONJ are reported to include extractions, root canal treatment, periodontal infections, periodontal surgery, and dental implant surgery, yet some cases appear to be idiopathic with spontaneous exposures.[157] In a retrospective evaluation of patients treated with IV bisphosphonates for metastatic bone cancer from 1996 to 2006, Estilo et al[73] found that type of cancer, duration of bisphosphonate therapy, sequential IV bisphosphonate treatment with pamidronate followed by zoledronate, comorbid osteoarthritis, rheumatoid arthritis, and benign hematologic conditions were significantly associated with an increased likelihood of ONJ. In their study, systemic administration of corticosteroids was not found to be associated with an increased risk BRONJ.[73]

Patients being treated for cancer with IV bisphosphonates are at greater risk than patients being treated for osteoporosis with oral bisphosphonates. Dental health care providers should evaluate patients carefully, consider the risks, communicate with medical health care providers, inform patients, and consider treatment options and risks carefully.

Not surprisingly, the bone-preserving action of bisphosphonates has been studied and advocated for use in the prevention of bone loss from periodontal disease.[232] Several animal studies have shown that bisphosphonates, applied topically or administered systemically, have the potential to prevent alveolar bone loss caused by periodontitis.* The use of bisphosphonates in bone regeneration has been proposed as well.[232] Although some studies have demonstrated bone preservation with low doses of bisphosphonate, higher doses and longer administration may have a neutral or detrimental effect on bone loss caused by periodontitis.[34,41] In a 2 to 3 year follow-up report of 4 female patients with periodontitis treated with etidronate (200 mg daily for periods of 2 weeks with 10-week "off drug" periods), the potential of this agent to prevent periodontal bone loss was reported.[229] In a 2-year randomized, placebo-controlled clinical trial of 335 patients treated with alendronate (70 mg once weekly), no significant difference in alveolar bone loss or alveolar bone density was found.[120] Interestingly, alendronate was found to significantly reduce bone loss relative to controls in a subset of this group (i.e., patients with low mandibular bone mineral density at baseline), suggesting that the effect may be more perceptible in cases with less bone mass or less bone density. In another clinical trial of 24 patients (12 experimental and 12 control), alendronate was shown to have a significant positive effect (bone preserving) on bone density in jaws.[68] Bone mineral density of the maxilla and mandible was measured for all patients using dual energy x-ray absorptiometry (DEXA) at baseline and following 6 months of treatment (10 mg daily for 6 months).

Corticosteroids

In humans, systemic administration of cortisone and ACTH appears to have no effect on the incidence or severity of gingival and periodontal disease. However, renal transplant patients receiving immunosuppressive therapy (prednisone or methylprednisone, azathioprine, or cyclophosphamide) have significantly less gingival inflammation than control subjects with similar amounts of plaque.[20,125,180,236135]

*References 41, 101, 165, 167, 178, 219, 251.

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Exogenous cortisone may have an adverse effect on bone quality and physiology. The systemic administration of cortisone in experimental animals results in osteoporosis of alveolar bone.[95] There was capillary dilation and engorgement with hemorrhage into the periodontal ligament and gingival connective tissue, as well as a degeneration and reduction in the number of collagen fibers in the periodontal ligament and increased destruction of the periodontal tissues associated with inflammation.[95]

Stress increases circulating endogenous cortisol levels through stimulation of the adrenal glands (hypothalamic-pituitary-adrenal axis). This increased exposure to endogenous cortisol may have adverse effects on the periodontium by diminishing the immune response to periodontal bacteria (see section on psychosocial stress).

OTHER SYSTEMIC CONDITIONS

Osteoporosis

Osteoporosis is a disease characterized by low bone mass and structural deterioration leading to an increased risk of bone fracture that affects approximately 10 million people in the US with a higher predilection for females (80%) as compared to males (20%). An additional 34 million individuals in the US are estimated to have osteopenia or low bone density. Loss of bone mass and the incidence of osteoporosis increases with age for both men and women, with women affected earlier than men. The rate of bone loss is greatest for women during the perimenopausal years when estrogen levels decrease.

A bone mineral density (BMD) test is used to measure an individual's bone mass. BMD is measured using a DEXA scan. The DEXA value, or T-score, is a comparison of the patient's BMD to that of a healthy 30-year-old adult with peak bone mass. The World Health Organization (WHO) defines osteoporosis and osteopenia by measures of "standard deviations" as compared to that of a normal healthy young adult. Thus a T-score between +1 and –1 is considered normal, whereas a T-score between –1 and –2.5 indicates low bone density or osteopenia and a T-score less than –2.5 (i.e., more than 2.5 standard deviations below normal) is diagnostic for osteoporosis (Table 27-2).

One of the most significant consequences of osteoporosis is the increased risk of bone fracture. All bones affected by osteoporosis are susceptible to fracture, but fractures of the pelvis and vertebrae carry the most serious risk of morbidity and mortality. Studies have reported that approximately 20% of patients with hip fractures die within a year and 50% experience significant disability (e.g., inability to walk unassisted).[137]

Although it is logical to surmise a relationship between osteoporosis and periodontitis, it has been a challenging hypothesis to prove. One of the difficulties is the fact that both osteoporosis and periodontitis are chronic, multifactorial diseases that result in bone loss, and bone loss in each condition is exacerbated by local and systemic factors.[179] Gender, genetic predisposition, inactivity, deficient diets (e.g., deficient in calcium or vitamin D), alcohol, smoking, hormones, and medications put individuals at risk for osteoporosis, with some of these factors putting them at risk for the progression of periodontitis as well. Variations in populations studied, measurements used to assess disease, and study design have also hindered the ability to clearly define a relationship between osteoporosis and periodontitis.

Attempts to use tooth loss as an indirect measure of periodontitis in individuals with osteoporosis have offered mixed conclusions. Several studies have reported greater tooth loss, more alveolar bone loss and edentulism in individuals with osteoporosis.[58,64,107,260] However, some other studies have suggested that tooth loss is not correlated to osteoporosis or BMD.[27,66,168] One large study (1365 Caucasian postmenopausal females) investigating systemic BMD (lumbar spine and proximal femur) and tooth loss, found no significant correlation.[66] Part of the challenge and the reason for conflicting results in these studies may be attributed to differences in measurement methods. Another problem in translating any tooth loss study conclusions to the question of whether osteoporosis contributes to periodontitis is that the cause of tooth loss in these studies is often unknown (i.e., tooth loss may or may not be related to periodontal disease).

Studies that attempt to correlate osteoporosis with periodontitis are fraught with similar, if not greater, challenges. Again, some studies have concluded or suggested that osteoporosis does contribute to the progression of periodontitis,[117,130,169,234] whereas others refute this conclusion.[69,147] One study by Elders et al failed to show a correlation between BMD (lumbar BMD) and clinical parameters of periodontitis in a dentate group of 46- to 55-year-old females.[69] They used intraoral periodontal examinations (probing depth, bleeding on probing, and missing teeth) and bitewing radiographs to assess clinical parameters of periodontitis and interproximal alveolar bone loss. This study included edentulous subjects (286) as well, but no significant difference in BMD was found between edentulous (60) and dentate (226) subjects, suggesting that neither periodontitis nor tooth loss was related to osteoporosis. The mean age of subjects in this study was relatively young, which may have contributed to the lack of correlation.[88]

Most studies citing a positive correlation between osteoporosis or BMD and periodontitis are cross-sectional studies of postmenopausal females. Klemetti et al evaluated 227 healthy postmenopausal women aged 48 to 56 years and found that women with higher skeletal BMD were more likely to retain teeth with periodontitis (deep periodontal pockets) than those with osteoporosis.[130] Tezal et al concluded that skeletal BMD (measured by DEXA at multiple sites) is related to alveolar bone loss and to a lesser extent to clinical attachment loss, implicating postmenopausal osteopenia as a risk indicator for periodontal disease in 70 postmenopausal white women aged 51 to 78 years.[234] Periodontal parameters included probing depth, supragingival plaque, calculus, bleeding on probing, clinical attachment loss, and interproximal alveolar bone loss. Analysis was adjusted for age, age at menopause, estrogen supplementation, cigarette smoking, body mass, and supragingival plaque. Inagaki et al evaluated the association between periodontal conditions, tooth loss, and metacarpal BMD (m-BMD) in a cross-sectional study of 356 Japanese women (171 premenopausal, mean age 37.9 years and 185 postmenopausal, mean age 63.3 years) and concluded that periodontal status and tooth loss after menopause could be a useful indicator of m-BMD

TABLE 27-2 Definitions of Osteoporosis and Osteopenia Indicated by T-Scores

T-Score	Bone Mineral Density (BMD) Category
≥ –1	Normal
–1 to –2.5	Low (osteopenia)
≤ –2.5	Osteoporosis

Ranges of T-scores are based on standard deviations outside of normal, which is based on the BMD of a healthy 30-year-old adult.
Adapted from World Health Organization definitions.

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loss in Japanese women.[117] These cross-sectional studies are suggestive but do not consider the periodontal condition or existence of periodontitis before the onset of osteoporosis or loss of systemic bone mineral density.

The effect of estrogen deficiency and osteopenia/osteoporosis on periodontitis is not known but may be an important factor to consider. Reinhardt et al[196] conducted a 2-year, prospective, longitudinal study of postmenopausal women (59 with moderate/advanced periodontitis and 16 with no periodontitis) to evaluate the effect of estrogen (serum estradiol) levels on periodontitis in these patients. Clinical measurements, including supragingival plaque, bleeding on probing, and relative clinical attachment, were taken at baseline and every 6 months for 2 years. They concluded that estrogen deficiency and osteopenia/osteoporosis together are risk factors for alveolar bone loss in postmenopausal women with a history of periodontitis.[196] Lerner[138] also proposed that estrogen deficiency might play a significant role in the progression of periodontitis in osteopenic/osteoporotic women citing that the cytokines believed to be involved in inflammation-induced remodeling are very similar to those suggested to play crucial roles in postmenopausal osteoporosis. In patients with periodontal disease and concomitant postmenopausal osteoporosis, the possibility exists that the lack of estrogen influences the activities of bone cells and immune cells in such a way that the progression of alveolar bone loss will be enhanced.[138] In fact, there are numerous studies that report less risk for tooth loss and improved oral health in postmenopausal women on hormone replacement therapy.* More prospective, longitudinal studies evaluating the effect of estrogen and osteopenia/osteoporosis on periodontitis are needed to improve our understanding.

For more information on other systemic conditions, and to view Figures 27-31 to 27-33, please visit the companion Expert CONSULT website at www.expertconsult.com.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

*References 7, 17, 24, 104, 105, and 228.

SCIENCE TRANSFER

Dentists need to appreciate the wide range of systemic conditions that have periodontal implications to modify the treatment of affected patients, and in some cases, the dentist may be the first doctor to diagnose systemic disease based on its oral presentation. Recurrent periodontal abscesses and accentuated gingival inflammation and periodontal bone loss occur more frequently in diabetic patients. Some cases of leukemia have been first diagnosed in the dental office because of the abnormal patterns of gingival hyperplasia, necrosis, and bleeding caused by the associated thrombocytopenia, which can result in the unusual sign of internal bleeding and ecchymosis of the gingival tissues.

Other diseases associated with deficient polymorphonuclear leukocyte function, such as Papillon Lefèvre syndrome, Down syndrome, and Chédiak-Higashi syndrome, present with aggressive periodontitis.

Hormonal changes of the estrogen progesterone complex can accentuate plaque-induced gingivitis. This is most profound when it occurs in pregnancy, but cyclic menstrual changes, puberty, and the use of hormonal-based contraceptives can make gingivitis more severe.

Nutritional deficiencies need to be severe and prolonged before any dramatic periodontal manifestations are observed, and this includes vitamin C dietary deficiency, which only causes significant changes to gingival health in the late stages of scurvy.

Osteoporosis does not have dramatic periodontal implications, but edentulous alveolar processes may be more affected than alveolar bone supporting the teeth. Recent widespread utilization of bisphosphonate therapy has focused attention on bisphosphonate-induced osteonecrosis of the jaw (BIONJ), which can occur spontaneously or follow surgical procedures such as tooth extraction, periodontal surgery, or implant surgery. The risk for this is particularly high in patients subjected to intravenous administration, and these patients need to be managed by dental specialists. Oral bisphosphonates carry a lower risk of osteonecrotic sequelae, but even these patients may be better treated by nonsurgical therapy.

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CHAPTER 28

Impact of Periodontal Infection on Systemic Health

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CHAPTER OUTLINE

PATHOBIOLOGY OF PERIODONTITIS
FOCAL INFECTION THEORY REVISITED
EVIDENCE-BASED CLINICAL PRACTICE
SUBGINGIVAL ENVIRONMENT AS RESERVOIR OF BACTERIA
PERIODONTAL DISEASE AND MORTALITY
PERIODONTAL DISEASE AND CORONARY HEART DISEASE/ATHEROSCLEROSIS
Effect of Periodontal Infection
Role of Periodontal Disease in Myocardial or Cerebral Infarction
PERIODONTAL DISEASE AND STROKE (online only)
Periodontal Infection Associated with Stroke (online only)
PERIODONTAL DISEASE AND DIABETES MELLITUS
Periodontal Infection Associated with Glycemic Control in Diabetes
PERIODONTAL DISEASE AND PREGNANCY OUTCOME (online only)
Bacterial Vaginosis (online only)
Role of Periodontitis (online only)
PERIODONTAL DISEASE AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE (online only)
PERIODONTAL DISEASE AND ACUTE RESPIRATORY INFECTIONS (online only)
PERIODONTAL MEDICINE IN CLINICAL PRACTICE
Periodontal Disease and Systemic Health
Patient Education
SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some text, figures, references, and tables may be out of numeric order in this printed chapter.

Knowledge of the pathogenesis of periodontal diseases has evolved markedly over the last 50 years.[55] Periodontal disease is an inflammatory disease initiated by bacterial pathogens. Environmental, physical, social, and host stresses may affect and modify disease expression through a multitude of pathways. Certain systemic conditions can affect the initiation and progression of gingivitis and periodontitis (see Chapters 24 and 27). Systemic disorders affecting neutrophil, monocyte/macrophage, and lymphocyte function result in altered production or activity of host inflammatory mediators.[55,86] These alterations may manifest clinically as early onset of periodontal destruction or a more rapid rate of destruction than would occur in the absence of such disorders.

Evidence has also shed light on the converse side of the relationship between systemic health and oral health, that is, the potential effects of inflammatory periodontal diseases on a wide range of organ systems. This field of periodontal medicine addresses the following important questions:

- Can the inflammatory response to bacterial infection of the *periodontium* have an effect remote from the oral cavity?
- Is periodontal infection a risk factor for systemic diseases or conditions that affect human health?

PATHOBIOLOGY OF PERIODONTITIS

Our understanding of the pathogenesis of periodontitis has changed remarkably over the last 30 years.[55,87,90] Nonspecific accumulation of bacterial plaque was once thought to be the cause of periodontal destruction, but it is now recognized that periodontitis is an infectious disease associated with a small number of

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predominantly gram-negative microorganisms that exist in a subgingival biofilm.[34] Furthermore, the importance of the host in disease initiation and progression is clearly recognized. Although pathogenic bacteria are necessary for periodontal disease, they are not sufficient alone to cause the disease. A susceptible host is also imperative. In a host who has relatively low susceptibility to disease, bacterial pathogens may have no clinical effect. This may be due to a particularly effective host immunoinflammatory response that eliminates pathogenic organisms while minimizing destruction of native tissues. Conversely, in a host with relatively high disease susceptibility, marked destruction of periodontal tissues may result.

Recognition of the importance of host susceptibility opens a door to understanding the differences in the onset, natural history, and progression of periodontitis seen throughout the scientific literature. Because of differences in host susceptibility, not all individuals are equally vulnerable to the destructive effects of periodontal pathogens and the immunoinflammatory response to those organisms. Thus patients may not necessarily have similar disease expression despite the presence of similar bacteria. Likewise, the response to periodontal treatment may vary depending on the wound-healing capacity and susceptibility of the host to further disease progression. The importance of host susceptibility is clearly evident in the medical literature. For example, respiratory tract pathogens may have minimal effect on many individuals, but in a susceptible host, such as an elderly patient, these same pathogens may cause life-threatening respiratory tract illnesses.

There are many systemic conditions that can modify the host's susceptibility to periodontitis. For example, patients with immune suppression may not be able to mount an effective host response to subgingival microorganisms, resulting in more rapid and severe periodontal destruction. Conversely, individuals with a significant increase in production of proinflammatory mediators may respond to periodontal pathogens with an exuberant inflammatory response that results in destruction of periodontal tissues. Although the potential impact of many systemic disorders on the periodontium is well documented, recent evidence suggests that periodontal infection may significantly enhance the risk for certain systemic diseases or alter the natural course of systemic conditions.[75,104] Conditions in which the influences of periodontal infection are documented include coronary heart disease (CHD) and CHD-related events such as angina and infarction, atherosclerosis, stroke, diabetes mellitus, preterm labor, low-birth-weight delivery, and respiratory conditions such as chronic obstructive pulmonary disease[75,89] (Box 28-1).

BOX 28-1 Organ Systems and Conditions Possibly Influenced by Periodontal Infection

Cardiovascular/Cerebrovascular System
Atherosclerosis
Coronary heart disease (CHD)
Angina
Myocardial infarction (MI)
Cerebrovascular accident (stroke)

Endocrine System
Diabetes mellitus

Reproductive System
Preterm low-birth-weight (LBW) infants
Preeclampsia

Respiratory System
Chronic obstructive pulmonary disease (COPD)
Acute bacterial pneumonia

FOCAL INFECTION THEORY REVISITED

Research in the area of periodontal medicine marks a resurgence in the concept of focal infection. In 1900, William Hunter, a British physician, first developed the idea that oral microorganisms were responsible for a wide range of systemic conditions that were not easily recognized as being infectious in nature.[82,116] He claimed that restoration of carious teeth instead of extraction resulted in the trapping of infectious agents under restorations. In addition to caries, pulpal necrosis, and periapical abscesses, Hunter also identified gingivitis and periodontitis as foci of infection. He advocated extraction of teeth with these conditions to eliminate the source of sepsis. Hunter believed that teeth were liable to septic infection primarily because of their structure and their relationship to alveolar bone. He stated that the degree of systemic effect produced by oral sepsis depended on the virulence of the oral infection and the individual's degree of resistance. He also believed that oral organisms had specific actions on different tissues and that these organisms acted by producing toxins, resulting in low-grade "subinfection," which produced systemic effects over prolonged periods. Finally, Hunter believed that the connection between oral sepsis and resulting systemic conditions could be shown by removal of the causative sepsis through tooth extraction and observation of the improvement in systemic health. Because it explained a wide range of disorders for which there was no known explanation at the time, Hunter's theory became widely accepted in Britain and eventually the United States, leading to wholesale extraction of teeth.

The focal infection theory fell into disrepute in the 1940s and 1950s when widespread extraction, often of the entire dentition, failed to reduce or eliminate the systemic conditions to which the supposedly infected dentition had been linked.[116] The theory, while offering a possible explanation for perplexing systemic disorders, had been based on very little, if any, scientific evidence. Hunter and other advocates of the theory were unable to explain how focal oral sepsis produced these systemic maladies. They were also unable to elucidate possible interactive mechanisms between oral and systemic health. Furthermore, the suggested intervention of tooth extraction often had no effect on the systemic conditions for which patients sought relief. However, Hunter's ideas did encourage extensive research in microbiology and immunology.

EVIDENCE-BASED CLINICAL PRACTICE

Many of the precepts of the focal infection theory are being revived today in light of recent research demonstrating links between oral and systemic health. However, in order for the "hypothesis not to fall into disrepute for a second time, there must be no unsubstantiated attributions, no theories without evidence."[82] Today's era of evidence-based medicine and dentistry provides an excellent environment in which to examine the possible relationships between oral infection and systemic disorders. To establish a relationship between conditions A and B, different levels of evidence must be examined. All scientific evidence is not given the same weight.[43,78,83] The stronger the evidence, the more likely it is that a true relationship exists between the conditions. Table 28-1 describes these various levels of evidence.

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TABLE 28-1 Evaluation of Evidence

Type of Evidence	Strength of Evidence	Description
Case report	+/–	• Provides relatively weak retrospective anecdotal evidence. • May suggest further study needed.
Cross-sectional study	+	• Compares groups of subjects at a single point in time. • Stronger than case report. • Fairly easy to conduct. • Relatively inexpensive to conduct.
Longitudinal study	++	• Follows groups of subjects over time. • Stronger than cross-sectional study. • Studies with control group much stronger than studies without controls. • More difficult and expensive to conduct.
Intervention trial	+++	• Examines effects of some intervention. • Studies with control group (i.e., placebo) much stronger than studies without controls. • Strongest form of evidence is randomized controlled intervention trial. • Difficult and expensive to conduct.
Systematic review	++++	• Systematically evaluates evidence from multiple studies, especially randomized controlled trials (RCTs). • Uses clearly defined guidelines for selection of evidence to be included or excluded from the review. • Examines for heterogeneity in the overall data, indicating variations in study design, sample populations, assessment methodologies.

For example, in examining the relationship between elevated cholesterol levels and CHD-related events, the literature might initially consist entirely of case reports or similar anecdotal information in which individual patients with recent myocardial infarction (MI) are found to have elevated cholesterol levels. These anecdotal reports suggest a possible relationship between elevated cholesterol and MI, but the evidence is weak. The case reports may lead to *cross-sectional studies* in which a large subject population is examined to determine whether those individuals who had an MI have higher cholesterol levels than other individuals (control subjects) who did not have an MI. Ideally, these cross-sectional studies are controlled for other potential causes or factors associated with MI such as age, gender, and smoking history. In other words, the subjects with a previous MI would be retrospectively "matched" with subjects of similar age, gender, and smoking history. Then their cholesterol levels would be examined for similarities or differences. Significantly higher cholesterol level in subjects with a previous MI compared with those without MI offers stronger evidence than case reports, and it further substantiates a possible link between elevated cholesterol and MI.

Even stronger evidence is provided by *longitudinal studies,* in which subject populations are examined over time. For example, a group of subjects might periodically have cholesterol levels evaluated over several years. If individuals with elevated cholesterol levels have a significantly higher rate of MI over time compared with subjects with normal cholesterol levels, even stronger evidence is available to substantiate the link between cholesterol and MI. Finally, *intervention trials* may be designed to alter the potentially causative condition and to determine the effect of this change on the resultant condition. For example, patients with elevated cholesterol may be divided into two groups: a group who uses a cholesterol-lowering drug or diet and a control group who has no intervention. These two groups might also be compared with a third group with normal cholesterol levels. Over time, the rate of MI in each group would be determined. If the group receiving the cholesterol-lowering regimen has a significantly lower rate of MI than the group with continued elevations in cholesterol level, strong evidence of a link between cholesterol and MI would be established.

Finally, the highest level of evidence is the systematic review. A systematic review is not a standard literature review in which the articles selected for review are based on the desires and search methods chosen by the author, often for convenience. In a systematic review, the topic in question is selected before the review begins. For example, the authors may state the question as, "Compared to subjects not taking cholesterol-lowering medications, do subjects taking such medications demonstrate a difference in the rate of myocardial infarction?" A specific search strategy is then determined to reveal as much potential data as possible to answer the stated question. The authors state specifically why research papers were included or excluded from the review. If possible, the data are subjected to metaanalysis, a statistical method that combines the results of multiple studies that address a similar research hypotheses. This provides a more robust evaluation of the overall data than one can glean from individual research articles.

At each level of evidence, it is important to determine whether a biologically plausible link exists between conditions A and B. For example, if case reports, cross-sectional studies, longitudinal studies, and intervention trials all support the link between cholesterol levels and MI, the following questions remain:

- How is cholesterol related to myocardial infarction?
- What are the mechanisms by which cholesterol affects the cardiovascular system and thus increases the risk for infarction?

These studies evaluate the mechanisms by which conditions A and B might be linked and provide explanatory data that further substantiate the association between the two conditions.

The focal infection theory, as proposed and defended in the early part of the twentieth century, was based on almost no evidence. Only the occasional case report and other anecdotes were

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available to substantiate the theory. Although explanatory mechanisms were proposed, none was validated with scientific research. Unfortunately, this theory predated current concepts of evidence-based clinical practice, leading to unnecessary extraction of millions of teeth. Currently, in reexamining the potential associations between oral infections and systemic conditions, it is important to determine what evidence (1) is available, (2) is still needed to substantiate the associations, and (3) validates the possible mechanisms of association. This chapter reviews current knowledge relating periodontal infection to overall systemic health.

SUBGINGIVAL ENVIRONMENT AS RESERVOIR OF BACTERIA

The subgingival microbiota in patients with periodontitis provides a significant and persistent gram-negative bacterial challenge to the host, one that is met by a potent immunoinflammatory response.[84] These organisms and their products, such as *lipopolysaccharide* (LPS), have ready access to the periodontal tissues and to the circulation via the sulcular epithelium, which is frequently ulcerated and discontinuous. Even with treatment, complete eradication of these organisms is difficult, and their reemergence is often rapid. The total surface area of pocket epithelium in contact with subgingival bacteria and their products in a patient with generalized moderate periodontitis has been estimated to be approximately the size of the palm of an adult hand, with even larger areas of exposure in cases of more advanced periodontal destruction.[90] Bacteremias are common after mechanical periodontal therapy and also occur frequently during normal daily function and oral hygiene procedures.[29,57,76] Just as the periodontal tissues mount an immunoinflammatory response to bacteria and their products, systemic challenge with these agents also induces a major vascular response.[23,40,93] This host response may offer explanatory mechanisms for the interactions between periodontal infection and a variety of systemic disorders.

PERIODONTAL DISEASE AND MORTALITY

The ultimate medical outcome measure is mortality. A number of studies suggest an increased mortality rate is associated with inflammatory periodontal diseases.[25,28,49,97] The Normative Aging Study examined 2280 healthy men every 3 years for more than 30 years after baseline clinical, radiographic, laboratory, and electrocardiographic examinations. A subset of this population was examined in the Veterans Affairs (VA) Dental Longitudinal Study to determine age-related changes in the oral cavity and to identify risk factors for oral disease. Clinical examinations were performed, and alveolar bone level measurements determined from full-mouth radiographs. The mean percentage of alveolar bone loss and the mean probing depth were determined for each subject.

A study of data from this subject population sought to determine whether periodontal disease status was a significant predictor of mortality independent of other baseline characteristics within the population.[28] From the original sample of 804 dentate, medically healthy subjects, a total of 166 died during the study. Periodontal status at the baseline examination was a significant predictor of mortality independent of other factors such as smoking, alcohol use, cholesterol levels, blood pressure, family history of heart disease, education level, and body mass. For those subjects with the most alveolar bone loss, averaging more than 21% alveolar bone loss at baseline, the risk of dying during the follow-up period was 70% higher than for all other subjects. Interestingly, alveolar bone loss increased the risk of mortality more than smoking (52% increased risk), a well-known risk factor for mortality. A later follow-up of these same subjects confirmed a higher incidence of CHD-related events such as MI and unstable angina among men under the age of 60 with alveolar bone loss than those without bone loss.[25] In these studies, periodontitis preceded and increased the risk of mortality. However, this only establishes an association, not causation. It is possible that periodontal disease reflects other health behaviors not evaluated in this study, rather than acting as a specific cause of mortality. In other words, patients with poor periodontal health may also have other risk factors that increase mortality rate (e.g., smoking).

In examining research that suggests oral health status as a possible risk factor for systemic conditions, it is important to recognize when other known risk factors for those systemic conditions have been accounted for in the analysis. Host susceptibility factors that place individuals at risk for periodontitis may also place them at risk for systemic diseases such as cardiovascular disease. In these patients the association may actually be between the risk factors rather than between the diseases. For example, periodontitis and cardiovascular disease share such risk factors as smoking, age, race, male gender, and stress. Genetic risk factors may also be shared.[54] In the VA Dental Longitudinal Study, smoking was an independent risk factor for mortality. When examining the data to determine if periodontal status was a risk factor, smoking status and other known risk factors for mortality were removed from the equation to allow independent evaluation of periodontal status. Other studies support the association between poor oral health and an increased risk for mortality.[97] In a prospective longitudinal study of subjects with type 2 diabetes, those with severe periodontitis had 3.2 times the risk of death from ischemic heart disease or kidney disease compared to subjects without periodontitis or with only slight periodontitis after adjusting for other risk factors including age, sex, duration of diabetes, glycemic control, macroalbuminuria, body mass index, serum cholesterol concentration, hypertension, and current smoking.[97]

PERIODONTAL DISEASE AND CORONARY HEART DISEASE/ATHEROSCLEROSIS

To further explore the periodontal disease and CHD/atherosclerosis association, investigators have studied specific systemic disorders and medical outcomes to determine their relationship to periodontal status. CHD-related events are a major cause of death. MI has been associated with acute systemic bacterial and viral infections, and MI is sometimes preceded by influenzalike symptoms.[70,106] Is it possible that oral infection is similarly related to MI? Traditional risk factors, such as smoking, dyslipidemia, hypertension, and diabetes mellitus, do not explain the presence of coronary atherosclerosis in a large number of patients. Localized infection resulting in a chronic inflammatory reaction has been suggested as a mechanism underlying CHD in these individuals.[77]

In cross-sectional studies of patients with acute MI or confirmed CHD compared with age- and gender-matched control patients, MI patients had significantly worse dental health (periodontitis, periapical lesions, caries, or pericoronitis) than did controls.[48,66,69] This association between poor dental health and MI was independent of known risk factors for heart disease, such as age, cholesterol levels, hypertension, diabetes, and smoking. Because atherosclerosis is a major determinant of CHD-related events, dental health has also been related to coronary atheromatosis. Mattila et al[67] performed oral radiographic examinations and diagnostic coronary angiography on men with known CHD and found significant correlation between the severity of dental disease and the degree of coronary atheromatosis. This relationship remained significant after accounting for other known risk factors

for coronary artery disease (CAD). Similarly, Malthaner et al[65] found an increased risk of angiographically defined CAD in subjects with greater bone loss and attachment loss; however, after adjusting for other known cardiovascular risk factors, the relationship between periodontal status and CAD was no longer statistically significant. There is evidence that the extent of periodontal disease may be associated with CHD. For example, there may be a greater risk for CHD-related events, such as MI, when periodontitis affects a greater number of teeth in the mouth, compared with subjects having periodontitis at fewer teeth.[5]

Cross-sectional studies thus suggest a possible link between oral health and CHD; however, such studies cannot determine causality in this relationship. Rather, dental diseases may be indicators of general health practices. For example, periodontal disease and CHD are both related to lifestyle and share numerous risk factors, including smoking, diabetes, and low socioeconomic status. Bacterial infections have significant effects on endothelial cells, blood coagulation, lipid metabolism, and monocytes/macrophages. The research of Mattila et al[66] showed that dental infections were the only factors, other than the classic and well-recognized coronary risk factors, that were associated independently with the severity of coronary atherosclerosis.

Longitudinal studies provide compelling data on this relationship. In a 7-year follow-up study of the patients from the study of Mattila and colleagues, dental disease was significantly related to the incidence of new fatal and nonfatal coronary events, as well as to overall mortality.[68] In a prospective study of a national sample of adults, subjects with periodontitis had a 25% increase in the risk for CHD compared with those with no or minimal periodontal disease, after adjusting for other known risk factors.[24] Among younger males (age 25 to 49), periodontitis increased the risk of CHD by 70%. The level of oral hygiene was also associated with heart disease. Patients with poor oral hygiene, as indicated by higher debris and calculus scores, had a twofold increased risk for CHD.

In another large prospective study, 1147 men were followed for 18 years.[9] During that time, 207 men (18%) developed CHD. When periodontal status at baseline was related to the presence or absence of CHD-related events during follow-up, a significant relationship was found. Subjects with greater than 20% mean bone loss had a 50% increased risk of CHD compared with those with up to 20% bone loss. The extent of sites with probing depth greater than 3 mm was strongly related to the incidence of CHD. Subjects with probing depths greater than 3 mm on at least half their teeth had a twofold increased risk, whereas those with probing depths greater than 3 mm on all teeth had more than a threefold increased risk of CHD. This study and others in which the periodontal condition is known to have preceded the CHD-related events support the concept that periodontal disease is a risk factor for CHD, independent of other classic risk factors. Not all studies, however, support this concept; some show little independent effect of periodontal status on the risk for CHD, after adjusting for commonly accepted cardiovascular risk factors.[44,45] It is particularly difficult to control for smoking as a confounding variable in these studies because it is such an important risk factor for both periodontal disease and cardiovascular disease. This confounding influence of smoking makes it difficult to clarify the significance of the relationship between the diseases.

Perhaps the best evidence available comes from *systematic reviews* of studies examining the relationship between periodontal infection and cardiovascular diseases. Janket et al[47] performed a metaanalysis of periodontal disease as a risk factor for future cardiovascular events and found an overall 19% increased risk of such events in individuals with periodontitis. The increase in risk was greater (44%) in people under age 65. Although this increased risk is fairly modest, the extensive prevalence of periodontal disease in the population may increase the significance of the risk from a public health perspective. An extensive systematic review by Scannapieco et al[98] concluded that a moderate degree of evidence exists to support an association between periodontal disease and atherosclerosis, MI, and cardiovascular disease but that causality is unclear. Importantly, insufficient evidence exists to show that treatment of periodontal disease has any impact on the risk of heart disease. Intervention trials are needed to make this determination.

Effect of Periodontal Infection

Periodontal infection may affect the onset or progression of atherosclerosis and CHD through both causal and noncausal mechanisms (i.e., by both direct and indirect pathways). Periodontitis and atherosclerosis both have complex etiologic factors, combining genetic and environmental influences. In addition to smoking, the diseases share many risk factors and have distinct similarities in basic pathogenic mechanisms.

Ischemic Heart Disease. Ischemic heart disease is associated with the processes of atherogenesis and thrombogenesis (Figure 28-1). Increased viscosity of blood may promote major ischemic heart disease and cerebrovascular accident (stroke) by increasing the risk of thrombus formation.[62] Fibrinogen is a major factor in promoting this hypercoagulable state. *Fibrinogen* is the precursor to fibrin, and increased fibrinogen levels increase blood viscosity. Increased plasma fibrinogen is a recognized risk factor for cardiovascular events and peripheral vascular disease[63] (Figure 28-2). Elevated white blood cell (WBC) count is also a predictor of heart disease and stroke, and circulating leukocytes may promote occlusion of blood vessels. Coagulation factor VIII/von Willebrand factor (vWF) has likewise been associated with the risk of ischemic heart disease.[94]

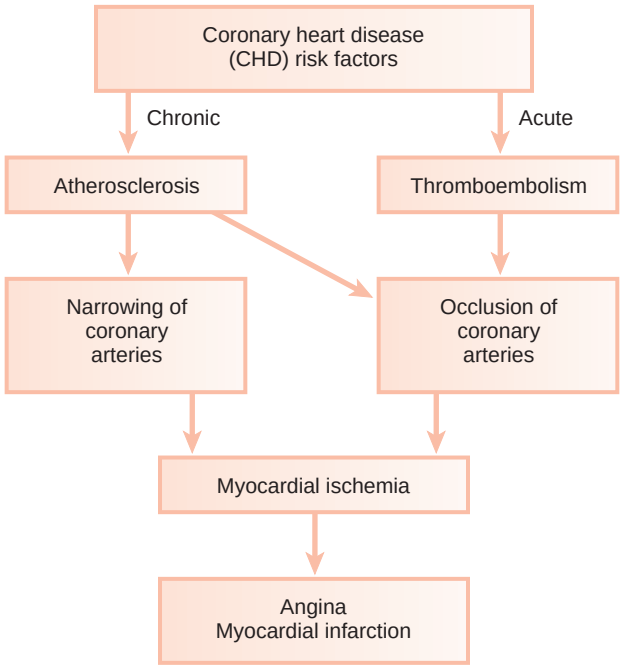

Figure 28-1 Acute and chronic pathways to ischemic heart disease. Coronary heart disease (CHD)-related events, such as angina or myocardial infarction (MI), may be precipitated by either pathway or both pathways.

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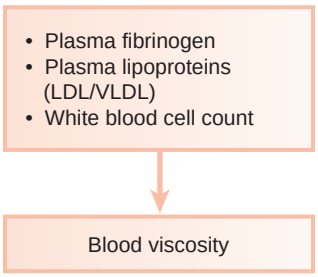

Figure 28-2 Factors affecting blood viscosity in health.

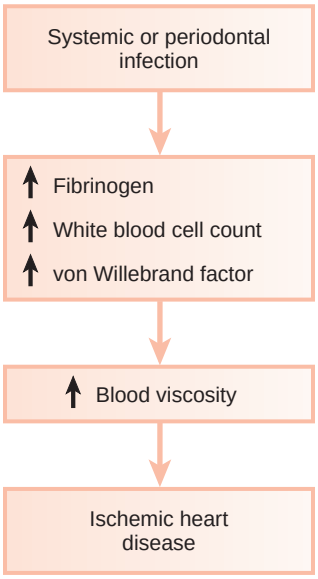

Figure 28-3 Effect of infection on blood viscosity. Increased plasma fibrinogen and von Willebrand factor cause hypercoagulability. When combined with increased white blood cell count, blood viscosity increases, increasing the risk of coronary ischemia.

Systemic Infections. Systemic infections are known to induce a hypercoagulable state and to increase blood viscosity (Figure 28-3). Fibrinogen levels and WBC counts are often increased in patients with periodontal disease.[15,56] Individuals with poor oral health may also have significant elevations in coagulation factor VIII/vWF antigen, increasing the risk of thrombus formation. Thus periodontal infection may also promote increased blood viscosity and thrombogenesis, leading to an increased risk for central and peripheral vascular disease.

Daily Activity. Routine daily activities, such as mastication and oral hygiene procedures, result in frequent bacteremia with oral organisms.[57] Periodontal disease may predispose the patient to an increased incidence of bacteremia, including the presence of virulent gram-negative organisms associated with periodontitis. An estimated 8% of all cases of infective endocarditis are associated with periodontal or dental disease, without a preceding dental procedure.[26] The periodontium, when affected by periodontitis, also acts as a reservoir of *endotoxin* (LPS) from gram-negative organisms. Endotoxin can pass readily into the systemic circulation during normal daily function, inducing damage to the vascular endothelium and precipitating many negative cardiovascular effects. In a study of the incidence of endotoxemia after simple chewing, subjects with periodontitis were four times more likely to have endotoxin present in the bloodstream than subjects without periodontitis. furthermore, the concentration of endotoxin present was more than fourfold greater in those with periodontitis than in healthy subjects.[29]

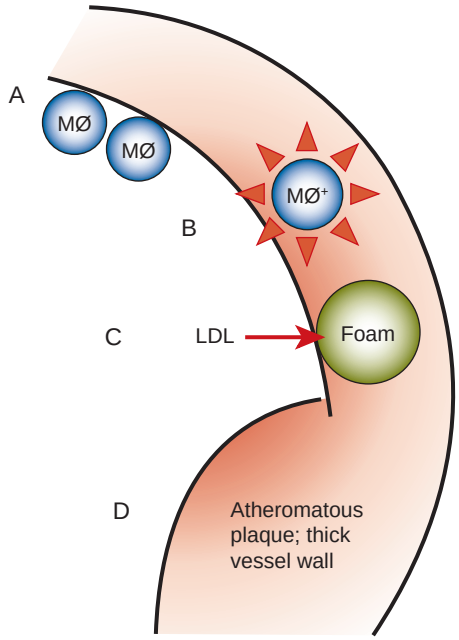

Figure 28-4 Pathogenesis of atherosclerosis. **A,** Monocytes/macrophages adhere to vascular endothelium. **B,** Monocytes/macrophages penetrate into arterial media, producing proinflammatory cytokines and growth factors. **C,** Ingestion of oxidized low-density lipoprotein (LDL) enlarges monocytes to form foam cells. **D,** Smooth muscle proliferation and plaque formation thicken vessel wall and narrow lumen. *MØ+,* Hyperinflammatory monocyte/macrophage phenotype.

Thrombogenesis. Platelet aggregation plays a major role in thrombogenesis, and most cases of acute MI are precipitated by thromboembolism. Oral organisms may be involved in coronary thrombogenesis. Platelets selectively bind some strains of *Streptococcus sanguis*, a common component of supragingival plaque, and *Porphyromonas gingivalis*, a pathogen closely associated with periodontitis.[38,39] Aggregation of platelets is induced by the *platelet aggregation–associated protein* (PAAP) expressed on some strains of these bacteria.[95] In animal models, intravenous infusion of PAAP-positive bacterial strains resulted in alterations of heart rate, blood pressure, cardiac contractility, and electrocardiogram (ECG) readings consistent with MI. Platelet accumulation also occurred in the lungs, leading to tachypnea. No such changes were seen with infusion of PAAP-negative strains. PAAP-positive bacteria caused aggregation of circulating platelets, resulting in formation of thromboemboli and the resultant cardiac and pulmonary changes. Thus periodontitis-associated bacteremia with certain strains of *S. sanguis* and *P. gingivalis* may promote acute thromboembolic events through interaction with circulating platelets.

Atherosclerosis. Atherosclerosis is a focal thickening of the arterial *intima,* the innermost layer lining the vessel lumen, and the *media,* the thick layer under the intima consisting of smooth muscle, collagen, and elastic fibers (Figure 28-4).[95] Early in the formation of atherosclerotic plaques, circulating monocytes adhere to the vascular endothelium. This adherence is mediated through several adhesion molecules on the endothelial cell surface, including intercellular adhesion molecule-1 (ICAM-1), endothelial leukocyte adhesion molecule-1 (ELAM-1), and vascular cell adhesion

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molecule-1 (VCAM-1).[11,52] These adhesion molecules are upregulated by a number of factors, including bacterial LPS, prostaglandins, and proinflammatory cytokines. After binding to the endothelial cell lining, monocytes penetrate the endothelium and migrate under the arterial intima. The monocytes ingest circulating low-density lipoprotein (LDL) in its oxidized state and become engorged, forming foam cells characteristic of atheromatous plaques. Once within the arterial media, monocytes may also transform to macrophages. A host of proinflammatory cytokines, such as interleukin-1 (IL-1), tumor necrosis factor alpha (TNF-α), and prostaglandin E2 (PGE_2), are then produced, which propagate the atheromatous lesion. Mitogenic factors, such as fibroblast growth factor and platelet-derived growth factor, stimulate smooth muscle and collagen proliferation within the media, thickening the arterial wall.[63] Atheromatous plaque formation and thickening of the vessel wall narrow the lumen and dramatically decrease blood flow through the vessel.[95] Arterial thrombosis often occurs after an atheromatous plaque ruptures. Plaque rupture exposes circulating blood to arterial collagen and tissue factor from monocytes/macrophages that activate platelets and the coagulation pathway. Platelet and fibrin accumulation forms a thrombus that may occlude the vessel, resulting in ischemic events such as angina or MI. The thrombus may separate from the vessel wall and form an embolus, which may also occlude vessels, again leading to acute events such as MI or cerebral infarction (stroke).

Role of Periodontal Disease in Myocardial or Cerebral Infarction

In animal models, gram-negative bacteria and associated LPS cause infiltration of inflammatory cells into the arterial wall, proliferation of arterial smooth muscle, and intravascular coagulation. These changes are identical to those seen in naturally occurring atheromatosis. Patients with periodontitis are at increased risk for thickening of the walls of major coronary arteries.[8] In several studies of atheromas obtained from humans during endarterectomy, more than half the lesions contained periodontal pathogens, and many atheromas contained multiple different periodontal species.[14,36,121] Periodontal diseases result in chronic systemic exposure to products of these organisms. Low-level bacteremia may initiate host responses that alter coagulability, endothelial and vessel wall integrity, and platelet function, resulting in atherogenic changes and possible thromboembolic events (Figure 28-5).

Research has clearly shown a wide variation in host response to bacterial challenge. Some individuals with heavy plaque accumulation and high proportions of pathogenic organisms appear relatively resistant to bone and attachment loss. Others develop extensive periodontal destruction in the presence of small amounts of plaque and low proportions of putative pathogens. Patients with abnormally exuberant inflammatory responses often have a hyperinflammatory monocyte/macrophage phenotype ($MØ^+$). Monocytes/macrophages from these individuals secrete significantly increased levels of proinflammatory mediators (e.g., IL-1, TNF-α, PGE_2) in response to bacterial LPS compared with patients with a normal monocyte/macrophage phenotype. Patients with aggressive periodontitis, refractory periodontitis, and type 1 diabetes mellitus often possess the $MØ^+$ phenotype,[10] which appears to be under both genetic and environmental control. The monocyte/macrophage cell line is intimately involved in the pathogenesis of both periodontal disease and atherosclerosis. Diet-induced elevations in serum LDL levels upregulate monocyte/macrophage responsiveness to bacterial LPS. Thus elevated LDL levels, a known risk factor for atherosclerosis and CHD, may increase secretion of destructive and inflammatory cytokines by

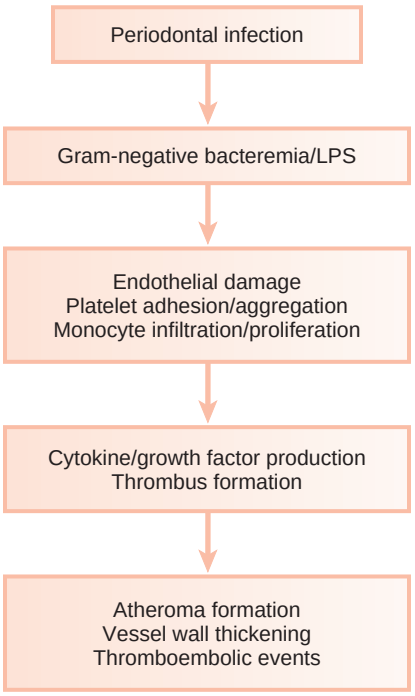

Figure 28-5 Influence of periodontal infection on atherosclerosis. Periodontal pathogens and their products result in damage to vascular endothelium. Monocytes/macrophages enter the vessel wall, producing cytokines that further increase inflammatory response and propagate the atheromatous lesion. Growth factor production leads to smooth muscle proliferation in the vessel wall. Damaged endothelium also activates platelets, resulting in platelet aggregation and potentiating thromboembolic events. *LPS,* Lipopolysaccharide.

monocytes/macrophages. This may result not only in propagation of atheromatous lesions but also in enhanced periodontal destruction in the presence of pathogenic organisms. This is one example of a potential shared mechanism in the pathogenesis of cardiovascular and periodontal diseases. The presence of an $MØ^+$ phenotype may place patients at risk for both CHD and periodontitis (Figure 28-6). Periodontal infections may contribute to atherosclerosis and thromboembolic events by repeatedly challenging the vascular endothelium and arterial wall with bacterial LPS and proinflammatory cytokines. Vascular monocytes/macrophages in patients with an $MØ^+$ phenotype meet this challenge with an abnormally elevated inflammatory response that may directly contribute to atherosclerosis and may precipitate thromboembolic events.[75]

Cardiovascular diseases are increasingly recognized as having a major systemic inflammatory component, further emphasizing possible similarities with inflammatory periodontal diseases.[95] As such, detection of systemic inflammatory markers plays an increasingly important role in risk assessment for vascular events such as MI and cerebral infarction. Acute-phase proteins, such as *C-reactive protein* (CRP) and fibrinogen, are produced in the liver in response to inflammatory or infectious stimuli and act as inflammatory markers.[93] CRP induces monocytes/macrophages to produce tissue factor, which stimulates the coagulation pathway and increases blood coagulability. Increased fibrinogen levels may contribute to this process. CRP also stimulates the complement cascade, further exacerbating inflammation.

Elevations in serum CRP and fibrinogen levels are well-accepted risk factors for cardiovascular disease.[92,93] Recent efforts have focused on periodontitis as a potential trigger for systemic inflammation. Serum CRP and fibrinogen levels are often elevated

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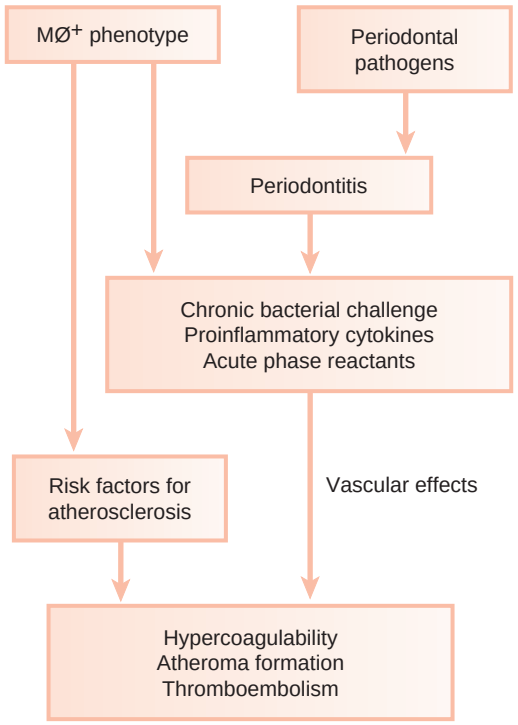

Figure 28-6 Cardiovascular and periodontal consequences of hyperresponsive monocyte/macrophage phenotype. In combination with other risk factors, the MØ+ phenotype predisposes to both atherosclerosis and periodontitis. Bacterial products and inflammatory mediators associated with periodontitis affect vascular endothelium, monocytes/ macrophages, platelets, and smooth muscle and may increase blood coagulability. This may further increase atherosclerosis and result in thromboembolism and ischemic events.

in subjects with periodontitis compared with subjects without periodontitis.[19,59,117] These acute-phase proteins may act as intermediary steps in the pathway from periodontal infection to cardiovascular disease (see Figures 28-5 and 28-6). Thus periodontal diseases may have both direct effects on the major blood vessels (e.g., atheroma formation) and indirect effects that stimulate changes in the cardiovascular system (e.g., elevation of systemic inflammatory responses).

Interesting supportive evidence for these mechanisms can be derived from intervention trials in which serum levels of inflammatory mediators and markers are assessed before and after treatment aimed at decreasing periodontal inflammation. For example, in subjects with chronic periodontitis, serum levels of IL-6 and CRP are reduced after scaling and root planing.[20] Inflammatory periodontal disease is also associated with altered vascular endothelial function compared to periodontally healthy individuals.[21] Altered vascular endothelial function is a major risk factor for acute thrombotic events. After scaling and root planing, with a resultant decrease in periodontal inflammation, markers of vascular health also improve significantly over time.[21,112] Functional assessment of vascular endothelial function also returns to normal after scaling and root planing.[102,112] These results suggest that periodontal inflammation adversely affects the health of the vascular endothelium, whereas a reduction in inflammation improves endothelial health. Whether these changes directly impact the risk of acute cardiovascular events remains to be determined in prospective, controlled clinical intervention trials over long periods of time; these studies do not currently exist.

BOX 28-2 Complications of Diabetes Mellitus

1. Retinopathy
2. Nephropathy
3. Neuropathy
4. Macrovascular disease
5. Altered wound healing
6. Periodontal disease

From Löe H: *Diabetes Care* 16(suppl 1):329, 1993.

For more information on periodontal disease and stroke, please visit the companion Expert CONSULT website at www.expertconsult.com.

PERIODONTAL DISEASE AND DIABETES MELLITUS

The relationship between diabetes mellitus and periodontal disease has been extensively examined. It is clear from epidemiologic research that diabetes increases the risk for and severity of periodontal diseases.[58] The biologic mechanisms through which diabetes influences the periodontium are discussed in Chapter 27. The increased prevalence and severity of periodontitis typically seen in patients with diabetes, especially those with poor metabolic control, led to the designation of periodontal disease as the "sixth complication of diabetes."[58] In addition to the five "classic" complications of diabetes (Box 28-2), the American Diabetes Association (ADA) has officially recognized that periodontal disease is common in patients with diabetes, and the Association's Standards of Care include taking a history of current or past dental infections as part of the physician's examination.[3] In fact, the ADA's 2009 Standards for Medical Care include assessment of a patient's history of "dental disease" as part of the recommended medical history in diabetic patient evaluation.[2] In addition, the Standards specifically recommend the physician refer patients with diabetes to a dentist for oral examination.

Although many studies have examined the effects of diabetes on the periodontium, fewer have examined the effect of periodontal infection on control of diabetes.[73] Such studies are difficult to perform because of the influence of ongoing medical management on diabetes control during the study. The following questions remain:

- Does the presence or severity of periodontal disease affect the metabolic state in diabetic patients?
- Does periodontal treatment aimed at reducing the bacterial challenge and minimizing inflammation have a measurable effect on glycemic (blood glucose) control?

In a longitudinal study of patients with type 2 diabetes, severe periodontitis was associated with significant worsening of glycemic control over time.[108] Individuals with severe periodontitis at the baseline examination had a greater incidence of worsening glycemic control over a 2- to 4-year period than did those without periodontitis at baseline. In this study, periodontitis is known to have preceded the worsening of glycemic control. Periodontitis has also been associated with the classic complications of diabetes. Diabetic adults with severe periodontitis at baseline had a significantly greater incidence of kidney and macrovascular complications over the subsequent 1 to 11 years than did diabetic adults with only gingivitis or mild periodontitis.[111] This was true despite both groups having similar glycemic control. One or more cardiovascular

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complications occurred in 82% of patients with severe periodontitis versus 21% of patients without severe periodontitis. Again, severe periodontitis preceded the onset of clinical diabetic complications in these subjects.

In diabetic patients with periodontitis, periodontal therapy may have beneficial effects on glycemic control.[73] This may be especially true for patients with relatively poor glycemic control and more advanced periodontal destruction before treatment. Fifty years ago, the potential benefits of periodontal therapy were first described in young adults with type 1 diabetes and severe periodontitis.[115,116] Treatment with scaling and root planing, surgery, selected tooth extraction, and systemic antibiotics resulted in decreased insulin demand. In a more recent case series evaluating scaling and root planing combined with systemic doxycycline therapy for 2 weeks, a small group of type 1 diabetic patients with improved periodontal health also had significant improvement in glycemic control (Figure 28-7).[80] Conversely, those individuals who demonstrated little beneficial clinical effect from periodontal treatment had no change in glycemic control. It is common to observe wide interindividual variability in responses to various medical management approaches in patients with type 1 diabetes. Similarly, wide variability in the impact of periodontal therapy on glycemic control can be expected in the type 1 diabetic population. For example, a study of periodontal therapy in 65 people with type 1 diabetes and chronic periodontitis showed highly variable responses.[110] While there was an overall improvement in periodontal health after therapy, approximately 35% of subjects had an improvement in glycemic control after treatment, 37% had no significant change, and 28% showed a worsening of glycemic control. In this study, subjects were divided into those with better baseline glycemic control (hemoglobin A1c [HbA1c] values <8.5%) and those with poorer glycemic control (HbA1c ≥ 8.5%). Interestingly, twice as many subjects showed improved glycemic control among those whose glycemic control before periodontal treatment was poor compared to those among whom baseline glycemic control was good. Thus, the clinician may anticipate a greater glycemic response to periodontal therapy among those type 1 diabetes patients whose glycemic control is relatively poor than among those whose glycemic control is already good before periodontal treatment.

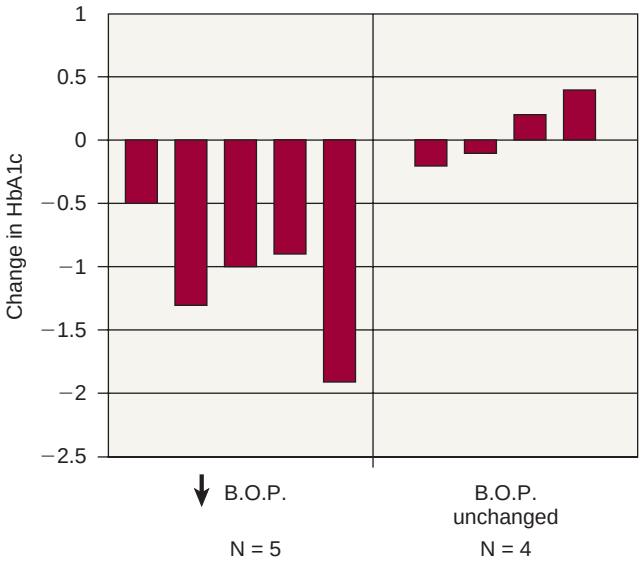

Figure 28-7 Periodontal treatment: effects on glycemic control. In five patients, reductions in periodontal inflammation after mechanical therapy combined with systemic doxycycline were accompanied by improved glycemic control (decreased glycated hemoglobin values, *HbA1c*). In four patients with no improvement in periodontal health, no improvement in glycemic control occurred. *B.O.P.,* Bleeding on probing (a measure of periodontal inflammation). *(Adapted from Miller LS, Manwell MA, Newbold D, et al:* J Periodontol *63:843, 1992.)*

In a classic placebo-controlled study of individuals with poorly controlled type 2 diabetes and severe periodontitis, scaling and root planing combined with systemic doxycycline for 14 days was compared with similar treatment combined with systemic placebo.[35] All patient groups had significant improvements in periodontal status, with reduced probing depths and bleeding on probing. Those treated with doxycycline had a greater reduction in the prevalence of *P. gingivalis*, which was more sustained over time. The doxycycline-treated patients also demonstrated significant improvement in glycemic control 3 months after treatment, which gradually reverted to baseline levels at 6 months. Placebo-treated subjects had no significant improvement in glycemic control. These studies suggest that the combination of subgingival mechanical debridement and systemic doxycycline may result in short-term improvement in glycemia in diabetic patients with severe periodontitis and poor metabolic control. Conversely, individuals with moderately well-controlled or well-controlled diabetes and periodontitis who are treated by mechanical therapy alone may demonstrate no significant changes in glycemic control, despite improvement in their periodontal condition.

In studies of subjects treated by mechanical therapy without adjunctive use of antibiotics, significant changes in glycemic control are less common.[1,16,105] Many patients in these studies had relatively good glycemic control before treatment, so less impact on metabolic control might be expected. However, some studies show improvement in glycemic control after scaling and root planing without antibiotics. In one such study, a group of well-controlled type 2 diabetic subjects with only gingivitis or mild periodontitis received scaling and root planing without antibiotics.[53] Three months after treatment, the subjects showed a 50% reduction in bleeding on probing that was accompanied by a significant improvement in glycemic control. A group of control subjects who received no periodontal treatment showed no change in glycemic control 3 months later, as one might expect given the continued presence of periodontal inflammation.

Although routine use of systemic antibiotics in treatment of chronic periodontitis is not justified, patients with poorly controlled diabetes and severe periodontitis may constitute one patient group for whom such therapy is appropriate. Antibiotics remain an adjunct to the necessary mechanical removal of plaque and calculus. The mechanisms by which adjunctive antibiotics may induce positive changes in glycemic control when combined with mechanical debridement are unknown at this time. Systemic antibiotics may eliminate residual bacteria after scaling and root planing, further decreasing the bacterial challenge to the host. Tetracyclines are also known to suppress glycation of proteins and to decrease activity of tissue-degrading enzymes such as matrix metalloproteinases (MMPs). These changes may contribute to improvement in metabolic control of diabetes. A low-dose form of doxycycline (20 mg) has been introduced with the specific purpose of reducing the production of collagen-degrading MMPs (see Chapter 48). This dose has no antibiotic effects and does not induce antimicrobial resistance with long-term use. Because diabetes is associated with greatly elevated production of collagenase, low-dose doxycycline has been used in treatment of periodontitis in diabetic subjects. Further research is indicated to determine the adjunctive benefit of such host modulation therapies in the diabetic population because little published evidence exists today.

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Periodontal Infection Associated with Glycemic Control in Diabetes

An understanding of the effects of other infections is useful in delineating the mechanisms by which periodontal infection influences glycemia. It is well known that systemic inflammation plays a major role in insulin sensitivity and glucose dynamics. As discussed previously, periodontal diseases can induce or perpetuate an elevated systemic chronic inflammatory state, as reflected in increased serum CRP, IL-6, and fibrinogen levels seen in many people with periodontitis.[20,59] Inflammation induces insulin resistance, and such resistance often accompanies systemic infections. For example, acute nonperiodontal bacterial and viral infections have been shown to increase insulin resistance and aggravate glycemic control.[96,120] This occurs in individuals with and without diabetes. Systemic infections increase tissue resistance to insulin through a variety of mechanisms, preventing glucose from entering target cells, causing elevated blood glucose levels, and requiring increased pancreatic insulin production to maintain normoglycemia. Insulin resistance may persist for weeks or even months after the patient has recovered clinically from their illness. In the individual with type 2 diabetes, who already has significant insulin resistance, further tissue resistance to insulin induced by infection may considerably exacerbate poor glycemic control. In type 1 patients, normal insulin doses may be inadequate to maintain good glycemic control in the presence of infection-induced tissue resistance. It is possible that chronic gram-negative periodontal infections may also result in increased insulin resistance and poor glycemic control.[34] In patients with periodontitis, persistent systemic challenge with periodontopathic bacteria and their products may act similar to well-recognized systemic infections (Figure 28-8). This mechanism would explain the worsening of glycemic control associated with severe periodontitis. Periodontal treatment designed to decrease the bacterial insult and reduce inflammation might restore insulin sensitivity over time, resulting in improved metabolic control. The improved glycemic control seen in several studies of periodontal therapy would support such a hypothesis.

For more information on periodontal disease and pregnancy outcome, and to view Figures 28-9 and 28-10, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on periodontal disease and chronic obstructive pulmonary disease, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on periodontal disease and acute respiratory infections, please visit the companion Expert CONSULT website at www.expertconsult.com.

PERIODONTAL MEDICINE IN CLINICAL PRACTICE

The concept of periodontal diseases as localized entities affecting only the teeth and supporting apparatus is oversimplified. Rather than being confined to the periodontium, inflammatory periodontal diseases may have wide-ranging systemic effects. In most persons, these effects may be relatively inconsequential or at least not clinically evident. In susceptible individuals, however, periodontal infection may act as an independent risk factor for systemic disease and may be involved in the basic pathogenic mechanisms of these conditions. Furthermore, periodontal infection may exacerbate existing systemic disorders.

Periodontal Disease and Systemic Health

Proper use of the knowledge of potential relationships between periodontal disease and systemic health requires the dental professional to recognize the oral cavity as one of many interrelated organ systems. An infection the size of one's palm on the leg of a pregnant woman would be a major concern to the patient and her health

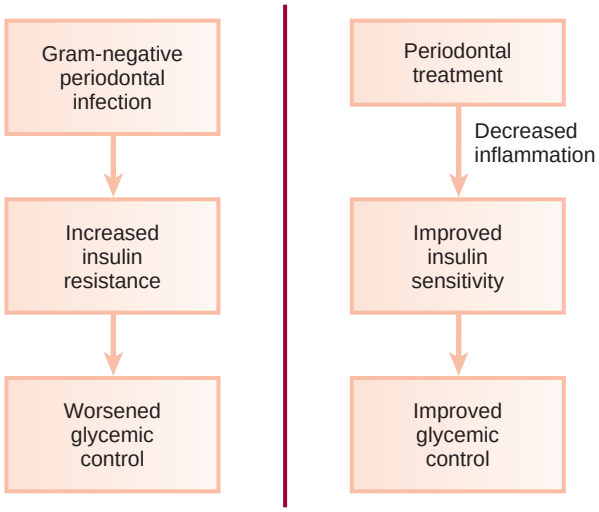

Figure 28-8 Potential effects of periodontal infection and periodontal therapy on glycemia in patients with diabetes.

SCIENCE TRANSFER

Clinicians have the responsibility to treat periodontal disease not only to maintain oral health but also to reduce the risk of systemic diseases related to active periodontal inflammation. The relationship between periodontal disease and arteriosclerosis is particularly important. Patients with periodontal disease are at greater risk for arteriosclerotic coronary artery disease (CAD) than patients with healthy periodontal tissues. There is also a connection between periodontal disease and thromboembolic events such as stroke. Diabetic patients may have improved diabetic status once their active periodontal disease is treated.

Dental clinicians need to inform patients of these risks and can play a role in enhancing the quality and longevity of life for their patients. Also, dentists need to communicate with physicians so that susceptible patients are evaluated for periodontal disease and receive appropriate treatment. Once successful periodontal therapy is completed, appropriate long-term maintenance therapy is essential to consolidate the reduction of the risk of systemic diseases.

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care provider, given the potential negative consequences of this localized infection on fetal and maternal health. A similar suppurating infection on the foot of a person with diabetes would be cause for immediate evaluation and aggressive treatment, knowing the effects of such infections on metabolic control of diabetes.

Periodontal infection must be viewed in a similar manner. Periodontitis is a gram-negative infection often resulting in severe inflammation, with potential intravascular dissemination of microorganisms and their products throughout the body. However, periodontitis tends to be a "silent" disease until destruction results in acute oral symptoms. Most patients, as well as many medical professionals, do not recognize the potential infection that may exist within the oral cavity.

Patient Education

Patient education is a priority. Only 30 years ago, the factors involved in CHD were unclear. At present, however, it would be difficult to find an individual who was unfamiliar with the link between cholesterol and heart disease. This change was precipitated by research clearly demonstrating the increased risk for heart disease in individuals with high cholesterol levels, followed by intensive education efforts to spread the message from the scientific community to the public at large. It is important to recognize that high cholesterol levels have not been shown to *cause* heart disease in all individuals, but rather significantly increase the *risk* of disease. Cholesterol has also been demonstrated to have a biologically plausible role in the pathogenesis of CHD.

Similarly, patient education efforts in the realm of periodontal medicine must emphasize the inflammatory nature of periodontal infections, the increased risk for systemic disease associated with the infection, and the biologically plausible role periodontal infection may play in systemic disease. Few individuals had their cholesterol levels evaluated until the knowledge of the link between cholesterol and heart disease became widespread. Likewise, increased appreciation of the potential effects of periodontal infection on systemic health may result in increased patient demand for periodontal evaluation.

Enhanced community awareness may be derived from newspapers, magazines, and other lay sources. However, the most reliable origin of information should be the dental and medical professions through daily contact with patients. The pregnant woman usually knows that infections may adversely affect her pregnancy. Individuals with diabetes generally know that infections impair glycemic control. However, many of these patients do not know that occult periodontal infections can have the same effect as more clinically evident infections. The dentist is responsible for diagnosing periodontal infections, providing appropriate treatment, and preventing disease recurrence or progression. Because many medical professionals are unfamiliar with the oral cavity and oral health research, dentists must reach out to the medical community to improve patient care through education and communication.[74] Likewise, patients must be educated in disease prevention. Just as patients know that lowering cholesterol levels may decrease their risk for heart disease, prevention of periodontal infection should be emphasized. A physician would be remiss if he or she did not provide education on decreasing cholesterol, losing weight, and ceasing a smoking habit to a patient at risk for CHD. Likewise, controlling the risk factor of periodontal infection requires the dentist to emphasize personal and professional preventive measures focused on thorough oral hygiene and regular recall.

SUMMARY

Does periodontal disease *cause* CHD, COPD, or adverse pregnancy outcomes? This question may only be answered based on the evidence currently available, with the full knowledge that conclusions may change as future evidence dictates. Periodontal disease may increase the risk for many systemic disorders. Biologically plausible mechanisms support the role of periodontal infection in these conditions, but periodontal infection should not be presented as the cause of such systemic diseases any more than cholesterol is said to cause heart disease. Periodontal infection is one of many potential risk factors for a number of systemic conditions. Fortunately, it is a readily modifiable risk factor, unlike age, gender, and genetic influences.

The focal infection theory of the early twentieth century was widely and appropriately discredited when treatment based on the theory, consisting almost solely of tooth extraction, had no effect on the underlying diseases that oral sepsis supposedly caused. Similarly, the clinical utility of our current knowledge base is only now evolving. Future research will further delineate the role of periodontal infection in systemic health. The associations between periodontal infection and conditions, such as LBW delivery, diabetes, cardiovascular and cerebrovascular diseases, and respiratory diseases, may be further substantiated. Longitudinal studies and intervention trials are needed before any causative role can be assigned.

The emerging field of periodontal medicine offers new insights into the concept of the oral cavity as one system interconnected with the whole human body. For many years the dental profession has recognized the effects of systemic conditions on the oral cavity. Only now, however, are dental professionals beginning to understand more fully the impact of the periodontium on systemic health.

Finally, it is important to appreciate the differences between the science and art of dental practice as it relates to periodontal medicine. Science is based, in general, on means and standard deviations (or standard errors). Thus science may determine that, on average, diabetic patients with periodontal disease have poorer glycemic control than diabetic patients who are periodontally healthy. However, the patient sitting in the dental chair at any given point in time may or may not be a "mean" patient, that is, she may demonstrate the same relationship that science has determined to exist as an average within the population, or she may not. She may lie somewhere within or outside the standard deviation. She may have extremely poor glycemic control that is directly related to her extensive periodontal inflammation, or she may have average glycemic control, or she may have good glycemic control. The clinical practice of periodontal medicine recognizes that while using an evidence-based approach is absolutely key to the modern dentistry, each patient is an individual and he or she may not always fit the average determined by science.

References and Suggested Reading can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 29

Oral Malodor

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CHAPTER OUTLINE

SEMANTICS
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Conversion of Volatile Sulfur Compounds
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SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some text, figures, references, and tables may be out of numeric order in this printed chapter.

SEMANTICS

Breath odor can be defined as the subjective perception after smelling someone's breath. It can be pleasant, unpleasant or even disturbing, if not repulsive. If unpleasant, the terms *breath malodor, halitosis, bad breath,* or *fetor ex ore* can be applied. These terms, however, are not synonymous with *oral malodor,* which has its origin in the oral cavity. This is not always the case for all breath malodors. The term "oral malodor" is thus too restrictive.

Breath malodor should not be confused with the momentarily disturbing odor caused by food intake (e.g., garlic, onions, and certain spices), smoking, or medication (e.g., metronidazole) because these odors do not reveal a health problem. The same is true for "morning" bad breath, as habitually experienced on awakening. This malodor is caused by a decreased salivary flow and increased putrefaction during the night and spontaneously disappears after breakfast or oral hygiene measures. A persistent breath malodor, by definition, does reflect some pathology.

Epidemiology

Breath malodor is a common complaint among the general population. It has a significant socioeconomic impact but was neglected until recently by scientists and clinicians and is hardly covered in the medical curricula. Halitosis can lead to personal discomfort and social embarrassment and is still one of the biggest taboos in society. Almost $1 billion a year is spent in the United States on deodorant-type mouth (oral) rinses, mints, and related over-the-counter products to manage bad breath.[56] It would be preferable to spend this money on a proper diagnosis and etiologic care instead of short-term and even inefficient masking attempts.

For more information on the epidemiology of oral malodor, please visit the companion Expert CONSULT website at www.expertconsult.com.

CLASSIFICATION

There are three main categories of halitosis: genuine halitosis, pseudo-halitosis, and halitophobia. *Genuine halitosis* is the term that is used when the breath malodor really exists and can be diagnosed organoleptically or by measurement of the responsible compounds. When an obvious breath malodor cannot be perceived, but the patient is convinced that he or she suffers from it, this is called *pseudo-halitosis.* If the patient still believes that there is bad breath after treatment of genuine halitosis or diagnosis of pseudo-halitosis, one considers halitophobia, which is a recognized psychiatric condition.[130]

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ETIOLOGY

In the vast majority, breath malodor originates from the oral cavity. Gingivitis, periodontitis, and especially tongue coating are the predominant causative factors.[20,78,79,131,132]

For more information on the etiology of oral malodor, please visit the companion Expert CONSULT website at www.expertconsult.com.

In general, one can identify two pathways for bad breath. The first one involves an increase of certain metabolites in the blood circulation (e.g., due to a systemic disease), which will escape via the alveoli of the lungs during breathing (blood-gas exchange). The second pathway involves an increase of either the bacterial load or the amount of substrates for these bacteria at one of the lining surfaces of the oropharyngeal cavity, the respiratory tract, or the esophagus. All types of infections, ulcerations, or tumors at one of the previously mentioned areas can thus lead to bad breath. The most commonly involved bacteria are *Porphyromonas gingivalis, Prevotella intermedia/nigrescens, Aggregatibacter actinomycetemcomitans* (previously *Actinobacillus actinomycetemcomitans*), *Campylobacter rectus, Fusobacterium nucleatum, Peptostreptococcus micros, Tannerella forsythia, Eubacterium* spp,, and spirochetes.

In a special patient category, subjects imagine they have breath malodor; this is called *imaginary breath odor* or *halitophobia.*[75] The latter has been associated with obsessive-compulsive disorders and hypochondria.

For more information on the etiology of oral malodor, please visit the companion Expert CONSULT website at www.expertconsult.com.

For oral malodor, the unpleasant smell of the breath mainly originates from VSCs, especially hydrogen sulfide (H_2S), methyl-mercaptan (CH_3SH), and less important dimethyl sulfide [$(CH_3)_2S$], as first discovered by Tonzetich.[117] However, in certain conditions (e.g., when the saliva dries out on the mucosal surfaces), other compounds in mouth air may also play a role such as diamines (e.g., putrescine, cadaverine), indole, skatole, and volatile organic acids like butyric or propionic acid.[33] Most of these compounds result from the proteolytic degradation by oral microorganisms of peptides present in saliva (sulfur-containing or non–sulfur-containing amino acids (Figure 29-1), shed epithelium, food debris, gingival crevicular fluid (GCF), interdental plaque, postnasal drip, and blood. In particular, gram-negative, anaerobic bacteria possess such proteolytic activity.

For the extraoral causes of halitosis, other compounds besides the VSCs may be involved, which have not all been identified yet.[50] Bad smelling metabolites can be formed/absorbed at any place in the body (e.g., the liver, the gut) and be transported by the bloodstream to the lungs. Exhalation of these volatiles in the alveolar air then causes halitosis, at least when the concentrations of the bad smelling metabolites are sufficiently high. The crevicular fluid reflects the circulating molecules in the blood and can thus also play a relevant role but due to the small amount probably not a very dominant one. The extraoral causes are much more difficult to detect, although they can sometimes be recognized by a typical odor. Uncontrolled diabetes mellitus can be associated with a sweet odor of ketones, liver disease can be revealed by a sulfur odor, and kidney failure can be characterized by a fishy odor because of the presence of dimethylamine and trimethylamine.[83]

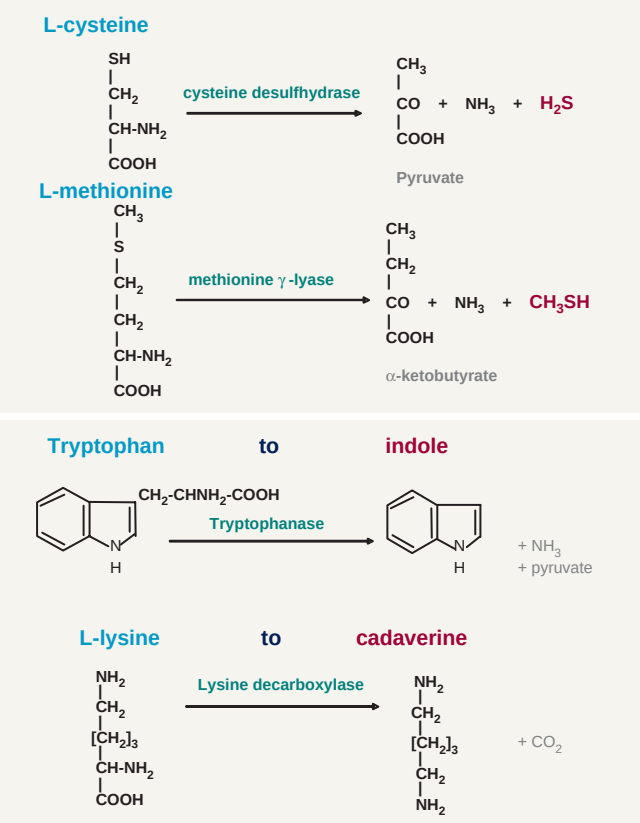

Figure 29-1 Proteolytic degradation by oral microorganisms of four amino acids (two containing sulfur and two not containing sulfur) to malodorous compounds.

Intraoral Causes

Tongue and Tongue Coating. The dorsal tongue mucosa, with an area of 25 cm^2, shows a very irregular surface topography.[16,108] The posterior part exhibits a number of oval cryptolymphatic units, which roughen the surface of this area. The anterior part is even rougher because of the high number of papillae: the filiform papillae with a core of 0.5 mm in length, a central crater and uplifted borders; the fungiform papillae, 0.5 to 0.8 mm in length; the foliate papillae, located at the edge of the tongue, separated by deep folds; and the vallate papillae, 1 mm in height and 2 to 3 mm in diameter. These innumerable depressions in the tongue surface are ideal niches for bacterial adhesion and growth, sheltered from cleaning actions.[18,131] Moreover, desquamated cells and food remnants also remain trapped in these retention sites and consequently can be putrefied by the bacteria.[5] A fissurated tongue (deep fissures on dorsum, also called *scrotal tongue* or *lingua plicata*) and a hairy tongue (lingua villosa) have an even rougher surface (Figure 29-2).

The accumulation of food remnants intermingled with exfoliated cells and bacteria causes a coating on the tongue dorsum. The latter cannot be easily removed because of the retention offered by the irregular surface of the tongue dorsum (see Figure 29-2). As such, the two factors essential for putrefaction are united. Several investigators have identified the dorsal posterior surface of the tongue as the primary source of breath malodor.[5,15,18,99] Indeed, high correlations have been reported between tongue coating and odor formation.[5,18,63,131]

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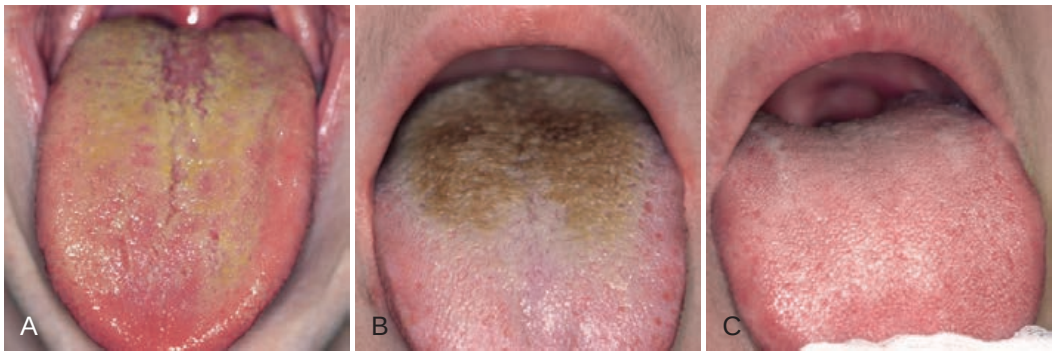

Figure 29-2 Different clinical pictures of heavily coated tongues.

Studies also suggest that oral malodor is associated with the total bacterial load of anaerobic bacteria in both saliva and tongue coating.[89]

Periodontal Infections. Several studies have shown a relationship between periodontitis and oral malodor. However, not all patients with gingivitis and/or periodontitis complain about bad breath, and there is some disagreement in the literature as to what extent oral malodor and periodontal disease are related.[5,98,110] Bacteria associated with gingivitis and periodontitis are indeed able to produce VSCs.*

For more information on periodontal infections, please visit the companion Expert CONSULT website at www.expertconsult.com.

Several studies have shown that the VSC levels in the mouth correlate positively with the depth of periodontal pockets (the deeper the pocket, the more bacteria, particularly anaerobic species) and that the amount of VSCs in breath increases with the number, depth, and bleeding tendency of the periodontal pockets.[15,77,132] VSCs aggravate the periodontitis process by increasing the permeability of the pocket and mucosal epithelium and therefore exposing the underlying connective tissues of the periodontium to bacterial metabolites. Moreover, methylmercaptan enhances interstitial collagenase production, interleukin-1 (IL-1) production by mononuclear cells, and cathepsin B production, thus further mediating connective tissue breakdown.[54,91] It was also shown that human gingival fibroblasts developed an affected cytoskeleton when exposed to methyl mercaptan.[8,91] The same gas-altered cell proliferation and migration. VSCs are also known to impede wound healing. Thus, when periodontal surgery is planned, especially the insertion of implants, clinicians should recognize this pathologic role of VSCs.

Some studies, however, have shown that when the presence of tongue coating is taken into account, the correlation between periodontitis and oral malodor is much lower, indicating that tongue coating remains a key factor for halitosis. The prevalence of tongue coating is six times higher in patients with periodontitis, and the same bacterial species associated with periodontal disease can also be found in large numbers on the dorsum of the tongue, particularly when tongue coating is present.[131] The reported association between periodontitis and oral malodor may thus primarily be due to the effects of periodontal disease on tongue coating. This theory is supported by a number of studies that have not found evidence for a strong direct association between periodontitis and risk of oral malodor.[5,110]

Other relevant malodorous pathologic manifestations of the periodontium are pericoronitis (the soft tissue "cap" being retentive for microorganisms and debris), major recurrent oral ulcerations, herpetic gingivitis, and necrotizing gingivitis/periodontitis. Microbiologic observations indicate that ulcers infected with gram-negative anaerobes (i.e., *Prevotella* and *Porphyromonas* species) are significantly more malodorous than noninfected ulcers.[6]

Dental Pathologies. Possible causes within the dentition are deep carious lesions with food impaction and putrefaction, extraction wounds filled with a blood clot, and purulent discharge leading to important putrefaction. Interdental food impaction in large interdental areas and crowding of teeth favor food entrapment and accumulation of debris. Acrylic dentures, especially when kept continuously in the mouth at night or not regularly cleaned, can also produce a typical smell. The denture surface facing the gingiva is porous and retentive for bacteria, yeasts, and debris, which are all factors that cause putrefaction.

Dry Mouth. Saliva has an important cleaning function in the oral cavity. Patients with xerostomia often present with large amounts of plaque on teeth and an extensive tongue coating. The increased microbial load and the escape of VSCs as gases when saliva is drying up explain the strong breath malodor.[46] Several studies link stress with VSC levels, but it is not clear whether this can simply be explained by a reduction of salivary flow.[52,84] Other causes of xerostomia are medication,[64] alcohol abuse,[28] Sjögren's syndrome (a common autoimmune rheumatic disease),[58] and diabetes.[127]

Extraoral Causes

Ear-Nose-Throat.

For more information on extraoral causes of oral malodor, please visit the companion Expert CONSULT website at www.expertconsult.com.

PHYSIOLOGY OF MALODOR DETECTION

The breath of a person contains up to 150 different molecules.[81,120,121] The characteristics of the expired molecules determine whether we can smell them or not. Some gases can cause a striking odor at very low concentrations, whereas others need to be present in much higher quantities. The perception of the molecules depends on the following factors[34]:

*References 46, 68, 70, 78, 79, and 117.

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TABLE 29-1 Odor Threshold and Odor Power of Key Malodorous Compounds

Compound	ODOR THRESHOLDS		ODOR POWER	VOLATILITY (HENRY'S CONSTANTS)
	Greenman* ($mol.dm^{-3}$)	Devos† ($mol.dm^{-3}$)	Greenman* (x increase to "up" score 1 unit)	Volatility (Kcc) in H_2O at 20° C
Butyrate	2.3×10^{-10}	2.4×10^{-10}	10-fold	3.9×10^{4}
Isovalerate	1.8×10^{-11}	2.5×10^{-11}	42-fold	2.5×10^{4}
Skatole	7.2×10^{-13}	1.0×10^{-12}	8-fold	4.1×10^{5}
Trimethylamine	1.8×10^{-11}	1.5×10^{-11}	96-fold (low affinity)	2.0×10^{2}
Putrescine	9.1×10^{-10}	1.0×10^{-9}	27-fold	4.1×10^{1}
CH_3SCH_3	5.9×10^{-8}	5.0×10^{-8}	10-fold	1.7×10^{1}
H_2S	6.4×10^{-10}	5.0×10^{-10}	4.8-fold	$\approx 1.7 \times 10^{1}$
CH_3SH	1.0×10^{-11}	1.0×10^{-11}	7.2-fold	$\approx 1.7 \times 10^{1}$

*Greenman J, Duffield J, Spencer P, et al: *J Dent Res* 83:81, 2004.
†Devos M, Patte F, Roualt J, et al: *Standardized human olfactory thresholds,* Oxford, 1990, IRL Press at Oxford University Press.
Kcc, Unit for Henry's Law constant.

TABLE 29-2 Odor Scores at the Back of the Hand

	Start	1 minute	3 minutes	10 minutes
Hydrogen sulfide	3	1	0	0
Methylmercaptan	3	1	0	0
Acetic acid	3	4	0	0
Propionic acid	3	4	0	0
Butyric acid	4	4	2	2
Valeric acid	4	4	3	2
Indole	4	4	4	2
Skatole	4	4	4	2
Putrescine	4	4	3	1
Cadaverine	4	4	2	1

1. The odor itself *(olfactory response)* can be pleasant, unpleasant, or even repulsive.
2. Each particular molecule has its specific concentration before it can be detected *(threshold concentration)*.
3. The *odor power* is the extent of concentration that is necessary to increase the odor score with one unit.
4. The *volatility* of the compound: malodorous molecules only express themselves when they become volatile.
5. The *substantivity:* the capacity of the molecule to stay present and thus to remain the cause of smell.

Table 29-1 provides an overview of these essential characteristics for the key malodorous compounds, clearly highlighting large interproduct variations. The odor power is the strongest for hydrogen sulfide and methylmercaptan; if the concentration of these products increases fivefold to tenfold, the odor will receive a higher organoleptic rating. For some other compounds, increases of 25 to 100 times are needed to reach a similar effect. Skatole and methyl mercaptan are detected at the lowest concentrations (low threshold). The three sulfur molecules have the lowest volatility (i.e., will escape the liquid phase first).[34]

In a study of Kleinberg and Codipilly, aqueous solutions of oral odoriferous volatiles were placed on the skin of the back of the hand.[46] Afterward, odor scores were given (organoleptic score, cfr. supra). The results are shown in Table 29-2. All metabolites caused an explicit odor, which decreased in intensity over time. Some molecules disappeared very fast (e.g., hydrogen sulfide and methylmercaptan), whereas others produced a bad smell for a longer period of time (e.g., indole and skatole, for 10 minutes and longer).

For more information on the physiology of malodor detection, please visit the companion Expert CONSULT website at www.expertconsult.com.

DIAGNOSIS OF MALODOR

Medical History

The proper diagnostic approach to a malodor patient starts with a thorough questioning about the medical history. Asking about all the relevant pathologies for breath malodor just discussed is not time-consuming; it may save time and expenses to achieve a proper differential diagnosis. As often repeated, "listen to the patient and the patient will tell you the diagnosis." It should take place at the clinician's desk in private and before any clinical examination (not in a dental chair). This will encourage the proper confidence needed for these patients. The patient's history should be discretely and intermittently noted. The clinician should ask about the frequency (e.g., every month), the time of appearance during the day (e.g., after meals can indicate a stomach hernia), the time when the problem first appeared, whether others (nonconfidants) have identified the problem (to exclude imaginary breath odor), which medications are taken, and whether there are possible contributing factors such as mouth breathing, dry mouth, allergies, and nasal problems.

Clinical and Laboratory Examination

Self-Examination. It can be worthwhile to involve the patient in monitoring the results of therapy by self-examination, especially when an intraoral cause has been identified. For example, this can motivate the patient to continue the oral hygiene instructions. The following self-testing can be used:

- Smelling a metallic or nonodorous plastic spoon after scraping the back of the tongue.
- Smelling a toothpick after introducing it in an interdental area.

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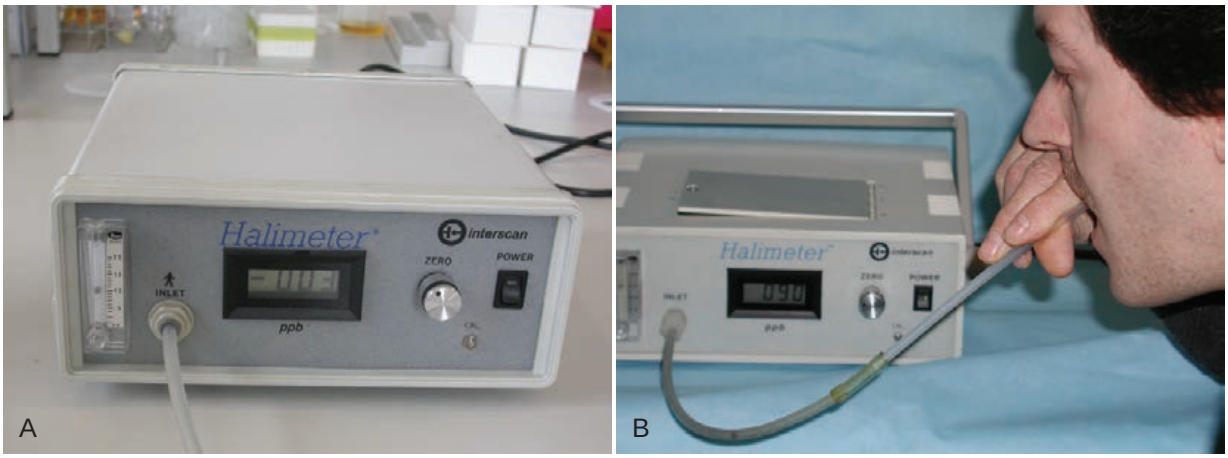

Figure 29-4 **A,** Portable sulfide monitor. **B,** Patient holds the aspirating tube in his mouth. Digital reading reveals the total amount of volatile sulfur compounds (VSCs) in parts per billion (ppb).

- Smelling saliva spit in a small cup or spoon (especially when allowed to dry for a few seconds so that putrefaction odors can escape from the liquid).
- Licking the wrist and allowing it to dry (reflects the saliva contribution to malodor).

Removing the odorous substances from the body allows a less emotional and thus more objective assessment. Smelling one's own breath by expiring in the hands kept in front of the mouth is not relevant because the nose gets used to the odor and the smell of the skin and soaps used for handwashing may interfere. Moreover, studies have shown that self-assessment of oral malodor is notoriously unreliable and one should be careful with the information obtained from the patient.[23]

Oropharyngeal Examination. The oropharyngeal examination includes inspection of deep carious lesions, interdental food impaction, wounds, bleeding of the gums, periodontal pockets, tongue coating, dry mouth, and the tonsils and pharynx (for tonsillitis and pharyngitis).

Organoleptic Rating. Even though devices are available, the organoleptic assessment by a judge is still the "gold standard" in the examination of breath malodor. It is the easiest and most often used method because it gives a reflection of the everyday situation when halitosis is noticed. Moreover, the human nose can smell 10,000 different odors.[39] In an organoleptic evaluation, a trained and preferably calibrated "judge" sniffs the expired air and assesses whether it is unpleasant by using an intensity rating, normally from 0 to 5, as proposed by Rosenberg and McCulloch.[101] It is thus solely based on the olfactory organs of the clinician: 0 = no odor present, 1 = barely noticeable odor, 2 = slight but clearly noticeable odor, 3 = moderate odor, 4 = strong offensive odor, and 5 = extremely foul odor.

For more information on the organoleptic rating and to view Figure 29-3, please visit the companion Expert CONSULT website at www.expertconsult.com.

Portable Volatile Sulfur Monitor. The portable volatile sulfur monitor (Halimeter, Interscan, Chatsworth, CA) is an electronic device that analyzes the concentration of hydrogen sulfide and methyl mercaptan but without discriminating them (Figure 29-4). The mouth air is aspirated by inserting a drinking straw fixed on the flexible tube of the instrument. The straw is kept about 2 cm behind the lips, without touching any surface, while the subject keeps the mouth slightly open and breathes through the nose. The sulfur meter uses a voltametric sensor that generates a signal when exposed to sulfur-containing gases.

Figure 29-5 Gas chromatography machinery, including thermal desorber *(TD)* to release molecules trapped in special collectors); gas chromatograph (*GC)* for separation of molecules; and mass spectrometer (*MS)* for identification of molecules.

For more information on the Portable Volatile Sulfur Monitor, please visit the companion Expert CONSULT website at www.expertconsult.com.

Gas Chromatography. A gas chromatography device can analyze air, saliva, or crevicular fluid (Figure 29-5). About 100 compounds have been isolated from the headspace of saliva and tongue coating, from ketones to alkanes and sulfur-containing compounds to phenyl compounds.[14] In the expired air of a person, approximately 150 compounds can be found.[81,120] The most important advantage of the technique (together with mass spectrometry) is that it can detect virtually any compound when using adequate materials and conditions. Moreover, it has a very high sensitivity and specificity.

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For more information on gas chromatography and to view Figure 29-6, please visit the companion Expert CONSULT website at www.expertconsult.com.

Dark-Field or Phase-Contrast Microscopy. Gingivitis and periodontitis are typically associated with a higher incidence of motile organisms and spirochetes, so shifts in these proportions allow monitoring of therapeutic progress. Another advantage of direct microscopy is that the patient becomes aware of bacteria being present in plaque, tongue coating, and saliva. Too often, patients confuse plaque with food remnants.

Saliva Incubation Test. The analysis of the headspace above incubated saliva by gas chromatography reveals next to VSCs also other compounds like *indole, skatole,* lactic acid, *methylamine, diphenylamine, cadaverine,* putrescine, urea, ammonia, *dodecanol,* and *tetradecanol.* By adding some proteins, such as lysine or cysteine, the production of respectively cadaverine or hydrogen sulfide is dramatically increased. Organoleptic evaluation (or assessment of the VSCs) of the saliva headspace offers promising perspectives for monitoring treatment results.[85] It is a less invasive test, especially for the patient, than smelling breath in front of the oral cavity.

Electronic Nose. Electronic noses identify the specific components of an odor and analyze its chemical makeup. They consist of a mechanism for chemical detection, such as an array of electronic sensors, and a mechanism for pattern recognition. They are smaller, less expensive, and easier to use than for example gas chromatography but can only be developed for specific applications if the important metabolites are already known. An artificial nose that has the same capacities as the human nose would be ideal. Currently, although significant improvements still need to be made, the first trials thus far have been promising.[115]

TREATMENT OF ORAL MALODOR

The treatment of oral malodor (thus of intraoral origin) should preferably be cause related. Because oral malodor is caused by the metabolic degradation of available proteins to malodorous gases by certain oral microorganisms, the following general treatment strategies can be applied:

- Mechanical reduction of intraoral nutrients (substrates) and microorganisms
- Chemical reduction of oral microbial load
- Rendering malodorous gases nonvolatile
- Masking the malodor

Treatment should be centered on reducing the bacterial load/micronutrients by effective mechanical oral hygiene procedures, including tongue scraping. Periodontal disease should be treated and controlled, and as an auxiliary aid, oral rinses containing chlorhexidine and other ingredients may further reduce the oral malodor. If breath malodor persists after these approaches, other sources of the malodor, such as the tonsils, lung disease, gastrointestinal disease, and metabolic abnormalities (e.g., diabetes) should be investigated.

Issues related to oral malodor emphasize the clinician's need for good diagnostic skills and stipulate an appreciation of chemistry. First, the etiology of breath malodor can be from a large variety of intraoral and extraoral causes, and much can be gained from a careful patient history before any oral or extraoral examination. Second, knowledge of the potential volatile substances and gases (e.g., VSCs) and their formation, sources, power, substantivity, and dilution capacity allows for treatment options and rational therapeutic interventions. Furthermore, this knowledge allows the dental clinician to help predict the outcome of therapeutic interventions, such as the short-term effectiveness of masking oral malodor and the longer-term effect of mechanically and chemically reducing the oral microbial load.

Mechanical Reduction of Intraoral Nutrients and Microorganisms

Because of the extensive accumulation of bacteria on the dorsum of the tongue, tongue cleaning should be emphasized.[12,105,131] Previous investigations demonstrated that tongue cleaning reduces both the amount of coating (and thus bacterial nutrients), as well as the number of bacteria and thereby improves oral malodor effectively.[18,31,32,37,90] Other reports indicated that the reduction of the microbial load on the tongue after cleaning is negligible and that the malodor reduction probably results from the reduction of the bacterial nutrients.[60,89]

Cleaning of the tongue can be carried out with a normal toothbrush, but preferably with a tongue scraper if a coating is established.[74,76] Tongue cleaning using a tongue scraper reduced the halitosis levels with 75% after 1 week.[76] This should be gentle cleaning to prevent soft tissue damage. It is best to clean as far backward as possible; the posterior portion of the tongue has the most coating.[100] Tongue cleaning should be repeated until almost

SCIENCE TRANSFER

The most common cause of oral malodor is bacterial metabolism in the oral cavity. Many of the foul-smelling molecules come from the anaerobic metabolism of proteins with subsequent increases in volatile sulfur-containing compounds (VSC) including hydrogen sulfide. The bacteria present in the biofilm attached to teeth, particularly those seen in the subgingival plaque of patients, play a major role in VSC production. Bacterial coating of the tongue are also important contributors to oral malodor.

Much of oral malodor can be controlled by periodontal therapy aimed at plaque removal and pocket reduction, coupled with an effective patient centered daily oral hygiene routine that includes toothbrushing and tongue scraping. The use of antimicrobal mouthwashes, such as those containing chlorhexidine, is also an important contributor to control of oral malodor.

Although electronic devices can detect some sulfur-containing compounds and gas chromotography can give an even wider and accurate measurement of up to 150 compounds, the use of the clinician's nose to rate oral malodor on a scale of 0 to 5 is probably all that is needed to diagnose and treat oral malodor. Patients are generally unreliable in detecting oral malodor in their own mouths and so clinicans and friends and family can play a role in giving feedback on the status of bad breath.

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no coating material can be removed.[13] Gagging reflexes often are elicited, especially when using brushes[89]; practice helps to prevent this.[11] It can also be helpful to pull the tongue out with a gauze pad. Tongue cleaning has the additional benefit of improving taste sensation.[89,129]

Interdental cleaning and toothbrushing are essential mechanical means of dental plaque control. Both remove residual food particles and organisms that cause putrefaction. Clinical studies have shown that exclusively brushing the teeth has no appreciable influence on the concentration of VSCs.[114] In a short-term study, a combination of tooth and tongue brushing or toothbrushing alone had a beneficial effect on bad breath for up to 1 hour (73% and 30% reduction in VSCs, respectively).[118]

Because periodontitis can cause chronic oral malodor, professional periodontal therapy is needed.[5,15,77,132] A one-stage, full-mouth disinfection, combining scaling and root planing with the application of chlorhexidine, reduced the organoleptic malodor levels up to 90%.[87] In a recent study of the same authors, initial periodontal therapy had only a weak impact on the VSC levels, except when combined with a mouthrinse containing chlorhexidine.[85]

Chewing gum may control bad breath temporarily because it can stimulate salivary flow.[93] The salivary flow itself also has a mechanical cleaning capability. Not surprisingly, therefore, subjects with extremely low salivary flow rate have higher VSC ratings and tongue coating scores than those with normal saliva production.[49] Waler[128] showed that chewing of a gum without any active ingredient can reduce halitosis modestly.

On the other hand, clinicians can use bacterial nutrients to provoke malodor, to prove an intraoral origin of the bad breath for example, or to test the efficacy of different oral rinses. Rinsing with amino acids (e.g., cysteine challenge test) can result in a dramatic increase in hydrogen sulfide[47] (see Figure 29-1).

Chemical Reduction of Oral Microbial Load

Mouth rinsing has become a common practice in patients with oral malodor.[29] The active ingredients in oral rinses are usually antimicrobial agents such as chlorhexidine, cetylpyridinium chloride (CPC), essential oils, chlorine dioxide, hydrogen peroxide, and triclosan. All these agents have only a temporary reducing effect on the total number of microorganisms in the oral cavity.

Chlorhexidine. Chlorhexidine is considered the most effective antiplaque and antigingivitis agent.[1-4,43] Its antibacterial action can be explained by disruption of the bacterial cell membrane by the chlorhexidine molecules, increasing its permeability and resulting in cell lysis and death.[43,53] Because of its strong antibacterial effects and superior substantivity in the oral cavity, chlorhexidine rinsing provides significant reduction in VSC levels and organoleptic ratings.[9,102,103,126,134]

For more information on chlorhexidine, please visit the companion Expert CONSULT website at www.expertconsult.com.

Unfortunately, as mentioned in some trials, chlorhexidine at concentrations ≥0.2% also has some disadvantages such as the increased tooth and tongue staining, bad taste, and some temporary reduction in taste sensation.[25]

Essential Oils. Previous studies evaluated the short-term effect (3 hours) of a Listerine rinse (which contains essential oils) compared with a placebo rinse.[82] Listerine was found to be only moderately effective against oral malodor (±25% reduction versus 10% for placebo of VSCs after 30 minutes) and caused a sustained reduction in the levels of odorigenic bacteria. Similar VSC reductions were found after rinsing for 4 days.[9]

Chlorine Dioxide. Chlorine dioxide (ClO_2) is a powerful oxidizing agent that can eliminate bad breath by oxidation of hydrogen sulfide, methylmercaptan, and the amino acids, methionine and cysteine. Studies demonstrated that single use of a ClO_2–containing oral rinse slightly reduces mouth odor.[26,27]

Two-Phase Oil-Water Rinse. Rosenberg et al[102] designed a two-phase oil-water rinse containing CPC. The efficacy of oil-water-CPC formulations is thought to result from the adhesion of a high proportion of oral microorganisms to the oil droplets, which is further enhanced by the CPC. A twice-daily rinse with this product (before bedtime and in the morning) showed reductions in both VSC levels and organoleptic ratings. These reductions were superior to Listerine and significantly superior to a placebo.[51,102]

Triclosan. Triclosan, a broad-spectrum antibacterial agent, has been found to be effective against most oral bacteria and has a good compatibility with other compounds used for oral home care. A pilot study demonstrated that an experimental mouth rinse containing 0.15% triclosan and 0.84% zinc produced a stronger and more prolonged reduction in mouth odor than a Listerine rinse.[92] The anti-VSC effect of triclosan, however, seems strongly dependent on the solubilizing agents.[133]

Aminefluoride/Stannous Fluoride. The association of aminefluoride with stannous fluoride (AmF/SnF_2) resulted in encouraging reductions of morning breath odor, even when oral hygiene is insufficient.[88]

Hydrogen Peroxide. Suarez et al[114] reported that rinsing with 3% hydrogen peroxide (H_2O_2) produced impressive reductions (±90%) in sulfur gases that persisted for 8 hours.

Oxidizing Lozenges. Greenstein et al[36] reported that sucking a lozenge with oxidizing properties reduces tongue dorsum malodor for 3 hours. This antimalodor effect may be caused by the activity of dehydroascorbic acid, which is generated by peroxide-mediated oxidation of ascorbate present in the lozenges.

Conversion of Volatile Sulfur Compounds

Metal Salt Solutions. Metal ions with affinity for sulfur are efficient in capturing the sulfur-containing gases. Zinc is an ion with two positive charges (Zn^{++}), which will bind to the twice–negatively loaded sulfur radicals, and thus can reduce the expression of the VSCs. The same applies for other metal ions such as mercury and copper. Clinically, the VSC inhibitory effect was CuCl2 > SnF2 > ZnCl2. In vitro, the inhibitory effect was $HgCl_2 = CuCl_2 = CdCl_2 > ZnCl_2 > SnF_2 > SnCl_2 > PbCl_2$.[135]

Compared with other metal ions, Zn^{++} is relatively nontoxic and noncumulative and gives no visible discoloration. Thus, Zn^{++} has been one of the most-studied ingredients for the control of oral malodor.[128,135] Schmidt and Tarbet[107] already reported that a rinse containing zinc chloride was remarkably more effective than a saline rinse (or no treatment) in reducing the levels of both VSCs (±80% reduction) and organoleptic scores (±40% reduction) for 3 hours.

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As mentioned, Halita, a rinse containing 0.05% chlorhexidine, 0.05% CPC and 0.14% zinc lactate, has been even more efficient than a 0.2% chlorhexidine formulation in reducing the VSC levels and organoleptic ratings.[86,126] The special effect of Halita may result from the VSC conversion ability of zinc, besides its antimicrobial action. The combination Zn^{++} and chlorhexidine seems to act synergistically.[134]

Toothpastes. Baking soda dentifrices have been shown to confer a significant odor-reducing benefit for time periods up to 3 hours.[7,70] The mechanisms by which baking soda produces its inhibition of oral malodor might be related to its bactericidal effects and its transformation of VSCs to a nonvolatile state.

Gerlach et al[30] compared the antimalodor efficacy of three different toothpastes and reported a slightly better outcome, especially for an SnF_2-containing paste (±50% reduction), when compared to water (±35% reduction). In a study by Hoshi and van Steenberghe,[40] a zinc citrate/triclosan toothpaste applied to the tongue dorsum appeared to control morning breath malodor for 4 hours. If the flavor oil was removed, however, the antimalodor efficacy of the active ingredients decreased. Another clinical study reported an up to 41% reduction in VSC levels after 7 days' use of a dentifrice containing triclosan and a copolymer, but the benefit compared with a placebo was relatively small (17% reduction).[71] Similar reductions were also found in two other more recent studies.[41,72]

Chewing Gum. Chewing gum can be formulated with antibacterial agents, such as fluoride or chlorhexidine, thus helping reduce oral malodor through both mechanical and chemical approaches. Tsunoda et al[119] investigated the beneficial effect of chewing gum containing tea extracts for its deodorizing mechanism. *Epigallocatechin* (EGCg) is the main deodorizing agent among the tea catechins. The chemical reaction between EGCg and CH_3SH results in a nonvolatile product. Waler[128] compared different concentrations of zinc in a chewing gum and found that a 2-mg Zn^{++} acetate–containing chewing gum that remained in the mouth for 5 minutes resulted in an immediate reduction in the VSC levels of up to 45%, but the long-term effect was not mentioned.

Masking the Malodor

Treatments with rinses, mouth sprays, and lozenges containing volatiles with a pleasant odor have only a short-term effect.[93,94] Typical examples are the mint-containing lozenges.

Another pathway is to increase the solubility of malodorous compounds in the saliva by increasing the secretion of saliva; a larger volume allows the retention of larger volumes of soluble VSCs.[48] The latter can also be achieved by ensuring a proper liquid intake or by using a chewing gum; chewing triggers the periodontal-parotid reflex, at least when the lower (pre)molars are still present.

SUMMARY

Breath malodor has important socioeconomic consequences and can reveal important diseases. A proper diagnosis and determination of the etiology allow initiation of the proper etiologic treatment. Although tongue coating and (less frequently) periodontitis and gingivitis are by far the most common causes of malodor, a clinician cannot take the risk of overlooking other, more challenging diseases. This can be done by a multidisciplinary consultation or if this is not feasible a trial therapy to deal quickly with intraoral causes (e.g., the full-mouth one-stage disinfection, including the use of the proper mouthrinses, tongue scrapers, and toothpastes). For more detailed information, the reader is encouraged to consult van Steenberghe[124] and recent review papers.[96,106,125]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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PART 6

Treatment of Periodontal Disease

Henry H. Takei

Periodontal treatment requires an interrelationship between the care of the periodontium and other phases of dentistry. The concept of total treatment is based on the elimination of gingival inflammation and the factors that lead to it (e.g., plaque accumulation favored by calculus and pocket formation, inadequate restorations, and areas of food impaction).

Total treatment requires consideration of systemic aspects, including the possibility of interaction of periodontal disease with other diseases, systemic adjuncts to local treatment, and special precautions in patient management necessitated by systemic conditions. It may also entail consideration of functional aspects for the establishment of optimal occlusal relationships for the entire dentition.

All these aspects are embodied in a master plan, which consists of a rational sequence of dental procedures that includes periodontal and other measures necessary to create a well-functioning dentition in a healthy periodontal environment.

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CHAPTER 30

Clinical Diagnosis

Henry H. Takei and Fermin A. Carranza

CHAPTER OUTLINE

FIRST VISIT
- Overall Appraisal of the Patient
- Medical History
- Dental History
- Intraoral Radiographic Survey
- Casts
- Clinical Photographs
- Review of the Initial Examination

SECOND VISIT
- Oral Examination
- Examination of the Teeth and Implants
- Examination of the Periodontium

LABORATORY AIDS TO CLINICAL DIAGNOSIS (online only)

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Proper diagnosis is essential to intelligent treatment. Periodontal diagnosis should first determine whether disease is present; then identify its type, extent, distribution, and severity; and finally provide an understanding of the underlying pathologic processes and its cause. Part 3, Sections 1 and 2 provide a detailed description of the different diseases that can afflict the periodontium. In general, they fall into the following three broad categories.

1. The gingival diseases (Box 30-1)
2. The various types of periodontitis (Table 30-1)
3. The periodontal manifestations of systemic diseases (see Chapter 27)

Periodontal diagnosis is determined after careful analysis of the case history and evaluation of the clinical signs and symptoms, as well as the results of various tests (e.g., probing, mobility assessment, radiographs, blood tests, and biopsies).

The interest should be in the patient who has the disease and not simply in the disease itself. Diagnosis must therefore include a general evaluation of the patient and consideration of the oral cavity.

Diagnostic procedures must be systematic and organized for specific purposes. It is not enough to assemble facts. The findings must be pieced together so that they provide a meaningful explanation of the patient's periodontal problem. The following is a recommended sequence of procedures for the diagnosis of periodontal diseases.

FIRST VISIT

Overall Appraisal of the Patient

From the first meeting, the clinician should attempt an overall appraisal of the patient. This includes consideration of the patient's mental and emotional status, temperament, attitude, and physiologic age.

Medical History

Most of the medical history is obtained at the first visit and can be supplemented by pertinent questioning at subsequent visits. The health history can be obtained verbally by questioning the patient and recording his or her responses on a blank piece of paper or by means of a printed questionnaire the patient completes. Figure 30-1 is the medical questionnaire form recommended by the American Dental Association.

The importance of the medical history should be clearly explained because patients often omit information that they cannot relate to their dental problems. The patient should be made aware of (1) the possible role that some systemic diseases, conditions, or behavioral factors may play in the cause of periodontal disease; (2) the presence of conditions that may require special precautions or modifications in the treatment procedure (see Chapter 37); and (3) the possibility that oral infections may have a powerful influence

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TABLE 30-1 Features of Types of Periodontitis

Parameter	Chronic	Aggressive	Prepubertal	LAP	NUP
Age (years)	35+	20–35	<11	11–19	15–35
Calculus	Moderate to abundant	Scanty to moderate	Scanty	Moderate	Scanty
Disease progression	Slow	Rapid	Rapid	Rapid	Rapid
Distribution	Generalized; associated with etiologic factors	Generalized; no consistent pattern	Primary molars and incisors	First molars and incisors, and no more than two other teeth	?
Prevalence	US: >50% Sri Lanka: 81%	US: 4%-5% Sri Lanka: 11%	?	<0.50%	?
Racial predilection	No	No	No	More common in blacks	No
Familial tendency	No	?	Yes	Yes	?
Gender distribution	More severe in men	?	?	?	?
PMN/macrophage defects	No	Yes	Yes	Yes	Yes
Association with systemic problems	No	Some cases	Yes	Yes	Yes
Response to therapy	Very good	Variable	Poor	Good	Variable

Prevalence in adolescents 14 to 17 years of age in the United States.
LAP, Localized aggressive periodontitis (formerly "localized juvenile periodontitis" [LJP]); *NUP,* necrotizing ulcerative periodontitis; *PMN,* polymorphonuclear leukocyte; *US,* United States.

BOX 30-1 Gingival Diseases

Chronic marginal gingivitis
Acute necrotizing ulcerative gingivitis
Acute herpetic gingivostomatitis
Allergic gingivitis
Gingivitis associated with skin diseases
Gingivitis associated with endocrine-metabolic disturbances
Gingivitis associated with hematologic-immunologic disturbances
Gingival enlargement associated with medications
Gingival tumors

on the occurrence and severity of a variety of systemic diseases and conditions (see Chapter 28)

The medical history should include reference to the following:

1. Is the patient under the care of a physician and if so, what is the nature and duration of the problem and the therapy? The name, address, and telephone number of the physician should be recorded, since direct communication with him or her may be necessary.
2. Details on hospitalizations and operations, including diagnosis, kind of operation, and untoward events, such as anesthetic, hemorrhagic, or infectious complications, should be provided.
3. A list of all medications being taken and whether they were prescribed or obtained over-the-counter. All the possible effects of these medications should be carefully analyzed to determine their effect, if any, on the oral tissues and also to avoid administering medications that would interact adversely with them. Special inquiry should be made regarding the dosage and duration of therapy with anticoagulants and corticosteroids. Patients taking the family of drugs called bisphosphanates (e.g., Actonel, Fosamax, Boniva, Aredia, and Zometa), which are often prescribed for patients with osteoporosis, should be cautioned of possible problems related to osteonecrosis of the jaw after undergoing any form of oral surgery involving the bone.
4. History should be taken of all medical problems (cardiovascular, hematologic, endocrine, etc), including infectious diseases, sexually transmitted diseases, and high-risk behavior for human immunodeficiency virus (HIV) infection. Chapter 19 discusses the subject of HIV infections.
5. Any possibility of occupational disease should be noted.
6. Abnormal bleeding tendencies, such as nosebleeds, prolonged bleeding from minor cuts, spontaneous ecchymoses, tendency toward excessive bruising, and excessive menstrual bleeding, should be cited. These symptoms should be correlated with the medications the patient is taking.
7. History of allergy should be taken, including hay fever, asthma, sensitivity to foods, or sensitivity to drugs, such as aspirin, codeine, barbiturates, sulfonamides, antibiotics, procaine, and laxatives, and to dental materials such as eugenol or acrylic resins.
8. Information is needed regarding the onset of puberty and for females, menopause, menstrual disorders, hysterectomy, pregnancies, and miscarriages.
9. Family medical history should be taken, including bleeding disorders and diabetes.

Dental History

Current Illness. Some patients may be unaware of any problems, but many may report bleeding gums; loose teeth; spreading of the teeth with the appearance of spaces where none existed before; foul taste in the mouth; and an itchy feeling in the gums, relieved by digging with a toothpick. There may also be pain of varied types and duration, including constant, dull, gnawing pain; dull pain after eating; deep, radiating pains in the jaws; acute throbbing pain; sensitivity when chewing; sensitivity to hot and cold; burning sensation in the gums; and extreme sensitivity to inhaled air.

A preliminary oral examination is done to explore the source of the patient's chief complaint and to determine whether immediate emergency care is required. If this is the case, the problem is addressed after consideration of the medical history (see Chapters 41 and 42).

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Health History Form

ADA
American Dental Association
www.ada.org

E-mail: Today's Date:

As required by law, our office adheres to written policies and procedures to protect the privacy of information about you that we create, receive or maintain. Your answers are for our records only and will be kept confidential subject to applicable laws. Please note that you will be asked some questions about your responses to this questionnaire and there may be additional questions concerning your health. This information is vital to allow us to provide appropriate care for you. This office does not use this information to discriminate.

Name: (Last First Middle) | Home Phone: *Include area code* ( ) | Business/Cell Phone: *Include area code* ( )

Address: (Mailing address) | City: | State: | Zip:

Occupation: | Height: | Weight: | Date of birth: | Sex: M F

SS# or Patient ID: | Emergency Contact: | Relationship: | Home Phone: ( ) | Cell Phone: ( ) *Include area codes*

If you are completing this form for another person, what is your relationship to that person?

Your Name Relationship

Do you have any of the following diseases or problems: *(Check DK if you Don't Know the answer to the question)*	Yes	No	DK
Active tuberculosis	☐	☐	☐
Persistent cough greater than a 3 week duration	☐	☐	☐
Cough that produces blood	☐	☐	☐
Been exposed to anyone with tuberculosis	☐	☐	☐

If you answer yes to any of the 4 items above, please stop and return this form to the receptionist.

Dental Information *For the following questions, please mark (X) your responses to the following questions.*

	Yes	No	DK
Do your gums bleed when you brush or floss?	☐	☐	☐
Are your teeth sensitive to cold, hot, sweets or pressure?	☐	☐	☐
Does food or floss catch between your teeth?	☐	☐	☐
Is your mouth dry?	☐	☐	☐
Have you had any periodontal (gum) treatments?	☐	☐	☐
Have you ever had orthodontic (braces) treatment?	☐	☐	☐
Have you had any problems associated with previous dental treatment?	☐	☐	☐
Is your home water supply fluoridated?	☐	☐	☐
Do you drink bottled or filtered water?	☐	☐	☐
If yes, how often? Circle one: DAILY / WEEKLY / OCCASIONALLY			
Are you currently experiencing dental pain or discomfort?	☐	☐	☐

	Yes	No	DK
Do you have earaches or neck pains?	☐	☐	☐
Do you have any clicking, popping or discomfort in the jaw?	☐	☐	☐
Do you brux or grind your teeth?	☐	☐	☐
Do you have sores or ulcers in your mouth?	☐	☐	☐
Do you wear dentures or partials?	☐	☐	☐
Do you participate in active recreational activities?	☐	☐	☐
Have you ever had a serious injury to your head or mouth?	☐	☐	☐

Date of your last dental exam:
What was done at that time?

Date of last dental x-rays:

What is the reason for your dental visit today?

How do you feel about your smile?

Medical Information *Please mark (X) your response to indicate if you have or have not had any of the following diseases or problems.*

	Yes	No	DK
Are you now under the care of a physician?	☐	☐	☐

Physician Name: Phone: *Include area code* ( )

Address/City/State/Zip:

Are you in good health?	☐	☐	☐
Has there been any change in your general health within the past year?	☐	☐	☐

If yes, what condition is being treated?

Date of last physical exam:

	Yes	No	DK
Have you had a serious illness, operation or been hospitalized in the past 5 years?	☐	☐	☐

If yes, what was the illness or problem?

Are you taking or have you recently taken any prescription or over the counter medicine(s)?	☐	☐	☐

If so, please list all, including vitamins, natural or herbal preparations and/or diet supplements:

_____
_____
_____
_____

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Form S500

SAMPLE

Figure 30-1 Medical history form from the American Dental Association.

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Medical Information

Please mark (X) your response to indicate if you have or have not had any of the following diseases or problems.

(Check DK if you Don't Know the answer to the question)	Yes	No	DK
Do you wear contact lenses?	☐	☐	☐
Joint Replacement. Have you had an orthopedic total joint (hip, knee, elbow, finger) replacement?	☐	☐	☐
Date: _____ If yes, have you had any complications?_____			
Are you taking or scheduled to begin taking either of the medications, alendronate (Fosamax®) or risedronate (Actonel®) for osteoporosis or Paget's disease?	☐	☐	☐
Since 2001, were you treated or are you presently scheduled to begin treatment with the intravenous bisphosphonates (Aredia® or Zometa®) for bone pain, hypercalcemia or skeletal complications resulting from Paget's disease, multiple myeloma or metastatic cancer?	☐	☐	☐
Date Treatment began: _____			

	Yes	No	DK
Do you use controlled substances (drugs)?	☐	☐	☐
Do you use tobacco (smoking, snuff, chew, bidis)?	☐	☐	☐
If so, how interested are you in stopping? (Circle one) VERY / SOMEWHAT / NOT INTERESTED			
Do you drink alcoholic beverages?	☐	☐	☐
If yes, how much alcohol did you drink in the last 24 hours? _____			
If yes, how much do you typically drink In a week? _____			
WOMEN ONLY Are you:			
Pregnant?	☐	☐	☐
Number of weeks: _____			
Taking birth control pills or hormonal replacement?	☐	☐	☐
Nursing?	☐	☐	☐

Allergies - Are you allergic to or have you had a reaction to: To all **yes** responses, specify type of reaction.	Yes	No	DK
Local anesthetics	☐	☐	☐
Aspirin	☐	☐	☐
Penicillin or other antibiotics	☐	☐	☐
Barbiturates, sedatives, or sleeping pills	☐	☐	☐
Sulfa drugs	☐	☐	☐
Codeine or other narcotics	☐	☐	☐
Metals	☐	☐	☐
Latex (rubber)	☐	☐	☐
Iodine	☐	☐	☐
Hay fever/seasonal	☐	☐	☐
Animals	☐	☐	☐
Food	☐	☐	☐
Other	☐	☐	☐

Please mark (X) your response to indicate if you have or have not had any of the following diseases or problems.

	Yes	No	DK
Artificial (prosthetic) heart valve	☐	☐	☐
Previous infective endocarditis	☐	☐	☐
Damaged valves in transplanted heart	☐	☐	☐
Congenital heart disease (CHD)			
Unrepaired, cyanotic CHD	☐	☐	☐
Repaired (completely) in last 6 months	☐	☐	☐
Repaired CHD with residual defects	☐	☐	☐

Except for the conditions listed above, antibiotic prophylaxis is no longer recommended for any other form of CHD.

	Yes	No	DK
Cardiovascular disease.	☐	☐	☐
Angina	☐	☐	☐
Arteriosclerosis	☐	☐	☐
Congestive heart failure	☐	☐	☐
Damaged heart valves	☐	☐	☐
Heart attack	☐	☐	☐
Heart murmur	☐	☐	☐
Low blood pressure	☐	☐	☐
High blood pressure	☐	☐	☐
Other congenital heart defects	☐	☐	☐
Mitral valve prolapse	☐	☐	☐
Pacemaker	☐	☐	☐
Rheumatic fever	☐	☐	☐
Rheumatic heart disease	☐	☐	☐
Abnormal bleeding	☐	☐	☐
Anemia	☐	☐	☐
Blood transfusion	☐	☐	☐
If yes, date:_____			
Hemophilia	☐	☐	☐
AIDS or HIV infection	☐	☐	☐
Arthritis	☐	☐	☐
Autoimmune disease	☐	☐	☐
Rheumatoid arthritis	☐	☐	☐
Systemic lupus erythematosus.	☐	☐	☐
Asthma	☐	☐	☐
Bronchitis	☐	☐	☐
Emphysema	☐	☐	☐
Sinus trouble	☐	☐	☐
Tuberculosis	☐	☐	☐
Cancer/Chemotherapy/ Radiation Treatment	☐	☐	☐
Chest pain upon exertion	☐	☐	☐
Chronic pain	☐	☐	☐
Diabetes Type I or II	☐	☐	☐
Eating disorder	☐	☐	☐
Malnutrition	☐	☐	☐
Gastrointestinal disease	☐	☐	☐
G.E. Reflux/persistent heartburn	☐	☐	☐
Ulcers	☐	☐	☐
Thyroid problems	☐	☐	☐
Stroke	☐	☐	☐
Glaucoma	☐	☐	☐
Hepatitis, jaundice or liver disease	☐	☐	☐
Epilepsy	☐	☐	☐
Fainting spells or seizures	☐	☐	☐
Neurological disorders	☐	☐	☐
If yes, specify:_____			
Sleep disorder	☐	☐	☐
Mental health disorders	☐	☐	☐
Specify:_____			
Recurrent infections	☐	☐	☐
Type of infection:_____			
Kidney problems	☐	☐	☐
Night sweats	☐	☐	☐
Osteoporosis	☐	☐	☐
Persistent swollen glands in neck	☐	☐	☐
Severe headaches/ migraines	☐	☐	☐
Severe or rapid weight loss	☐	☐	☐
Sexually transmitted disease	☐	☐	☐
Excessive urination	☐	☐	☐

Has a physician or previous dentist recommended that you take antibiotics prior to your dental treatment? ☐ ☐ ☐

Name of physician or dentist making recommendation: | Phone:

Do you have any disease, condition, or problem not listed above that you think I should know about? ☐ ☐ ☐
Please explain:

NOTE: Both doctor and patient are encouraged to discuss any and all relevant patient health issues prior to treatment.

I certify that I have read and understand the above and that the information given on this form is accurate. I understand the importance of a truthful health history and that my dentist and his/her staff will rely on this information for treating me. I acknowledge that my questions, if any, about inquiries set forth above have been answered to my satisfaction. I will not hold my dentist, or any other member of his/her staff, responsible for any action they take or do not take because of errors or omissions that I may have made in the completion of this form.

Signature of Patient/Legal Guardian: | Date:

FOR COMPLETION BY DENTIST

Comments:_____

SAMPLE

Figure 30-1, cont'd

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The dental history should include reference to the following:

1. Visits to the dentist, including frequency, date of the most recent visit, nature of the treatment, and oral prophylaxis or cleaning by a dentist or hygienist, including frequency and date of most recent cleaning.
2. The patient's oral hygiene regimen, including toothbrushing frequency, time of day, method, type of toothbrush and dentifrice, and interval at which brushes are replaced. Other methods for mouth care, such as mouthwashes, interdental brushes or other devices, water irrigation, and dental floss.
3. Any orthodontic treatment, including duration and approximate date of termination.
4. If the patient is experiencing pain in the teeth or in the gingiva, the manner in which the pain is provoked, its nature and duration, and the manner in which it is relieved.
5. Gingival bleeding, including when first noted; whether it occurs spontaneously, on brushing or eating, at night, or with regular periodicity; whether it is associated with the menstrual period or other specific factors; and the duration of the bleeding and the manner in which it is stopped.
6. A bad taste in the mouth and areas of food impaction.
7. Do the teeth feel "loose" or insecure? Is there difficulty in chewing? Any tooth mobility should be recorded.
8. The patient's general dental habits such as grinding or clenching of the teeth during the day or at night. Do the teeth or jaw muscles feel "sore" in the morning? Are there other habits such as tobacco smoking or chewing, nail biting, or biting on foreign objects?
9. History of previous periodontal problems, including the nature of the condition and if previously treated, the type of treatment received (surgical or nonsurgical) and approximate period of termination of previous treatment. If, in the opinion of the patient, the present problem is a recurrence of previous disease, what does he or she think caused it?
10. Does the patient wear any removable prosthesis? Does the prosthesis enhance or is it a detriment to the existing dentition or the surrounding soft tissues?
11. Does the patient have implants replacing any of the missing teeth?

Intraoral Radiographic Survey

The radiographic survey should consist of a minimum of 14 intraoral films and four posterior bite-wing films (Figure 30-2).

Panoramic radiographs are a simple and convenient method of obtaining a survey view of the dental arch and surrounding structures (Figure 30-3). They are helpful for the detection of developmental anomalies, pathologic lesions of the teeth and jaws, and fractures as well as dental screening examinations of large groups.

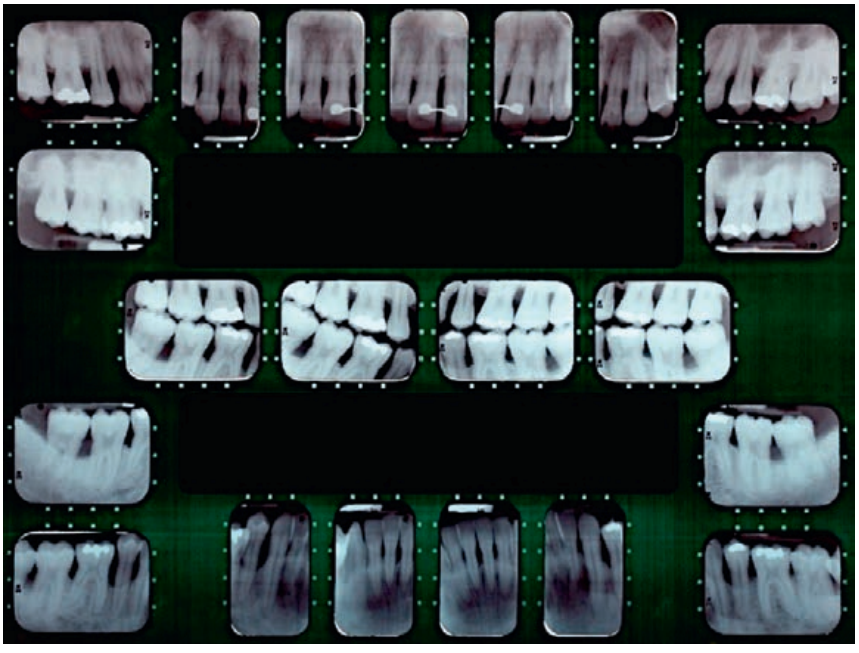

Figure 30-2 Full-mouth intraoral radiographic series (16 periapical films and four bite-wing films) used as an adjunct in periodontal diagnosis.

Figure 30-3 Panoramic radiograph showing temporomandibular joints and "cystic" spaces in the jaw. Areas of periodontal bone loss are not seen in detail. (Compare with Figure 30-2.)

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They provide an informative overall radiographic picture of the distribution and severity of bone destruction in periodontal disease, but *a complete intraoral series is required for periodontal diagnosis and treatment planning.* Chapter 31 gives a detailed description of radiographic interpretation in periodontics.

Casts

Casts from dental impressions are useful adjuncts in the oral examination. They indicate the position of the gingival margins (recession) and the position and inclination of the teeth, proximal contact relationships, and food impaction areas. In addition, they provide a view of the lingual-cuspal relationships. Casts are important records of the dentition before it is altered by treatment. Finally, casts also serve as visual aids in discussions with the patient and are useful for pretreatment and posttreatment comparisons, as well as for reference at recall visits. They are also helpful to determine the position of implant placement if the case will require their use.

Clinical Photographs

Color photographs are useful for recording the appearance of the tissue before and after treatment. Photographs cannot always be relied on for comparing subtle color changes in the gingiva, but they do depict gingival morphologic changes. With the advent of digital clinical photography, record keeping for mucogingival problems, such as areas of gingival recession, frenum involvement, and papilla loss, has become important.

Review of the Initial Examination

If no emergency care is required, the patient is dismissed and instructed as to when to report for the second visit. Before this visit, a correlated examination is made of the radiographs, photographs, and casts to relate the radiographic changes to unfavorable conditions represented on the casts. The casts are checked for evidence of abnormal wear, plunger cusps, uneven marginal ridges, malposed or extruded teeth, crossbite relationships, or other conditions that could cause occlusal disharmony or food impaction. Such areas are marked on the casts to serve as a reference during the detailed examination of the oral cavity. The radiographs, photographs, and casts are valuable diagnostic aids; however, it is *the clinical findings in the oral cavity that constitute the basis for diagnosis.*

SECOND VISIT

Oral Examination

Oral Hygiene. The cleanliness of the oral cavity is appraised in terms of the extent of accumulated food debris, plaque, and tooth surface stains (Figure 30-4). Disclosing solution may be used to detect plaque that would otherwise be unnoticed. The amount of plaque detected, however, is not necessarily related to the severity of the disease present. For example, aggressive periodontitis is a destructive type of periodontitis in which plaque is minimal. Qualitative assessments of plaque are more meaningful, and their value in diagnosis is discussed in Chapter 23.

Oral Malodor. Oral malodor, also termed *fetor ex ore, fetor oris,* or *halitosis,* is foul or offensive odor emanating from the oral cavity. Mouth odors may be of diagnostic significance, and their origin may be either oral or extraoral (remote).[62] Chapter 29 discusses in detail the problems related to oral malodor.

Examination of the Oral Cavity. The entire oral cavity should be carefully examined. The examination should include the lips, floor of the mouth, tongue, palate, and oropharyngeal region, as well as the quality and quantity of saliva. Although findings may not be related to the periodontal problem, the dentist should detect all pathologic changes present in the mouth. Textbooks in oral medicine and oral diagnosis cover these topics in detail.

Figure 30-4 Poor oral hygiene. Gingival inflammation associated with plaque, materia alba, and calculus.

Examination of Lymph Nodes. Because periodontal, periapical, and other oral diseases may result in lymph node changes, the diagnostician should routinely examine and evaluate head and neck lymph nodes. Lymph nodes can become enlarged and/or indurated as a result of an infectious episode, malignant metastases, or residual fibrotic changes.

Inflammatory nodes become enlarged, palpable, tender, and fairly immobile. The overlying skin may be red and warm. Patients are often aware of the presence of "swollen glands." Primary herpetic gingivostomatitis, necrotizing ulcerative gingivitis (NUG), and acute periodontal abscesses may produce lymph node enlargement. After successful therapy, lymph nodes return to normal in a matter of days or a few weeks.

Examination of the Teeth and Implants

The teeth are examined for caries, poor restorations, developmental defects, anomalies of tooth form, wasting, hypersensitivity, and proximal contact relationships. The stability, position, and number of implants and their relationship to the adjacent natural dentition is also examined.

Wasting Disease of the Teeth. Wasting is defined as any gradual loss of tooth substance characterized by the formation of smooth, polished surfaces, without regard to the possible mechanism of this loss. The forms of wasting are erosion, abrasion, and attrition.[46,64]

Erosion, also called *corrosion,* is a sharply defined wedge-shaped depression in the cervical area of the facial tooth surface.[53] The long axis of the eroded area is perpendicular to the vertical axis of the tooth. The surfaces are smooth, hard, and polished. Erosion generally affects a group of teeth. In the early stages, it may be confined to the enamel, but it generally extends to involve the underlying dentin, as well as the cementum.

The etiology of erosion is not known. Decalcification by acidic beverages[44] or citrus fruits, combined with the effect of acid salivary secretion are suggested causes. Sognnaes[70] refers to these lesions as *dentoalveolar ablations* and attributes them to forceful frictional actions between the oral soft tissues and the adjacent hard tissues. In patients with erosion, the salivary pH, buffering capacity, and calcium and phosphorus content have been reported as normal, with the mucin level elevated.[43]

Abrasion refers to the loss of tooth substance induced by mechanical wear other than that of mastication. Abrasion results in saucer-shaped or wedge-shaped indentations with a smooth,

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Figure 30-5 Abrasion attributed to aggressive toothbrushing. Involvement of the roots is followed by undermining of the enamel.

shiny surface. Abrasion starts on exposed cementum surfaces rather than on the enamel and extends to involve the dentin of the root. A sharp "ditching" around the cemento-enamel junction appears to be the result of the softer cemental surface, as compared with the much harder enamel surface.

Continued exposure to the abrasive agent, combined with decalcification of the enamel by locally formed acids, may result in loss of enamel, followed by loss of the dentin of the crown.

Toothbrushing[24] with an abrasive dentifrice (Figure 30-5) and the action of clasps are frequently mentioned, but aggressive toothbrushing is the most common cause.[34] Tooth position (facial) is also a major factor in the abrasive loss of the root surface. The degree of tooth wear from toothbrushing depends on the abrasive effect of the dentifrice and the angle of brushing.[41,42] Horizontal brushing at right angles to the vertical axis of the teeth results in the severest loss of tooth substance. Occasionally, abrasion of the incisal edges occurs as a result of habits such as holding objects (e.g., a bobby pin or tacks) between the teeth.

Attrition is occlusal wear resulting from functional contacts with opposing teeth. Such physical wear patterns may occur on incisal, occlusal, and approximal tooth surfaces. A certain amount of tooth wear is physiologic, but accelerated wear may occur when abnormal anatomic or unusual functional factors are present.

Occlusal or incisal surfaces worn by attrition are called *facets*. When active tooth grinding occurs, the enamel rods are fractured and become highly reflective to light.[80] Thus shiny, smooth, and curviplanar facets are usually the best indicator of ongoing frictional activity. If dentin is exposed, a yellowish brown discoloration is frequently present (Figure 30-6). Facets vary in size and location depending on whether they are produced by physiologic or abnormal wear.[12,78] At least one significant wear facet has been reported in 92% of adults,[67] and facet prevalence approaches universality.[10,79] Facets are usually not sensitive to thermal or tactile stimulation.

Facets generally represent functional or parafunctional wear, as well as iatrogenic dental treatment through coronoplasty (occlusal adjustment). Coronoplasty, however, does not appear to contribute to higher ratings of wear.[68] Excessive wear may result in obliteration of the cusps and the formation of either a flat or a cuneiform (cupped-out) occlusal surface. Contrary to earlier thought, attrition in young adults from modern societies is not age related.[16,68] This suggests that a significant amount of attrition, when present in young adults, is unlikely to occur from functional wear[36] and is probably the result of bruxing activity.[68] Attrition has been correlated with age when older adults are considered.[7,66]

The angle of the facet on the tooth surface is potentially significant to the periodontium. Horizontal facets tend to direct forces on the vertical axis of the tooth, to which the periodontium can adapt most effectively. Angular facets direct occlusal forces laterally

Figure 30-6 Occlusal wear. Flat, shiny, discolored surfaces produced by occlusal wear.

and increase the risk of periodontal damage. However, gradual attrition may be compensated for by continuous tooth eruption without alveolar bone growth and is characterized by a lack of inflammatory changes on the alveolar bone surfaces.[75]

Another mechanism of tooth wear that has been studied recently is called *abfraction* and results from occlusal loading surfaces causing tooth flexure and mechanical microfractures and tooth substance loss in the cervical area.[26]

These four mechanisms of tooth wear (corrosion, abrasion, attrition, and abfraction) can combine with each other, resulting in increased degree of tooth wear.

Dental Stains. These are pigmented deposits on the teeth. They should be carefully examined to determine their origin (see Chapter 22).

Hypersensitivity. Root surfaces exposed by gingival recession may be hypersensitive to thermal changes or tactile stimulation. Patients often direct the clinician to the sensitive areas. These may be located by gentle exploration with a probe or cold air.

Proximal Contact Relations. Open contacts allow food impaction. The tightness of contacts should be checked by means of clinical observation and with dental floss. Abnormal contact relationships may also initiate occlusal changes such as a shift in the median line between the central incisors, with labial flaring of the maxillary canine, buccal, or lingual displacement of the posterior teeth, and an uneven relationship of the marginal ridges. Teeth opposite an edentulous site may supererupt, thus opening the proximal contacts.

Tooth Mobility. All teeth have a slight degree of physiologic mobility, which varies for different teeth and at different times of the day.[50,54] It is greatest on arising in the morning and progressively decreases. The increased mobility in the morning is attributed to slight extrusion of the tooth because of limited occlusal contact during sleep. During the waking hours, mobility is reduced by chewing and swallowing forces, which intrude the teeth in the sockets. These 24-hour variations are less marked in persons with a healthy periodontium than in those with occlusal habits such as bruxism and clenching.

Single-rooted teeth have more mobility than multirooted teeth, with incisors having the most. Mobility is primarily in a

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horizontal direction, although some axial mobility occurs, to a lesser degree.[52]

Tooth mobility occurs in the following two stages:

1. The initial or intrasocket stage is where the tooth moves within the confines of the periodontal ligament. This is associated with viscoelastic distortion of the ligament and redistribution of the periodontal fluids, interbundle content, and fibers.[35] This initial movement occurs with forces of about 100 lb and is of the order of 0.05 to 0.10 mm (50 to 100 µm).[47]
2. The secondary stage occurs gradually and entails elastic deformation of the alveolar bone in response to increased horizontal forces.[49] When a force of 500 g is applied to the crown, the resulting displacement is about 100 to 200 µm for incisors, 50 to 90 µm for canines, 8 to 10 µm for premolars, and 40 to 80 µm for molars.[47]

When a force such as that applied to teeth in occlusion is discontinued, the teeth return to their original position in two stages: the first is an immediate, springlike elastic recoil; the second is a slow, asymptomatic recovery movement. The recovery movement is pulsating and is apparently associated with the normal pulsation of the periodontal vessels, which occurs in synchrony with the cardiac cycle.[48]

Many attempts have been made to develop mechanical or electronic devices for the precise measurement of tooth mobility.[48,51,53,67] Even though standardization of grading mobility would be helpful in diagnosing periodontal disease and in evaluating the treatment outcome, these devices are not widely used. As a general rule, mobility is graded clinically by holding the tooth firmly between the handles of two metallic instruments or with one metallic instrument and one finger (Figure 30-7). An effort then is made to move it in all directions. Abnormal mobility most often occurs faciolingually. Mobility is graded according to the ease and extent of tooth movement as follows:

- Normal mobility
- Grade I: Slightly more than normal.
- Grade II: Moderately more than normal.
- Grade III: Severe mobility faciolingually and/or mesiodistally, combined with vertical displacement

Mobility beyond the physiologic range is termed *abnormal* or *pathologic*. It is pathologic in that it exceeds the limits of normal mobility values; however, the periodontium is not *necessarily* diseased at the time of examination.

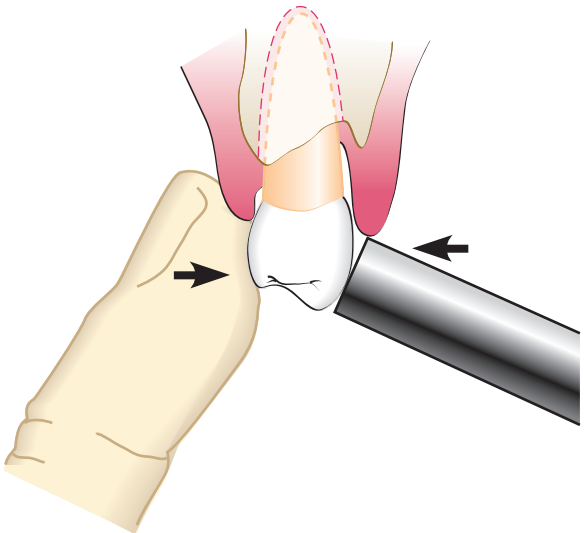

Figure 30-7 Tooth mobility checked with a metal instrument and one finger.

Increased mobility is caused by one or more of the following factors:

1. *Loss of tooth support (bone loss)* can result in mobility. The amount of mobility depends on the severity and distribution of bone loss at individual root surfaces, the length and shape of the roots, and the root size compared with that of the crown.[53] A tooth with short, tapered roots is more likely to loosen than one with normal-size or bulbous roots with the same amount of bone loss. One should carefully evaluate post orthodontic cases for possible apical shortening of the root, which may lead to excessive mobility. Because bone loss usually results from a combination of factors and does not occur as an isolated finding, the severity of tooth mobility does not necessarily correspond to the amount of bone loss.
2. *Trauma from occlusion* (i.e., injury produced by excessive occlusal forces or incurred because of abnormal occlusal habits such as bruxism and clenching) is a common cause of tooth mobility. Mobility is also increased by hypofunction. Mobility produced by trauma from occlusion occurs initially as a result of resorption of the cortical layer of bone, leading to reduced fiber support, and later as an adaptation phenomenon resulting in a widened periodontal space.
3. *Extension of inflammation* from the gingiva or from the periapex into the periodontal ligament results in changes that increase mobility. The spread of inflammation from an acute periapical abscess may increase tooth mobility in the absence of periodontal disease.
4. *Periodontal surgery* temporarily increases tooth mobility immediately after the intervention and for a short period.[56–59]
5. Tooth mobility is increased in *pregnancy* and is sometimes associated with the *menstrual cycle* or the use of *hormonal contraceptives*. This is unrelated to periodontal disease and occurs presumably because of physicochemical changes in the periodontal tissues.
6. *Pathologic processes of the jaws* that destroy the alveolar bone and/or the roots of the teeth can also result in mobility. Osteomyelitis and tumors of the jaws belong in this category.

One study[22] has suggested that pockets around mobile teeth harbor higher proportions of *Campylobacter rectus* and *Peptostreptococcus micros* and possibly of *Porphyromonas gingivalis* than nonmobile teeth. This hypothesis needs further verification.

Trauma from Occlusion. *Trauma from occlusion* refers to *tissue injury* produced by occlusal forces, not to the occlusal forces themselves (see Chapter 15). The criterion that determines that an occlusion is traumatic is whether it causes damage in the periodontal tissues; therefore the diagnosis of trauma from occlusion is made from the condition of the periodontal tissues. The periodontal findings are then used as a guide for locating the responsible occlusal relationships.

Periodontal findings that suggest the presence of trauma from occlusion include excessive tooth mobility, particularly in teeth showing radiographic evidence of a widened periodontal space; vertical or angular bone destruction; infrabony pockets; and pathologic migration, especially of the anterior teeth (see following discussion).

Pathologic Migration of the Teeth. Alterations in tooth position should be carefully noted, particularly with a view

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Figure 30-8 Periodontal disease with pathologic migration of the anterior teeth. **A,** Clinical photograph. **B,** Radiographic view.

toward identifying abnormal forces, a tongue-thrusting habit, or other habits that may be contributing factors (see Chapter 15). Premature tooth contacts in the posterior region that deflect the mandible anteriorly contribute to destruction of the periodontium of the maxillary anterior teeth and to pathologic migration (Figure 30-8; see also Figure 15-10, *A* and *B*). The loss of posterior teeth can lead to the facial "flaring" of the maxillary anterior dentition. This is due to the increased trauma that the mandibular anterior dentition places against the palatal surface of the maxillary anterior dentition. Pathologic migration of anterior teeth in young persons may be a sign of localized aggressive (juvenile) periodontitis.

Sensitivity to Percussion. Sensitivity to percussion is a feature of acute inflammation of the periodontal ligament. Gentle percussion of a tooth at different angles to the long axis often aids in localizing the site of inflammatory involvement.

Dentition with the Jaws Closed. Examination of the dentition with the jaws closed can detect conditions, such as irregularly aligned teeth, extruded teeth, improper proximal contacts, and areas of food impaction, all of which may favor plaque accumulation.

Excessive *overbite,* seen most often in the anterior region, may cause impingement of the teeth on the gingiva and food impaction, followed by gingival inflammation, gingival enlargement, and pocket formation. The real significance of the detrimental effect of excessive anterior overbite on gingival health is still controversial.[2]

In *open-bite* relationships, abnormal vertical spaces exist between the maxillary and mandibular teeth. The condition occurs most often in the anterior region, although posterior open bite is occasionally seen. Reduced mechanical cleansing by the passage of food may lead to accumulation of plaque, debris, calculus formation, and extrusion of teeth.

In *crossbite,* the normal relationship of the mandibular teeth to the maxillary teeth is reversed, with the maxillary teeth being lingual to the mandibular teeth. Crossbite may be bilateral or unilateral, or it may affect only a pair of antagonists. Trauma from occlusion, food impaction, spreading of the mandibular teeth, and associated gingival and periodontal disturbances may be caused by crossbite.

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Functional Occlusal Relationships. Examination of functional occlusal relationships is an important part of the diagnostic procedure. Dentitions that appear normal when the jaws are closed may present marked functional abnormalities. Systematic procedures for the detection and correction of functional abnormalities are presented in Chapter 49.

Examination of the Periodontium

The periodontal examination should be systematic, starting in the molar region in either the maxilla or the mandible and proceeding around the arch. This prevents overemphasis of unusual findings at the expense of other conditions that although less striking, may be equally important. It is important to detect the earliest signs of gingival and periodontal disease.

Charts to record the periodontal and associated findings provide a guide for a thorough examination and record of the patient's condition (Figure 30-9, *A* and *B*). They are also used to evaluate the response to treatment and for comparison at recall visits. However, excessively complicated mouth charting may lead to a frustrating maze of minutiae rather than clarification of the patient's problem.

In the last decade, electronic clinical records have been developed and are increasingly being used by general dentists and periodontists. Most systems provide a rapid and easy access to information and allow the incorporation of digital clinical and radiographic images.[63] Computerized dental examination systems using high-resolution graphics and voice-activated technology permit easy retrieval and comparison of data.[11]

Plaque and Calculus. There are many methods available for assessing plaque and calculus accumulation.[18] The presence of supragingival plaque and calculus can be directly observed and the amount measured with a calibrated probe. For the detection of subgingival calculus, each tooth surface is carefully checked to the level of the gingival attachment with a no. 17 or no. 3A explorer (Figure 30-10). Warm air may be used to deflect the gingiva and aid in visualization of the calculus.

Although the radiograph may sometimes reveal heavy calculus deposits interproximally and even on the facial and lingual surfaces, it cannot be relied on for the thorough detection of calculus.

Gingiva. The gingiva must be dried before accurate observations can be made (Figure 30-11). Light reflection from moist gingiva obscures detail. In addition to visual examination and exploration with instruments, firm but gentle palpation should be used for detecting pathologic alterations in normal resilience, as well as for locating areas of exudate.

Features of the gingiva to consider are: color, size, contour, consistency, surface texture, position, ease of bleeding, and pain (see Chapter 8). Any deviation from the norm should be evaluated and not overlooked. The distribution of gingival disease and its acute or chronic nature should also be noted.

Clinically gingival inflammation can produce two basic types of tissue response: edematous and fibrotic. *Edematous* tissue response is characterized by a smooth, glossy, soft, red gingiva. In the *fibrotic* tissue response, some of the characteristics of normalcy persist; the gingiva is more firm, stippled, and opaque, it is usually thicker, and the margin appears rounded.

Use of Clinical Indices in Dental Practice. There has been a tendency to extend the use of indices originally designed for epidemiologic studies into dental practice (see Chapter 5). Of all the indices proposed, the gingival index and the sulcus bleeding index appear to be the most useful and most easily transferred to clinical practice.

The *gingival index* (Löe and Silness) provides an assessment of the gingival inflammatory status that and can be used in practice to compare gingival health before and after phase I therapy or before and after surgical therapy. It can also be used to compare the gingival status at recall visits. Attaining good intraexaminer and interexaminer calibration is imperative in the dental office.

The *sulcus bleeding index* (Mühlemann and Son) provides an objective, easily reproducible assessment of the gingival status. It is extremely useful for detecting early inflammatory changes and the presence of inflammatory lesions located at the base of the periodontal pocket, an area inaccessible to visual examination. Patients can easily understand this index; therefore it can be used to enhance the patient's motivation for plaque control.

Periodontal Pockets. Examination for periodontal pockets must include their presence and distribution on each tooth surface, pocket depth, level of attachment on the root, and type of pocket (suprabony or infrabony).

Signs And Symptoms. Although probing is the only reliable method of detecting pockets, clinical signs, such as color changes (bluish red marginal gingiva or bluish red vertical zone extending from the gingival margin to the attached gingiva); a "rolled" edge separating the gingival margin from the tooth surface; or an enlarged, edematous gingiva, may suggest their presence. The presence of bleeding, suppuration, and loose, extruded teeth may also denote the presence of a pocket (Figures 30-12 to 30-15).

Periodontal pockets are generally painless but may give rise to symptoms such as localized or sometimes radiating pain or sensation of pressure after eating, which gradually diminishes. A foul taste in localized areas, sensitivity to hot and cold, and toothache in the absence of caries is also sometimes present.

Detection Of Pockets. The only accurate method of detecting and measuring periodontal pockets is careful exploration with a periodontal probe. Pockets are not detected by radiographic examination. The periodontal pocket is a soft tissue change. Radiographs indicate areas of bone loss in which pockets may be suspected, but they do not show pocket presence or depth and consequently they show no difference before and after pocket elimination unless bone has been modified.

Gutta percha points or calibrated silver points[29] can be used with the radiograph to assist in determining the level of attachment of periodontal pockets (Figure 30-16). They may be used effectively for individual pockets or in clinical research, but their routine use throughout the mouth would be difficult to manage. Clinical examination and probing are more direct and efficient. See Chapter 31 for detailed information regarding radiographic examination.

Pocket Probing. There are two different pocket depths: (1) the biologic or histologic depth and (2) the clinical or probing depth[28] (Figure 30-17).

The *biologic depth* is the distance between the gingival margin and the base of the pocket (the coronal end of the junctional epithelium). This can be measured only in carefully prepared and adequately oriented histologic sections. The *probing depth* is the distance to which a probe penetrates into the pocket. Probes presently used are described in Chapter 45.

Probe penetration can vary, depending on the force of introduction, the shape and size of the probe tip, the direction of penetration, resistance of the tissues, convexity of the crown, and the degree of tissue inflammation.[4] Several studies have been made to

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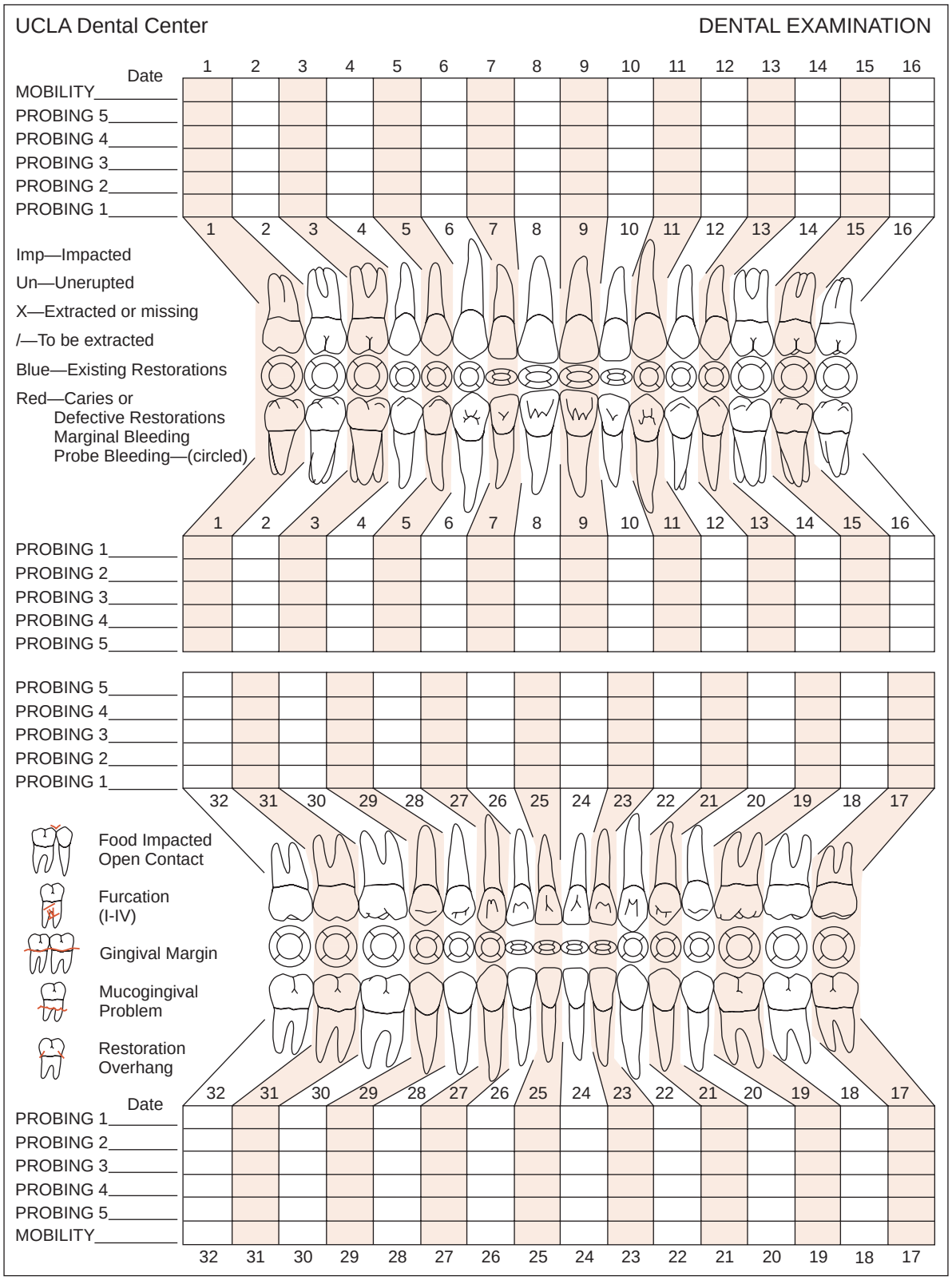

Figure 30-9 **A,** UCLA periodontal chart. **B,** Computerized diagram showing various periodontal parameters. (Courtesy University of California, Los Angeles School of Dentistry.)

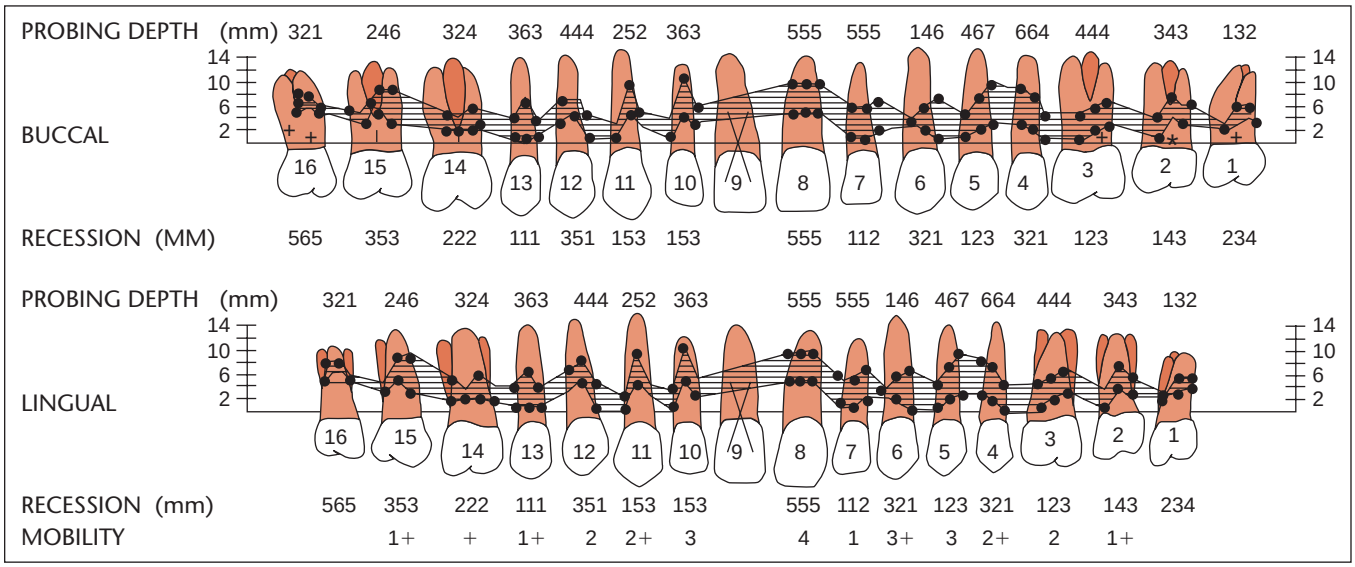

Figure 30-9, cont'd

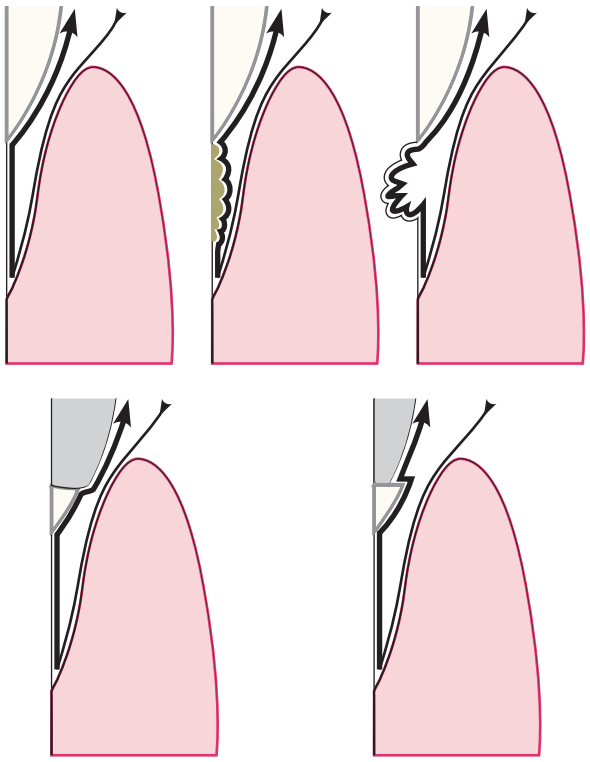

Figure 30-10 *Top left,* Detection of smoothness of various irregularities on the root surface with outward motion of a probe or explorer. *Top center,* Calculus. *Top right,* Caries. *Bottom left and right,* Irregular margins of restorations.

Figure 30-11 Normal gingiva with incipient gingivitis in tooth #7. Normal surface features are better revealed by drying gingiva.

Figure 30-12 Periodontal pockets around mandibular anterior teeth, showing rolled margins, edematous inflammatory changes, and abundant calculus and plaque.

determine the depth of penetration of a probe in a sulcus or pocket. Armitage and colleagues[6] used beagle dogs to evaluate the penetration of a probe using a standardized force of 25 g. They reported that in healthy gingiva the probe penetrated the epithelium to about two-thirds of its length; in gingivitis, it stopped 0.1-mm short of its apical end; and in periodontitis, the probe tip consistently went past the most apical cells of the junctional epithelium (Figure 30-18).

In human periodontal pockets, the probe tip penetrates to the most coronal intact fibers of the connective tissue attachment.[38,71] The depth of penetration of the probe in the connective tissue apical to the junctional epithelium in a periodontal pocket is about 0.3 mm.[38,65,71] This is important in evaluating differences in probing

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Figure 30-13 Periodontal pocket with vertical discolored zone extending to the alveolar mucosa.

Figure 30-14 Periodontal pocket between maxillary central incisors produced bluish discoloration extending apically. Probing reveals presence of deep pocket.

Figure 30-15 Severe generalized gingival inflammation. Note the dark hue in the marginal areas of the central incisors, which is caused in part by dark subgingival calculus and a deep pocket.

Figure 30-16 Blunted silver points assist in locating the base of pocket.

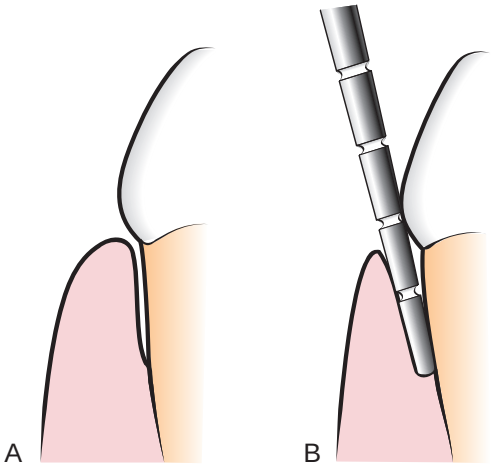

Figure 30-17 **A,** Biologic or histologic pocket depth is the actual distance between the gingival margin and the attached tissues (bottom of pocket). **B,** Probing or clinical pocket depth is the depth of penetration of the probe.

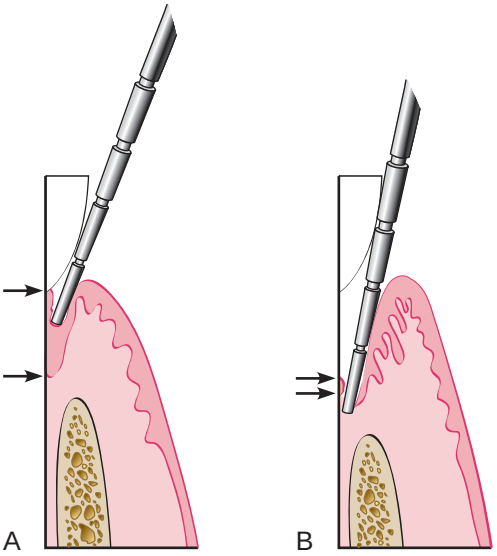

Figure 30-18 **A,** In a normal sulcus with a long junctional epithelium (between *arrows*), the probe penetrates about one third to one half the length of the junctional epithelium. **B,** In a periodontal pocket with a short junctional epithelium (between *arrows*), the probe penetrates beyond the apical end of the junctional epithelium.

depth before and after treatment, as the reduction in probe penetration may be a result of reduced inflammatory response rather than gain in attachment.[37,39]

The probing forces have been explored by several investigators[19,76,77]; forces of 0.75 N have been found to be well tolerated and accurate.[73] Interexaminer error (depth discrepancies between examiners) was reported to be as much as 2.1 mm, with an average of 1.5 mm, in the same areas.[28]

Probing Technique. The probe should be inserted parallel to the vertical axis of the tooth and "walked" circumferentially around each surface of each tooth to detect the areas of deepest penetration (Figure 30-19).

In addition, special attention should be directed to detecting the presence of interdental craters and furcation involvements. To detect an *interdental crater,* the probe should be placed obliquely

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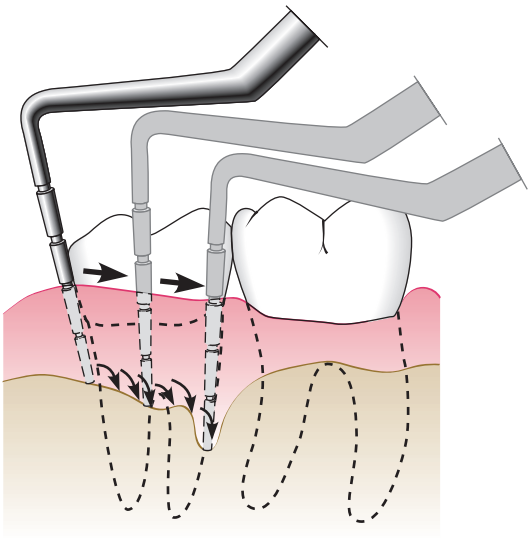

Figure 30-19 "Walking" the probe to explore the entire pocket.

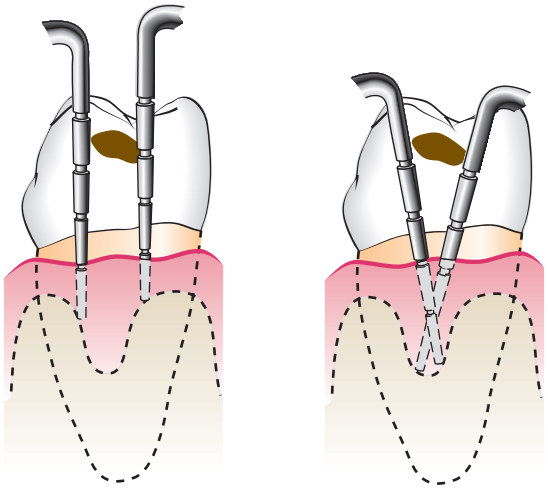

Figure 30-20 Vertical insertion of the probe *(left)* may not detect interdental craters; oblique positioning of the probe *(right)* reaches the depth of the crater.

from both the facial and lingual surfaces so as to explore the deepest point of the pocket located beneath the contact point (Figure 30-20). In multirooted teeth, the possibility of *furcation involvement* should be carefully explored. The use of specially designed probes (e.g., Nabers probe) allows an easier and more accurate exploration of the horizontal component of furcation lesions (Figure 30-21).

Level of Attachment Versus Pocket Depth. *Pocket depth* is the distance between the base of the pocket and the gingival margin. It may change from time to time even in untreated periodontal disease because of changes in the position of the gingival margin, and therefore it may be unrelated to the existing attachment of the tooth.

The level of attachment, on the other hand, is the distance between the base of the pocket and a fixed point on the crown such as the cementoenamel junction (CEJ). Changes in the level of attachment can be the result of gain or loss of attachment and afford a better indication of the degree of periodontal destruction

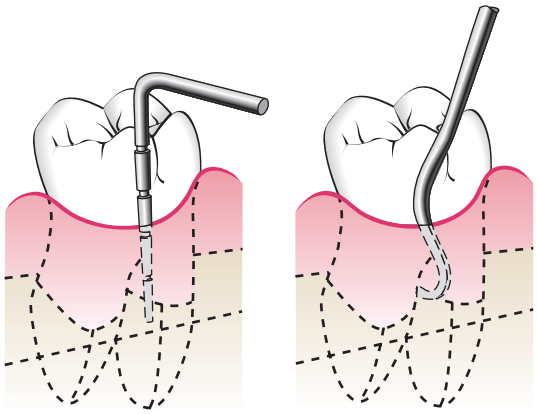

Figure 30-21 Exploring with a periodontal probe *(left)* may not detect furcation involvement; specially designed instruments (Nabers probe) *(right)* can enter the furcation area.

(or gain). *Shallow pockets attached at the level of the apical third of the root connote more severe destruction than deep pockets attached at the coronal third of the roots* (see Chapter 13 and Figures 13-17 to 13-19).

Determining The Level of Attachment. When the *gingival margin is located on the anatomic crown,* the level of attachment is determined by subtracting from the depth of the pocket the distance from the gingival margin to the CEJ. If both are the same, the loss of attachment is zero.

When the gingival margin coincides with the CEJ, the loss of attachment equals the pocket depth.

When the gingival margin is located apical to the CEJ, the loss of attachment is greater than the pocket depth. Therefore the distance between the CEJ and the gingival margin should be added to the pocket depth. Drawing the gingival margin on the chart where pocket depths are entered helps clarify this important point.[69]

Bleeding on Probing. The insertion of a probe to the bottom of the pocket elicits bleeding if the gingiva is inflamed and the pocket epithelium is atrophic or ulcerated. Noninflamed sites rarely bleed. In most cases, bleeding on probing is an earlier sign of inflammation than gingival color changes[45] (see Chapter 8). However, color changes may present without bleeding on probing.[24] Depending on the severity of inflammation, bleeding can vary from a tenuous red line along the gingival sulcus to profuse bleeding.[1] If periodontal treatment is successful, bleeding on probing will cease.[3]

To test for bleeding after probing, the probe is carefully introduced to the bottom of the pocket and gently moved laterally along the pocket wall. Sometimes bleeding appears immediately after removal of the probe; other times it may be delayed for a few seconds. Therefore the clinician should recheck for bleeding 30 to 60 seconds after probing.

As a single test, bleeding on probing is not a good predictor of progressive attachment loss; however, its absence is an excellent predictor of periodontal stability.[4] When bleeding is present in multiple sites of advanced disease, bleeding on probing is a good indicator of progressive attachment loss.[4,20] Armitage analyzed the literature on this subject up to 1996, performing a metaanalysis of the various papers and concluded that the presence of bleeding on probing in a "treated and maintained patient population" is an important risk predictor for increased loss of attachment.[4]

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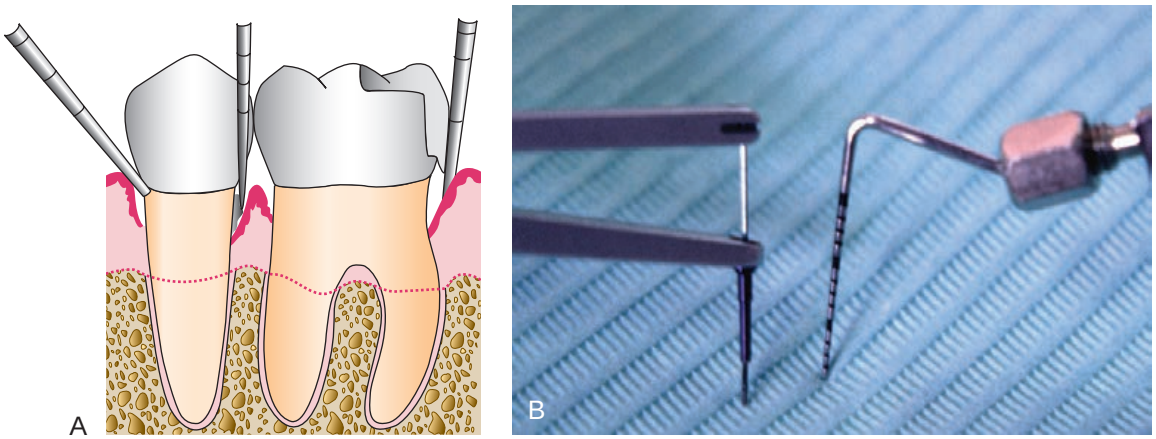

Figure 30-22 **A,** Limitations in periodontal probing. **B,** Probing pressure caused by probe angulation, presence of subgingival calculus, and presence of overhanging restorations. (**A** courtesy Dr. J. Frontan.)

Insertion of a soft wooden interdental stimulator in the interdental space produces a similar bleeding response[3] and can be used by the patient to self-examine the gingiva for the presence of inflammation.[13]

When to Probe. Probing of pockets is done at various times for diagnosis and for monitoring the course of treatment and maintenance. The initial probing of moderate or advanced cases is usually hampered by the presence of heavy inflammation and abundant calculus and cannot be done very accurately. Probing at this stage is also difficult as the result of the discomfort and pain that occurs when the gingival tissues are inflamed.

The purpose of this initial probing, together with the clinical and radiographic examination, is done to determine whether the tooth can be saved or should be extracted. After the patient has performed an adequate plaque control for some time and calculus has been removed, the major inflammatory changes disappear and an accurate probing of the pockets can be performed. The purpose of this second probing is to accurately establish the level of attachment and degree of involvement of roots and furcations. Data obtained from this probing provides valuable information for treatment decisions.

Later in periodontal treatment, probings are done to determine changes in pocket depth and to ascertain healing progress after different procedures.

Probing Around Implants. Since periimplantitis can create pockets around implants, probing around them becomes part of examination and diagnosis. To prevent scratching of the implant surface, plastic periodontal probes should be used instead of the steel probes used for the natural dentition.

Automatic and Electronic Periodontal Probing. The use of the periodontal probe is the classic method to detect pocket depth and loss of attachment. However, it presents some problems in terms of reproducibility of the measurements. Accuracy and reproducibility depend not only on root morphology and tissue changes but also, very importantly, on the probing technique, the probing force, the size of the probe, the angle of insertion of the probe, and the precision of probe calibration (Figure 30-22).

Probing Force. One of the main problems in reproducibility has been the variation of probing force. The development of pressure-sensitive probes has helped overcome this. It has been shown that with forces of up to 30 g, the tip of the probe remains within the junctional epithelium,[5] whereas forces of up to 50 g are necessary to reach the bone level.[31]

Figure 30-23 Automated periodontal probes: Florida Probe System. Integration of direct electronic measurements with constant probing force with computer storage and online data readout.

Probe Angulation. Standardization of the probe tip (<1 mm) and the addition of registration stents to maintain reproducible probe angulation have been used to overcome this error.[8] New, commercially available computer-assisted technology has been used to improve probing accuracy and reproducibility. In addition to more accurate measurement, the data derived from automatic probing can become part of the electronic record. Once incorporated, clinical changes and patterns of disease activity can be easily determined. These records also provide feedback to the patient. The Florida Probe System[2] (Figure 30-23) consists of a probe handpiece, digital readout, foot switch, computer interface, and computer. The end of the probe is 0.4 mm, and it reciprocates through a sleeve that provides a reference by which measurements are made.

These measurements are made electronically and transferred automatically when the foot switch is pressed or a voice command is given. Constant probing force is provided by coil springs inside the probe handpiece and digital readout (Figure 30-24). This method combines the advantages of a constant probing force with precise electronic measurement and computer storage of data, thus

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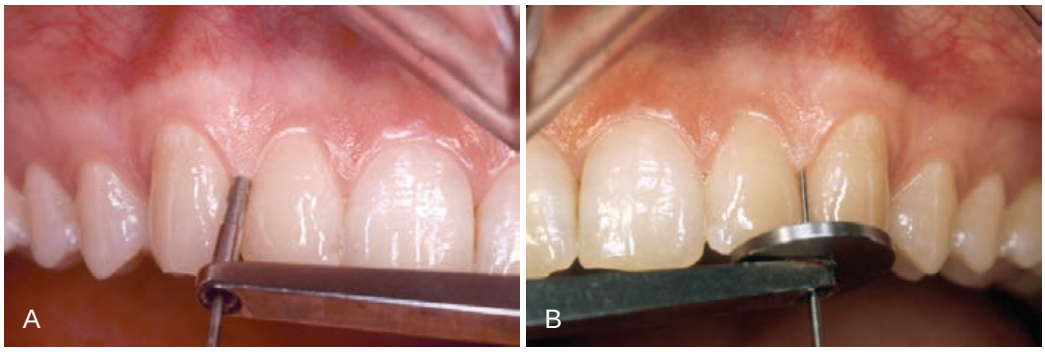

Figure 30-24 Florida Probe System. **A,** Handpiece for assessing probing pocket depths. **B,** Handpiece for assessing relative clinical attachment levels.

eliminating the potential errors associated with visual reading and the need for an assistant to record the measurements.

Several studies have been made comparing the Florida Probe with conventional probing. The automatic probe appears to underestimate deep probing depths[55] but show less variability than conventional probing.[15,61,81] The automatic probe also has the problem of providing little tactile sensitivity thus making it more difficult to "walk" the probe.

Other electronic systems, such as the Interprobe and the Periprobe, provide constant probing force and computer storage of data, but their reproducibility is only slightly better than conventional methods.[61,81]

The lack of standardization between probings by different individuals and at different times by the same individual depend not only on root morphology and tissue changes but also on the probing technique, the probing force, the size of the probe, the angle of insertion of the probe, and precision of the probe calibration. For measurements of clinical attachment loss, it is necessary to identify the location of the CEJ, and this offers another hurdle for the standardization of measurements.

Determination of Disease Activity. Currently, there are no accurate methods to determine activity or inactivity of a lesion. Inactive lesions may show little or no bleeding on probing and minimal amounts of gingival fluid. Active lesions bleed more readily on probing and have large amounts of fluid and exudates although active and nonactive sites may show no differences in bleeding on probing, even in patients with aggressive periodontitis.[31] The bacterial flora in a healthy gingival sulcus, as revealed by dark-field microscopy, consists mostly of coccoid cells. The bacterial flora of a periodontal pocket shows a greater number of spirochetes and motile bacteria.[25]

For the determination of pocket depth or attachment levels to provide information on whether the lesion is in an active or inactive state, measurements taken at different times have to be compared. The precise assessment and comparison of the clinical attachment level (CAL) at different intervals of time can determine whether the attachment is being lost, which indicates that the lesion is active. The exact measurement of this important clinical parameter is done from the CEJ to the bottom of the pocket and therefore requires to determine exactly and reproducibly the location of this landmark.

The Florida Probe provides a means of recording relative CAL changes over time. A later model of the probe has a modified sleeve with a prominent 0.125-mm edge to facilitate a "catch" of the CEJ. The width of this edge is considered small enough not to interfere with probing-depth measurements, thus providing concurrent measurements of CAL and pocket depth.[32,66] Of course, this measurement does not predict the future but only shows what happened in the period of time between the two measurements. See Chapters 23 and 25 for other methods of determination of disease activity still in a developmental stage. The precise determination of disease activity will have a direct influence on diagnosis, prognosis, and therapy. The goals of therapy may change, depending on the state of the periodontal lesion.

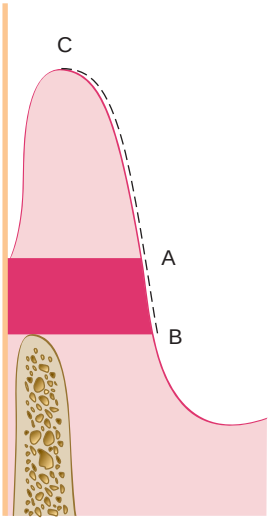

Figure 30-25 Shaded area shows the attached gingiva, which extends between the projection on the external surface of the bottom of the pocket *(A)* and the mucogingival junction *(B)*. The keratinized gingiva may extend from the mucogingival junction (*B*) to the gingival margin (*C*).

Amount of Attached Gingiva. It is important to establish the relation between the bottom of the pocket and the mucogingival line. The width of the attached gingiva is the distance between the mucogingival junction and the projection on the external surface of the bottom of the gingival sulcus or the periodontal pocket. It should not be confused with the width of the keratinized gingiva, because the latter also includes the marginal gingiva (Figure 30-25).

The width of the attached gingiva is determined by subtracting the sulcus or pocket depth from the total width of the gingiva (gingival margin to mucogingival line). This is done by stretching the lip or cheek to demarcate the mucogingival line while the pocket is being probed (Figure 30-26). The amount of attached gingiva is generally considered to be insufficient when stretching of the lip or cheek induces movement of the free gingival margin.

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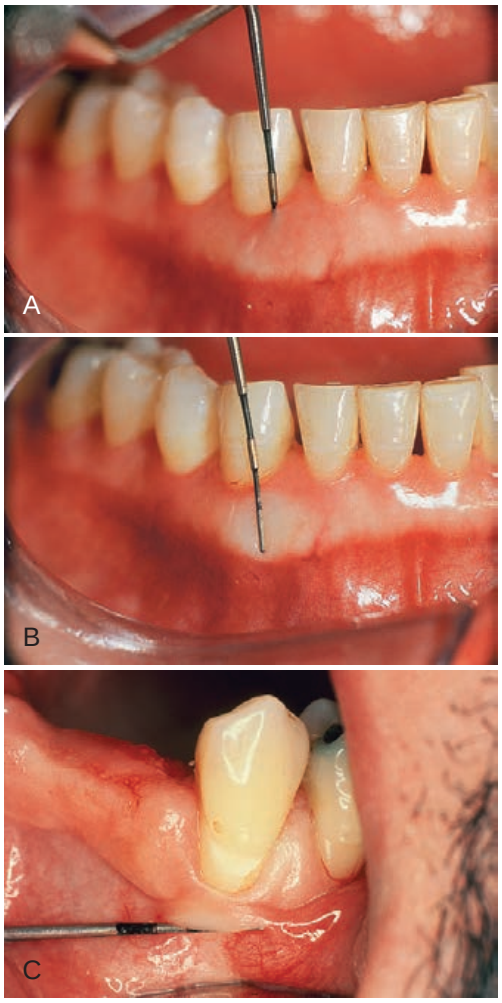

Figure 30-26 To determine the width of the attached gingiva, the pocket is probed **(A),** and then the probe is placed on the outer surface **(B)** while the lip (or cheek) is extended to demarcate the mucogingival line. **C,** Another method to demarcate the mucogingival line is pushing the lip (cheek) coronally.

Other methods used to determine the amount of attached gingiva include pushing the adjacent mucosa coronally with a dull instrument or painting the mucosa with Schiller's potassium iodide solution, which stains keratin.

Degree of Gingival Recession. During periodontal examination, it is necessary to record the data regarding the amount of gingival recession. This measurement is taken with a periodontal probe from the CEJ to the gingival crest, and it is drawn on the patient's chart (see Chapter 8).

Alveolar Bone Loss. Alveolar bone levels are evaluated by clinical and radiographic examination. Probing is helpful for determining (1) the height and contour of the facial and lingual bones obscured on the radiograph by the roots and (2) the architecture of the interdental bone. Transgingival probing, performed after the area is anesthetized, is a more accurate method of evaluation and provides additional information on bone architecture[23,34,73] (see Chapter 60).

Palpation. Palpating the oral mucosa in the lateral and apical areas of the tooth may help locate the origin of radiating pain that the patient cannot localize. Infection deep in the periodontal tissues and the early stages of a periodontal abscess may also be detected by palpation.

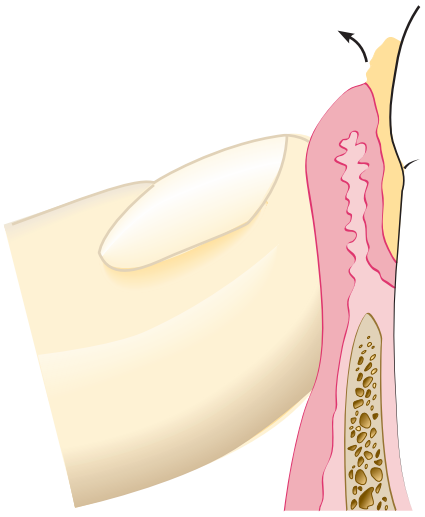

Figure 30-27 Purulent exudate expressed from a periodontal pocket by digital pressure.

Suppuration. The presence of an abundant number of neutrophils in the gingival fluid transforms it into a purulent exudate.[4] Several studies[9,10,13,30] have evaluated the association between suppuration and the progression of periodontitis and reported that this sign is present in a very low percentage of diseased sites (3% to 5%).[4] Therefore it is not by itself a good indicator.

Clinically, the presence of exudate in a periodontal pocket is determined by placing the ball of the index finger along the lateral aspect of the marginal gingiva and applying pressure in a rolling motion toward the crown (Figure 30-27). Visual examination without digital pressure is not enough. The purulent exudate is formed in the inner pocket wall, and therefore the external appearance may give no indication of its presence. Exudate formation does not occur in all periodontal pockets, but digital pressure often reveals it in pockets where its presence is not suspected.

Periodontal Abscess. A periodontal abscess is a localized accumulation of exudate within the gingival wall of a periodontal pocket (see Chapter 13). Periodontal abscesses may be acute or chronic.

The *acute periodontal abscess* appears as an ovoid elevation of the gingiva along the lateral aspect of the root (Figures 30-28 to 30-30). The gingiva is edematous and red, with a smooth, shiny surface. The shape and consistency of the elevated area vary; the area may be domelike and relatively firm, or pointed and soft. In most cases, exudate may be expressed from the gingival margin with gentle digital pressure.

The acute periodontal abscess is accompanied by symptoms such as throbbing, radiating pain and tenderness of the gingiva to palpation. Other symptoms may include sensitivity of the tooth to palpation; tooth mobility and lymphadenitis; and less frequently, systemic effects such as fever, leukocytosis, and malaise. Occasionally, the patient may have symptoms of an acute periodontal abscess without any notable clinical lesion or radiographic changes.

The *chronic periodontal abscess* usually presents a sinus that opens onto the gingival mucosa along the length of the root. There may be a history of intermittent exudation. The orifice of the sinus may

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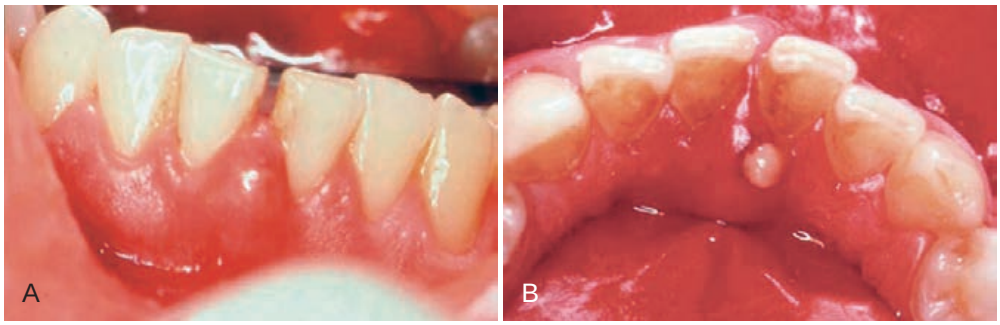

Figure 30-28 A, Facial view of acute periodontal abscess between the lower central incisors. **B,** Lingual view of the same patient with a suppurating draining sinus.

Figure 30-29 Acute periodontal abscess in the wall of a deep pocket in the lingual surface of lower premolars.

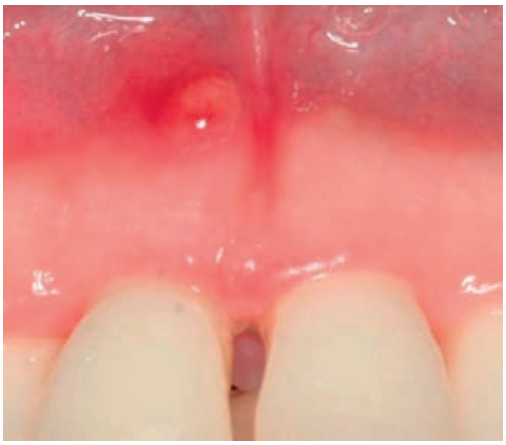

Figure 30-31 Nodular mass at the orifice of a draining sinus.

Figure 30-30 Acute periodontal abscess associated with a deep periodontal pocket in palatal area of first and second upper molars. Note how the fibrotic character of the palatal tissue masks the typical changes of the abscess.

appear as a difficult-to-detect pinpoint opening, which, when probed, reveals a sinus tract leading deep into the periodontium. The sinus may be covered by a small, pink, beadlike mass of granulation tissue. The chronic periodontal abscess is usually asymptomatic. However, the patient may report episodes of dull, gnawing pain; slight elevation of the tooth; and a desire to bite down and grind the tooth. The chronic periodontal abscess often undergoes acute exacerbations, with all the associated symptoms.

Diagnosis of the periodontal abscess requires correlation of the history and clinical and radiographic findings. The suspected area should be probed carefully along the gingival margin in relation to each tooth surface to detect a channel from the marginal area to the deeper periodontal tissues. Continuity of the lesion with the gingival margin is clinical evidence that the abscess is periodontal.

The abscess is not necessarily located on the same surface of the root as the pocket from which it is formed. A pocket at the facial surface may give rise to a periodontal abscess interproximally. It is common for a periodontal abscess to be located at a root surface other than that along which the pocket originated because drainage is more likely to be impaired when a pocket follows a tortuous course.

In children a sinus orifice along the lateral aspect of a root is usually the result of periapical infection of a deciduous tooth. In the permanent dentition, such an orifice may be caused by a periodontal abscess, as well as by apical involvement. The orifice may be patent and draining, or it may be closed and appear as a red, nodular mass (Figure 30-31). Exploration of such masses with a probe usually reveals a pinpoint orifice that communicates with an underlying sinus.

Periodontal Abscess and Gingival Abscess. The principal differences between the periodontal abscess and the gingival abscess are location and history (see Chapter 13). The gingival abscess is confined to the marginal gingiva, and it often occurs in previously disease-free areas (Figure 30-32). It is usually an acute inflammatory response to forcing of foreign material into the gingiva. The periodontal abscess involves the supporting periodontal structures and generally occurs in the course of chronic destructive periodontitis.

Periodontal Abscess and Periapical Abscess. Several characteristics can be used as guidelines in differentiating a periodontal abscess from a periapical abscess. If the tooth is nonvital, the lesion is most likely periapical. However, a previously nonvital tooth can have a deep periodontal pocket that can abscess. Moreover, a deep periodontal pocket can extend to the apex and cause pulpal involvement and necrosis.

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Figure 30-32 Gingival abscess between upper lateral incisor and canine.

An apical abscess may spread along the lateral aspect of the root to the gingival margin. However, when the apex and lateral surface of a root are involved by a single lesion that can be probed directly from the gingival margin, the lesion is more likely to have originated as a periodontal abscess.

Radiographic findings are helpful in differentiating between a periodontal and a periapical lesion (see Chapter 31). Early acute periodontal and periapical abscesses present no radiographic changes. Ordinarily, a radiolucent area along the lateral surface of the root suggests the presence of a periodontal abscess, whereas apical rarefaction suggests a periapical abscess. However, acute periodontal abscesses that show no radiographic changes often cause symptoms in teeth with long-standing, radiographically detectable periapical lesions that are not contributing to the patient's complaint. Clinical findings, such as the presence of extensive caries, pocket formation, lack of tooth vitality, and the existence of continuity between the gingival margin and the abscess area, often prove to be of greater diagnostic value than radiographic appearance.

A draining sinus on the lateral aspect of the root suggests periodontal rather than apical involvement; a sinus from a periapical lesion is more likely to be located further apically. However, sinus location is not conclusive. In many instances, particularly in children, the sinus from a periapical lesion drains on the side of the root rather than at the apex (see Chapter 51).

For more information on laboratory aids to clinical diagnosis, please visit the companion Expert CONSULT website at www.expertconsult.com.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

All patients should have a comprehensive periodontal evaluation that is recorded in the chart. This, at a minimum, includes probing data on six surfaces on each tooth, as well as recording gingival recession and bleeding on probing. Other parameters that need to be recorded include mobility, furcation involvement, mucogingival deficiencies, plaque scores, open contacts, and examination of functional occlusal relationships. A comprehensive medical and dental history must accompany the clinical protocol. A full-mouth series of radiographs taken within the last 2 years with any needed updates is necessary to achieve a correct diagnosis and prepare the appropriate treatment plan.

Periodontal screening and recording systems have little value in managing patients in a private practice because they do not give sufficient specific data for individual patient treatment planning.

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CHAPTER 31

Radiographic Aids in the Diagnosis of Periodontal Disease

Sotirios Tetradis, Fermin A. Carranza, Robert C. Fazio, and Henry H. Takei

CHAPTER OUTLINE

NORMAL INTERDENTAL BONE
RADIOGRAPHIC TECHNIQUES
BONE DESTRUCTION IN PERIODONTAL DISEASE
Bone Loss
Pattern of Bone Destruction
RADIOGRAPHIC APPEARANCE OF PERIODONTAL DISEASE
Periodontitis
Interdental Craters
Furcation Involvement
Periodontal Abscess
Clinical Probing
Localized Aggressive Periodontitis
Trauma from Occlusion
ADDITIONAL RADIOGRAPHIC CRITERIA (online only)
SKELETAL DISTURBANCES MANIFESTED IN THE JAWS (online only)
DIGITAL INTRAORAL RADIOGRAPHY
ADVANCED IMAGING MODALITIES

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Some figures may be out of numeric order in this printed chapter.

Radiographs are valuable for diagnosis of periodontal disease, estimation of severity, determination of prognosis, and evaluation of treatment outcome. *However, radiographs are an adjunct to the clinical examination, not a substitute for it.*

Radiographs demonstrate changes in calcified tissue; they do not reveal current cellular activity but rather reflect the effects of past cellular experience on the bone and roots.

NORMAL INTERDENTAL BONE

Evaluation of bone changes in periodontal disease is based mainly on the appearance of the interdental bone because the relatively dense root structure obscures the facial and lingual bony plates. The interdental bone normally is outlined by a thin, radiopaque line adjacent to the periodontal ligament (PDL) and at the alveolar crest, referred to as the *lamina dura* (Figure 31-1). *Because the lamina dura represents the cortical bone lining the tooth socket, the shape and position of the root and changes in the angulation of the x-ray beam produce considerable variations in its appearance.*[15]

The width and shape of the interdental bone and the angle of the crest normally vary according to the convexity of the proximal tooth surfaces and the level of the cementoenamel junction (CEJ) of the approximating teeth.[29] The faciolingual diameter of the bone is related to the width of the proximal root surface. The angulation of the crest of the interdental septum is generally parallel to a line between the CEJs of the approximating teeth (see Figure 31-1). When there is a difference in the level of the CEJs, the crest of the interdental bone appears angulated rather than horizontal.

RADIOGRAPHIC TECHNIQUES

In conventional radiographs, periapical and bite-wing projections offer the most diagnostic information and are most commonly used in the evaluation of periodontal disease. To properly and accurately depict periodontal bone status, proper techniques of exposure and development are required. The bone level, pattern of bone destruction, PDL space width,[33] as well as the radiodensity, trabecular pattern, and marginal contour of the interdental bone, vary by modifying exposure and development time, type of film, and x-ray angulation.[17] Standardized, reproducible techniques are required to obtain reliable radiographs for pretreatment and posttreatment comparisons.[16,24,31]

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Prichard[23] established the following four criteria to determine adequate angulation of periapical radiographs:

1. The radiograph should show the tips of molar cusps with little or none of the occlusal surface showing.
2. Enamel caps and pulp chambers should be distinct.
3. Interproximal spaces should be open.
4. Proximal contacts should not overlap unless teeth are out of line anatomically.

For periapical radiographs, the long-cone paralleling technique most accurately projects the alveolar bone level[8] (Figure 31-2). The bisection-of-the-angle technique elongates the projected image, making the bone margin appear closer to the crown; the level of the facial bone is distorted more than that of the lingual. Inappropriate horizontal angulation results in tooth overlap, changes the shape of the interdental bone image, alters the radiographic width of the PDL space and the appearance of the lamina dura, and may distort the extent of furcation involvement (see Figure 31-2).[19]

Periapical radiographs frequently do not reveal the correct relationship between the alveolar bone and the CEJ. This is particularly true in cases in which a shallow palate or floor of the mouth does not allow ideal placement of the periapical film. Bite-wing projections offer an alternative that better images periodontal bone levels. For bite-wing radiographs, the film is placed behind the crowns of the upper and lower teeth parallel to the long axis of the teeth. The x-ray beam is directed through the contact areas of the teeth and perpendicular to the film.[35] Thus the projection geometry of the bite-wing films allows the evaluation of the relationship between the interproximal alveolar crest and the CEJ without distortion (Figures 31-3 and 31-4). If the periodontal bone loss is severe and the bone level cannot be visualized on regular bite-wing radiographs, films can be placed vertically to cover a larger area of the jaws (Figure 31-5). More than two vertical bite-wing films might be necessary to cover all the interproximal spaces of the area of interest.

Figure 31-1 Crest of interdental bone normally parallel to a line drawn between the cementoenamel junction of adjacent teeth *(arrow)*. Note also the radiopaque lamina dura around the roots and interdental bone.

BONE DESTRUCTION IN PERIODONTAL DISEASE

Early destructive changes of bone that do not remove sufficient mineralized tissue cannot be captured on radiographs.[3,4,25] Therefore slight radiographic changes of the periodontal tissues suggest that the disease has progressed beyond its earliest stages. *The earliest signs of periodontal disease must be detected clinically.*

Bone Loss

The radiographic image tends to underestimate the severity of bone loss.[28,32] The difference between the alveolar crest height and the radiographic appearance ranges from 0 mm to 1.6 mm,[27] mostly accounted for by x-ray angulation.

Amount. Radiographs are an indirect method for determining the amount of bone loss in periodontal disease; they image the amount of remaining bone rather than the amount lost. The amount of bone lost is estimated to be the difference between the physiologic bone level and the height of the remaining bone.

The distance from the CEJ to the alveolar crest has been analyzed by several investigators.[12,14,27] Most studies, conducted in adolescents, suggest a distance of 2 mm to reflect normal periodontium; this distance may be greater in older patients.

Distribution. The distribution of bone loss is an important diagnostic sign. It points to the location of destructive local factors

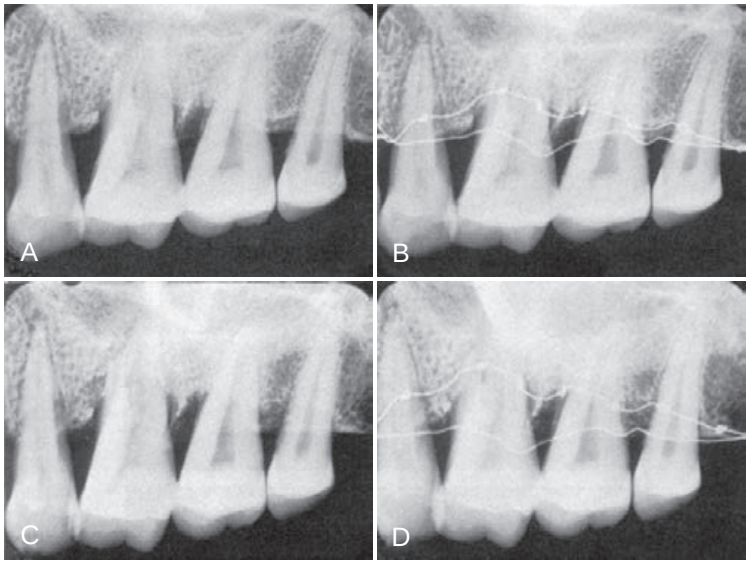

Figure 31-2 Comparison of long-cone paralleling and bisection-of-the-angle techniques. **A,** Long-cone paralleling technique, radiograph of dried specimen. **B,** Long-cone paralleling technique, same specimen as **A.** Smooth wire is on margin of the facial plate and knotted wire is on the lingual plate to show their relative positions. **C,** Bisection-of-the-angle technique, same specimen as **A** and **B. D,** Bisection-of-the-angle technique, same specimen. Both bone margins are shifted toward the crown, the facial margin (smooth wire) more than the lingual margin (knotted wire), creating the illusion that the lingual bone margin has shifted apically. *(Courtesy Dr. Benjamin Patur, Hartford, CT.)*

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in different areas of the mouth and in relation to different surfaces of the same tooth.

Pattern of Bone Destruction

In periodontal disease the interdental bone undergoes changes that affect the lamina dura, crestal radiodensity, size and shape of the medullary spaces, and height and contour of the bone. Height of interdental bone may be reduced, with the crest perpendicular to the long axis of the adjacent teeth (*horizontal* bone loss; Figure 31-6), or angular or arcuate defects (*angular,* or *vertical,* bone loss; Figure 31-7) could form (see Chapter 14).

Radiographs do not indicate the internal morphology or depth of craterlike defects. Also, radiographs do not reveal the extent of involvement on the facial and lingual surfaces. Bone destruction of facial and lingual surfaces is masked by the dense root structure, and bone destruction on the mesial and distal root surfaces may be partially hidden by superimposed anatomy, such as a dense mylohyoid ridge (Figure 31-8). In most cases, it can be assumed that

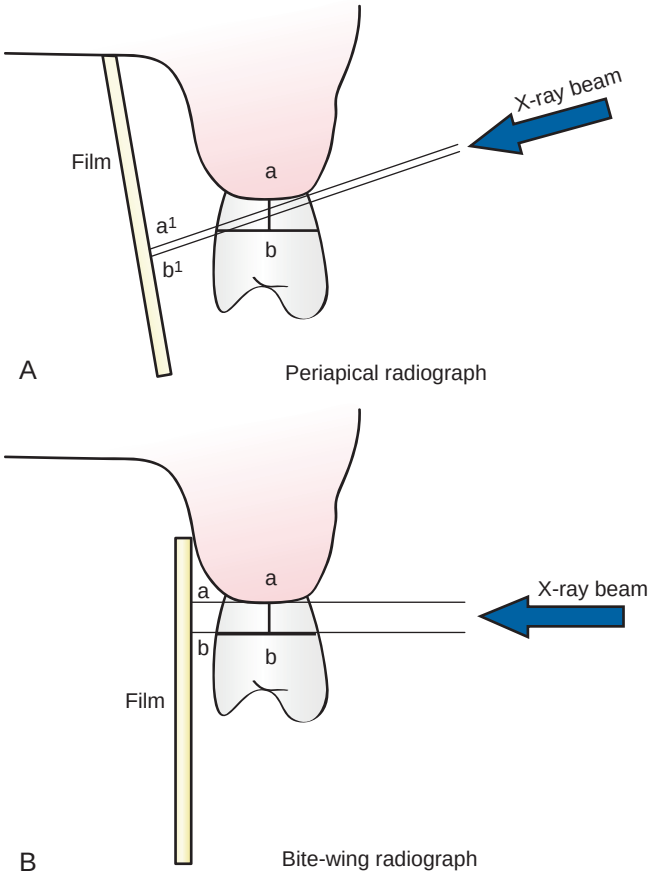

Figure 31-3 Schematic diagram of periapical **(A)** and bite-wing **(B)** radiographs. Angulation of the x-ray beam and the film on the periapical radiograph distort the distance between the alveolar crest and the cementoenamel junction (CEJ) (compare a-b versus a^1-b^1). In contrast, the projection geometry of the bite-wing radiograph allows a more accurate depiction (a^2-b^2) of the distance between the alveolar crest and the CEJ (a-b).

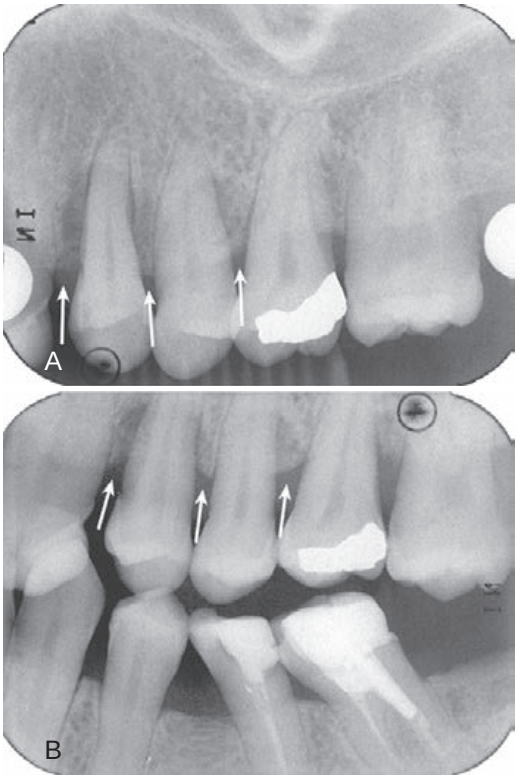

Figure 31-4 Periapical **(A)** and bite-wing **(B)** radiographs from full-mouth series of patient with periodontitis. The periapical film clearly underestimates the amount of bone loss *(white arrows).* Because of appropriate projection geometry, the alveolar crest height is accurately depicted on the bite-wing radiograph *(white arrows).*

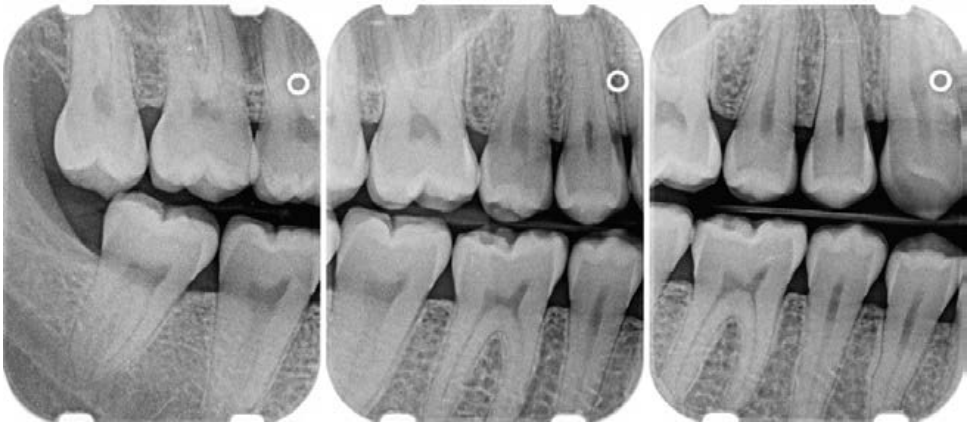

Figure 31-5 Vertical bite-wing films can be used to cover a larger area of the alveolar bone.

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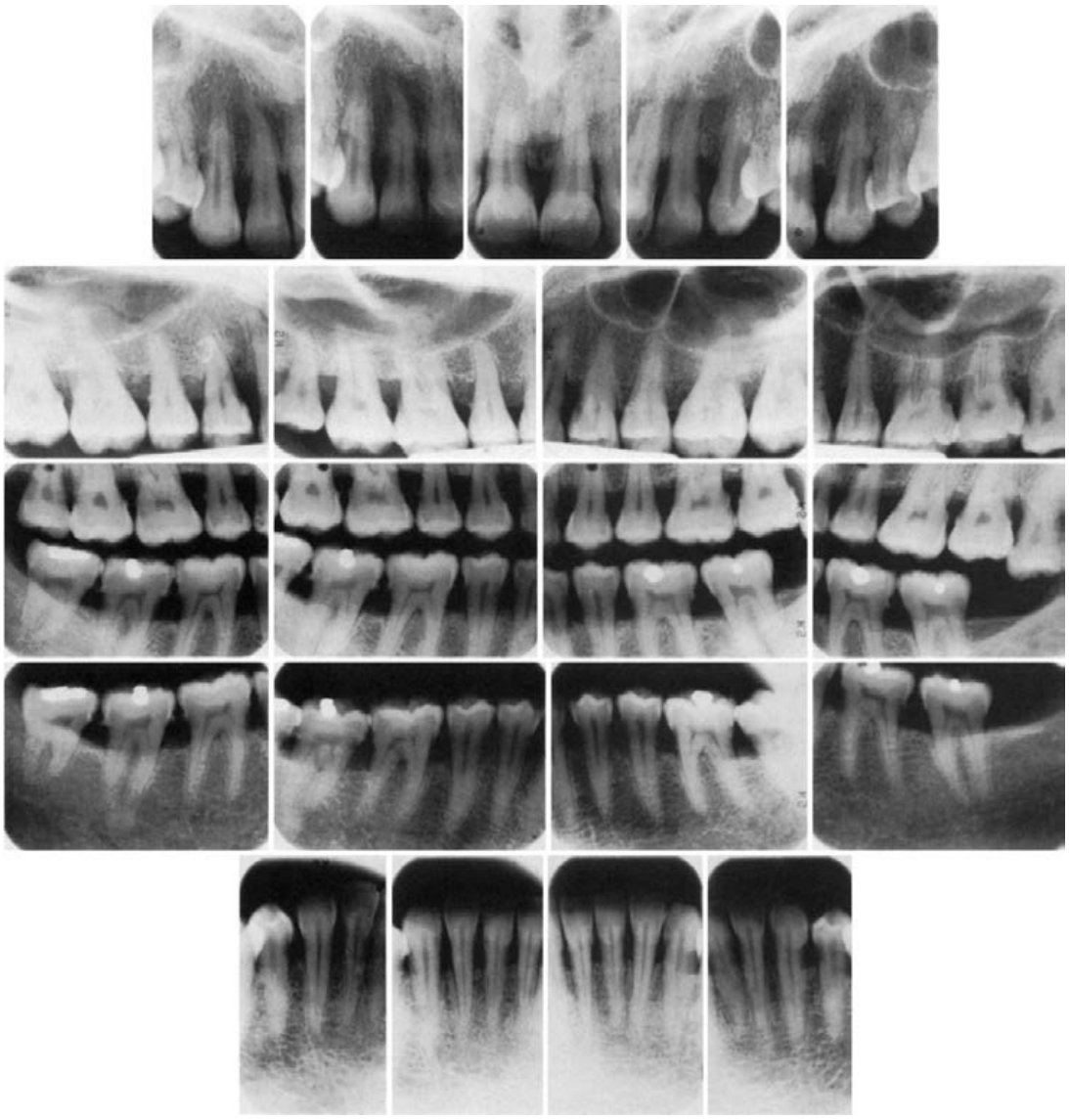

Figure 31-6 Generalized horizontal bone loss.

Figure 31-7 Angular bone loss on first molar with involvement of the furcation.

Figure 31-8 Angular bone loss on mandibular molar partially obscured by dense mylohyoid ridge.

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bone losses seen interdentally continue in either the facial or the lingual aspect, creating a troughlike lesion.

Dense cortical facial and lingual plates of interdental bone obscure destruction of the intervening cancellous bone. Thus a deep craterlike defect between the facial and lingual plates might not be depicted on conventional radiographs. To record destruction of the interproximal cancellous bone radiographically, the cortical bone must be involved. A reduction of only 0.5 to 1.0 mm in the thickness of the cortical plate is sufficient to permit radiographic visualization of destruction of the inner cancellous trabeculae.[20]

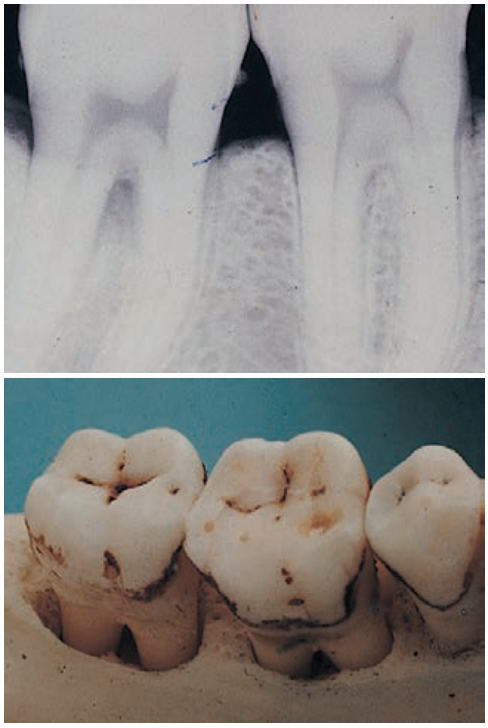

Figure 31-9 Interdental lesion that extends to the facial or lingual surfaces in a troughlike manner.

Interdental bone loss may continue facially and/or lingually to form a troughlike defect that could be difficult to appreciate radiographically. These lesions may terminate on the radicular surface or may communicate with the adjacent interdental area to form one continuous lesion (Figure 31-9).

Figure 31-10 shows two adjacent interdental lesions connecting on the radicular surface to form one interconnecting osseous lesion. Along with clinical probing of these lesions, the use of a radiopaque pointer placed in these radicular defects will demonstrate the extent of the bone loss.

RADIOGRAPHIC APPEARANCE OF PERIODONTAL DISEASE

Periodontitis

Radiographic changes in periodontitis follow the pathophysiology of periodontal tissue destruction and include the following:

1. *Fuzziness and disruption of lamina dura* crestal cortication continuity is the earliest radiographic change in periodontitis (Figure 31-11, *A* and *B*) and results from bone resorption activated by extension of gingival inflammation into the periodontal bone. Depicting these early changes depends greatly on the radiographic technique, as well as on anatomic variations (thickness and density of interdental bone, position of adjoining teeth). No correlation has been found between crestal lamina dura in radiographs and the presence or absence of clinical inflammation, bleeding on probing,

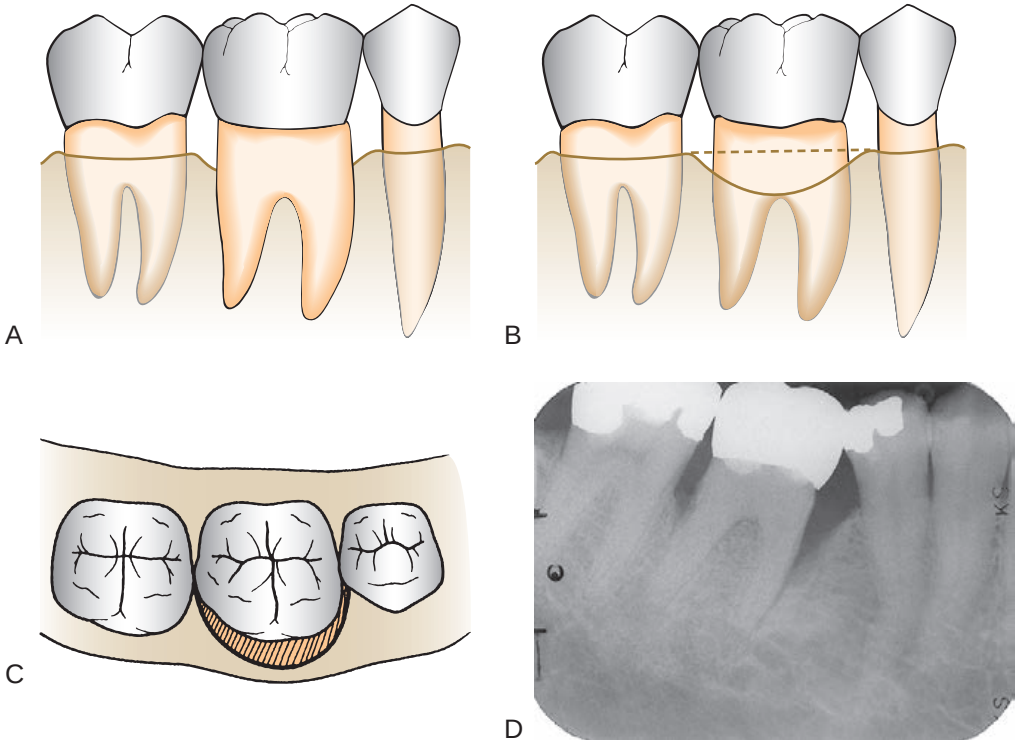

Figure 31-10 **A,** Interdental mesial and distal lesions. **B,** Facial or lingual outlines of actual lesion. **C,** Occlusal view of lesion. **D,** Radiograph of mesial and facial lesions.

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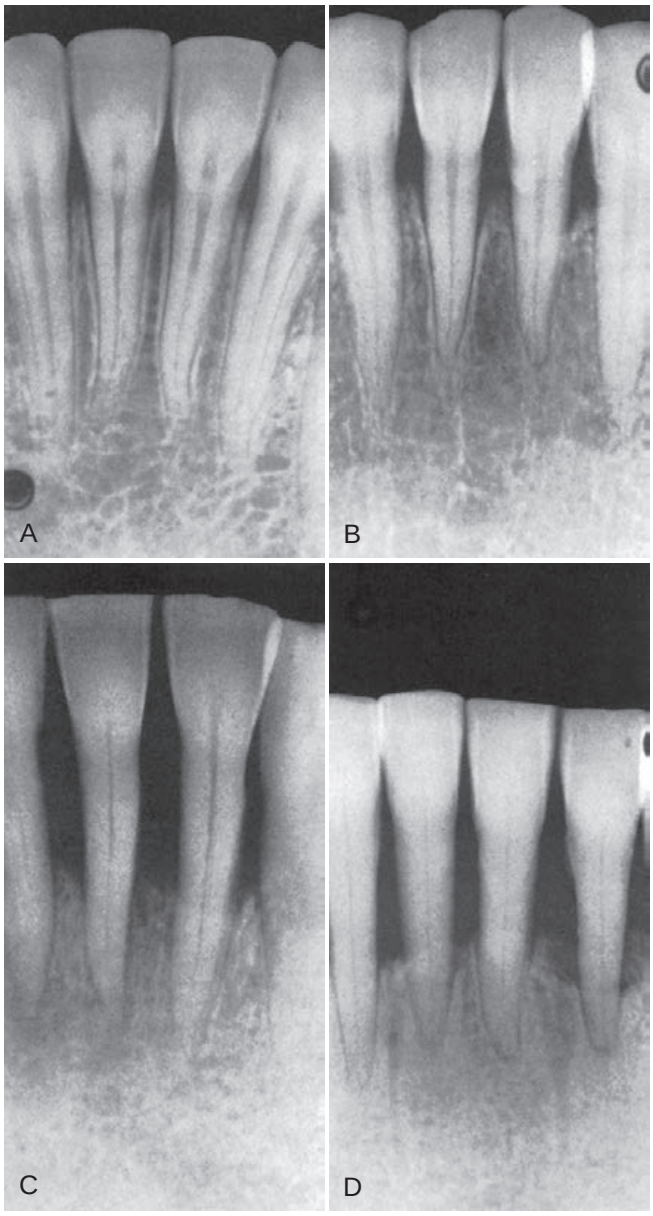

Figure 31-11 Radiographic changes in periodontitis. **A,** Normal appearance of interdental bone. **B,** Fuzziness and a break in the continuity of the lamina dura at the crest of the bone distal to the central incisor *(left)*. There are wedge-shaped radiolucent areas at the crest of the other interdental bone. **C,** Radiolucent projections from the crest into the interdental bone indicate extension of destructive processes. **D,** Severe bone loss.

periodontal pockets, or loss of attachment.[11,26] Therefore it can be concluded that the presence of an intact crestal lamina dura may be an indicator of periodontal health, whereas its absence lacks diagnostic relevance.[2]

2. Continued periodontal bone loss and widening of the periodontal space results in *a wedge-shaped radiolucency* at the mesial or distal aspect of the crest (Figure 31-11, *B*). The apex of the area is pointed in the direction of the root.
3. Subsequently, the destructive process extends across the alveolar crest thus *reducing the height* of the interdental bone. As increased osteoclastic activity results in increased bone resorption along the endosteal margins of the medullary spaces, the remaining interdental bone can appear partially eroded (Figure 31-11, *C*).

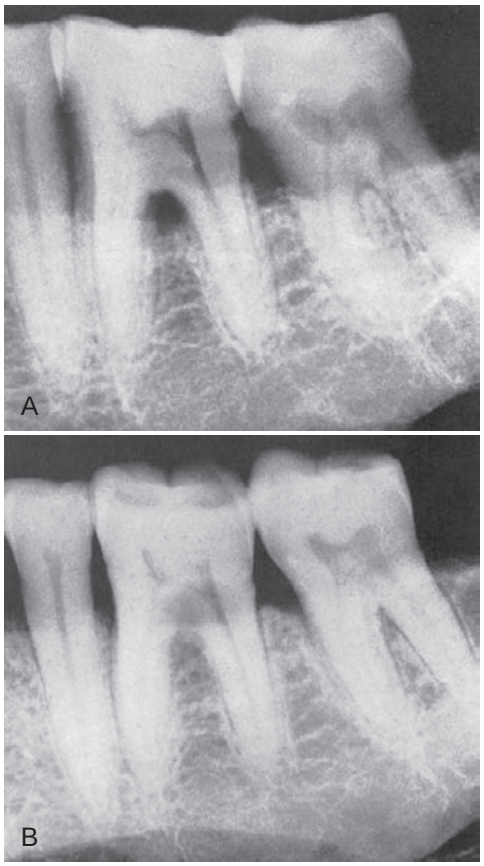

Figure 31-12 **A,** Furcation involvement indicated by triangular radiolucency in bifurcation area of mandibular first molar. The second molar presents only a slight thickening of the periodontal space in the bifurcation area. **B,** Same area as **A,** different angulation. The triangular radiolucency in the bifurcation of the first molar is obliterated, and involvement of the second molar bifurcation is apparent.

4. The height of the interdental septum is progressively reduced by the extension of inflammation and the resorption of bone (Figure 31-11, *D*).

Interdental Craters

Interdental craters are seen as irregular areas of reduced density on the alveolar bone crests.[28] Craters are generally not sharply demarcated but gradually blend with the rest of the bone. Conventional radiographs do not accurately depict the morphology or depth of interdental craters, which sometimes appear as vertical defects.

Furcation Involvement

Definitive diagnosis of furcation involvement is made by clinical examination, which includes careful probing with a specially designed probe (e.g., Nabers). Radiographs are helpful, but root superimposition, caused by anatomic variations and/or improper technique, can obscure radiographic representation of furcation involvement. As a general rule, bone loss is greater than it appears in the radiograph. A tooth may present marked bifurcation involvement in one film (Figure 31-12, *A*) but appear to be uninvolved in another (Figure 31-12, *B*). Radiographs should be taken at different angles to reduce the risk of missing furcation involvement.

The recognition of a large, clearly defined radiolucency in the furcation area is easy to identify (see Figure 31-12, *A*), but less clearly defined radiographic changes are often overlooked. To assist

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Figure 31-13 Early furcation involvement suggested by fuzziness in the bifurcation of the mandibular first molar, particularly when associated with bone loss on the roots.

Figure 31-14 Furcation involvement of mandibular first and second molars indicated by thickening of periodontal space in furcation area. The furcation of the third molar is also involved, but the thickening of the periodontal space is partially obscured by the external oblique line.

in the radiographic detection of furcation involvement, the following diagnostic criteria are suggested:

1. The slightest radiographic change in the furcation area should be investigated clinically, especially if there is bone loss on adjacent roots (Figure 31-13).
2. Diminished radiodensity in the furcation area in which outlines of bony trabeculae are visible suggests furcation involvement (Figure 31-14).
3. Whenever there is marked bone loss in relation to a single molar root, it may be assumed that the furcation is also involved (Figure 31-15).

Periodontal Abscess

The typical radiographic appearance of the periodontal abscess is a discrete area of radiolucency along the lateral aspect of the root (Figures 31-16 and 31-17). *However, the radiographic picture is often not characteristic* (Figure 31-18). This can be due to the following:

1. *The stage of the lesion.* In the early stages the acute periodontal abscess is extremely painful but presents no radiographic changes.
2. *The extent of bone destruction and the morphologic changes of the bone.*
3. *The location of the abscess.* Lesions in the soft tissue wall of a periodontal pocket are less likely to produce radiographic changes than those deep in the supporting tissues. Abscesses on the facial or lingual surface are obscured by the radiopacity of the root; interproximal lesions are more likely to be visualized radiographically.

Figure 31-15 Furcation involvement of the first molar partially obscured by the radiopaque lingual root. The horizontal line across the distobuccal root demarcates the apical portion *(arrow),* which is covered by bone, from the remainder of the root, where the bone has been destroyed.

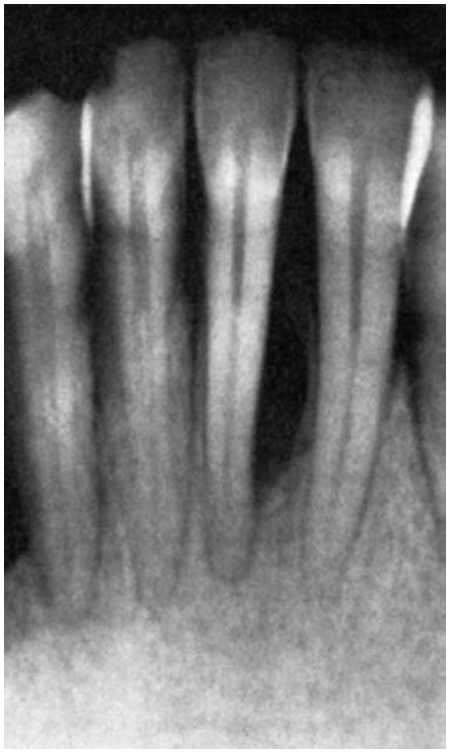

Figure 31-16 Radiolucent area on lateral aspect of root with chronic periodontal abscess.

Therefore radiographs alone cannot provide final diagnosis of a periodontal abscess but need to be accompanied by careful clinical examination.

Clinical Probing

Regenerative and resective flap designs and incisions require prior knowledge of the underlying osseous topography. Careful probing of these pocket areas after scaling and root planing often require local anesthesia and definitive radiographic evaluation of the osseous lesions. Radiographs taken with periodontal probes or

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Figure 31-17 Typical radiographic appearance of periodontal abscess on right central incisor.

other indicators (e.g., Hirschfeld pointers) placed into the anesthetized pocket show the true extent of the bone lesion. As indicated previously, the attachment level on the radicular surface or interdental lesions with thick facial or lingual bone cannot be visualized in the radiograph. The use of radiopaque indicators is an efficient diagnostic aid (Figure 31-19).

Localized Aggressive Periodontitis

Localized aggressive (formerly "localized juvenile") periodontitis is characterized by the following:

1. Initially, bone loss in the maxillary and mandibular incisor and/or first molar areas, usually bilaterally, resulting in vertical, arclike destructive patterns (Figure 31-20).
2. As the disease progresses, loss of alveolar bone may become generalized but remains less pronounced in the premolar areas.

Trauma from Occlusion

Trauma from occlusion can produce radiographically detectable changes in the thickness of the lamina dura, morphology of the

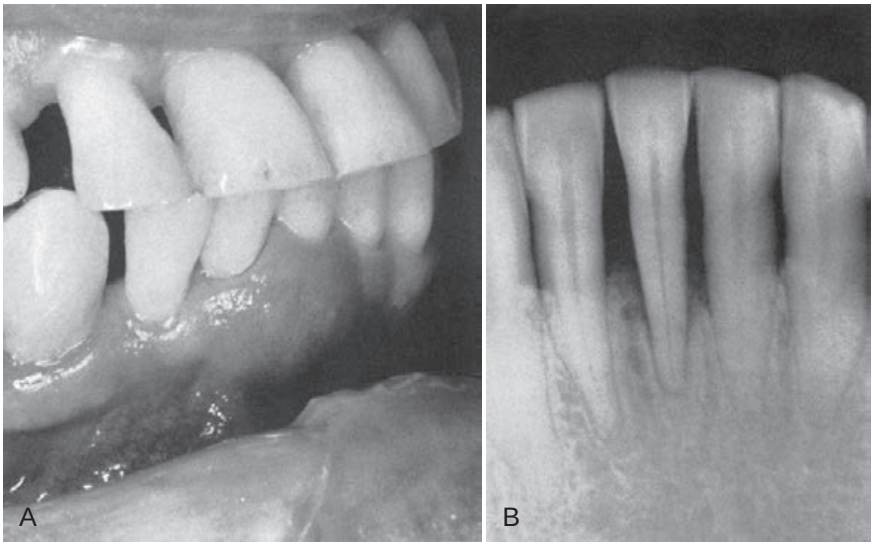

Figure 31-18 Chronic periodontal abscess. **A,** Periodontal abscess in the right central and lateral incisor area. **B,** Extensive bone destruction and thickening of the periodontal ligament space around the right central incisor.

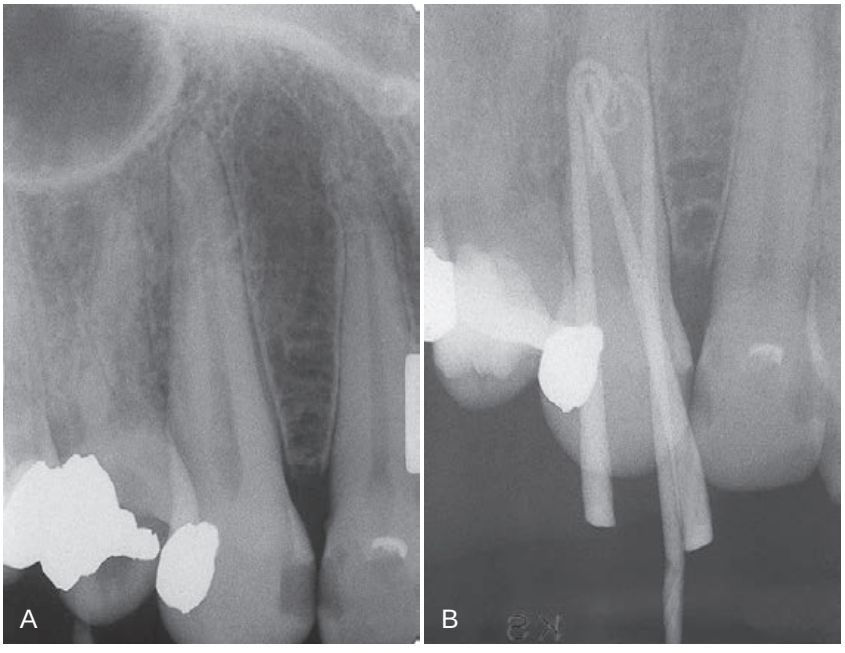

Figure 31-19 **A,** Radiograph of maxillary cuspid. This view does not show facial bone loss. **B,** Radiograph of same maxillary cuspid as **A,** with gutta percha points placed in the facial pocket to indicate bone loss.

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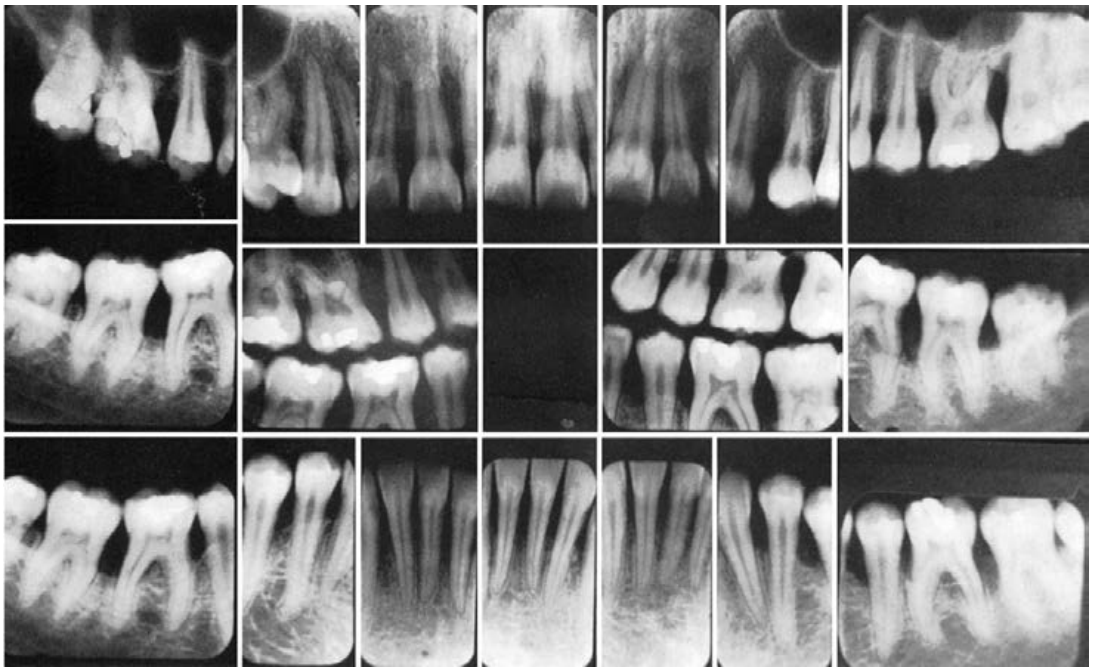

Figure 31-20 Localized aggressive periodontitis. The accentuated bone destruction in the anterior and first molar areas is considered to be characteristic of this disease.

alveolar crest, width of the PDL space, and density of the surrounding cancellous bone.

Traumatic lesions manifest more clearly in faciolingual aspects because mesiodistally the tooth has the added stability provided by the contact areas with adjacent teeth. Therefore slight variations in the proximal surfaces may indicate greater changes in the facial and lingual aspects. *The radiographic changes listed next are not pathognomonic of trauma from occlusion and must be interpreted in combination with clinical findings,* particularly tooth mobility, presence of wear facets, pocket depth, and analysis of occlusal contacts and habits.

The *injury phase* of trauma from occlusion produces a loss of the lamina dura that may be noted in apices, furcations, and marginal areas. This loss of lamina dura results in widening of the PDL space (Figure 31-21). *The repair phase* of trauma from occlusion results in an attempt to strengthen the periodontal structures to better support the increased loads. Radiographically, this is manifested by a widening of the PDL space, which may be generalized or localized.

Although microscopic measurements have determined normal variations in the PDL space width along the root surface, these are generally not detected radiographically. Thus, when seen on the radiographs, PDL space variations in width suggest that the tooth is being subjected to increased forces. Successful attempts to reinforce the periodontal structures by widening of the PDL space is accompanied by increased width of the lamina dura and sometimes by condensation of the perialveolar cancellous bone.

More advanced traumatic lesions may result in deep angular bone loss, which, when combined with marginal inflammation, may lead to intrabony pocket formation. In terminal stages, these lesions extend around the root apex, producing a wide, radiolucent periapical image (cavernous lesions).

Root resorption may also result from excessive forces on the periodontium, particularly those caused by orthodontic appliances. Although trauma from occlusion produces many areas of root resorption, these areas are usually of a magnitude insufficient to be detected radiographically.

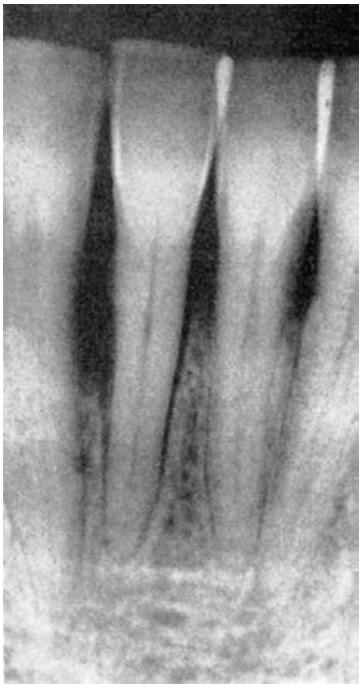

Figure 31-21 Widened periodontal space caused by trauma from occlusion. Note the increased density of the surrounding bone caused by new bone formation in response to increased occlusal forces.

For more information on additional radiographic criteria, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on skeletal disturbances manifested in the jaws, please visit the companion Expert CONSULT website at www.expertconsult.com.

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SCIENCE TRANSFER

Periapical radiographic examination should be part of each patient's periodontal evaluation and should be coupled with a detailed recording of pocket depths, gingival margin location, and bleeding on probing. Radiographic evaluation should be updated every 2 years. Periapical radiographs often underestimate the amount of periodontal bone loss, and early changes are usually not detected. Significant interdental bone loss can occur and may not be detectable on periapical radiographs because the density of the intact buccal and lingual or palatal bone plates obscure changes that occur as the result of periodontitis. Comparison of periapical radiographs of the same area taken at different times will only be reliable in documenting dramatic changes in bone levels since variations in angulation of the beam, placement of the film, and development of the image make accurate measurements taken over time very difficult and unreliable.

Recent use of three-dimensional radiographic techniques with cone beam computed tomography (CBCT) gives a much more accurate picture of periodontal bone loss than two-dimensional radiographs and will be more widely used as this technology becomes available in more clinics.

DIGITAL INTRAORAL RADIOGRAPHY

Intraoral digital radiographs offer several advantages over conventional film-based radiographs in clinical dentistry. The number of dentists using intraoral digital radiographs increases steadily, and some practices are now completely digital. Telediagnosis, videoconferencing, rapid image transmission among dentists and third parties, a seamless electronic patient record, and integration with computer-aided diagnostic software are areas in which digital information is revolutionizing dentistry.[36,37]

Two major digital intraoral systems are currently available.[18] The first system uses charge-coupled devices (CCDs) or complementary metal oxide semiconductor (CMOS) receptors as detectors. These detectors are placed in the patient's mouth and are linked by a wire to the computer. On radiation exposure, virtually in real time, the radiographic image appears on a computer screen. The detector is then moved to the next position, and so on, until the whole area of interest is imaged. The second system uses photostimulable phosphor (PSP) plates as detectors. PSP plates resemble film with one of the sides lined with a PSP coating. When interacting with x-rays, PSP stores energy, which it then releases on stimulation by light of an appropriate wavelength. PSP plates are placed and exposed similar to regular film. The exposed plates are placed on a plate scanner and scanned by a laser beam, and the radiographic image appears on the computer screen.[18]

Once captured and displayed, computer software can be used to enhance the digital image and increase its diagnostic efficacy.[21] Exposure adjustment provides a balanced image that utilizes the complete scale of gray levels and does not suppress diagnostic information (Figure 31-27, compare *A* and *B*). A sharpening (edge enhancement) filter increases the definition and separation of adjacent structures (Figure 31-27, compare *B* and *C*). Inversion filters provide a negative of the image that sometimes might reveal disease not seen on the positive image (Figure 31-27, *D*). The ability to magnify the image allows close examination of the area of interest and detection of occult disease (Figure 31-27, *E*). Different image qualities allow better detection of dental disease. For example, high-contrast images favor caries detection, whereas low-contrast images permit a more accurate depiction of the alveolar crest. Digital images can be adjusted such that the contrast of the interproximal contacts can be enhanced for caries detection while the remainder of the image is not altered (Figure 31-27, *F*).

Advantages of intraoral digital radiography include the speed of image capture and display; low x-ray exposure; ability to manipulate the image and maximize diagnostic efficacy; use of digital tools, such as linear, angular, and density measurements; improved patient education; ease of storage, transfer, and copying; and seamless integration with electronic patient record management or other software. Most studies conclude that the aid provided by digital radiographs in the diagnosis of common dental diseases such as caries is similar to that of conventional radiographs.

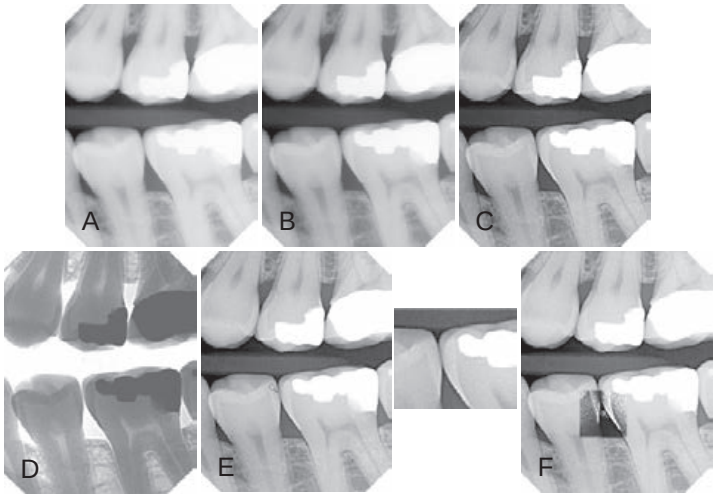

Figure 31-27 Various enhancement features help optimize the appearance of the radiographic image to improve diagnosis. Brightness and contrast of an underexposed image **(A)** is adjusted **(B)** to utilize the full gray scale. **C,** Sharpness of the image is then increased using "clear view" feature. D, "Invert" feature provides a negative of the image. **E,** "Zooming" function allows close examination of the interproximal space. **F,** Contrast of the interproximal area is maximized to aid in the diagnosis of possible caries. *(Courtesy Dexis Digital Diagnostic Imaging, Alpharetta, GA.)*

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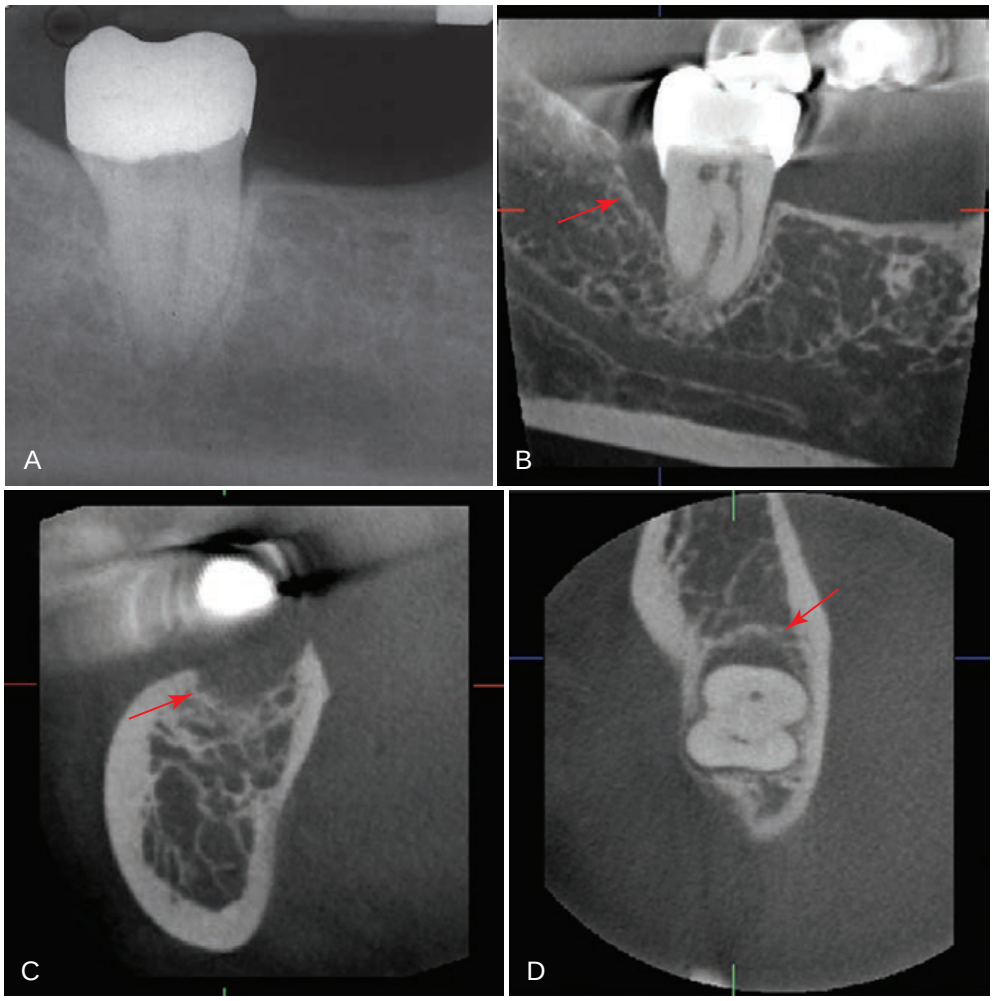

Figure 31-28 Periapical radiograph **(A)** and sagittal **(B),** cross-sectional **(C),** and axial **(D)** cone-beam computed tomography (CBCT) sections of tooth #31. No pathology is detected on the periapical radiograph. However, CBCT images clearly illustrate a deep vertical three-wall defect on the distal surface of #31 *(red arrow).*

However, the proper use of digital intraoral radiographs requires familiarity with the digital nature of the images and an understanding of the principles of image manipulation by computer software.[35] Although contrast and brightness adjustments can be used to refine the image, they cannot correct a grossly overexposed or underexposed projection. The ease of digital image acquisition should not replace correct detector placement and exposure techniques. Image enhancement should be used with caution and should be task oriented. For example, increased contrast might aid in caries diagnosis but lead to underestimation of the alveolar bone crest height. If properly used, intraoral digital radiography offers great benefits in the diagnosis of dental disease.[35]

ADVANCED IMAGING MODALITIES

In the last decade, cone beam computed tomography (CBCT) has revolutionized the field of oral and maxillofacial imaging. CBCT technology and its use in evaluation of the implant patient are discussed in Chapter 71. However, CBCT finds application in almost every diagnostic task of clinical dentistry, including evaluation of periodontal and periapical structures. CBCT offers many advantages over conventional radiography, including the accurate three-dimensional imaging of teeth and supporting structures. Although not recommended for every dental patient, CBCT avoids the problems of geometric superimposition and unpredictable magnification and can provide valuable diagnostic information in periodontal evaluation.[31]

As discussed earlier, periapical and bite-wing radiographs provide information mostly for the interdental bone. However, a three-wall defect that preserves the buccal and/or lingual cortices can be difficult to diagnose, and the buccal, lingual and furcational periodontal bone levels are hard to evaluate in conventional radiographs. When clinical examination raises concerns for such areas, CBCT imaging can add diagnostic value. Figure 31-28 illustrates the CBCT diagnostic advantage in the evaluation of a three wall defect in the distal surface of tooth #31. Periapical radiograph (Figure 31-28, *A*) presents normal bone height at the mesial and distal surface of #31. CBCT sections (Figure 31-28, *A* to *C*) clearly demonstrate a three-wall defect on the distal surface of #31. Note that the buccal and lingual cortical plates are well preserved. For this reason, the periodontal defect is not depicted by the periapical radiograph.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 32

Clinical Risk Assessment

Karen F. Novak and M. John Novak

CHAPTER OUTLINE

DEFINITIONS
RISK FACTORS FOR PERIODONTAL DISEASE
Tobacco Smoking
Diabetes
Pathogenic Bacteria and Microbial Tooth Deposits
RISK DETERMINANTS/BACKGROUND CHARACTERISTICS FOR PERIODONTAL DISEASE
Genetic Factors
Age
Gender
Socioeconomic Status
Stress
RISK INDICATORS FOR PERIODONTAL DISEASE (online only)
Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome
Osteoporosis
Infrequent Dental Visits
RISK MARKERS/PREDICTORS FOR PERIODONTAL DISEASE (online only)
Previous History of Periodontal Disease
Bleeding on Probing
CLINICAL RISK ASSESSMENT FOR PERIODONTAL DISEASE (online only)
CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

DEFINITIONS

Risk assessment is defined by numerous components.[2,29] Risk is the probability that an individual will develop a specific disease in a given period. The risk of developing the disease will vary from individual to individual.

Risk factors may be environmental, behavioral, or biologic factors that, when present, increase the likelihood that an individual will develop the disease. Risk factors are identified through longitudinal studies of patients with the disease of interest. Exposure to a risk factor or factors may occur at a single point in time; over multiple, separate points in time; or continuously. However, to be identified as a risk factor, the exposure must occur before disease onset. Interventions often can be identified and when implemented, can help modify risk factors.

The term *risk determinant/background characteristic*, which is sometimes substituted for the term *risk factor*, should be reserved for those risk factors that cannot be modified.

Risk indicators are *probable* or *putative* risk factors that have been identified in cross-sectional studies but not confirmed through longitudinal studies.

Risk predictors/markers, although associated with increased risk for disease, do not cause the disease.

These factors also are identified in cross-sectional and longitudinal studies.

Box 32-1 lists elements of these categories of risk for periodontal disease.

RISK FACTORS FOR PERIODONTAL DISEASE

Tobacco Smoking

Tobacco smoking is a well-established risk factor for periodontitis.[2,32] A direct relationship exists between smoking and the prevalence of periodontal disease (see Chapter 26). This association is independent of other factors such as oral hygiene or age.[18] Studies comparing the response to periodontal therapy in smokers, previous smokers, and nonsmokers have shown that smoking has a negative impact on the response to therapy. However, former smokers respond similarly to nonsmokers.[3] These studies demonstrate the

BOX 32-1 Categories of Risk Elements for Periodontal Disease

Risk Factors
Tobacco smoking
Diabetes
Pathogenic bacteria
Microbial tooth deposits

Risk Determinants/Background Characteristics
Genetic factors
Age
Gender
Socioeconomic status
Stress

Risk Indicators
HIV/AIDS
Osteoporosis
Infrequent dental visits

Risk Markers/Predictors
Previous history of periodontal disease
Bleeding on probing

HIV, Human immunodeficiency virus; *AIDS,* acquired immunodeficiency syndrome.

therapeutic impact of intervention strategies on patients who smoke (see Chapter 26).

Diabetes

Diabetes is a clear risk factor for periodontitis.[2] Epidemiologic data demonstrate that the prevalence and severity of periodontitis are significantly higher in patients with type 1 or type 2 diabetes mellitus than in those without diabetes, and that the level of diabetic control is an important variable in this relationship (see Chapter 5).

Pathogenic Bacteria and Microbial Tooth Deposits

It is well documented that accumulation of bacterial plaque at the gingival margin results in the development of gingivitis and that the gingivitis can be reversed with the implementation of oral hygiene measures.[25] These studies demonstrate a causal relationship between accumulation of bacterial plaque and gingival inflammation. However, a causal relationship between *plaque accumulation* and *periodontitis* has been more difficult to establish. Often, patients with severe loss of attachment have minimal levels of bacterial plaque on the affected teeth, indicating that the *quantity* of plaque is not of major importance in the disease process. However, although quantity may not indicate risk, there is evidence that the composition, or *quality,* of the complex plaque biofilm is of importance.

In terms of quality of plaque, three specific bacteria have been identified as etiologic agents for periodontitis: *Aggregatibacter actinomycetemcomitans* (formerly *Actinobacillus actinomycetemcomitans*), *Porphyromonas gingivalis,* and *Tannerella forsythia* (formerly *Bacteroides forsythus*).[11] *P. gingivalis* and *T. forsythia* are often found in chronic periodontitis, whereas *A. actinomycetemcomitans* is often associated with aggressive periodontitis. Cross-sectional and longitudinal studies support the delineation of these three bacteria as risk factors for periodontal disease. Additional evidence that these organisms are causal agents includes the following[16]:

1. Their elimination or suppression impacts the success of therapy.
2. There is a host response to these pathogens.
3. Virulence factors are associated with these pathogens.
4. Inoculation of these bacteria into animal models induces periodontal disease.

Although not completely supported by these criteria for causation, moderate evidence also suggests that *Campylobacter rectus, Eubacterium nodatum, Fusobacterium nucleatum, Prevotella intermedia/nigrescens, Peptostreptococcus micros, Streptococcus intermedius, and Treponema denticola* are etiologic factors in periodontitis.[11]

Therefore the quantity of plaque present may not be as important as the quality of the plaque in determining risk for periodontitis.

Anatomic factors, such as furcations, root concavities, developmental grooves, cervical enamel projections, enamel pearls, and bifurcation ridges, may predispose the periodontium to disease as a result of their potential to harbor bacterial plaque and present a challenge to the clinician during instrumentation. Similarly, the presence of subgingival and overhanging margins can result in increased plaque accumulation, increased inflammation, and increased bone loss. Although not clearly defined as risk factors for periodontitis, anatomic factors and *restorative factors* that influence plaque accumulation may play a role in disease susceptibility for specific teeth.[7]

The presence of *calculus,* which serves as a reservoir for bacterial plaque, has been suggested as a risk factor for periodontitis. Although the presence of some calculus in healthy individuals receiving routine dental care does not result in significant loss of attachment, the presence of calculus in other groups of patients, such as those not receiving regular care and patients with poorly controlled diabetes, can have a negative impact on periodontal health.[29]

RISK DETERMINANTS/BACKGROUND CHARACTERISTICS FOR PERIODONTAL DISEASE

Genetic Factors

Evidence indicates that genetic differences between individuals may explain why some patients develop periodontal disease and others do not. Studies conducted in twins have shown that genetic factors influence clinical measures of gingivitis, probing pocket depth, attachment loss, and interproximal bone height.[26-28] The familial aggregation seen in localized and generalized aggressive periodontitis also is indicative of genetic involvement in these diseases (see Chapter 18).

Kornman et al[21] demonstrated that alterations in specific genes encoding the inflammatory cytokines interleukin-1α (IL-1α) and interleukin-1β (IL-1β) were associated with severe chronic periodontitis in nonsmoking subjects.[21] However, results of other studies have shown limited association between these altered genes and the presence of periodontitis. Overall, it appears that changes in the IL-1 genes may be only one of several genetic changes involved in the risk for chronic periodontitis. Therefore, although the alteration in the IL-1 genes may be a valid marker for periodontitis in defined populations, its usefulness as a genetic marker in the general population may be limited.[20]

Immunologic alterations, such as neutrophil abnormalities,[17] monocytic hyperresponsiveness to lipopolysaccharide stimulation

in patients with localized aggressive periodontitis,[37] and alterations in the monocyte/macrophage receptors for the Fc portion of antibody,[20,47] also appear to be under genetic control. In addition, genetics play a role in regulating the titer of the protective immunoglobulin G2 (IgG2) antibody response to *A. actinomycetemcomitans* in patients with aggressive periodontitis[15] (see Chapter 24).

Age

Both the prevalence and severity of periodontal disease increase with age.[8,31,32] It is possible that degenerative changes related to aging may increase susceptibility to periodontitis. However, it also is possible that the attachment loss and bone loss seen in older individuals are the result of prolonged exposure to other risk factors over a person's life, creating a cumulative effect over time. In support of this, studies have shown minimal loss of attachment in aging subjects enrolled in preventive programs throughout their lives.[33,34] Therefore it is suggested that periodontal disease is not an inevitable consequence of the aging process and that aging alone does not increase disease susceptibility. However, it remains to be determined whether changes related to the aging process, such as intake of medications, decreased immune function, and altered nutritional status, interact with other well-defined risk factors to increase susceptibility to periodontitis.

Evidence of loss of attachment may have more consequences in younger patients. The younger the patient, the longer the patient has for exposure to causative factors. In addition, aggressive periodontitis in young individuals often is associated with an unmodifiable risk factor such as a genetic predisposition to disease.[32] Therefore young individuals with periodontal disease may be at greater risk for continued disease as they age.

Gender

Gender plays a role in periodontal disease.[2] Surveys conducted in the United States since 1960 demonstrate that men have more loss of attachment than women.[42,44,45] In addition, men have poorer oral hygiene than women, as evidenced by higher levels of plaque and calculus.[1,43,45] Therefore gender differences in prevalence and severity of periodontitis appear to be related to preventive practices rather than any genetic factor.

Socioeconomic Status

Gingivitis and poor oral hygiene can be related to lower socioeconomic status (SES).[2,42,44] This can most likely be attributed to decreased dental awareness and decreased frequency of dental visits compared with more educated individuals with higher SES. After adjusting for other risk factors, such as smoking and poor oral hygiene, lower SES alone does not result in increased risk for periodontitis (see Chapter 5).

Stress

The incidence of necrotizing ulcerative gingivitis increases during periods of emotional and physiologic stress, suggesting a link between the two.[10,38] Emotional stress may interfere with normal immune function[6,40] and may result in increased levels of circulating hormones, which can affect the periodontium.[36] Stressful life events, such as bereavement and divorce, appear to lead to a greater prevalence of periodontal disease,[13] and an apparent association exists between psychosocial factors and risk behaviors such as smoking, poor oral hygiene, and chronic periodontitis.[9] Adult patients with periodontitis who are resistant to therapy are more stressed than those who respond to therapy.[5] Individuals with financial strain, distress, depression, or inadequate coping mechanisms have more severe loss of attachment.[12] Although epidemiologic data on the relationship between stress and periodontal disease are limited, stress may be a putative risk factor for periodontitis.[32]

For more information on risk indicators, risk markers/predictors, and risk assessment for periodontal disease, please visit the companion Expert CONSULT website at www.expertconsult.com.

CONCLUSION

Risk assessment involves identifying elements that either may predispose a patient to developing periodontal disease or may influence the progression of disease that already exists. In either case, these patients may require modification of their prognosis and treatment plan. In addition to an evaluation of the factors contributing to their risk, these patients should be educated concerning their risk, and when appropriate, suitable intervention strategies should be implemented.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

The presence of dental plaque over time is the major risk factor for developing periodontitis. The qualitative bacterial makeup of the biofilm also can affect the risk of having periodontal disease. Smoking not only increases the risk of a patient having periodontal bone loss but also negatively affects treatment outcomes. Smoking cessation can overcome the bad effects on treatment prognosis. Smoking patients should be involved in smoking cessation programs so that their periodontal prognosis is optimized. Several genetic factors, including alteration of the genes responsible for interleukin-1 (IL-1) levels, do affect the risk of periodontal disease but apparently do not predict treatment outcomes.

Diabetes and acquired immunodeficiency syndrome (AIDS) are also systemic factors that increase the risk of periodontal destruction. The role of osteoporosis is not clearly documented, and a patient's age plays a role mainly by the increased time the plaque bacteria has in contact with periodontal tissues. Many older patients involved in a lifelong preventive periodontal program show minimal loss of attachment.

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CHAPTER 33

Determination of Prognosis

Karen F. Novak and Henry H. Takei

CHAPTER OUTLINE

DEFINITIONS
TYPES OF PROGNOSIS
Overall Versus Individual Tooth Prognosis
FACTORS IN DETERMINATION OF PROGNOSIS
Overall Clinical Factors
Systemic and Environmental Factors
Local Factors
Prosthetic and Restorative Factors
RELATIONSHIP BETWEEN DIAGNOSIS AND PROGNOSIS
Prognosis for Patients with Gingival Disease
Prognosis for Patients with Periodontitis
REEVALUATION OF PROGNOSIS after PHASE I THERAPY

DEFINITIONS

The *prognosis* is a prediction of the probable course, duration, and outcome of a disease based on a general knowledge of the pathogenesis of the disease and the presence of risk factors for the disease. It is established after the diagnosis is made and before the treatment plan is established. The prognosis is based on specific information about the disease and the manner in which it can be treated, but it also can be influenced by the clinician's previous experience with treatment outcomes (successes and failures) as they relate to the particular case. It is important to note that determination of prognosis is a dynamic process. As such, the prognosis initially assigned should be reevaluated after completion of all phases of therapy, including periodontal maintenance.

Prognosis is often confused with the term *risk*. Risk generally deals with the likelihood that an individual will develop a disease in a specified period (see Chapter 5). *Risk factors* are those characteristics of an individual that put the person at increased risk for developing a disease (see Chapter 32). In contrast, *prognosis* is the prediction of the course or outcome of a disease. *Prognostic factors* are characteristics that predict the outcome of disease once the disease is present. In some cases, risk factors and prognostic factors are the same. For example, patients with diabetes or patients who smoke are more at risk for acquiring periodontal disease, and once they have it, they generally have a worse prognosis.

TYPES OF PROGNOSIS

Although some factors may be more important than others when assigning a prognosis[34,35] (Box 33-1), consideration of each factor may be beneficial to the clinician.

Historically, prognosis classification schemes have been designed based on studies evaluating tooth mortality.[2,3,25,33,34] One scheme[25,34] assigns the following classifications:

Good prognosis: Control of etiologic factors and adequate periodontal support ensure the tooth will be easy to maintain by the patient and clinician.

Fair prognosis: Approximately 25% attachment loss and/or Class I furcation involvement (location and depth allow proper maintenance with good patient compliance).

Poor prognosis: 50% attachment loss, Class II furcation involvement (location and depth make maintenance possible but difficult).

Questionable prognosis: >50% attachment loss, poor crown-to-root ratio, poor root form, Class II furcations (location and depth make access difficult) or Class III furcation involvements; >2+ mobility; root proximity.

Hopeless prognosis: Inadequate attachment to maintain health, comfort, and function.

It should be recognized that good, fair, and hopeless prognoses in this classification system could be established with a reasonable degree of accuracy. However, poor and questionable prognoses were likely to change to other categories because they depend on a large number of factors that can interact in an unpredictable number of ways.[8,14,47]

In contrast to schemes based on tooth mortality, Kwok and Caton[25] have proposed a scheme based on "the probability of obtaining stability of the periodontal supporting apparatus." This scheme is based on the probability of disease progression as related to local and systemic factors (see Box 33-1). Although some of these factors may affect disease progression more than others, consideration of each factor is important in assigning a prognosis. This scheme is as follows:

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BOX 33-1 Factors to Consider when Determining a Prognosis

Overall Clinical Factors
Patient age
Disease severity
Plaque control
Patient compliance

Systemic and Environmental Factors
Smoking
Systemic disease or condition
Genetic factors
Stress

Local Factors
Plaque and calculus
Subgingival restorations

Anatomic Factors
Short, tapered roots
Cervical enamel projections
Enamel pearls
Bifurcation ridges
Root concavities
Developmental grooves
Root proximity
Furcation involvement
Tooth mobility

Prosthetic and Restorative Factors
Abutment selection
Caries
Nonvital teeth
Root resorption

Favorable prognosis: Comprehensive periodontal treatment and maintenance will stabilize the status of the tooth. Future loss of periodontal support is unlikely.

Questionable prognosis: Local and/or systemic factors influencing the periodontal status of the tooth may or may not be controllable. If controlled, the periodontal status can be stabilized with comprehensive periodontal treatment. If not, future periodontal breakdown may occur.

Unfavorable prognosis: Local and/or systemic factors influencing the periodontal status cannot be controlled. Comprehensive periodontal treatment and maintenance are unlikely to prevent future periodontal breakdown.

Hopeless prognosis: The tooth must be extracted.

Because periodontal stability is assessed on a regular basis using clinical measures, it may be more useful in making treatment decisions and prognosis predictions than trying to determine the likelihood that the tooth will be lost.

In many of these cases, it may be advisable to establish a *provisional prognosis* until phase I therapy is completed and evaluated. The provisional prognosis allows the clinician to initiate treatment of teeth that have a doubtful outlook in the hope that a favorable response may tip the balance and allow teeth to be retained. The reevaluation phase in the treatment sequence allows the clinician to examine the tissue response to scaling, oral hygiene, and root planing, as well as to the possible use of chemotherapeutic agents where indicated. The patient's compliance with the proposed treatment plan also can be determined.

Overall Versus Individual Tooth Prognosis

Prognosis can be divided into overall prognosis and individual tooth prognosis. The *overall prognosis* is concerned with the dentition as a whole. Factors that may influence the overall prognosis include patient age; current severity of disease; systemic factors; smoking; the presence of plaque, calculus, and other local factors; patient compliance; and prosthetic possibilities (see Box 33-1). The overall prognosis answers the following questions:

- Should treatment be undertaken?
- Is treatment likely to succeed?
- When prosthetic replacements are needed, are the remaining teeth able to support the added burden of the prosthesis?

The *individual tooth prognosis* is determined after the overall prognosis and is affected by it.[33] For example, in a patient with a poor overall prognosis, the dentist likely would not attempt to retain a tooth that has a questionable prognosis because of local conditions. Many of the factors listed under local factors and prosthetic and restorative factors in Box 33-1 have a direct effect on the prognosis for individual teeth, in addition to any overall systemic or environmental factors that may be present.

FACTORS IN DETERMINATION OF PROGNOSIS

Overall Clinical Factors

Patient Age. For two patients with comparable levels of remaining connective tissue attachment and alveolar bone, the prognosis is generally better for the older of the two. For the younger patient, the prognosis is not as good because of the shorter time frame in which the periodontal destruction has occurred; the younger patient may have an aggressive type of periodontitis, or disease progression may have increased because of systemic disease or smoking. In addition, although the younger patient would ordinarily be expected to have a greater reparative capacity, the occurrence of so much destruction in a relatively short period would exceed any naturally occurring periodontal repair.

Disease Severity. Studies have demonstrated that a patient's history of previous periodontal disease may be indicative of their susceptibility for future periodontal breakdown (see Chapter 5). Therefore the following variables should be carefully recorded because they are important for determining the patient's past history of periodontal disease: pocket depth, level of attachment, degree of bone loss, and type of bony defect. These factors are determined by clinical and radiographic evaluation (see Chapters 30 and 31).

The determination of the level of clinical attachment reveals the approximate extent of root surface that is devoid of periodontal ligament; the radiographic examination shows the amount of root surface still invested in bone. Pocket depth is less important than level of attachment because it is not necessarily related to bone loss. In general, a tooth with deep pockets and little attachment and bone loss has a better prognosis than one with shallow pockets and severe attachment and bone loss. However, deep pockets are a source of infection and may contribute to progressive disease.

Prognosis is adversely affected if the base of the pocket (level of attachment) is close to the root apex. The presence of apical disease as a result of endodontic involvement also worsens the prognosis. However, surprisingly good apical and lateral bone repair

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can sometimes be obtained by combining endodontic and periodontal therapy (see Chapter 51).

The prognosis also can be related to the height of remaining bone. Assuming bone destruction can be arrested, is there enough bone remaining to support the teeth? The answer is readily apparent in extreme cases, that is, when there is so little bone loss that tooth support is not in jeopardy (Figure 33-1), or when bone loss is so severe that the remaining bone is obviously insufficient for proper tooth support (Figure 33-2). Most patients, however, do not fit into these extreme categories. The height of remaining bone is usually somewhere in between, making bone level assessment alone insufficient for determining the overall prognosis.

The type of defect also must be determined. The prognosis for horizontal bone loss depends on the height of the existing bone because it is unlikely that clinically significant bone height regeneration will be induced by therapy. In the case of angular, intrabony defects, if the contour of the existing bone and the number of osseous walls are favorable, there is an excellent chance that therapy could regenerate bone to approximately the level of the alveolar crest.[45]

When greater bone loss has occurred on one surface of a tooth, the bone height on the less involved surfaces should be taken into consideration when determining the prognosis. Because of the greater height of bone in relation to other surfaces, the center of rotation of the tooth will be nearer the crown (Figure 33-3). This results in a more favorable distribution of forces to the periodontium and less tooth mobility.[49]

In dealing with a tooth with a questionable prognosis, the chances of successful treatment should be weighed against any benefits that would accrue to the adjacent teeth if the tooth under consideration were extracted. Strategic extraction of teeth was first proposed as a means of improving the overall prognosis of adjacent teeth and/or enhancing the prosthetic treatment plan.[9] It has now been expanded to include the extraction of teeth with a

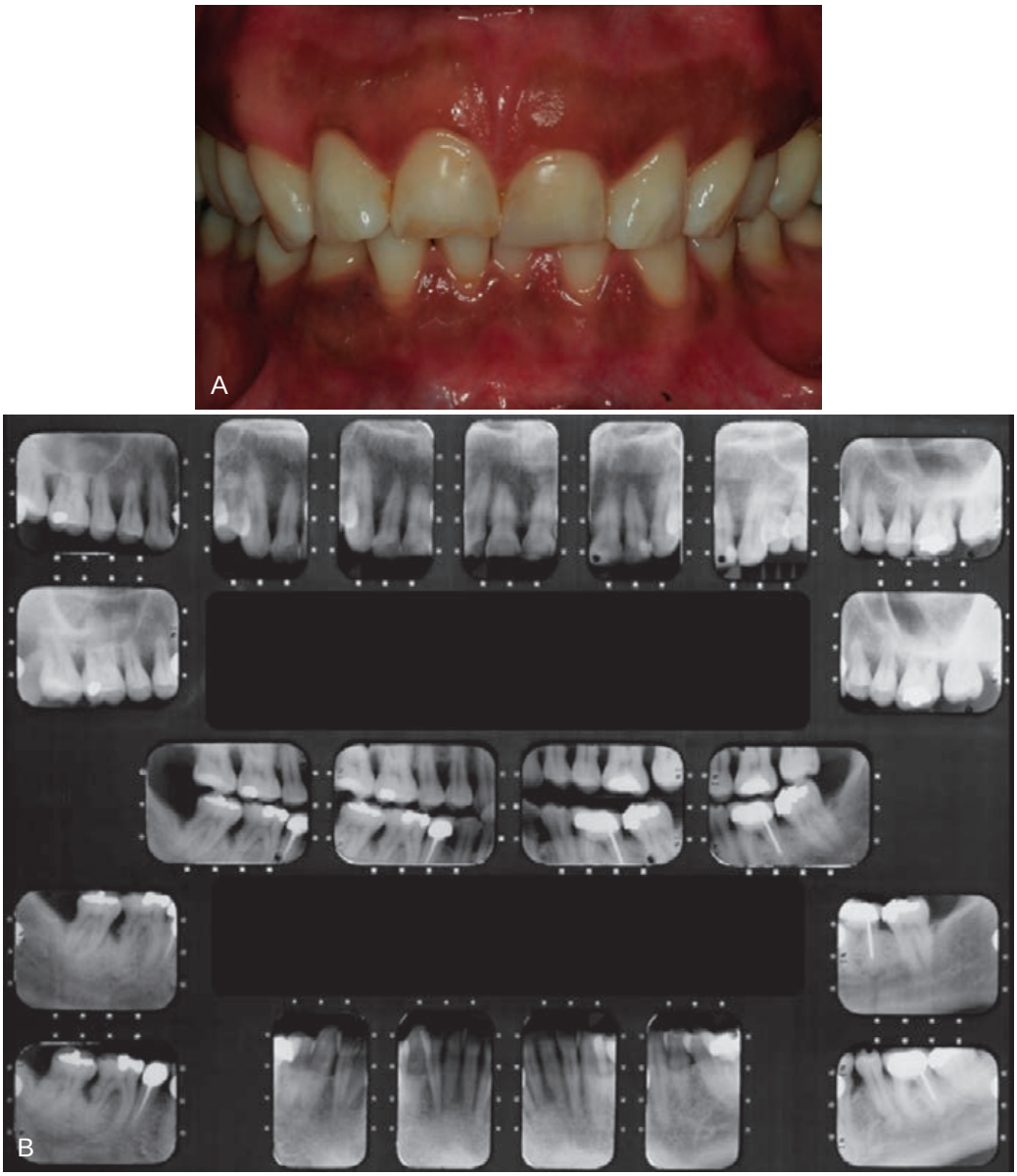

Figure 33-1 Chronic periodontitis in systemically healthy, nonsmoking 42-year-old male; overall prognosis good. **A,** Gingival inflammation, poor oral hygiene, and pronounced anterior overbite. **B,** Although local factors are present, the patient presents with adequate remaining bone support and a good prognosis, provided local factors can be controlled.

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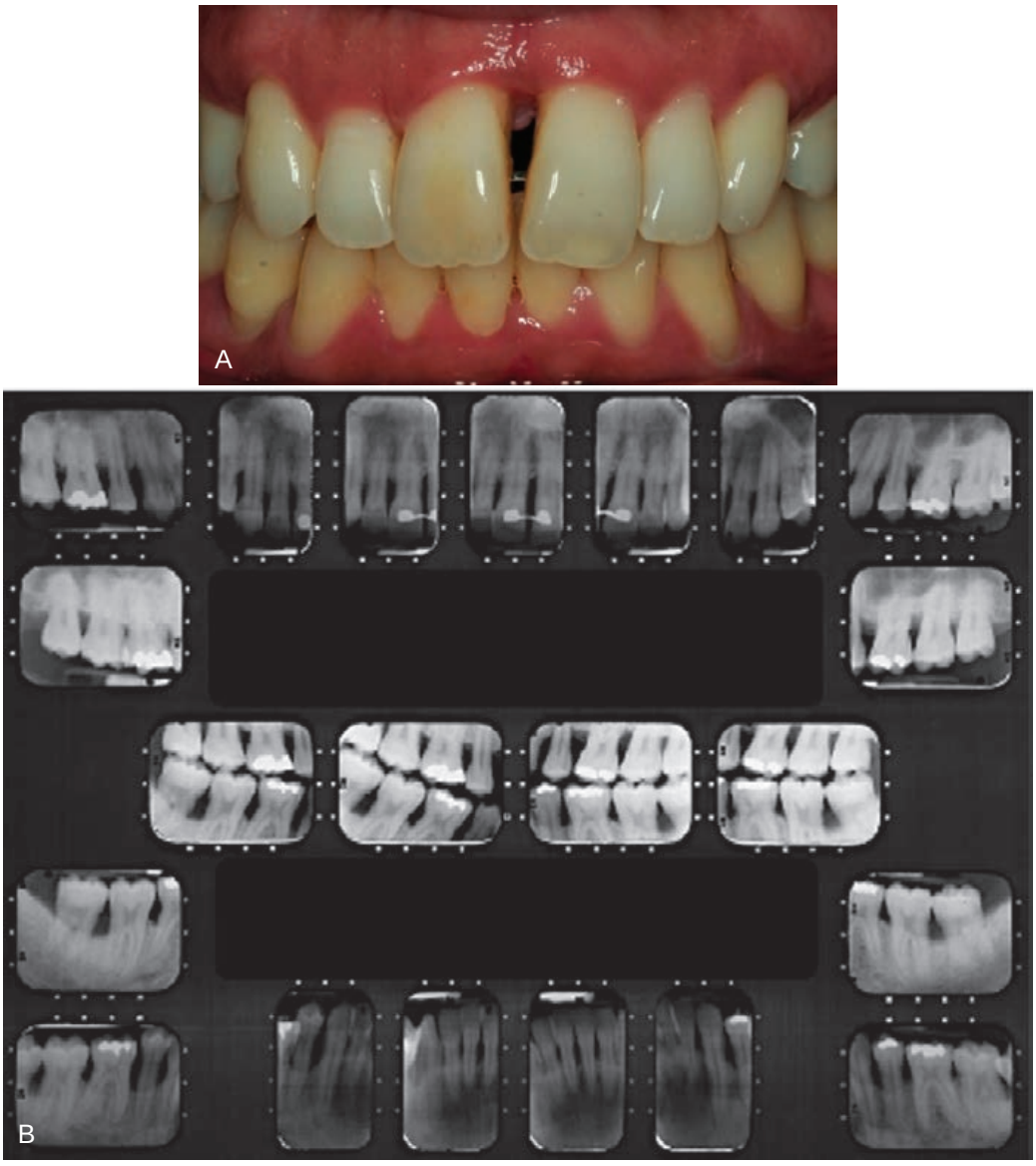

Figure 33-2 Localized aggressive periodontitis in 17-year-old girl; overall prognosis fair. **A,** Gingival inflammation, periodontal pockets, and pathologic migration. **B,** Severe bone destruction.

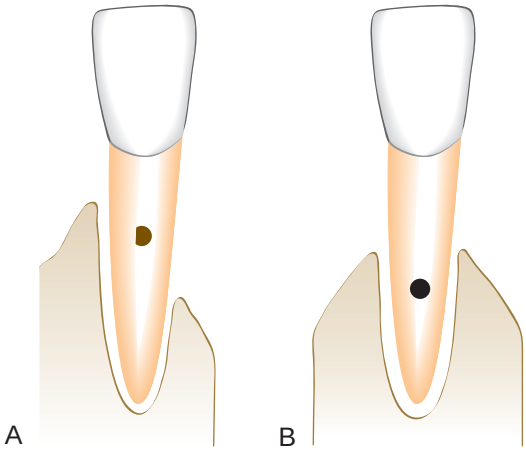

Figure 33-3 Prognosis for tooth **A** is better than for tooth **B,** despite less bone on one of the surfaces of A. Because the center of rotation of tooth **A** is closer to the crown, the distribution of occlusal forces to the periodontium is more favorable than in **B.**

questionable prognosis to enhance the likelihood of partial restoration of the bone supporting of the adjacent teeth (Figure 33-4 *A* to *D*) or successful implant placement. With the growing evidence of the long-term success of dental implants, it is proposed that a "watch and wait" approach may allow an area to deteriorate to the point that placing an implant is no longer a viable option. This means that the practitioner should weigh the potential success of one treatment option (extraction and implant placement) versus the other (periodontal therapy and maintenance) carefully when assigning a prognosis to questionable teeth.[20]

Plaque Control. Bacterial plaque is the primary etiologic factor associated with periodontal disease (see Chapter 23). Therefore effective removal of plaque on a daily basis by the patient is critical to the success of periodontal therapy and to the prognosis.

Patient Compliance and Cooperation. The prognosis for patients with gingival and periodontal disease is critically

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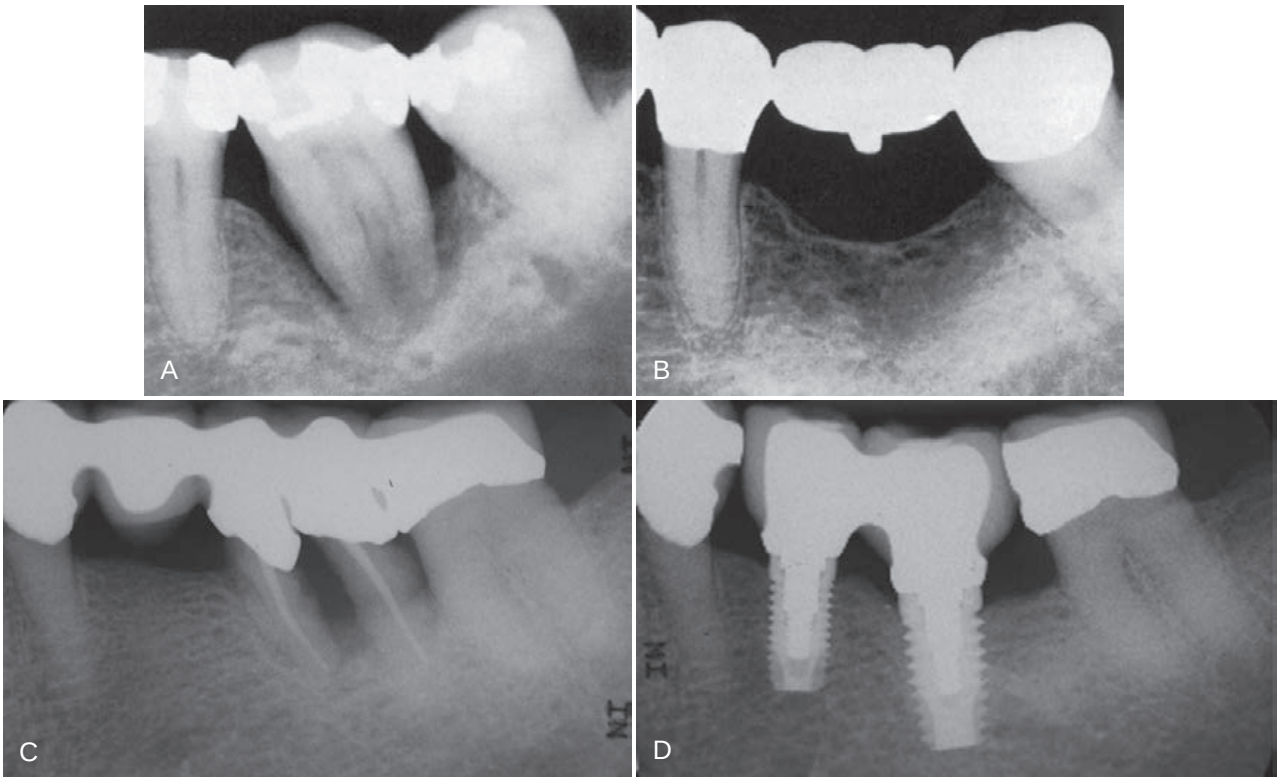

Figure 33-4 Extraction of severely involved tooth to preserve bone on adjacent teeth. **A,** Extensive bone destruction around the mandibular first molar. **B,** Radiograph made 8½ years after extraction of the first molar and replacement by a prosthesis. Note the excellent bone support. **C,** Extraction of periodontally involved bicuspid and molar. **D,** Implant replacement of both teeth. *(Courtesy of Dr. S. Angha, University of California, Los Angeles.)*

dependent on the patient's attitude, desire to retain the natural teeth, and willingness and ability to maintain good oral hygiene. Without these, treatment cannot succeed. Patients should be clearly informed of the important role they must play for treatment to succeed. If patients are unwilling or unable to perform adequate plaque control and to receive the timely periodic maintenance checkups and treatments that the dentist deems necessary, the dentist can (1) refuse to accept the patient for treatment or (2) extract teeth that have a hopeless or poor prognosis and perform scaling and root planing on the remaining teeth. The dentist should make it clear to the patient and in the patient record that further treatment is needed but will not be performed because of a lack of patient cooperation.

Systemic and Environmental Factors

Smoking. Epidemiologic evidence suggests that smoking may be the most important environmental risk factor impacting the development and progression of periodontal disease (see Chapter 5). Therefore it should be made clear to the patient that a direct relationship exists between smoking and the prevalence and incidence of periodontitis. In addition, patients should be informed that smoking affects not only the severity of periodontal destruction but also the healing potential of the periodontal tissues. As a result, patients who smoke do not respond as well to conventional periodontal therapy as patients who have never smoked.[43,44] Therefore the prognosis in patients who smoke and have slight to moderate periodontitis is generally fair to poor. In patients with severe periodontitis, the prognosis may be poor to hopeless.

However, it should be emphasized that smoking cessation can affect the treatment outcome and therefore the prognosis.[5,17] Patients with slight-to-moderate periodontitis who stop smoking can often be upgraded to a good prognosis, whereas those with severe periodontitis who stop smoking may be upgraded to a fair prognosis.

Systemic Disease or Condition. The patient's systemic background affects overall prognosis in several ways. For example, evidence from epidemiologic studies clearly demonstrates that the prevalence and severity of periodontitis are significantly higher in patients with type 1 and type 2 diabetes than in those without diabetes and that the level of control of the diabetes is an important variable in this relationship (see Chapter 5). Therefore patients at risk for diabetes should be identified as early as possible and informed of the relationship between periodontitis and diabetes. Similarly, patients diagnosed with diabetes must be informed of the impact of diabetic control on the development and progression of periodontitis. It follows that the prognosis in these cases depends on patient compliance relative to both medical and dental status. Well-controlled diabetic patients with slight-to-moderate periodontitis who comply with their recommended periodontal treatment should have a good prognosis. Similarly, in patients with other systemic disorders that could affect disease progression, prognosis improves with correction of the systemic problem.

The prognosis is questionable when surgical periodontal treatment is required but cannot be provided because of the patient's health (see Chapter 37). Incapacitating conditions that limit the patient's performance of oral procedures (e.g., Parkinson's disease) also adversely affect the prognosis. Newer "automated" oral hygiene devices, such as electric toothbrushes, may be helpful for these patients and may improve their prognosis (see Chapter 44).

Genetic Factors. Periodontal diseases represent a complex interaction between a microbial challenge and the host's response to that challenge, both of which may be influenced by environmental factors such as smoking. In addition to these external factors, evidence also indicates that genetic factors may play an important role in determining the nature of the host response.[18] Evidence for this type of genetic influence exists for patients with both chronic and aggressive periodontitis. Genetic polymorphisms in the interleukin-1 (IL-1) genes, resulting in increased production of IL-1β, have been associated with a significant increase in risk for severe, generalized, and chronic periodontitis.[24,37] It has been demonstrated that knowledge of the patient's IL-1 genotype and smoking status can aid the clinician in assigning a prognosis.[36] Genetic factors also appear to influence serum immunoglobulin G2 (IgG2) antibody titers and the expression of FcγRII receptors on the neutrophil, both of which may be significant in aggressive periodontitis.[18] Other genetic disorders, such as leukocyte adhesion deficiency type 1, can influence neutrophil function, creating an additional risk factor for aggressive periodontitis.[18] Finally, the familial aggregation that is characteristic of aggressive periodontitis indicates that additional, as yet unidentified genetic factors may be important in susceptibility to this form of disease (see Chapter 18).

The influence of genetic factors on prognosis is not simple. Although microbial and environmental factors can be altered through conventional periodontal therapy and patient education, genetic factors currently cannot be altered. However, detection of genetic variations linked to periodontal disease can potentially influence the prognosis in several ways. First, early detection of patients at risk because of genetic factors can lead to early implementation of preventive and treatment measures for these patients. Second, identification of genetic risk factors later in the disease or during the course of treatment can influence treatment recommendations, such as the use of adjunctive antibiotic therapy or increased frequency of maintenance visits. Third, identification of young individuals who have not been evaluated for periodontitis, but who are recognized as being at risk because of the familial aggregation seen in aggressive periodontitis, can lead to the development of early intervention strategies. In each of these cases, early diagnosis, intervention, and alterations in the treatment regimen may lead to an improved prognosis for the patient.

Stress. Physical and emotional stress, as well as substance abuse, may alter the patient's ability to respond to the periodontal treatment performed (see Chapter 5). These factors must be realistically faced when attempting to establish a prognosis.

Local Factors

Plaque and Calculus. The microbial challenge presented by bacterial plaque and calculus is the most important local factor in periodontal diseases. Therefore, in most cases, having a good prognosis depends on the ability of the patient and the clinician to remove these etiologic factors (see Chapters 22 and 23).

Subgingival Restorations. Subgingival margins may contribute to increased plaque accumulation, increased inflammation, and increased bone loss[4,40,48] when compared with supragingival margins. Furthermore, discrepancies in these margins (e.g., overhangs) can negatively impact the periodontium (see Chapter 22). The size of these discrepancies and duration of their presence are important factors in the amount of destruction that occurs. In general, however, a tooth with a discrepancy in its subgingival margins has a poorer prognosis than a tooth with well-contoured supragingival margins.

Anatomic Factors. Anatomic factors that may predispose the periodontium to disease and therefore affect the prognosis include short, tapered roots with large crowns; cervical enamel projections and enamel pearls; intermediate bifurcation ridges; root concavities; and developmental grooves. The clinician must also consider root proximity and the location and anatomy of furcations when developing a prognosis.

Prognosis is poor for teeth with short, tapered roots and relatively large crowns (Figure 33-5). Because of the disproportionate crown-to-root ratio and the reduced root surface available for periodontal support,[21] the periodontium may be more susceptible to injury by occlusal forces.

Cervical enamel projections (CEPs) are flat, ectopic extensions of enamel that extend beyond the normal contours of the cementoenamel junction.[32] CEPs extend into the furcation of 28.6% of mandibular molars and 17% of maxillary molars.[32] CEPs are most likely to be found on buccal surfaces of maxillary second molars.[16,50] Enamel pearls are larger, round deposits of enamel that can be located in furcations or other areas on the root surface.[39] Enamel pearls are seen less frequently (1.1% to 5.7% of permanent molars; 75% appearing in maxillary third molars[39]) than CEPs. An intermediate bifurcation ridge has been described in 73% of mandibular first molars, crossing from the mesial to the distal root at the midpoint of the furcation.[11] The presence of these enamel projections on the root surface interferes with the attachment apparatus and may prevent regenerative procedures from achieving their maximum potential. Therefore their presence may have a negative effect on the prognosis for an individual tooth.

Scaling with root planing is a fundamental procedure in periodontal therapy. Anatomic factors that decrease the efficiency of this procedure can have a negative impact on the prognosis. Therefore the morphology of the tooth root is an important consideration when discussing prognosis. *Root concavities* exposed through loss of attachment can vary from shallow flutings to deep depressions. They appear more marked on maxillary first premolars, the mesiobuccal root of the maxillary first molar, both roots of mandibular first molars, and the mandibular incisors[6,7] (Figures 33-6 and 33-7). Any tooth, however, can have a proximal concavity.[13] Although these concavities increase the attachment area and produce a root shape that may be more resistant to torquing forces, they also create areas that can be difficult for both the dentist and the patient to clean.

Other anatomic considerations that present accessibility problems are developmental grooves, root proximity, and furcation involvements. The presence of any of these can worsen the prognosis. *Developmental grooves,* which sometimes appear in the maxillary lateral incisors (palatogingival groove[51]; Figure 33-8) or in the lower incisors, create an accessibility problem.[10,15] They initiate on enamel and can extend a significant distance on the root surface, providing a plaque-retentive area that is difficult to instrument. These palatogingival grooves are found on 5.6% of maxillary lateral incisors and 3.4% of maxillary central incisors.[23] Similarly, *root proximity* can result in interproximal areas that are difficult for the clinician and patient to access. Finally, *access to the furcation area* is usually difficult to obtain. In 58% of maxillary and mandibular first molars, the furcation entrance diameter is narrower than the width of conventional periodontal curettes[7] (Figure 33-9). Maxillary first premolars present the greatest difficulties, and therefore their prognosis is usually unfavorable when the lesion reaches the mesiodistal furcation. Maxillary molars also present some difficulty; sometimes

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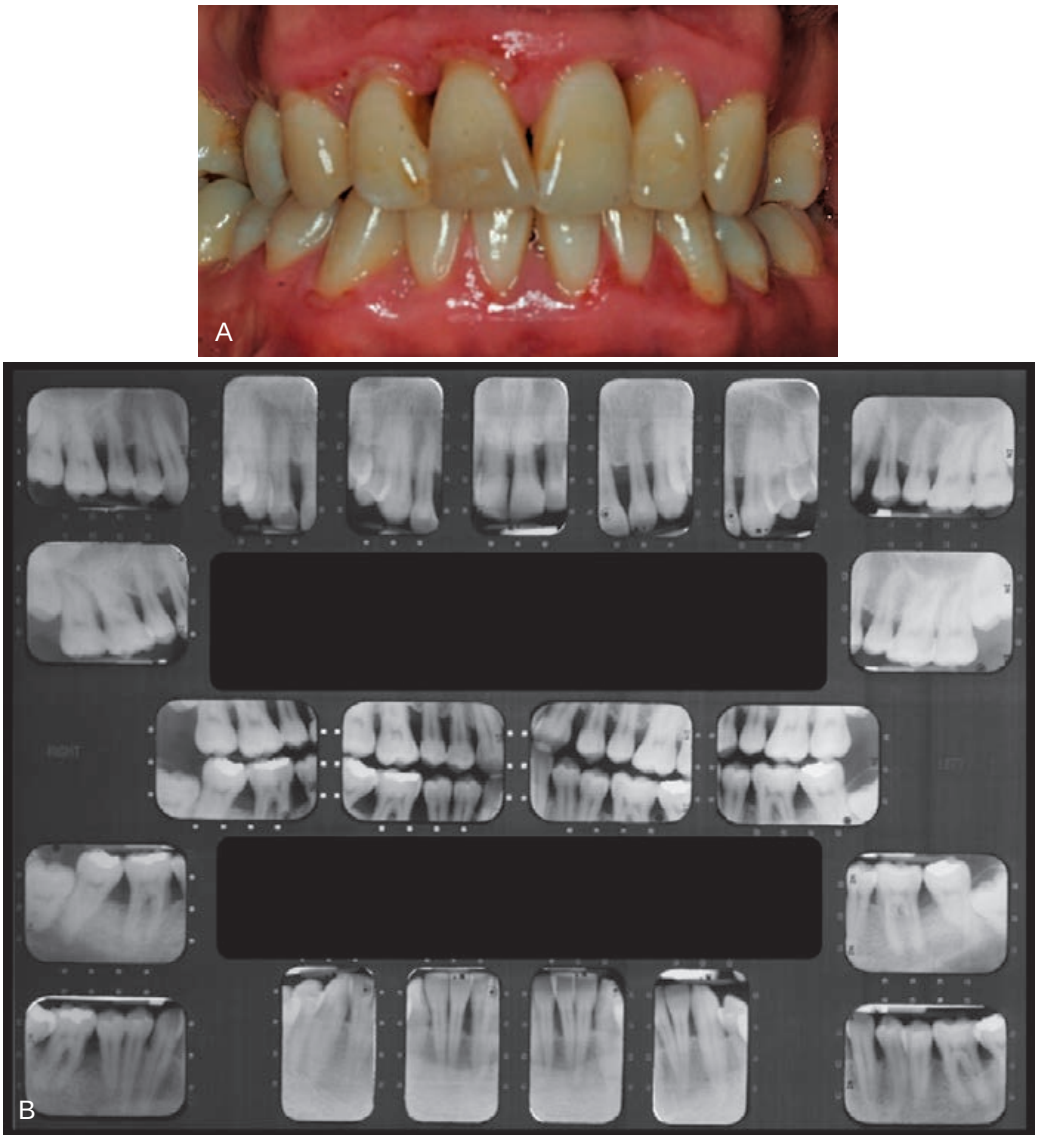

Figure 33-5 Generalized aggressive periodontitis and poor crown-to-root ratio in 24-year-old patient; overall prognosis poor. **A,** generalized periodontal attachment loss and pocket formation. **B,** Moderate to advanced bone destruction. The contrast between the well-formed crowns and the relatively short, tapered roots worsens the prognosis.

their prognosis can be improved by resecting one of the buccal roots (see Chapter 62), thereby improving access to the area. When mandibular first molars or buccal furcations of maxillary molars offer good access to the furcation area, their prognosis is usually better.

Tooth Mobility. The principal causes of tooth mobility are loss of alveolar bone, inflammatory changes in the periodontal ligament, and trauma from occlusion. Tooth mobility caused by inflammation and trauma from occlusion may be correctable.[38] However, tooth mobility resulting from loss of alveolar bone is not likely to be corrected. The likelihood of restoring tooth stability is inversely proportional to the extent to which mobility is caused by loss of supporting alveolar bone. A longitudinal study of the response to treatment of teeth with different degrees of mobility revealed that pockets on clinically mobile teeth do not respond as well to periodontal therapy as pockets on nonmobile teeth exhibiting the same initial disease severity.[12] Another study, however, in which ideal plaque control was attained, found similar healing in both hypermobile and firm teeth.[45] The stabilization of tooth mobility through the use of splinting may have a beneficial impact on the overall and individual tooth prognosis.

Prosthetic and Restorative Factors

The overall prognosis requires a general consideration of bone levels (evaluated radiographically) and attachment levels (determined clinically) to establish whether enough teeth can be saved either to provide a functional and aesthetic dentition or to serve as abutments for a useful prosthetic replacement of the missing teeth.

At this point, the overall prognosis and the individual tooth prognosis overlap because the prognosis for key individual teeth may affect the overall prognosis for prosthetic rehabilitation. For example, saving or losing a key tooth may determine whether other teeth are saved or extracted or whether the prosthesis used is fixed or removable (see Figure 33-4). When few teeth remain, the prosthodontic needs become more important, and sometimes periodontally treatable teeth may have to be extracted if they are not compatible with the design of the prosthesis.

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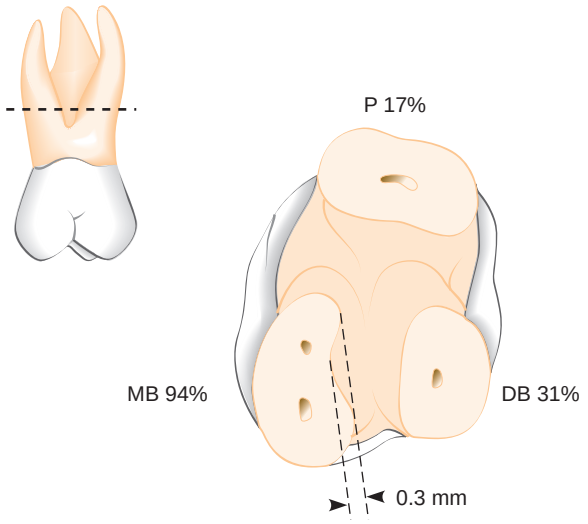

Figure 33-6 Root concavities in maxillary first molars sectioned 2 mm apical to the furca. The furcal aspect of the root is concave in 94% of the mesiobuccal *(MB)* roots, 31% of the distobuccal *(DB)* roots, and 17% of the palatal *(P)* roots. The deepest concavity is found in the furcal aspects of the mesiobuccal root (mean concavity, 0.3 mm). The furcal aspect of the buccal roots diverges toward the palate in 97% of teeth (mean divergence, 22 degrees). *(Redrawn from Bower RC:* J Periodontol *50:366, 1979.)*

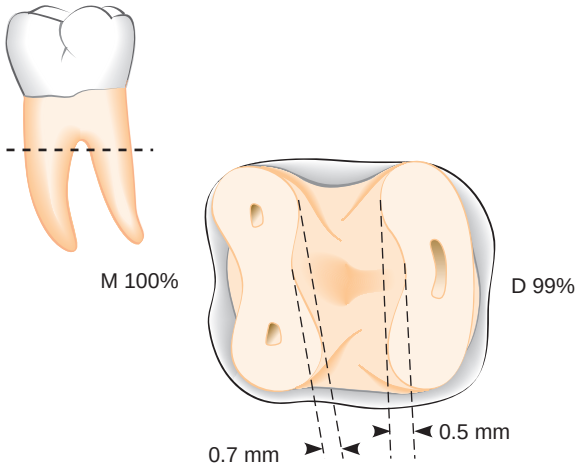

Figure 33-7 Root concavities in mandibular first molars sectioned 2 mm apical to the furca. Concavity of the furcal aspect was found in 100% of mesial *(M)* roots and 99% of distal *(D)* roots. Deeper concavity was found in the mesial roots (mean concavity, 0.7 mm). *(Redrawn from Bower RC:* J Periodontol *50:366, 1979.)*

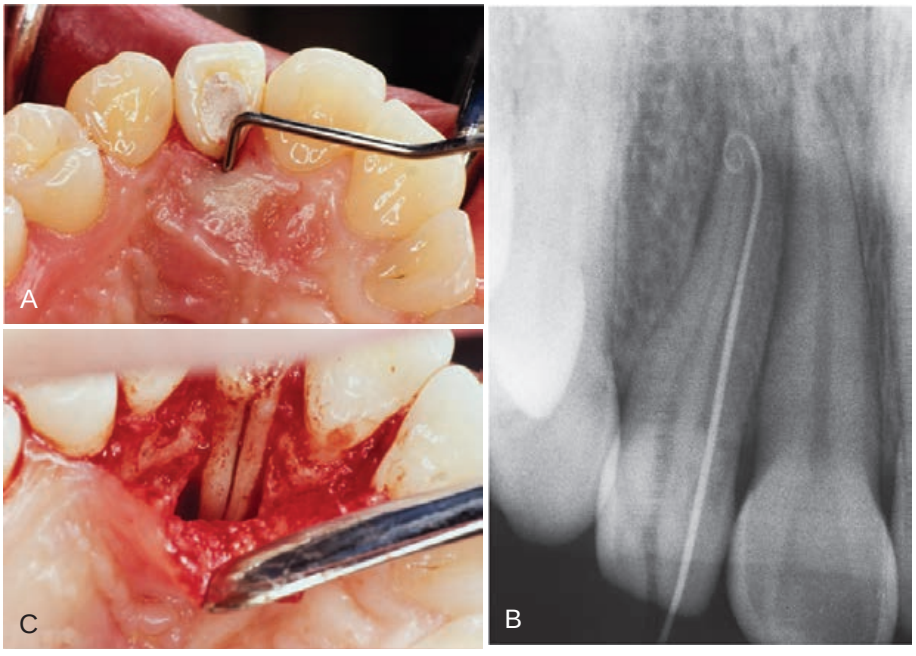

Figure 33-8 Palatogingival groove. **A,** Probe in place to indicate a deep pocket along the palatogingival groove. **B,** Radiograph with a gutta percha point placed in the pocket. **C,** The area is surgically opened. Note the palatogingival groove along the entire palatal portion of the root. *(Courtesy of Dr. Nadia Chugal, University of California, Los Angeles.)*

Teeth that serve as abutments are subjected to increased functional demands. More rigid standards are required when evaluating the prognosis of teeth adjacent to edentulous areas. A tooth with a post that has undergone endodontic treatment is more likely to fracture when serving as a distal abutment supporting a distal removable partial denture. Additionally, special oral hygiene measures must be instituted in these areas.

Caries, Nonvital Teeth, and Root Resorption. For teeth mutilated by extensive caries, the feasibility of adequate restoration and endodontic therapy should be considered before undertaking periodontal treatment. Extensive idiopathic root resorption or root resorption resulting from orthodontic therapy jeopardizes the stability of teeth and adversely affects the response to periodontal treatment. The periodontal prognosis of treated

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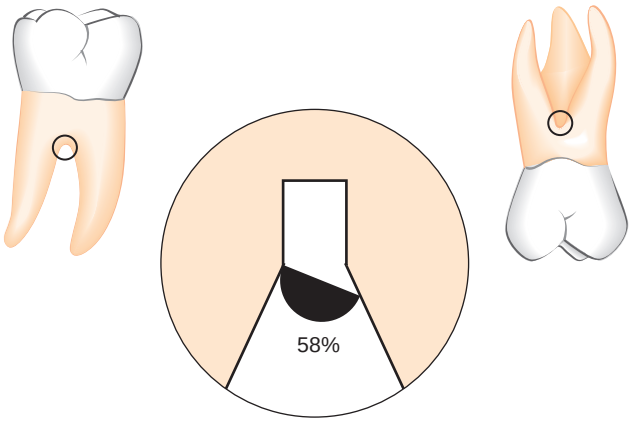

Figure 33-9 The furcation entrance is narrower than a standard curette in 58% of first molars. *(Redrawn from Bower RC:* J Periodontol *50:366, 1979.)*

nonvital teeth does not differ from that of vital teeth. New attachment can occur to the cementum of both nonvital and vital teeth.

RELATIONSHIP BETWEEN DIAGNOSIS AND PROGNOSIS

Many of the criteria used in the diagnosis and classification of the different forms of periodontal disease[1] (see Chapter 4) are also used in developing a prognosis (see Box 33-1). Factors such as patient age, severity of disease, genetic susceptibility, and presence of systemic disease are important criteria in the diagnosis of the condition, as well as important in developing a prognosis. These common factors suggest that for any given diagnosis, there should be an expected prognosis under ideal conditions. This section discusses the potential prognoses of the various periodontal diseases outlined in Chapter 4.

Prognosis for Patients with Gingival Disease

Dental Plaque–Induced Gingival Diseases

Gingivitis Associated with Dental Plaque Only. Plaque-induced gingivitis is a reversible disease that occurs when bacterial plaque accumulates at the gingival margin.[29,31] This disease can occur on a periodontium that has experienced no attachment loss or on a periodontium with nonprogressing attachment loss. In either case, the prognosis for patients with gingivitis associated with dental plaque only is good, provided all local irritants are eliminated, other local factors contributing to plaque retention are eliminated, gingival contours conducive to the preservation of health are attained, and the patient cooperates by maintaining good oral hygiene.

Plaque-Induced Gingival Diseases Modified by Systemic Factors. The inflammatory response to bacterial plaque at the gingival margin can be influenced by systemic factors, such as endocrine-related changes associated with puberty, menstruation, pregnancy, and diabetes, and the presence of blood dyscrasias. In many cases, the frank signs of gingival inflammation that occur in these patients are seen in the presence of relatively small amounts of bacterial plaque. Therefore the long-term prognosis for these patients depends not only on control of bacterial plaque but also on control or correction of the systemic factor(s).

Plaque-Induced Gingival Diseases Modified by Medications. Gingival diseases associated with medications include drug-influenced *gingival enlargement,* often seen with phenytoin, cyclosporine, and nifedipine and in oral contraceptive–associated gingivitis. In drug-influenced gingival enlargement, reductions in dental plaque can limit the severity of the lesions. However, plaque control alone does not prevent development of the lesions, and surgical intervention is usually necessary to correct the alterations in gingival contour. Continued use of the drug usually results in recurrence of the enlargement, even after surgical intervention (see Chapter 9). Therefore the long-term prognosis depends on whether the patient's systemic problem can be treated with an alternative medication that does not have gingival enlargement as a side effect.

In oral contraceptive–associated gingivitis, frank signs of gingival inflammation can be seen in the presence of relatively little plaque. Therefore, as seen in plaque-induced gingival diseases modified by systemic factors, the long-term prognosis in these patients depends on not only the control of bacterial plaque but also on the likelihood of continued use of the oral contraceptive.

Gingival Diseases Modified by Malnutrition. Although malnutrition has been suspected to play a role in the development of gingival diseases, most clinical studies have not shown a relationship between the two. One possible exception is severe vitamin C deficiency. In early experimental vitamin C deficiency, gingival inflammation and bleeding on probing were independent of plaque levels present. The prognosis in these patients may depend on the severity and duration of the deficiency and on the likelihood of reversing the deficiency through dietary supplementation.

Non–Plaque-Induced Gingival Lesions. Non–plaque-induced gingivitis can be seen in patients with a variety of bacterial, fungal, and viral infections.[19] Since the gingivitis in these patients is not usually attributed to plaque accumulation, prognosis depends on elimination of the source of the infectious agent. Dermatologic disorders, such as lichen planus, pemphigoid, pemphigus vulgaris, erythema multiforme, and lupus erythematosus, also can manifest in the oral cavity as atypical gingivitis (see Chapter 12). Prognosis for these patients is linked to management of the associated dermatologic disorder. Finally, allergic, toxic, and foreign body reactions, as well as mechanical and thermal trauma, can result in gingival lesions. Prognosis for these patients depends on elimination of the causative agent.

Prognosis for Patients with Periodontitis

Chronic Periodontitis. Chronic periodontitis is a slowly progressive disease associated with well-known local environmental factors.[27] It can present in a localized or generalized form (see Chapter 16). In cases in which the clinical attachment loss and bone loss are not very advanced (slight-to-moderate periodontitis), the prognosis is generally good, provided the inflammation can be controlled through good oral hygiene and the removal of local plaque-retentive factors (see Figure 33-1). In patients with more severe disease, as evidenced by furcation involvement and increasing clinical mobility, or in patients who are noncompliant with oral hygiene practices, the prognosis may be downgraded to fair to poor.

Aggressive Periodontitis. Aggressive periodontitis can present in a localized or a generalized form.[26] Two common features of both forms are (1) rapid attachment loss and bone destruction in an otherwise clinically healthy patient and (2) a familial aggregation. These patients often present with limited microbial

deposits that seem inconsistent with the severity of tissue destruction. However, the deposits that are present often have elevated levels of *Aggregatibacter actinomycetemcomitans* or *Porphyromonas gingivalis*. These patients also may present with phagocyte abnormalities and a hyperresponsive monocyte/macrophage phenotype. These clinical, microbiologic, and immunologic features would suggest that patients diagnosed with aggressive periodontitis would have a poor prognosis.

However, the clinician should consider additional specific features of the localized form of disease when determining the prognosis (see Figure 33-2). Localized aggressive periodontitis usually occurs around the age of puberty and is localized to first molars and incisors. The patient often exhibits a strong serum antibody response to the infecting agents, which may contribute to localization of the lesions (see Chapter 18). When diagnosed early, these cases can be treated conservatively with oral hygiene instruction and systemic antibiotic therapy,[42] resulting in an excellent prognosis. When more advanced disease occurs, the prognosis can still be good if the lesions are treated with debridement, local and systemic antibiotics, and regenerative therapy.[30,52]

In contrast, although patients with generalized aggressive periodontitis also are young patients (usually under age 30), they present with generalized interproximal attachment loss and a poor antibody response to infecting agents. Secondary contributing factors, such as cigarette smoking, are often present. These factors, coupled with the alterations in host defense seen in many of these patients, may result in a case that does not respond well to conventional periodontal therapy (scaling with root planing, oral hygiene instruction, and surgical intervention). Therefore these patients often have a fair, poor, or questionable prognosis, and the use of systemic antibiotics should be considered to help control the disease (see Chapter 47).

Periodontitis as Manifestation of Systemic Diseases. Periodontitis as a manifestation of systemic diseases can be divided into the following two categories[22,28]:

1. Periodontitis associated with hematologic disorders such as leukemia and acquired neutropenias.
2. Periodontitis associated with genetic disorders such as familial and cyclic neutropenia, Down syndrome, Papillon-Lefèvre syndrome, and hypophosphatasia.

Although the primary etiologic factor in periodontal diseases is bacterial plaque, systemic diseases that alter the ability of the host to respond to the microbial challenge presented may affect the progression of disease and therefore the prognosis for the case. For example, decreased numbers of circulating neutrophils (as in acquired neutropenias) may contribute to widespread destruction of the periodontium. Unless the neutropenia can be corrected, these patients present with a fair-to-poor prognosis. Similarly, genetic disorders that alter the way the host responds to bacterial plaque (as in leukocyte adhesion deficiency [LAD] syndrome) also can contribute to the development of periodontitis. Because these disorders generally manifest early in life, the impact on the periodontium may be clinically similar to generalized aggressive periodontitis. The prognosis in these cases will be fair to poor.

Other genetic disorders do not affect the host's ability to combat infections but still affect the development of periodontitis. Examples include hypophosphatasia, in which patients have decreased levels of circulating alkaline phosphatase, severe alveolar bone loss, and premature loss of deciduous and permanent teeth, and the connective tissue disorder Ehlers-Danlos syndrome, in which patients may present with the clinical characteristics of aggressive periodontitis. In both examples the prognosis is fair to poor.

Necrotizing Periodontal Diseases. Necrotizing periodontal disease can be divided into necrotic diseases that affect the gingival tissues exclusively (necrotizing ulcerative gingivitis [NUG]) and necrotic diseases that affect deeper tissues of the periodontium, resulting in loss of connective tissue attachment and alveolar bone (necrotizing ulcerative periodontitis [NUP]).[41,46] In NUG the primary predisposing factor is bacterial plaque. However, this disease is usually complicated by the presence of secondary factors such as acute psychologic stress, tobacco smoking, and poor nutrition, all of which can contribute to immunosuppression. Therefore superimposition of these secondary factors on a preexisting gingivitis can result in the painful, necrotic lesions characteristic of NUG. With control of both the bacterial plaque and the secondary factors, the prognosis for a patient with NUG is good. However, the tissue destruction in these cases is not reversible, and poor control of the secondary factors may make these patients susceptible to recurrence of the disease. With repeated episodes of NUG, the prognosis may be downgraded to fair.

The clinical presentation of NUP is similar to that of NUG, except the necrosis extends from the gingiva into the periodontal ligament and alveolar bone. In systemically healthy patients, this progression may have resulted from multiple episodes of NUG, or the necrotizing disease may occur at a site previously affected with periodontitis. In these patients, the prognosis depends on alleviating the plaque and secondary factors associated with NUG. However, many patients presenting with NUP are immunocompromised through systemic conditions, such as human immunodeficiency virus (HIV) infection. In these patients the prognosis depends on not only reducing local and secondary factors, but also on dealing with the systemic problem (see Chapter 40).

In advanced cases, prognosis may be better established after reviewing the effectiveness of phase I therapy.

Determination of the prognosis of a tooth or teeth can be difficult, particularly for teeth with previous disease. Many factors can influence disease progression and the response to therapy, and the specific influence of any one factor is unknown and likely different from one patient to another. In addition, each patient can respond differently at different times. All these issues make determination of a prognosis difficult.

Therefore the prognosis is often determined after initial treatment is provided, assuming a favorable outcome. The prognosis is delayed until after initial therapy because the etiology depends on the host response. During initial therapy, the patient's motivation and commitment, acknowledged as critical in all periodontal therapy, also can be determined, as well as the host response and healing capacity of the patient. Clearly, enhancing the host response to plaque's microbial challenge will significantly and positively influence the periodontal prognosis. Likewise, an *inability* to enhance the host response will negatively influence the prognosis. Either outcome, however, will allow the clinician to determine a more accurate prognosis.

REEVALUATION OF PROGNOSIS AFTER PHASE I THERAPY

A frank reduction in pocket depth and inflammation after phase I therapy indicates a favorable response to treatment and may suggest a better prognosis than previously assumed. If the inflammatory changes present cannot be controlled or reduced by phase I therapy, the overall prognosis may be unfavorable. In these patients, the prognosis can be directly related to the severity of inflammation. Given two patients with comparable bone destruction, the prognosis may be better for the patient with the greater

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SCIENCE TRANSFER

Because of the complexity and variability of a patient's periodontal disease and the individual systemic factors that influence prognosis, it is not possible to develop an accurate methodology for determining long-term success of therapy. The clinician needs to have experience with treating many different patients, as well as the ability to synthesize the data from each patient to develop a prognosis.

Determining the prognosis after assessing a patient's response to initial therapy will give a more accurate evaluation than the prognosis put together before treatment began. The patient's ability to maintain low levels of plaque is the most significant factor affecting treatment outcomes. Smoking has a negative influence on prognosis as does the presence of systemic diseases such as diabetes. Single-rooted teeth have a better prognosis than multirooted teeth with the same amount of periodontal destruction. Similarly, advanced tooth mobility, if untreated, can negatively affect prognosis.

In general, younger patients have a greater capacity for healing, repair, and regeneration than older patients, so that in patients with the same diagnosis, systemic status, plaque scores, and anatomic factors, the younger patients will respond better to periodontal treatment. This obviously refers to patients with the same prognosis, since patients with aggressive periodontitis who are frequently younger have a poorer prognosis than older patients with chronic periodontitis.

degree of inflammation because a larger component of that patient's bone destruction may be attributable to local irritants. In addition, phase I therapy allows the clinician an opportunity to work with the patient and the patient's physician to control systemic and environmental factors such as diabetes and smoking, which may have a positive effect on prognosis if adequately controlled.

The progression of periodontitis generally occurs in an episodic manner, with alternating periods of quiescence and shorter destructive stages (see Chapter 13). No methods are available at present to determine accurately whether a given lesion is in a stage of remission or exacerbation. Advanced lesions, if active, may progress rapidly to a hopeless stage, whereas similar lesions in a quiescent stage may be maintainable for long periods. Phase I therapy will, at least temporarily, transform the prognosis of the patient with an active advanced lesion, and the lesion should be reanalyzed after completion of phase I therapy.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 34

The Treatment Plan

Fermin A. Carranza and Henry H. Takei

CHAPTER OUTLINE

MASTER PLAN FOR TOTAL TREATMENT
Extracting or Preserving a Tooth
SEQUENCE OF THERAPEUTIC PROCEDURES
EXPLAINING TREATMENT PLAN TO THE PATIENT

After the diagnosis and prognosis have been established, the treatment is planned. The plan should encompass short- and long-term goals.

The *short-term goals* are the elimination of all infectious and inflammatory processes that cause periodontal and other oral problems that may hinder the patient's general health. Basically, the short-term goals are to bring the oral cavity to a state of health. This may require periodontal procedures, as well as other dental therapy, such as endodontics and correcting oral mucous membrane pathology. Referral to other dental and medical specialties will be necessary.

From a periodontal viewpoint, the short-term goals are important, since they consist of the elimination of gingival inflammation and correction of the conditions that cause and perpetuate it. These include not only elimination of root accretions but also pocket eradication or reduction and establishment of good gingival contours and mucogingival relationships conducive to good periodontal health. Restoration of carious areas and the correction of poor existing restorations may also be necessary.

The *long-term goals* are the reconstruction of a healthy dentition that fulfills all functional and esthetic requirements. Long-term planning involves consideration of prosthetic reconstruction of the dentition, which may require implant therapy, including surgical preparation of the implant site. Also, the need for orthodontic treatment should be evaluated. The financial impact of long-term treatment requires careful consideration and understanding by the patient. The age and medical health status of the patient must also be considered.

The treatment plan is the blueprint for case management. It includes all procedures required for the establishment and maintenance of oral health and involves the following decisions:

- Need for emergency treatment (pain, acute infections).
- Teeth that will require removal.
- Periodontal pocket therapy techniques (surgical or nonsurgical).
- Endodontic therapy.
- The need for occlusal correction, including orthodontic therapy.
- The use of implant therapy.
- The need for caries removal and the placement of temporary and final restorations.
- Prosthetic replacements that may be needed and which teeth will be abutments if a fixed prosthesis is used.
- Decisions regarding esthetic considerations in periodontal therapy.
- Sequence of therapy.

Unforeseen developments during treatment may necessitate modification of the initial treatment plan. However, *except for emergencies, no therapy should be initiated until a treatment plan has been established.*

MASTER PLAN FOR TOTAL TREATMENT

The aim of the treatment plan is total treatment, that is, the coordination of all the short- and long-term goals for the purpose of creating a well-functioning dentition in a healthy periodontal environment. The master plan of periodontal treatment encompasses different areas of therapeutic objectives for each patient according to his or her needs. It is based on the diagnosis, disease severity, risk factors, and other factors outlined in previous chapters.

Extracting or Preserving a Tooth

Periodontal treatment requires long-range planning. Its value to the patient is measured in years of healthy functioning of the entire dentition and not by the number of teeth retained at the time of treatment. *Treatment is directed to establishing and maintaining the health of the periodontium throughout the mouth rather than attempting spectacular efforts to "tighten loose teeth."*

In the past two decades, implant replacement of missing teeth has become a predictable course of therapy. Therefore attempts to

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save questionable teeth may jeopardize adjacent teeth and may lead to the loss of bone needed for implant therapy. Teeth on the borderline of a hopeless prognosis do not contribute to the overall usefulness of the dentition. Such teeth become sources of recurrent problem to the patient and detract from the value of the greater service rendered by the establishment of periodontal health in the remainder of the oral cavity.

Removal, retention, or temporary (interim) retention of one or more teeth is a very important part of the overall treatment plan. A tooth should be extracted under the following conditions:

- It is so mobile that function becomes painful.
- It can cause acute abscesses during therapy.
- There is no use for it in the overall treatment plan.

In some cases, a tooth can be retained temporarily, postponing the decision to extract until after treatment is completed. A tooth in this category can be retained under the following conditions:

- It maintains posterior stops; the tooth can be removed after treatment when it can be replaced by an implant or another type of prosthesis.
- It maintains posterior stops and may be functional after implant placement in adjacent areas. When the implant is restored, these teeth can be extracted.
- In the anterior esthetic zone, a tooth can be retained during periodontal therapy and removed when treatment is completed and a permanent restorative procedure can be performed. The retention of this tooth should not jeopardize the adjacent teeth. This approach avoids the need for temporary appliances during therapy.
- Extraction of hopeless teeth can also be performed during periodontal surgery of the adjacent teeth. This approach reduces the number of appointments needed for surgery in the same area.

In the formulation of the treatment plan, in addition to the proper function of the dentition, esthetic considerations play an important role. Different patients value esthetics differently, according to their age, gender, profession, and social status. The clinician should carefully evaluate and consider a final esthetic outcome of treatment that will be acceptable to the patient without jeopardizing the basic need of attaining health.

With the predictable use of implants, questionable teeth should be carefully evaluated as to whether their removal and replacement with an implant may be a better and more satisfactory course of therapy.

In complex cases, interdisciplinary consultation with other specialty areas is necessary before a final plan can be made. The opinion of orthodontists and prosthodontists is especially important for the final decision in these patients.

Occlusal evaluation and therapy may be necessary during treatment, which may necessitate planning for occlusal adjustment (see Chapters 49 and 50), orthodontics, and splinting. The correction of bruxism and other occlusal habits may also be necessary.

Systemic conditions should be carefully evaluated because they may require special precautions during the course of periodontal treatment. The tissue response to treatment procedures may be affected, or the preservation of periodontal health may be threatened after treatment is completed. The patient's physician should always be consulted when the patient presents with medical and systemic problems that may affect the periodontal therapy.

Supportive periodontal care is also of paramount importance for case maintenance. Such care entails all procedures for maintaining periodontal health after it has been attained. It consists of instruction in oral hygiene and recall therapy at regular intervals, according to the patient's needs.

SEQUENCE OF THERAPEUTIC PROCEDURES

Periodontal therapy is an inseparable part of dental therapy. The list of procedures presented in Box 34-1 includes periodontal procedures (in italics) and other procedures not considered to be within the province of the periodontist. These procedures are listed together in Box 34-1 to emphasize the close relationship of periodontal therapy with other phases of therapy performed by general dentists or other specialists.

The sequence in which these phases of therapy are performed may vary to some extent in response to the requirements of the case. However, the preferred sequence, which covers the vast majority of cases, is shown in Figure 34-1.

Although the phases of treatment have been numbered, the recommended sequence does not follow the numbers. Phase I, or the *nonsurgical phase,* is directed to the elimination of the etiologic

BOX 34-1 Phases of Periodontal Therapy

Preliminary Phase

Treatment of emergencies:

- Dental or periapical
- *Periodontal*
- Other

Extraction of hopeless teeth and provisional replacement if needed (may be postponed to a more convenient time)

Nonsurgical Phase (Phase I Therapy)

Plaque control and patient education:

- Diet control (in patients with rampant caries)
- *Removal of calculus and root planing*
- *Correction of restorative and prosthetic irritational factors*
- Excavation of caries and restoration (temporary or final, depending on whether a definitive prognosis for the tooth has been determined and the location of caries)
- *Antimicrobial therapy (local or systemic)*
- *Occlusal therapy*
- *Minor orthodontic movement*
- *Provisional splinting and prosthesis*

Evaluation of Response to Nonsurgical Phase

Rechecking:

- *Pocket depth and gingival inflammation*
- *Plaque and calculus, caries*

Surgical Phase (Phase II Therapy)

- Periodontal therapy, including placement of implants
- Endodontic therapy

Restorative Phase (Phase III Therapy)

- Final restorations
- Fixed and removable prosthodontic appliances
- Evaluation of response to restorative procedures
- Periodontal examination

Maintenance Phase (Phase IV Therapy)

Periodic rechecking:

- *Plaque and calculus*
- *Gingival condition (pockets, inflammation)*
- *Occlusion, tooth mobility*
- Other pathologic changes

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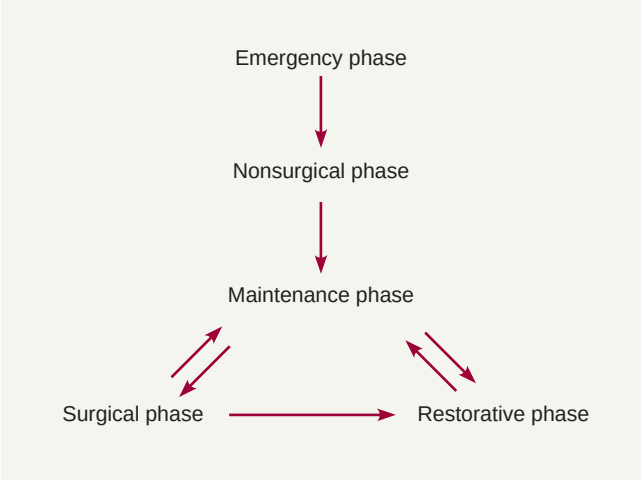

Figure 34-1 Preferred sequence of periodontal therapy.

factors of gingival and periodontal diseases. When successfully performed, this phase stops the progression of dental and periodontal disease.

Immediately after completion of phase I therapy, the patient should be placed on the *maintenance phase* (phase IV) to preserve the results obtained and prevent any further deterioration and recurrence of disease. While on the maintenance phase, with its periodic evaluation, the patient enters into the *surgical phase* (phase II) and the *restorative phase* (phase III) of treatment. These phases include periodontal surgery to treat and improve the condition of the periodontal and surrounding tissues. This may include regeneration of the gingiva and bone for function and esthetics, placement of implants, and restorative therapy.

EXPLAINING TREATMENT PLAN TO THE PATIENT

The following discussion includes suggestions for explaining the treatment plan to the patient.

Be specific. Tell your patient, "You have gingivitis" or "You have periodontitis," then explain exactly what these conditions are.

Avoid vague statements. Do not use statements such as, "You have trouble with your gums" or "Something should be done about your gums." Patients may not understand the significance of such statements and may disregard them.

Begin your discussion on a positive note. Talk about the teeth that can be retained and the long-term service they can be expected to render. Do not begin your discussion with the statement, "The following teeth have to be extracted." This creates a negative impression, which adds to the erroneous attitude of hopelessness the patient already may have regarding his or her mouth. Make it clear that every effort will be made to retain as many teeth as possible, but do not dwell on the patient's loose teeth. Emphasize that the important purpose of the treatment is to prevent the other teeth from becoming as severely diseased as the loose teeth.

Present the entire treatment plan as a unit. Avoid creating the impression that treatment consists of separate procedures, some or all of which may be selected by the patient. Make it clear that dental restorations and prostheses contribute as much to the health of the gingiva as the elimination of inflammation and periodontal pockets. Do not speak in terms of "having the gums treated and then taking care of the necessary restorations later" as if these were unrelated treatments.

Patients often seek guidance from the dentist with questions such as the following:

- "Are my teeth worth treating?"
- "Would you have them treated if you had my problem?"
- "Why don't I just go along the way I am until the teeth really bother me and then have them all extracted?"

Explain that "doing nothing" or holding onto hopelessly diseased teeth as long as possible is inadvisable for the following reasons:

1. Periodontal disease is a microbial infection, and research has clearly shown it to be an important risk factor for severe life-threatening diseases such as stroke, cardiovascular disease, pulmonary disease, and diabetes, as well as for premature low-birth-weight babies in women of childbearing age. Correcting the periodontal condition eliminates a serious potential risk of systemic disease, which in some cases ranks as high on the danger list as smoking.
2. It is not feasible to place restorations or fixed bridges on teeth with untreated periodontal disease because the usefulness of the restoration would be limited by the uncertain condition of the supporting structures.
3. Failure to eliminate periodontal disease not only results in the loss of teeth already severely involved, but also shortens the life span of other teeth. With proper treatment, these teeth can serve as the foundation for a healthy, functioning dentition.

Therefore the dentist should make it clear to the patient that if the periodontal condition is treatable, the best results are obtained by prompt treatment. If the condition is not treatable, the teeth should be extracted.

It is the dentist's responsibility to advise the patient of the importance of periodontal treatment. However, if treatment is to be successful, the patient must be sufficiently interested in retaining his or her natural teeth and to maintain the necessary oral hygiene. Individuals who are not particularly perturbed by the thought of losing their teeth are generally not good candidates for periodontal treatment.

SCIENCE TRANSFER

Treatment planning should not result in just a list of procedures but should be phased. The preliminary phase includes emergency treatment and is followed by the nonsurgical phase I, which leads to phase II, the surgical phase; then phase III, the restorative phase; and the final phase is the maintenance phase. Part of this approach to treatment planning is the incorporation of comprehensive reevaluation of periodontal status on the completion of each phase. This newly updated set of data on the patient's periodontal status is then used to validate or modify the original treatment plan.

Patient involvement is essential; clinicians need to develop good communication skills so that patients are informed and motivated to optimize their periodontal status and have their lives enhanced by a healthy functional dentition.

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CHAPTER 35

Rationale for Periodontal Treatment

Fermin A. Carranza and Henry H. Takei

CHAPTER OUTLINE

WHAT DOES PERIODONTAL THERAPY ACCOMPLISH?
Local Therapy
Systemic Therapy
FACTORS THAT AFFECT HEALING
Local Factors
Systemic Factors
HEALING AFTER PERIODONTAL THERAPY
Regeneration
Repair
New Attachment
PERIODONTAL RECONSTRUCTION

WHAT DOES PERIODONTAL THERAPY ACCOMPLISH?

The effectiveness of periodontal therapy is made possible by the remarkable healing capacity of the periodontal tissues. Periodontal therapy can restore chronically inflamed gingiva so that, from a clinical and structural point of view, it is almost identical with gingiva that has never been exposed to excessive plaque accumulation[20] (see Part 8).

Properly performed, periodontal treatment can eliminate pain, exudate, gingival inflammation,[27] and bleeding. It can also reduce periodontal pockets, eliminate infection, arrest the destruction of soft tissue and bone,[28] and reduce abnormal tooth mobility.[7] Other benefits are to establish optimal occlusal function, restore tissue destroyed by disease, reestablish physiologic gingival contour, and prevent the recurrence of disease, which will all help to maintain the natural dentition[24] (Figure 35-1).

Local Therapy

The cause of periodontitis and gingivitis is bacterial plaque accumulation on the tooth surface close to the gingival tissue. The accumulation of plaque can be favored by a variety of local factors, such as calculus, overhanging margins of restorations, and food impaction. *The removal of plaque and all of the factors that favor its accumulation is the primary goal in local therapy.*

Abnormal forces on the tooth can increase tooth mobility. The thorough elimination of plaque and the prevention of its formation can help maintain periodontal health, even if traumatic forces are allowed to persist.[18,19] However, the elimination of trauma may increase the chances for bone regeneration and the gain of attachment.[15] Although this point is not widely accepted,[25] it appears that creating occlusal relationships that are more tolerable to the periodontal tissues increases the margin of safety of the periodontium to the buildup of plaque, in addition to reducing tooth mobility. It should be remembered that total plaque elimination as obtained in experimental studies may not be possible in human subjects.

Systemic Therapy

Systemic therapy may be employed as an adjunct to local measures for specific purposes such as controlling systemic complications from acute infections or chemotherapy and preventing harmful effects of posttreatment bacteremia. The control of systemic diseases that aggravate the patient's periodontal condition is always a consideration so proper precautions can be instituted during therapy (see Chapters 37, 38, and 39).

Systemic therapy for treatment of the periodontal condition in conjunction with local therapy is indicated in patients with aggressive periodontitis. In these diseases, systemic antibiotics are used to eliminate the bacteria that invade the gingival tissues and can repopulate the pocket after scaling and root planing (see Chapters 40 and 47).

In addition, periodontal manifestations of systemic diseases (see Chapter 28) are treated primarily by other local measures.

In the late twentieth century, the concept of *host modulation* was introduced as a medical approach to periodontal treatment. The classic 1979 paper by Nyman, Schroeder, and Lindhe[23] reported that it was possible to block periodontal bone loss in animals with the aspirinlike drug indomethacin. Evidence was then presented that some *nonsteroidal antiinflammatory drugs* (NSAIDs), such as flurbiprofen and ibuprofen, can reduce the development of experimental gingivitis,[9] as well as the loss of alveolar bone in periodontitis.[11,33–35] These drugs are propionic acid derivatives and act by inhibiting the cyclooxygenase pathway of arachidonic acid metabolism, thereby reducing prostaglandin formation. These NSAIDs can be administered by mouth[13] or applied topically.[33]

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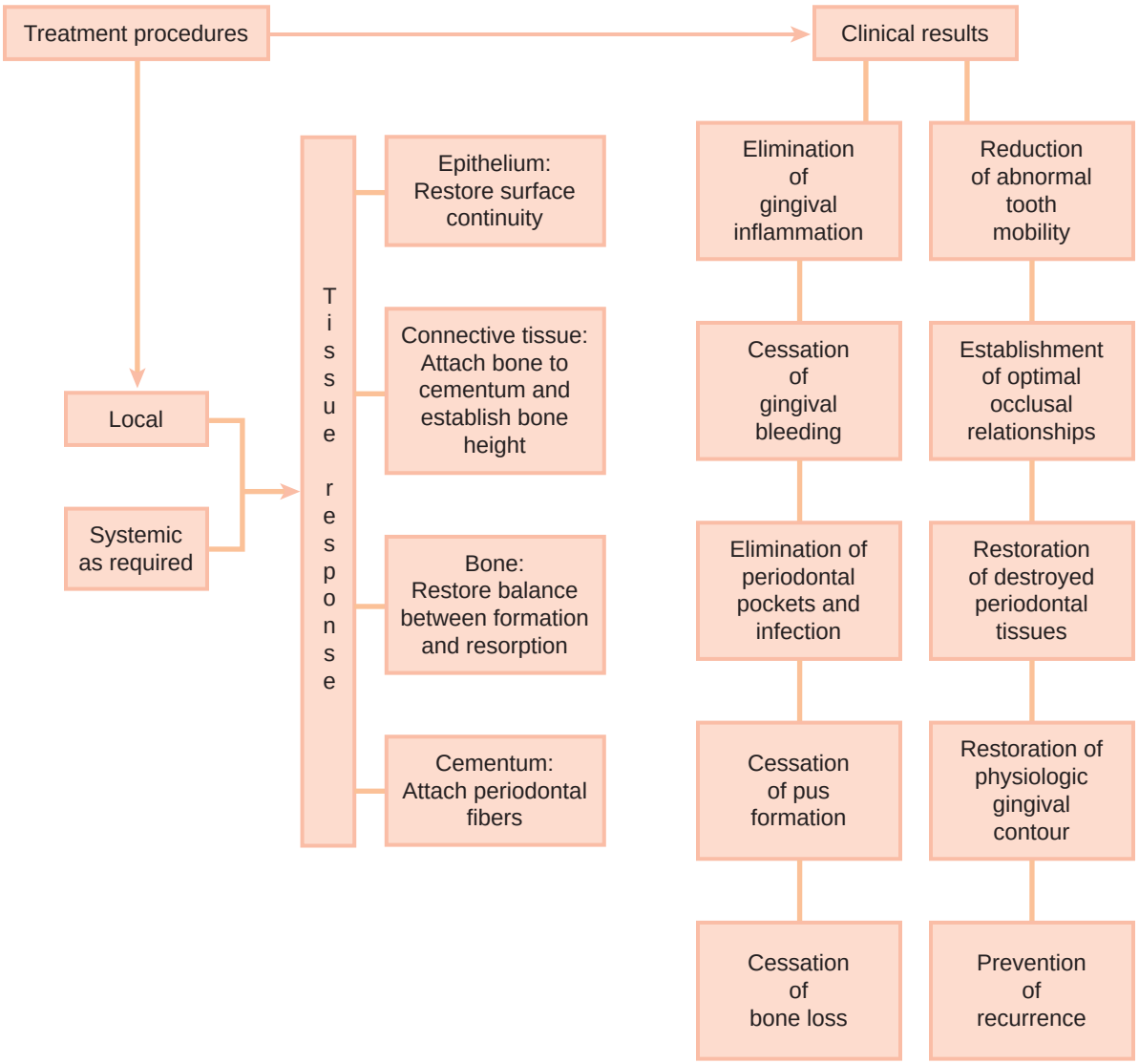

Figure 35-1 Tissue response and clinical results after periodontal treatment.

Another drug that has a strong inhibitory effect on bone resorption is alendronate, a *bisphosphonate,* which is currently used to treat metabolic diseases in humans, such as Paget's disease or hypercalcemia of malignancy, which result in bone resorption. Experimental studies in monkeys have shown that alendronate reduced the bone loss associated with periodontitis.[3,32]

Host modulation is still in its experimental stages, and protocols for its clinical use have not been established. However, current studies indicates that future treatment modalities may attempt not only to control the bacterial cause of the disease but also to suppress the self-destructive components of the host inflammatory response[11] (see Chapter 48).

FACTORS THAT AFFECT HEALING

In the periodontium, as elsewhere in the body, healing is affected by local and systemic factors.

Local Factors

Systemic conditions that impair healing may reduce the effectiveness of local periodontal treatment and should be corrected before or during local procedures. However, local factors, particularly plaque microorganisms, are the most common deterrents to healing after periodontal treatment.

Healing is also delayed by (1) excessive tissue manipulation during treatment, (2) trauma to the tissues, (3) the presence of foreign bodies, and (4) repetitive treatment procedures that disrupt the orderly cellular activity in the healing process. An adequate blood supply is needed for the increased cellular activity during healing. If the blood supply is impaired or insufficient, areas of necrosis will develop and delay the healing process.

Healing is improved by debridement (removal of degenerated and necrotic tissue), immobilization of the healing area, and pressure on the wound. The cellular activity in healing entails an increase in oxygen consumption, but healing of the gingiva is not accelerated by artificially increasing the oxygen supply beyond the normal requirements.[8]

Systemic Factors

The effects of systemic conditions on healing have been extensively documented in animal experiments but are less clearly defined in humans. Healing capacity diminishes with age,[4,10] probably because

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of the atherosclerotic vascular changes common in aging and the resulting reduction in blood circulation. Healing is delayed in patients with generalized infections and in those with diabetes and other debilitating diseases.

Healing is impaired by insufficient food intake; bodily conditions that interfere with the use of nutrients; and deficiencies in vitamin C,[1,31] proteins,[30] and other nutrients. However, the nutrient requirements of the healing tissues in minor wounds, such as those created by periodontal surgical procedures, are usually satisfied by a well-balanced diet.

Healing is also affected by hormones. Systemically administered glucocorticoids such as cortisone hinder repair by depressing the inflammatory reaction or by inhibiting the growth of fibroblasts, the production of collagen, and the formation of endothelial cells. Systemic stress,[29] thyroidectomy, testosterone, adrenocorticotropic hormone (ACTH), and large doses of estrogen suppress the formation of granulation tissue and impair healing.[4] Progesterone increases and accelerates the vascularization of immature granulation tissue[17] and appears to increase the susceptibility of the gingiva to mechanical injury by causing dilation of the marginal vessels.[12]

HEALING AFTER PERIODONTAL THERAPY

The basic healing processes are the same after all forms of periodontal therapy. These processes consist of the removal of degenerated tissue debris and the replacement of tissues destroyed by disease. This implies regeneration and repair of the periodontal structures but not necessarily a gain in attachment. Techniques to gain attachment and bone level are discussed in the sections on new attachment and periodontal reconstruction.

Regeneration

Regeneration is the natural renewal of a structure, produced by growth and differentiation of new cells and intercellular substances to form new tissues or parts. Regeneration occurs through growth from the same type of tissue that has been destroyed or from its precursor. In the periodontium, gingival epithelium is replaced by epithelium, and the underlying connective tissue and periodontal ligament are derived from connective tissue. Bone and cementum are replaced by connective tissue, which is the precursor of both. Undifferentiated connective tissue cells develop into osteoblasts and cementoblasts, which form bone and cementum.

Regeneration of the periodontium is a continuous physiologic process. Under normal conditions, new cells and tissues are constantly being formed to replace those that mature and die; this is termed *wear and tear repair.*[16] It is manifested by (1) mitotic activity in the epithelium of the gingiva and the connective tissue of the periodontal ligament, (2) the formation of new bone, and (3) the continuous deposition of cementum.

Regeneration is occurring even during destructive periodontal disease. Most gingival and periodontal diseases are chronic inflammatory processes and as such are *healing* lesions. Regeneration is part of healing. However, bacteria and bacterial products that perpetuate the disease process, along with the resulting inflammatory exudate, are injurious to the regenerating cells and tissues, thus preventing completion of the healing process.

By removing bacterial plaque and creating the conditions to prevent its new formation, periodontal treatment removes the obstacles to regeneration and enables the patient to benefit from the inherent regenerative capacity of the tissues. A brief "spurt" in regenerative activity occurs immediately after periodontal treatment, but no local treatment procedures promote or accelerate regeneration.

Repair

Repair simply restores the continuity of the diseased marginal gingiva and reestablishes a normal gingival sulcus at the same level on the root as the base of the preexisting periodontal pocket (Figure 35-2, *B*). This process, called *healing by scar,*[26] arrests bone destruction but does not result in gain of gingival attachment or bone height. This return of the destroyed periodontium to health involves regeneration and mobilization of epithelial and connective tissue cells into the damaged area and increased local mitotic divisions to provide sufficient numbers of cells (Figure 35-3).

For the diseased gingiva and attachment apparatus to regain (totally or partially) their level on the root (Figure 35-2, *C*), therapy must include special materials and techniques. If these are not used

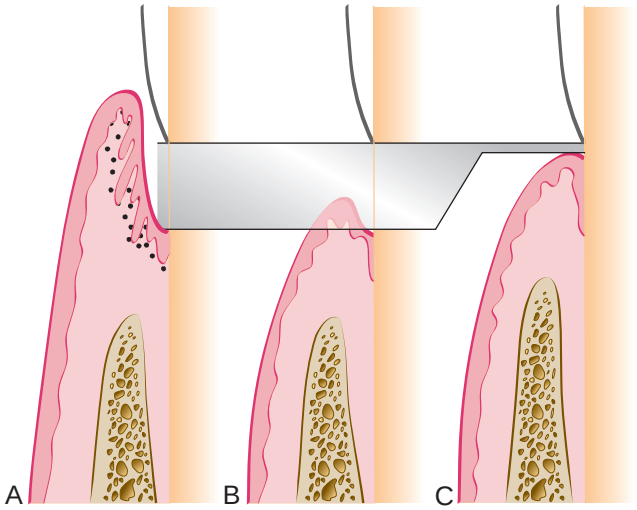

Figure 35-2 Two possible outcomes of pocket elimination. **A,** Periodontal pocket before treatment. **B,** Normal sulcus reestablished at the level of the base of the pocket. **C,** Periodontium restored on the root surface previously denuded by disease; this is called *new attachment.* Shaded areas show denudation caused by periodontal disease.

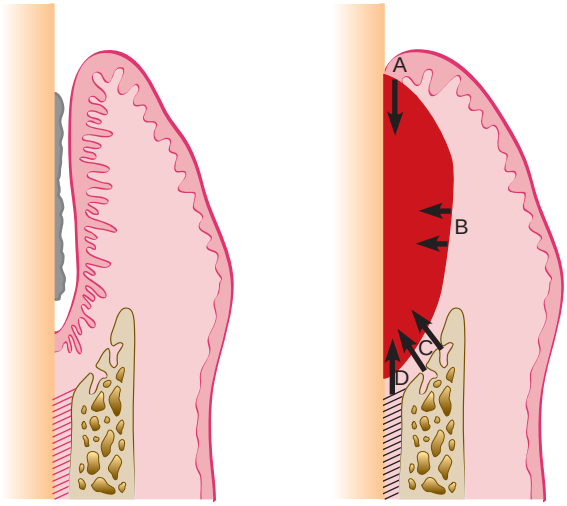

Figure 35-3 Sources of regenerating cells in the healing stages of a periodontal pocket. *Left,* Intrabony pocket. *Right,* After therapy the clot formed is invaded by cells from *A,* the marginal epithelium; *B,* the gingival connective tissue; *C,* the bone marrow; and *D,* the periodontal ligament.

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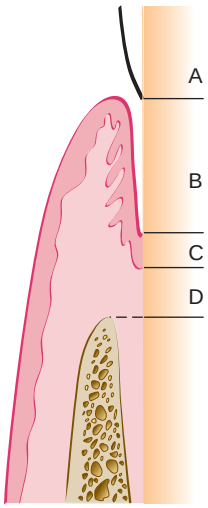

Figure 35-4 Enamel surface *(A)*. Area of cementum denuded by pocket formation *(B)*. Area of cementum covered by junctional epithelium *(C)*. Area of cementum apical to junctional epithelium *(D)*. The term *new attachment* refers to a new junctional epithelium and attached connective tissue fibers formed on zone *B*.

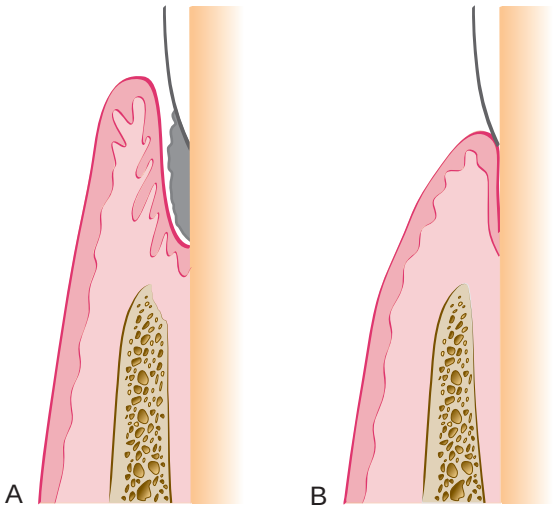

Figure 35-5 Epithelial adaptation after periodontal treatment. **A,** Periodontal pocket. **B,** After treatment. The pocket epithelium is closely adapted to but not attached to the root.

or are not successful, tissues undergo repair only, which involves regeneration of tissue to remodel the attachment apparatus but does not include regaining attachment level or new bone height. For this reason, we prefer to use the term *reconstruction of the periodontium* to refer to the crucial therapeutic techniques that seek to rebuild the periodontium and result in a significant gain of attachment and bone height (see Chapter 61).

New Attachment

New attachment is the embedding of new periodontal ligament fibers into new cementum and the attachment of the gingival epithelium to a tooth surface previously denuded by disease.

The critical phrase in this definition is "tooth surface previously denuded by disease" (Figure 35-4, area *B*). The attachment of the gingiva or the periodontal ligament to areas of the tooth from which they have been removed in the course of treatment (or during preparation of teeth for restorations) represents simple healing or reattachment of the periodontium, not new attachment.[14] The term *reattachment* refers to repair in areas of the root not previously exposed to the pocket such as after surgical detachment of the tissues or following traumatic tears in the cementum, tooth fractures, or the treatment of periapical lesions (Figure 35-4, area *D*).

Epithelial adaptation differs from new attachment in that it is the close apposition of the gingival epithelium to the tooth surface, with no gain in height of gingival fiber attachment. The pocket is not completely obliterated, although it may not permit passage of a probe (Figure 35-5).

However, studies have shown that these deep sulci lined by long, thin epithelium may be as resistant to disease as true connective tissue attachments.[2,21] The absence of bleeding or secretion on probing, the absence of clinically visible inflammation, and the absence of stainable plaque on the root surface when the pocket wall is deflected from the tooth may indicate that the "deep sulcus" persists in an inactive state, causing no further loss of attachment.[5,35] A posttherapy depth of 4 or even 5 mm may therefore be acceptable in these cases.

PERIODONTAL RECONSTRUCTION

As previously discussed, the term *periodontal reconstruction* refers to the process of regeneration of cells and fibers and remodeling of the lost periodontal structures that results in (1) gain of attachment level, (2) formation of new periodontal ligament fibers, and (3) a level of alveolar bone significantly coronal to that present before treatment.

A technique to attain these ideal results has been a constant but elusive goal of periodontal therapy for centuries.[6] Since the 1970s, renewed laboratory and clinical research efforts have resulted in new concepts and techniques that have moved us much closer to attaining this ideal result of therapy. Melcher[22] pointed out that the regeneration of the periodontal ligament is the key to periodontal reconstruction because it "provides continuity between the alveolar bone and the cementum and also because it contains cells that can synthesize and remodel the three connective tissues of the alveolar part of the periodontium." Chapter 61 discusses growth factors that help the maturation of these tissues.

During the healing stages of a periodontal pocket, the area is invaded by cells from four different sources (see Figure 35-3): oral epithelium, gingival connective tissue, bone, and periodontal ligament.

The final outcome of periodontal pocket healing depends on the sequence of events during the healing stages.[22] If the epithelium proliferates along the tooth surface before the other tissues reach the area, the result will be a long junctional epithelium. If the cells from the gingival connective tissue are the first to populate the area, the result will be fibers parallel to the tooth surface and remodeling of the alveolar bone with no attachment to the cementum. If bone cells arrive first, root resorption and ankylosis may occur. Finally, only when cells from the periodontal ligament proliferate coronally is there new formation of cementum and periodontal ligament.[22]

Several methods, based on different concepts and resulting in various techniques, have been recommended to improve the likelihood of gaining new attachment and increased bone levels, as discussed in Chapter 61.

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SCIENCE TRANSFER

The successful treatment of periodontal disease leads to enhancement of the patients quality of life by ensuring a healthy dentition that is functional and has good esthetic qualities. There is also increasing evidence that control of active periodontal disease leads to reduction in specific systemic diseases, such as arteriosclerosis, stroke, pulmonary infections, and diabetes, and the incidence of premature low-birth-weight babies. Clinicians can now advise patients that periodontal treatment has benefits beyond providing stable oral health and that there is a need for closer working relationships between dentists and physicians to expand the availability of periodontal therapy. This applies particularly to patients that have documented risk factors from these systemic health problems.

Surgical and nonsurgical periodontal procedures have a high success rate in obtaining periodontal health by a variety of tissue reactions, including repair and new attachment. There is a continuing improvement in success rates as new procedures and new biologic approaches are instituted, so that clinicians can have increasing confidence in therapeutic success. Maintenance of low levels of bacterial plaque after successful periodontal therapy is the essential element for long-term periodontal stability.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 36

Levels of Clinical Significance

Philippe P. Hujoel

CHAPTER OUTLINE

TANGIBLE VERSUS INTANGIBLE BENEFITS
SIZE OF THE TREATMENT EFFECT
DEFINING FOUR LEVELS OF CLINICAL SIGNIFICANCE
Clinical Significance Level 1
Clinical Significance Level 2
Clinical Significance Level 3
Clinical Significance Level 4
SUMMARY

In a study of periodontal tissue regeneration, investigators reported that a treatment that resulted in a gain of 1.2 mm in clinical attachment level and a reduction of 1 mm in probing pocket depth "may not have a great clinical impact."[4] Another group reported that a treatment that resulted in 0.0 mm gain in clinical attachment level and a reduction of 0.2 mm in probing depth had such clinical significance that it should "be used universally."[26] Different individuals will reach different decisions regarding what is meant by the term *clinical significance.* As a result, the term *clinically significant* has largely become a useless term, more useful to marketers than to clinicians.

The term *clinically significant* could be made more relevant by recognizing (1) the nature of the benefits (tangible/intangible) and (2) the size of the treatment effect (large/small).The presence or absence of these two criteria can be used for classifying clinical significance into four levels. Before doing so, each classification term is described.

TANGIBLE VERSUS INTANGIBLE BENEFITS

The clinical significance of a treatment depends on whether the benefits identified are tangible or intangible to the patient who undergoes the procedure.

Tangible benefits are those treatment outcomes that reflect how a patient feels, functions, or survives. The word "tangible" is defined as "capable of being precisely identified or realized by the mind." Examples of tangible benefits could include improved oral health–related quality of life,[13,14] a decrease in self-reported symptoms (e.g., bleeding) after brushing, prevention of tooth loss, or elimination of a painful periodontal abscess. These examples of treatment benefits can precisely be identified or realized by the *patient's mind,* that is, they are tangible. Tangible benefits can also be referred to as "clinically relevant" benefits or "clinically meaningful" benefits.

Intangible benefits cannot be realized or perceived by the patient's mind. Changes in probing attachment level as a result of scaling, changes in enamel mineralization level as a result of fluoride application, and changes in the size of a periapical radiolucency as a result of a root canal treatment are examples of changes that the patient's mind cannot identify or realize; thus they are described as intangible treatment benefits. Intangible treatment benefits can often be measured objectively by the clinician or by laboratory methods.

A first step in assessing the clinical significance of a treatment is to determine whether the documented treatment benefits are tangible or intangible. This distinction is important because intangible benefits often do not necessarily translate into tangible benefits. A medication that lowers elevated blood lipid levels (an intangible benefit) may shorten lifespan (a tangible patient harm).[19] A treatment that increases bone density (an intangible benefit) can increase fracture risk (a tangible patient harm).[10] A treatment that provides extensive periodontal bone regeneration (an intangible benefit) can lead to tooth loss (a tangible harm).[12]

A treatment that has been shown to provide tangible benefits has a higher level of clinical significance than a treatment for which only evidence of intangible benefits exists. The finding that implant-supported dentures improve quality of life[1] has a higher level of clinical significance than the finding that scaling increases probing attachment levels. The finding that an endodontic treatment reliably eliminates tooth pain has a higher level of clinical significance than the finding that chlorhexidine reduces *Streptococcus mutans* levels.

SIZE OF THE TREATMENT EFFECT

A second important criterion for assessing clinical significance is the size of the treatment effect. The size of the treatment effect is a comparison of the success rates of the experimental treatment and the control treatment. This comparison of treatments can be a subtraction of the success rates, a division of the success rates, or some other mathematic operation. The size of the treatment effect,

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The categorization of levels of clinical significance are based on the size of the treatment effect, the possibility of obtaining the clinical effect, and whether the clinical effect is tangible. Clinicians can often view large benefits by looking at epidemiologic data, whereas smaller benefits will be best documented by randomized controlled trials (RCTs).

Clinical judgment and experience are essential to identify results with a strong statistical significance but with only a minor change that is not clinically significant.

regardless of how it is calculated, has long been recognized as an important part of assessing clinical significance. The larger the likelihood of obtaining an expected benefit of a treatment (relative to a control treatment), the more clinically significant is the treatment. We suggest that if the odds ratio associated with the treatment comparison is 0.25 or smaller (when compared to the control), the size of the treatment effect may be considered large.

The likelihood of obtaining a treatment benefit (relative to the control) determines to a large extent the methodologic and analytic rigor required to establish treatment effectiveness. At one extreme, in all-or-none situations, reliable evidence may result from observations on a small number of patients. For example, no concurrent controlled trials were conducted to assess the effectiveness of general anesthesia. Determining effectiveness for treatments that achieve a dramatic and immediate effect is straightforward, and only essential scientific principles (e.g., consistency of observations across different operators) are considered sufficient evidence of treatment effectiveness. Reportedly, the words, "Gentleman, this is no humbug," were sufficient to convince an audience that general anesthesia was effective.

At another extreme, if the likelihood of obtaining an expected treatment benefit is small, extreme rigor in both design and analysis of controlled clinical trials is required. The benefit of mammography screening for early detection of breast cancer,[8] the benefit of one "clot-buster" drug over another after a myocardial event, and the benefit of local antibiotics in the treatment of periodontitis are all so small that large, randomized controlled trials are required to provide reliable evidence of whether small benefits indeed are associated with treatment.

The likelihood of obtaining a treatment benefit is a determinant of clinical significance; the larger the likelihood, the more confident a patient can feel that a treatment will be successful. Although it is possible to have a clear, unequivocal definition of what constitutes a tangible benefit associated with treatment, it is not possible to have a similar rigorous definition of what can be considered a large likelihood. We define a "large treatment effect" as an odds ratio of 0.25, which can reliably be identified using good epidemiologic methodology.[5,24]

DEFINING FOUR LEVELS OF CLINICAL SIGNIFICANCE

Based on the nature of the benefit (tangible/intangible) and the size of the treatment effect (large/small), four levels of clinical significance can be defined (Table 36-1). In order of decreasing significance levels, these are numbered from 1 to 4.

Clinical Significance Level 1

Treatments of clinical significance level 1 are the "magic bullets" or the "miracle cures" in which the treatment provides a tangible benefit and the size of the treatment effect is large. Examples of such treatments include the use of vitamin C to treat scurvy, bone marrow transplantation to treat leukemia, and dental implants to improve the oral health–related quality of life of edentulous individuals. In all three examples, the benefits of the treatment are tangible and the size of the treatment effect is large.

TABLE 36-1 Definition of Levels of Significance Based on Size and Nature of the Benefit

		Size of the Benefit	
Clinical significance		**Large**‡	**Small**§
Nature of the Benefit	*Tangible**	Level 1	Level 2
	Intangible†	Level 3	Level 4

*Tangible benefits are outcomes that directly measure how a patient feels, functions, or survives.
†Intangible benefits are outcomes that are not perceivable by the patient's mind.
‡A large benefit is defined as one that can reliably be identified using epidemiologic methodology.
§A small benefit is defined as one that requires the conduct of randomized controlled trials for reliable identification.

An understanding of biologic mechanisms of treatment actions is not required to establish that a treatment has clinical significance level 1. Lemon juice was identified as an effective method to prevent scurvy in 1601, but it was not until the beginning of the twentieth century that vitamin C was isolated.[21] Digitalis was discovered as a treatment for "dropsy" long before physicians became aware of the drug's cardiac effects.[25] Lithium is an effective drug for bipolar disorder, but its mechanism of action remains largely unknown.[23] In contrast, hormone replacement therapy (HRT), for which the biologic mechanisms explaining how the drug provided benefits were supposedly so well understood, resulted in more harm than good.[11]

Treatments of clinical significance level 1 are not always immediately accepted or widely used. It took the British Navy 264 years from the time of the observations of Captain James until a universal preventive policy was established to prevent scurvy.[3] This lack of appreciation for this treatment of clinical significance level 1 was unfortunate: "It is estimated that 5000 lives a year were needlessly lost from scurvy during this period: that is a total of nearly 800,000. In the 200 years from 1600 to 1800 nearly 1,000,000 men died of an easily preventable disease. There are in the whole of human history few more notable examples of official indifference and stupidity producing such disastrous consequence to human life."[20] Although it is easy to determine clinical significance level 1 in retrospect, it may be difficult to recognize at the time of discovery.

Clinical Significance Level 2

The term *clinical significance level 2* is used to describe treatments that have demonstrated a tangible benefit but for which the

likelihood of obtaining the benefit from treatment is small. As the size of the benefit of one therapy over another decreases in size, randomized controlled trials (RCTs), often large in size and rigorous in execution and analysis, are required to provide unequivocal evidence that the treatment provides tangible patient benefits. Examples of such treatments include the advantage of tissue plasminogen activator (t-PA) over streptokinase[9] and the benefits of penciclovir in the treatment of herpetic lesions.[18]

Determining the clinical relevance of treatments of clinical significance level 2 is an individual choice in which issues such as cost and side effects often play a more important role. For example, the mortality rate with t-PA is 6.3%, whereas the mortality rate with streptokinase is 7.3%.[9] In other words, there is a 1% increased chance for survival associated with t-PA. In the 1990s, when this treatment was introduced, the increased cost for t-PA was $2000. Is a 1% increased survival probability worth $2000? Different individuals, different governments, and different health insurance companies may decide differently on this important question. Indeed, some individuals may believe that if large RCTs are required to determine treatment effectiveness, the clinical significance of the treatment comes into question.

The use of penciclovir in the treatment of herpetic lesions provides another example of a drug of clinical significance level 2. When applying a 1% penciclovir cream, 70% of the patients report lesion healing by day 6. When applying placebo cream, 59% of the patients report lesion healing by day 6.[18] Is an 11% increased probability of lesion healing by day 6 of sufficient magnitude to refer to the treatment as "clinically relevant"? Once again, the answer to this question is highly individual; the cream may be worth its weight in gold for a young teenager when prom night is approaching, but it may be clinically irrelevant for a more sedate adult. By using the terminology "clinical significance level 2," the concept of small, tangible patient benefit can quickly be communicated without becoming trapped in meaningless discussions regarding the clinical relevance of small benefits.

Clinical Significance Level 3

Treatments of clinical significance level 3 are the magic bullets or miracle cures in the surrogate world in which the beneficial but intangible effects of treatment are so convincing that the need for RCTs may appear unethical. Examples of such treatments include the use of highly active antiretroviral therapy (HAART) in patients with acquired immunodeficiency syndrome (AIDS),[15] imatinib (Gleevec) in the treatment of chronic myeloid leukemia,[16] and chlorhexidine varnish in the prevention of caries.[22]

With a treatment that has the label of "clinical significance level 3," there is always the uncertainty of whether the intangible benefits translate into real, tangible patient benefits. It has been a common observation that the larger the effect size observed on the surrogate, the more likely the surrogate benefit translates into a real, tangible patient benefit.[6]

For certain treatments, such as HAART for AIDS or Gleevec for chronic leukemia, the opportunity may exist to avoid RCTs, and treatments of clinical significance level 3 can become treatments of clinical significance level 1 by means of epidemiologic studies in which the tangible benefits associated with the intangible surrogate changes are identified. For example, drastic changes in the viral load of human immunodeficiency virus (HIV) have been shown to lead to large reductions in the risk of AIDS and death. Using historic controls, it was shown that HAART treatment reduced the AIDS risk by 38% and the mortality risk by 34%.[15] A large surrogate benefit (clinical significance level 3) translated into a large survival benefit (clinical significance level 1).

However, assuming that large, intangible treatment benefits invariably translate into tangible treatment benefits remains a dangerous assumption, no matter how large the effect on the surrogate endpoint. A 40% chlorhexidine varnish used for the prevention of caries was reported to result in a 99.9% mutans streptococci reduction in all 20 subjects treated, and the streptococci stayed below detectable levels for at least 4 weeks in 9 subjects. In contrast, the placebo varnish-sealant led to only a 32% mutans streptococci reduction, and none of the 20 subjects had mutans streptococci below detectable levels for 4 weeks.[22] Based on these data, it was reported that, "Chlorzoin will wipe out dental decay much like smallpox." A subsequent RCT in 1240 children at high risk for caries did not translate into a reduction of large cavities in the teeth, the Chlorzoin group had 6.8 D3 lesions (standard deviation, 6.2), and the placebo group had 6.4 D3 lesions (standard deviation, 6.4), in other words, fewer lesions.[7] This example shows that even large, intangible treatment benefits do not always translate into tangible treatment benefits.

Clinical Significance Level 4

Treatments of clinical significance level 4 are those treatments that have reliable evidence from large RCTs of small, intangible treatment benefits. Because the treatment effects are small, epidemiologic studies are almost always incapable of identifying treatment of clinical significance level 4. In other words, rigorously conducted RCTs are a necessity to reliably identify a small surrogate benefit. Examples of treatments of clinical significance level 4 include those that cause a small decrease in lipid level, a small drop in blood pressure, or a small decrease in pocket depth. Large leaps of faith are often required to jump from the observations that small changes in surrogate endpoints translate into real, tangible benefits.

The use of clofibrate to lower lipid levels is an example of a drug of clinical significance level 4. Clofibrate reduced the mean cholesterol levels from 324 mg to 224 mg and the mean value of triglycerides from 271 to 125 (which can be argued are "not so small" mean changes).[2] Clofibrate was the most widely prescribed lipid-lowering agent in the United States, but the uncertainty remained whether clofibrate actually provided a tangible patient benefit. Advertisements that were widely used in medical journals accurately reflected the clinical uncertainty surrounding use of this drug. A text box within the advertisement stated "It has not been established whether the drug-induced lowering of serum cholesterol or lipid levels has a detrimental, beneficial, or no effect on the mortality and the morbidity due to atherosclerosis or coronary heart disease. Several years will be required before current investigations will yield an answer to this question." A subsequent World Health Organization (WHO) cooperative trial on clofibrate revealed the wisdom of this disclaimer. The trial results showed that clofibrate resulted in excess mortality of 47%, providing yet another example of a misleading surrogate.[19]

Treatment of clinical significance level 4 may cause more harm than good, and there is debate whether the drug-approval process should be changed.[17] If such a change were to occur, it could have significant consequences for periodontal therapies because most approved periodontal therapies are clinical significance level 4, and information on their long-term safety and lack of harm is minimal.

SUMMARY

Two important determinants of clinical significance are the nature of benefit (tangible versus intangible) and the likelihood for obtaining the benefit (when compared to the control treatment). These two characteristics can be used to define 4 hierarchic levels of

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clinical significance. Treatments that provide a tangible patient benefit (levels 1 and 2) are of greater value and should correspond to a higher level of clinical significance than treatments with evidence of only intangible benefits (levels 3 and 4). Similarly, treatments with a large likelihood for clinical improvement (levels 1 and 3) are clinically more significant than treatments with a small likelihood for clinical improvement (levels 2 and 4). Providing 4 hierarchical levels of clinical significance may help clinicians and patients communicate more effectively regarding the clinical significance of a treatment. In particular, the dental clinician should inform his periodontal patients that no unequivocal evidence is available that periodontal treatments provide a tangible patient benefit. We hope that we provide tangible benefits to our patients, but the RCTs are lacking to ensure that we really do. Hopefully, the RCTs that have been asked for since the 1960s regarding the effect of periodontal therapy on tooth loss will be conducted in the coming years.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 37

Periodontal Treatment of Medically Compromised Patients

Perry R. Klokkevold, Brian L. Mealey, and Joan Otomo-Corgel

CHAPTER OUTLINE

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- Ischemic Heart Diseases (online only)
- Congestive Heart Failure (online only)
- Cardiac Pacemakers and Implantable Cardioverter-Defibrillators (online only)
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ENDOCRINE DISORDERS
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MEDICATIONS AND CANCER THERAPIES
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PROSTHETIC JOINT REPLACEMENT
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Some tables and boxes may be out of numeric order in this printed chapter.

Many patients seeking dental care have significant medical conditions that may alter both the course of their oral disease and the therapy provided. Older periodontal patients will have a greater likelihood of having underlying disease. Therefore the therapeutic responsibility of the clinician includes identification of the patient's medical problems to formulate proper treatment plans. Thorough medical histories are paramount.[82] If significant findings are unveiled, consultation with or referral of the patient to an appropriate physician may be indicated. This ensures correct patient management and provides medicolegal coverage to the clinician.

This chapter covers common medical conditions and associated periodontal management. Review of each topic area is general, and the reader is encouraged to consult other references for more detailed coverage of specific disorders. Understanding these conditions will enable the clinician to treat the total patient, not merely the periodontal reflection of underlying disease.

CARDIOVASCULAR DISEASES

Cardiovascular diseases are the most prevalent category of systemic disease in the United States (US) and many other countries, and they are more common with increasing age.[109] Health histories should be closely scrutinized for cardiovascular problems. These conditions include hypertension, angina pectoris, myocardial infarction (MI), previous cardiac bypass surgery, previous cerebrovascular

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SCIENCE TRANSFER

In managing periodontal therapy in medically compromised patients, the clinician should always obtain a consultation from the patient's physician, who will determine the necessary precautions that should accompany periodontal treatment. Changes in recommendations for medically compromised patients are continually occurring. Recently, changes have been suggested for protecting patients with artificial joints from the bacteremias associated with periodontal and other invasive dental procedures. Currently, the American Association of Orthopedic Surgeons (AAOS) recommends systemic antibiotic coverage to accompany dental procedures associated with bacteremias for all patients with artificial joints. Dentists should follow the recommendations from the patient's orthopedic surgeon and utilize the appropriate protocol.

Dentists have the responsibility to understand the role of periodontal inflammation in accentuating certain systemic diseases (e.g., arteriosclerosis, diabetes, and preterm low-birth-weight infants. Thus all clinicians need to be cognizant of the systemic implications of periodontal diseases and their treatment and should stay up to date to provide accurate and detailed information during consultation with the patient's physician.

accident (CVA), congestive heart failure (CHF), presence of cardiac pacemakers or automatic cardioverter-defibrillators, and infective endocarditis (IE).

In most cases the patient's physician should be consulted, especially if stressful or prolonged treatment is anticipated. Short appointments and a calm, relaxing environment help minimize stress and maintain hemodynamic stability.

Hypertension

Hypertension, the most common cardiovascular disease, affects more than 50 million American adults, many of whom are undiagnosed.[40] In 2003, the National Heart, Lung and Blood Institute issued revised guidelines for evaluation and management of hypertension.[22,41,47] The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7) guidelines[22] simplified the classification of blood pressure (Table 37-1).

Compared with previous classification[3,6] schemes that focused on diastolic blood pressure (BP), the JNC-7 guidelines[22] emphasize the importance of systolic BP greater than 140 mm Hg. Systolic blood pressure greater than 140 mm Hg is considered a greater risk factor for cardiovascular disease than elevated diastolic pressure. JNC-7 also introduced a category known as *prehypertension* to replace the more innocuous terms "high normal" and "borderline" hypertension. People with systolic BP between 120 and 139 mm Hg or diastolic BP between 80 and 89 mm Hg are classified as "prehypertensive." Hypertension is now classified into only two categories versus three under past classification schemes for simplicity and because treatment for categories 2 and 3 was essentially the same. *Stage 1 hypertension* is defined by systolic pressure of 140 to 159 mm Hg or diastolic pressure of 90 to 99 mm Hg. *Stage 2 hypertension* is defined by a systolic pressure greater than 160 mm Hg or diastolic pressure greater than 100 mm Hg.

Hypertension is not diagnosed on a single elevated BP recording. Rather, classification is usually based on the average value of two or more BP readings taken at two or more appointments. The higher value of either the systolic or diastolic pressure determines the patient's classification. Patients with hypertension enter the dental practice every day and are particularly common among the older population seen in most periodontal practices. Evidence from

TABLE 37-1 Classification of Adult Blood Pressure

Classification	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Dental Treatment Modifications
Normal	<120	and <80	No changes in dental treatment.
Prehypertension	120–139	or 80–89	No changes in dental treatment. Monitor BP at each appointment.
Stage 1 hypertension	140–159	or 90–99	Inform patient of findings. Routine medical consultation/referral. Monitor BP at each appointment. No changes in dental treatment; minimize stress.
Stage 2 hypertension	≥160	≥100	Inform patient. Medical consultation/referral. Monitor BP at each appointment. If systolic BP is <180 and diastolic is <110, perform selective dental care (routine exam, prophylaxis, restorative nonsurgical endodontics and periodontics); minimize stress. If systolic BP ≥ 180 or diastolic ≥100, give immediate medical consultation/referral, and perform emergency dental care only (to alleviate pain, bleeding, infection)*; minimize stress. Consider stress reduction protocol.

BP, Blood pressure.
*Risk of providing emergency dental care must outweigh risk of possible hypertensive complications.[22,41,47]

the Framingham Heart Study revealed that people with normal BP at age 55 still have a 90% risk of becoming hypertensive later in life.[110]

Hypertension is divided into primary and secondary types. *Primary* (essential) *hypertension* occurs when no underlying pathologic abnormality can be found to explain the disease. Approximately 95% of all hypertensive patients have primary hypertension. The remaining 5% have *secondary hypertension,* in which an underlying etiology can be found and often treated. Examples of the conditions responsible for secondary hypertension are renal disease, endocrinologic changes, and neurogenic disorders.

In early hypertension, the patient may be asymptomatic. If not identified and diagnosed, hypertension may persist and increase in severity, leading eventually to coronary artery disease, angina, MI, CHF, CVA, or kidney failure.[54] The dental office can play a vital role in the detection of hypertension and maintenance care of the patient with hypertensive disease. The first dental office visit should include two BP readings spaced at least 10 minutes apart, which are averaged and used as a baseline. Before the clinician refers a patient to a physician because of elevated BP, readings should be taken at a minimum of two appointments, unless the measurements are extremely high (i.e., systolic pressure >180 mm Hg or diastolic pressure >100 mm Hg). The periodontal recall system is an ideal method for hypertension detection and monitoring. Almost three of every four adult patients with hypertension in the US do not control their BP well enough to attain the goal of systolic pressure less than 140 mm Hg and diastolic pressure less than 90 mm Hg.[19] Lack of compliance with antihypertensive therapy is the primary reason for this failure. Dentists can help achieve greater success in managing hypertension by taking BP readings at each periodontal recall visit.

Periodontal procedures should not be performed until accurate BP measurements and histories have been taken to identify those patients with significant hypertensive disease. The time of day also should be recorded since BP varies significantly throughout the day.[74] Table 37-1 outlines appropriate medical referral or consultation and dental treatment modifications, depending on the patient's stage of hypertension.

Dental treatment for hypertensive patients is generally safe as long as stress is minimized.[54,60] If a patient is currently receiving antihypertensive therapy, consultation with the physician may be warranted regarding the current medical status, medications, periodontal treatment plan, and patient management. Many physicians are not knowledgeable about the nature of specific periodontal procedures. The dentist must inform the physician regarding the estimated degree of stress, length of the procedures, and complexity of the individualized treatment plan. Morning dental appointments were once suggested for hypertensive patients. However, recent evidence indicates that BP generally increases around awakening and peaks at midmorning.[15,74,102] Lower BP levels occur in the afternoon; therefore afternoon dental appointments may be preferred.

No routine periodontal treatment should be given to a patient who is hypertensive and not under medical management. For patients with systolic BP greater than 180 mm Hg or diastolic BP greater than 110 mm Hg, treatment should be limited to emergency care until hypertension is controlled. Analgesics are prescribed for pain and antibiotics for infection. Acute infections may require surgical incision and drainage, although the surgical field should be limited because excessive bleeding may be seen with elevated BP.

When treating hypertensive patients, the clinician should not use a local anesthetic containing an epinephrine concentration greater than 1:100,000 nor should a vasopressor be used to control local bleeding. Local anesthesia without epinephrine may be used for short procedures (<30 minutes). In a patient with hypertensive disease, however, it is important to minimize pain by providing profound local anesthesia to avoid an increase in endogenous epinephrine secretion.[54,60]

The benefits of the small doses of epinephrine used in dentistry far outweigh the potential for hemodynamic compromise. The smallest possible dose of epinephrine should be used, and aspiration before injection of local anesthetics is critical. Intraligamentary injection is generally contraindicated because hemodynamic changes are similar to intravascular injection.[101] If the hypertensive patient exhibits anxiety, use of conscious sedation in conjunction with periodontal procedures may be warranted[107] (see Chapter 55).

Beta-adrenergic receptor antagonists, or β-blockers, are typically used to treat hypertension (Table 37-2). β-blockers are either *cardioselective,* blocking only β-1 cardiac receptors (β1 receptors), or *nonselective,* blocking both β-1 cardiac receptors and β-2 peripheral receptors (β2 receptors). Epinephrine, an α-adrenergic and β-adrenergic agonist, produces an increase in heart rate through direct stimulation of cardiac β-1 receptors. Epinephrine also stimulates α-adrenergic receptors, producing vasoconstriction of arteries, as well as β-2 receptors, causing vasodilation of skeletal muscle arterioles. Administration of local anesthetics containing epinephrine to patients taking nonselective β-blockers (e.g., propranolol, nadolol) may cause elevated BP.[119] Epinephrine-induced α-adrenergic stimulation results in vasoconstriction and increased BP. Because the patient's nonselective medication has blocked the β-2 receptors, epinephrine will not stimulate the normal compensatory β-2 receptor–induced vasodilation. This may result in dramatically increased BP, followed by reflex bradycardia mediated by the vagus nerve and carotid baroreceptors. The end result is a patient with severe hypertension and bradycardia, resulting in a dangerous decrease in vascular perfusion and possible death. Because of this potential complication, epinephrine-containing local anesthetics should be used cautiously and only in very small amounts in patients taking nonselective β-blockers, with careful monitoring of vital signs.[54,119]

The clinician should be aware of the many side effects of various antihypertensive medications. *Postural hypotension* is common and can be minimized by slow positional changes in the dental chair.[54]

TABLE 37-2 Nonselective and Selective β-Adrenergic Receptor Antagonists (β-Blockers)

Generic Name	Trade Name
Nonselective β-Blockers	
Carvedilol	Coreg
Carteolol hydrochloride	Cartrol
Nadolol	Corgard
Penbutolol sulfate	Levatol
Pindolol	Visken
Propranolol hydrochloride	Inderal; Inderal LA
Timolol maleate	Blocadren
Selective β-Blockers	
Acebutolol hydrochloride	Sectral
Atenolol	Tenormin
Betaxolol hydrochloride	Kerlone
Bisoprolol fumarate	Zebeta
Metoprolol tartrate	Lopressor
Metoprolol succinate	Toprol-XL

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Depression is a side effect of which many patients are unaware. Nausea, sedation, oral dryness, lichenoid drug reactions, and gingival overgrowth are associated with certain classes of antihypertensive agents.[60]

For more information on Cardiovascular Diseases, including ischemic heart diseases, congestive heart failure, and cardiac pacemakers and implantable cardioverter-defibrillators, please visit the companion Expert CONSULT website at www.expertconsult.com.

Infective Endocarditis

Infective endocarditis (IE) is a disease in which microorganisms colonize the damaged endocardium or heart valves.[35] Although the incidence of IE is low, it is a serious disease with a poor prognosis, despite modern therapy. The term *infective endocarditis* is preferred to the previous term *bacterial endocarditis* because the disease can also be caused by fungi and viruses. The organisms most often encountered in IE are α-hemolytic streptococci (e.g., *Streptococcus viridans*). However, nonstreptococcal organisms often found in the periodontal pocket have been increasingly implicated, including *Eikenella corrodens, Aggregatibacter actinomycetemcomitans, Capnocytophaga,* and *Lactobacillus species*.[14]

IE has been divided into acute and subacute forms. The *acute* form involves virulent organisms, generally nonhemolytic streptococci and strains of staphylococci, which invade normal cardiac tissue, produce septic emboli, and cause infections that run a rapid, generally fatal course. The *subacute* form, on the other hand, results from colony formation on damaged endocardium or heart valves by low-grade pathogenic organisms; the classic example is rheumatic carditis consequent to rheumatic fever.

Since the last American Heart Association (AHA) publication on prevention of IE in 1997,[25] many have questioned the efficacy of antimicrobial prophylaxis to prevent IE in patients who undergo dental or other procedures and have suggested that the AHA guidelines should be revised.[29,103] Members of the Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee of the AHA Council on Cardiovascular Disease in the Young, and a national and international group of experts on IE extensively reviewed data published on the prevention of IE. The committee concluded that only an extremely small number of IE cases might be prevented by antibiotic prophylaxis for dental procedures (even if such therapy was 100% effective). Consequently, the guidelines were changed and published in a 2008 report.[117] The new guidelines advise that IE prophylaxis should only be recommended for cardiac conditions with the highest risk of adverse outcome from IE (Box 37-1). For these patients, antibiotic prophylaxis is recommended for all dental procedures that involve manipulation of the gingival tissues, periapical tissues, or perforation of the oral mucosa. Antibiotic prophylaxis is not indicated for individuals on the basis of an increased lifetime risk of contracting IE.

The practice of periodontics is intimately concerned with the prevention of IE. However, bacteremia may occur even in the absence of dental procedures, especially in individuals with poor oral hygiene and significant periodontal inflammation. In fact, IE is much more likely to result from frequent exposure to random bacteremias associated with daily activities than caused by a dental procedure.[117] Thus the prevention of periodontal inflammation is paramount. The AHA states that patients who are at risk for IE should "establish and maintain the best possible oral health to reduce potential sources of bacterial seeding." To provide adequate preventive measures for IE, the clinician's major concern should be to reduce the microbial population in the oral cavity so as to minimize soft tissue inflammation and bacteremia.

BOX 37-1 Cardiac Conditions Associated with the Highest Risk of Adverse Outcome from Endocarditis for which Prophylaxis with Dental Procedures Is Recommended*

Infective Endocarditis Prophylaxis Recommended

Previous history of infective endocarditis

Prosthetic cardiac valves or prosthetic material used for cardiac valve repair

Congenital heart disease (CHD), with the following conditions:

- Unrepaired cyanotic CHD, including palliative shunts and conduits
- Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first six months after the procedure
- Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)

Cardiac transplantation recipients who develop cardiac valvulopathy

*Recommendations by the American Heart Association.[117]

Preventive measures to reduce the risk of IE should consist of the following:

1. *Define the susceptible patient.* A careful medical history will disclose the previously mentioned susceptible patients. Health questioning should cover history of all potential categories of risk. If any doubt exists, the patient's physician should be consulted.
2. *Provide oral hygiene instruction.* Oral hygiene should be practiced with methods that improve gingival health. In patients with significant gingival inflammation, oral hygiene should initially be limited to gentle procedures (i.e., oral rinses and gentle toothbrushing with a soft brush) to minimize bleeding. As gingival health improves, more aggressive oral hygiene may be initiated. Oral irrigators are generally not recommended because their use may induce bacteremia.[66] Susceptible patients should be encouraged to maintain the highest level of oral hygiene once soft tissue inflammation is controlled.
3. *During periodontal treatment, currently recommended antibiotic prophylactic regimens* (Table 37-3) *should be practiced with all high-risk patients.* If any doubt regarding susceptibility exists, the patient's physician should be consulted. In patients who have been receiving continuous oral penicillin for secondary prevention of rheumatic fever, penicillin-resistant α-hemolytic streptococci are occasionally found in the oral cavity. It therefore is recommended that an alternate regimen be followed instead. Likewise, if the periodontal patient is taking a systemic antibiotic as part of periodontal therapy, changes in the IE prophylaxis regimen may be indicated. For example, a patient currently taking a penicillin agent after regenerative therapy may be placed on azithromycin before the next periodontal procedure. Patients with early-onset forms of periodontitis often have high levels of *A. actinomycetemcomitans* in the subgingival plaque. This organism has been associated with IE and is often resistant to penicillins. Therefore, in patients with aggressive periodontitis who should be given prophylaxis, Slots et al[100] suggested using tetracycline, 250 mg, 4 times daily for 14 days to

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TABLE 37-3 Recommended Antibiotic Prophylaxis Regimens for Periodontal Procedures in Adults At Risk for Infective Endocarditis

Regimen	Antibiotic	Dosage*
Standard oral regimen	Amoxicillin	2.0 g 30–60 minutes before procedure
Alternate regimen for patients allergic to amoxicillin/penicillin	Clindamycin *or* Azithromycin or clarithromycin *or* Cephalexin or cefadroxil†	600 mg 30–60 minutes before procedure 500 mg 30–60 minutes before procedure 2.0 g 30–60 minutes before procedure
Patients unable to take oral medications	Ampicillin	2.0 g intramuscularly or intravenously within 30 minutes before procedure
Patients unable to take oral medications and allergic to penicillin	Clindamycin *or* Cefazolin*	600 mg intravenously within 30 minutes before procedure (must be diluted and injected slowly) 1.0 g intramuscularly or intravenously within 30 minutes before procedure

*Adult dosages listed. Children's dosages are lower.
†Cephalosporins should not be used in patients with immediate-type hypersensitivity reactions to penicillins (e.g., urticaria, angioedema, anaphylaxis).

eliminate or reduce *A. actinomycetemcomitans,* followed by the conventional prophylaxis protocol at the time of dental treatment.

4. *Periodontal treatment should be designed for susceptible patients to accommodate their particular degree of periodontal involvement.* The nature of periodontal therapy enhances the problems related to the prophylaxis of subacute IE. Patients are faced with long-term therapy, healing periods that extend beyond a 1-day antibiotic regimen, multiple visits, and procedures that easily elicit gingival bleeding.

The following guidelines should aid in the development of periodontal treatment plans for patients susceptible to IE:

- Periodontal disease is an infection with potentially wide-ranging systemic effects. In patients at risk for IE, every effort should be made to eliminate this infection. Teeth with severe periodontitis and a poor prognosis may require extraction. Teeth with less severe involvement in a motivated patient should be retained, treated, and maintained closely.
- All periodontal treatment procedures (including probing) require antibiotic prophylaxis; gentle oral hygiene methods are excluded. Pretreatment chlorhexidine rinses are recommended before all procedures, including periodontal probing, because these oral rinses significantly reduce the presence of bacteria on mucosal surfaces.[25]
- To reduce the number of visits required and thereby minimize the risk of developing resistant bacteria, numerous procedures may be accomplished at each appointment, depending on the patient's needs and ability to tolerate dental treatment.[54]
- When possible, allow at least 7 days between appointments (preferably 10 to 14 days). If this is not possible, select an alternative antibiotic regimen for appointments within a 7-day period.
- Evidence does not support or refute a need to place patients at risk for IE on extended antibiotic regimens after treatment.[54] Therefore patients who have had periodontal surgery are not generally placed on antibiotics for the first week of healing (unless there are specific indications to do so). If patients are placed on such regimens, the dosages are inadequate to prevent endocarditis during ensuing appointments. Therefore the standard prophylactic antibiotic dose is still needed. For example, if a patient was placed on 250 mg of amoxicillin 3 times a day for 10 days after periodontal surgery and was returning to the office for more treatment on the seventh day, the patient would still require a full 2.0-g dose of amoxicillin before that treatment. Alternatively, clindamycin or azithromycin could be used at the second appointment.
- Regular recall appointments, with an emphasis on oral hygiene reinforcement and maintenance of periodontal health, are extremely important for patients susceptible to IE.

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ENDOCRINE DISORDERS

Diabetes

The diabetic patient requires special precautions before periodontal therapy. The two major types of diabetes are *type 1* (formerly known as "insulin-dependent diabetes") and *type 2* (formerly called "non–insulin-dependent diabetes").[56] Over the past decade, the medical management of diabetes has changed significantly in an effort to minimize the debilitating complications associated with this disease.[2,7] Patients are more tightly managing their blood glucose levels (glycemia) through diet, oral agents, and insulin therapy.[55]

If the clinician detects intraoral signs of undiagnosed or poorly controlled diabetes, a thorough history is indicated.[78] The classic signs of diabetes include *polydipsia* (excessive thirst), *polyuria* (excessive urination), and *polyphagia* (excessive hunger, often with unexplained concurrent weight loss). If the patient has any of these signs or symptoms, or if the clinician's index of suspicion is high, further investigation with laboratory studies and physician consultation is indicated. Periodontal therapy has limited success in the presence of undiagnosed or poorly controlled diabetes.

If a patient is suspected of having undiagnosed diabetes, the following procedures should be performed:

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BOX 37-2 Diagnostic Criteria for Diabetes Mellitus

Diabetes mellitus may be diagnosed by any one of three different laboratory methods. Whichever method is used, it must be confirmed on a subsequent day by using any one of the following three methods.

1. Symptoms of diabetes plus casual (nonfasting) plasma glucose ≥200 mg/dl. Casual glucose may be drawn at any time of day without regard to time since the last meal. Classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.
2. Fasting plasma glucose ≥126 mg/dl. "Fasting" is defined as no caloric intake for at least 8 hours. (Normal fasting glucose is 70–100 mg/dl.)
3. Two-hour postprandial glucose ≥200 mg/dl during an oral glucose tolerance test.* The test should be performed using a glucose load containing the equivalent of 75 g of anhydrous glucose dissolved in water. (Normal 2-hour postprandial glucose is <140 mg/dl.)

Data from American Diabetes Association: *Diabetes Care* 26(suppl 1):5, 2003.
*The third method is not recommended for routine clinical use.

BOX 37-3 Laboratory Evaluation of Diabetes Control: Glycosylated Hemoglobin Assay (HbA1c)*

4%–6%	Normal
<7%	Good diabetes control
7%–8%	Moderate diabetes control
>8%	Action suggested to improve diabetes control

*American Diabetes Association guidelines.

1. Consult the patient's physician.
2. Analyze laboratory tests (Box 37-2): fasting blood glucose and casual glucose.[9]
3. Rule out acute orofacial infection or severe dental infection; if present, provide emergency care immediately.
4. Establish best possible oral health through nonsurgical debridement of plaque and calculus; institute oral hygiene instruction. Limit more advanced care until diagnosis has been established and good glycemic control obtained.

If a patient is known to have diabetes, it is critical that the level of glycemic control be established before initiating periodontal treatment. The fasting glucose and casual glucose tests provide "snapshots" of the blood glucose concentration at the time the blood was drawn; these tests reveal nothing about long-term glycemic control. The primary test used to assess glycemic control in a known diabetic individual is the *glycosylated* (or glycated) *hemoglobin* (Hb) *assay* (Box 37-3). Two different tests are available, the HbA1 and the HbA1c assay; the HbA1c is used more often.[55] This assay has been shown by a large international study to provide an accurate measure of the average blood glucose concentrations over the preceding 2 to 3 months.[63] Table 37-4 lists the average blood glucose concentrations for HbA1c values from that study, and Figure 37-2 is a simplified graphic representation of the data. The therapeutic goal for many patients is to achieve and maintain an HbA1c below 8%. Patients with relatively well-controlled diabetes (HbA1c < 8%) usually respond to therapy in a manner similar to nondiabetic individuals.[23,105,116] Poorly controlled patients (HbA1c > 10%) often have a poor response to treatment, with more postoperative complications and less favorable long-term results[56,105]

TABLE 37-4 Comparison of Hba1c Values to Estimated Average Glucose Measurements

HbA1c (%)	Estimated Average Glucose (mg/dl)
5.0	97
5.5	111
6.0	126
6.5	140
7.0	154
7.5	169
8.0	183
8.5	197
9.0	212
9.5	226
10.0	240
10.5	255
11.0	269
11.5	283
12.0	298

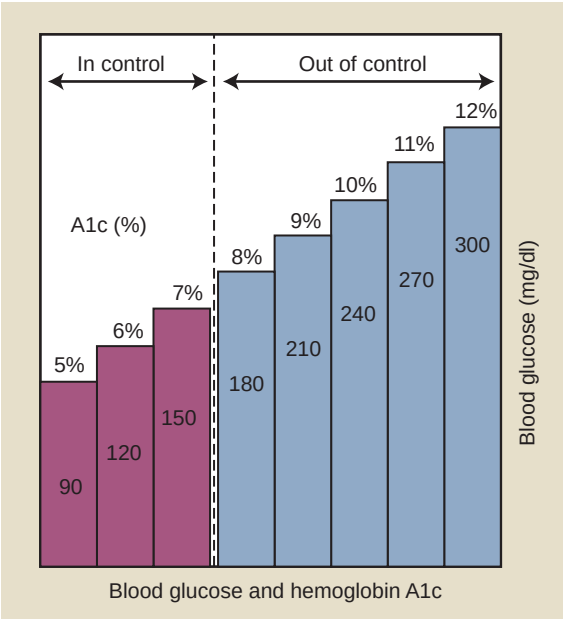

Figure 37-2 Graphic representation of HbA1c values and estimated average blood glucose.

(see Figure 27-3). Improvements in HBA1c values after periodontal therapy may provide an indication of the potential response.

As discussed in Chapter 27, periodontal infection may worsen glycemic control and should be managed aggressively. Diabetic patients with periodontitis should receive oral hygiene instructions, mechanical debridement to remove local factors, and regular maintenance. When possible, an HbA1c of less than 10% should be established before surgical treatment is performed. Systemic antibiotics are not needed routinely, although recent evidence indicates that tetracycline antibiotics in combination with scaling and root planing may positively influence glycemic control. If the patient has poor glycemic control and surgery is absolutely needed, prophylactic antibiotics may be given; penicillins are most often used for this purpose. Frequent reevaluation after active therapy is needed to assess treatment response and prevent recurrence of periodontitis.

Almost all diabetic patients use glucometers for immediate blood glucose self-monitoring. These devices use capillary blood from a simple fingerstick to provide blood glucose readings in seconds. Diabetic patients should be asked whether they have glucometers and how often they use them. Because these devices provide instantaneous assessment of blood glucose, they are highly beneficial in the dental office environment. The following guidelines should be observed:

1. Patients should be asked to bring their glucometer to the dental office at each appointment.
2. Patients should check their blood glucose before any long procedure to obtain a baseline level. Patients with blood glucose levels at or below the lower end of normal before the procedure may become hypoglycemic intraoperatively. It is advisable to have such a patient consume some carbohydrate before starting treatment. For example, if a 2-hour procedure is planned and the pretreatment glucose level is 70 mg/dl (lower end of normal range), providing 4 oz of juice preoperatively may help prevent hypoglycemia during treatment. If pretreatment glucose levels are excessively high, the clinician should determine whether or not the patient's glycemic control has been poor recently. This can be done by thorough patient questioning and by determining the most recent HbA1c values. If glycemic control has been poor over the preceding few months, the procedure may need to be postponed until better glycemic control is established. If glycemic control has been good, and the currently high glucometer reading is a fairly isolated event, the surgical procedure may proceed.
3. If the procedure lasts several hours, it is often beneficial to check the glucose level during the procedure to ensure that the patient does not become hypoglycemic.
4. After the procedure, the blood glucose can be checked again to assess fluctuations over time.
5. Any time the patient feels symptoms of hypoglycemia, blood glucose should be checked immediately. This may prevent onset of severe hypoglycemia, a medical emergency.

The most common dental office complication seen in diabetic patients taking insulin is symptomatic low blood glucose, or *hypoglycemia* (Box 37-4). Hypoglycemia is also associated with the use of numerous oral agents (Table 37-5). In patients receiving conscious sedation, the warning signs of an impending hypoglycemic episode may be masked, making the patient's glucometer one of the best diagnostic aids. Hypoglycemia does not usually occur until blood glucose levels fall below 60 mg/dl. However, in patients with poor glycemic control who have prolonged *hyperglycemia* (high blood glucose levels), a rapid drop in glucose can precipitate signs and symptoms of hypoglycemia at levels well above 60 mg/dl.

As medical management of diabetes has intensified over the last decade, the incidence of severe hypoglycemia has risen.[5] The

BOX 37-4 Signs and Symptoms of Hypoglycemia

Shakiness or tremors
Confusion
Agitation and anxiety
Sweating
Tachycardia
Dizziness
Feeling of "impending doom"
Unconsciousness
Seizures

TABLE 37-5 Oral Agents Used in Management of Diabetes

Agent	Action	Risk of Hypoglycemia
Sulfonylureas (first generation): Chlorpropamide Tolbutamide Tolazamide	Stimulate pancreatic insulin secretion	++
Sulfonylureas (second generation): Glyburide Glipizide	Stimulate pancreatic insulin secretion	+++
Sulfonylureas (third generation): Glimepiride	Stimulate pancreatic insulin secretion	+
Meglitinides: Repaglinide Nateglinide	Stimulate rapid pancreatic insulin secretion (different mechanism than sulfonylureas)	+
Biguanides: Metformin	Block production of glucose by liver; improve tissue sensitivity to insulin	–
Thiazolidinediones: Rosiglitazone Pioglitazone	Improve tissue sensitivity to insulin	–
a-Glucosidase inhibitors: Acarbose Miglitol	Slow absorption of some carbohydrates from gut, decreasing postprandial peaks in glycemia	–
Dipeptidyl peptidase-4 (DPP-4) inhibitors: Sitagliptin Saxagliptin	Inhibit the enzyme DPP-4; enable the pancreas to produce more insulin but only after food ingestion	–
Combination agents	Combine two different oral agents into single drug	Risk depends on which drugs are combined

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TABLE 37-6 Types of Insulin

Type	Classification	ACTIVITY		
		Onset	Peak	Duration
Lispro; Aspart; Glulisine	Rapid acting	15 minutes	30–90 minutes	<5 hours
Regular	Short acting	30–60 minutes	2–3 hours	4–12 hours
NPH	Intermediate acting	2–4 hours	4–10 hours	14–18 hours
Levemir	Long acting	1–2 hours	Relatively flat	up to 24 hours
Glargine	Long acting	6–8 hours	"Peakless" (has no peak in activity)	20–24+ hours

clinician should question diabetic patients about past episodes of hypoglycemia. Hypoglycemia is more common in patients with better glycemic control. When planning dental treatment, it is best to schedule appointments before or after periods of peak insulin activity. This requires knowledge of the pharmacodynamics of the drugs being taken by the diabetic patient. Patients taking insulin are at greatest risk, followed by those taking sulfonylurea agents. Metformin and thiazolidinediones generally do not cause hypoglycemia (see Table 37-5).

Insulins are classified as rapid-acting, short-acting, intermediate-acting, or long-acting agents (Table 37-6). The categories vary in their onset, peak, and duration of activity. It is important that the clinician establish exactly which insulins the diabetic patient takes, the amount, the number of times per day, and the time of the last dose. Periodontal treatment often can be timed to avoid peak insulin activity. Many diabetic patients take multiple injections each day, in which case it is difficult, if not impossible, to avoid peak insulin activity. Checking the pretreatment glucose with the patient's glucometer, checking again during long procedures, and checking again at the end of the procedure provides a better understanding of the patient's insulin pharmacodynamics and help prevent hypoglycemia.

If hypoglycemia occurs during dental treatment, therapy should be immediately terminated. If a glucometer is available, the blood glucose level should be checked. Treatment guidelines include the following[55]:

1. Provide approximately 15 g of oral carbohydrate to the patient:
   - 4 to 6 oz of juice or soda
   - 3 or 4 tsp of table sugar
   - Hard candy with 15 g of sugar
2. If the patient is unable to take food or drink by mouth, or if the patient is sedated:
   - Give 25 to 30 ml of 50% dextrose intravenous (IV), which provides 12.5 to 15.0 g of dextrose, or
   - Give 1 mg of glucagon IV (glucagon results in rapid release of stored glucose from the liver), or
   - Give 1 mg of glucagon intramuscularly or subcutaneously (if no IV access).

Emergencies resulting from hyperglycemia are rare in the dental office. They generally take days to weeks to develop. However, the glucometer may be used to rule out hyperglycemic emergencies such as diabetic ketoacidosis, a life-threatening event.

Because periodontal therapy may render the patient unable to eat for some time, adjustment in insulin or oral agent dosages may be required. It is absolutely critical that patients eat their normal meal before dental treatment. Taking insulin without eating is the primary cause of hypoglycemia. If the patient is restricted from eating before treatment (e.g., for conscious sedation), normal insulin doses will need to be reduced. As a general guideline, *well-controlled diabetic patients having routine periodontal treatment may take their normal insulin doses as long as they also eat their normal meal.* If the procedures are going to be particularly long, the insulin dose before treatment may need to be reduced. Likewise, if the patient will have dietary restrictions after treatment, insulin or sulfonylurea dosages may need to be reduced.

Consultation with the patient's physician is prudent and allows both practitioners to review the proposed treatment plan and determine any modifications needed. When periodontal surgery is indicated, it is usually best to limit the size of the surgical fields so that the patient will be comfortable enough to resume a normal diet immediately.

For more information on Endocrine Disorders, including thyroid and parathyroid disorders and adrenal insufficiency, and to see Box 37-5, please visit the companion Expert CONSULT website at www.expertconsult.com.

HEMORRHAGIC DISORDERS

Patients with a history of bleeding problems caused by disease or drugs should be managed to minimize risks of hemorrhage. Identification of these patients through the health history, clinical examination, and clinical laboratory tests is paramount. Health questioning should cover (1) history of bleeding after previous surgery or trauma, (2) past and present drug history, (3) history of bleeding problems among relatives, and (4) illnesses associated with potential bleeding problems.

Clinical examination should detect the presence of jaundice, ecchymosis, spider telangiectasia, hemarthrosis, petechiae, hemorrhagic vesicles, spontaneous gingival bleeding, and gingival hyperplasia. Laboratory tests should include methods to measure the hemostatic, coagulation, or lytic phases of the clotting mechanism, depending on clues regarding which phase is involved (Table 37-8). These tests include bleeding time, tourniquet test, complete blood cell count, prothrombin time (PT), partial thromboplastin time (PTT), and coagulation time.

Bleeding disorders may be classified as coagulation disorders, thrombocytopenic purpuras, or nonthrombocytopenic purpuras.

Coagulation Disorders

The main inherited coagulation disorders include hemophilia A, hemophilia B and von Willebrand's disease[78,88] (Table 37-9 online). *Hemophilia A* results in a deficiency of coagulation factor VIII, and the clinical severity of the disorder depends on the level of factor VIII remaining.[67] Patients with severe hemophilia who have less than 1% of normal factor VIII levels may have severe bleeding on the slightest provocation, whereas those with more moderate

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TABLE 37-8 Laboratory Tests for Bleeding Disorders

HEMOSTATIC TESTS			
Vascular	**Platelet**	**Coagulation**	**Lytic**
1. Tourniquet test *N:* 10 petechiae *Abn:* >10 petechiae	1. Platelet count *N:* 150,000–300,000/mm^3 *Abn:* Thrombocytopenia = <100,000/mm^3; clinical bleeding occurs at <80,000/mm^3; spontaneous bleeding occurs at <20,000/mm^3.	1. PT measures extrinsic and common pathways: factors I, II, V, VII, and X. *N:* 11–14 sec (depending on laboratory) measured against control PT reported as international normalized ratio (INR): *N:* INR = 1.0 *Abn:* INR >1.5	1. Euglobin clot lysis time *N:* <90 min *Abn:* >90 min
2. Bleeding time *N:* 1–6 min *Abn:* >6 min	2. Bleeding time 3. Clot retraction 4. Complete blood cell count	2. PTT measures intrinsic and common pathways: factors III, IX, XI, and low levels of factors I, II, V, X, and XII. *N:* 25–40 sec (depending on laboratory) measured against control *Abn:* >1.5 times normal 3. Clotting (coagulation) time *N:* 30–40 min *Abn:* >1 hr	
Clinical Disease Association			
Vascular (capillary) wall defect *Rule out:* Thrombocytopenia Purpuras Telangiectasia Aspirin or NSAID therapy Leukemia Renal dialysis	Thrombocytopenia *Rule out:* Vascular wall defect Acute/chronic leukemia Aplastic anemia Liver disease Renal dialysis	*All three tests:* Liver disease Warfarin therapy Aspirin or NSAID therapy Malabsorption syndrome or long-term antibiotic therapy (lack of vitamin K utilization) PT: Factor VII deficiency PTT: Hemophilia Renal dialysis	Increase in fibrinolytic activity

N, Normal; *Abn,* abnormal; *PT,* prothrombin time; *PTT,* partial thromboplastin time; *NSAID,* nonsteroidal antiinflammatory drug.

TABLE 37-9 Inherited Coagulation Disorders

Type	Prolonged	Normal	Treatment
Hemophilia A	PTT	PT Bleeding time	DDAVP (desmopressin) factor VIII concentrate or cryoprecipitate Fresh-frozen plasma Fresh whole blood ε-Aminocaproic acid (Amicar) Tranexamic acid
Hemophilia B	PTT	PT Bleeding time	Purified prothrombin complex concentrates Factor IX concentrates Fresh-frozen plasma
Von Willebrand's disease	Bleeding time PTT Variable factor VIII deficiency	PT Platelet count	DDAVP (desmopressin) factor VIII concentrate or cryoprecipitate

PT, Prothrombin time; *PTT,* partial thromboplastin time; *DDAVP,* 1-deamino-8-D-arginine vasopressin.

hemophilia (1% to 5% factor VIII) have less frequent spontaneous hemorrhage but still bleed with minimal trauma.[54] Patients with mild hemophilia (6% to 30% factor VIII) rarely bleed spontaneously but may still have hemorrhage after severe trauma or during surgical procedures. The clinician should consult the patient's physician before dental treatment to determine the risk for bleeding and treatment modifications required. To prevent surgical hemorrhage, factor VIII levels of at least 30% are needed.[54,67] Parenteral 1-deamino-8-D-arginine vasopressin (DDAVP; desmopressin) can be used to raise factor VIII levels twofold to threefold in

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patients with mild or moderate hemophilia. DDAVP has the significant advantage of avoiding the risk of viral disease transmission from factor VIII infusion and is considered the drug of choice in responsive patients. Most patients with moderate and severe hemophilia require infusion of factor VIII concentrate before surgical procedures. Before 1985, the risk of viral disease transmission from these infusions was high. In recent years, virally safe, highly purified monoclonal antibody or recombinant DNA factor VIII products have come into widespread use.

Hemophilia B, or Christmas disease, results in a deficiency of factor IX. The severity of the disorder depends on the relative amount of existing factor IX. Surgical therapy requires a factor IX level of 30% to 50% and is usually achieved by administration of purified prothrombin complex concentrates or factor IX concentrates.[67]

Von Willebrand's disease results from a deficiency of von Willebrand factor, which mediates adhesion of platelets to the injured vessel wall and is required for primary hemostasis. Von Willebrand factor also carries the coagulant portion of factor VIII in the plasma. The disorder has three major subtypes with a wide range of clinical severity. In fact, many cases of von Willebrand's disease go undiagnosed, and bleeding during dental treatment may be the first sign of the underlying disease. More severe forms require preoperative factor VIII concentrate or cryoprecipitate infusion. Patients with milder forms respond favorably to administration of DDAVP before periodontal surgery or tooth extraction.[67,68]

Periodontal treatment may be performed in patients with these coagulation disorders, provided that sufficient precautions are taken. Probing, scaling, and prophylaxis can usually be done without medical modification. More invasive treatment, such as local block anesthesia, root planing, or surgery, dictate prior physician consultation.

During treatment, local measures to ensure clot formation and stability are of major importance. Complete wound closure and application of pressure will reduce hemorrhage. Antihemostatic agents, such as oxidized cellulose or purified bovine collagen, may be placed over surgical sites or into extraction sockets. The antifibrinolytic agent *ε-aminocaproic acid* (Amicar), given orally or via IV, is a potent inhibitor of initial clot dissolution.[44] *Tranexamic acid* is a more potent antifibrinolytic agent than Amicar and has been shown to prevent excessive oral hemorrhage after periodontal surgery and tooth extraction.[77] It is available as an oral rinse and may be used either alone or in combination with systemic tranexamic acid for several days after surgery.[99]

Not all coagulation disorders are hereditary. Liver disease may affect all phases of blood clotting because most coagulation factors are synthesized and removed by the liver. Long-term alcohol abusers or chronic hepatitis patients often demonstrate inadequate coagulation. Coagulation may be impaired by vitamin K deficiency, often caused by malabsorption syndromes, or by prolonged antibiotic administration, which alters the intestinal microflora that produces vitamin K. Dental treatment planning for patients with liver disease should include the following:

1. Physician consultation.
2. Laboratory evaluations: PT, bleeding time, platelet count, and PTT (in patients in later stages of liver disease).
3. Conservative, nonsurgical periodontal therapy, whenever possible.
4. If surgery is required (may require hospitalization):
   - International normalized ratio (INR; PT) should generally be less than 2.0. For simple surgical procedures, INR less than 2.5 is generally safe.[42]
   - Platelet count should be *more* than 80,000/mm^3.

Anticoagulant Medications. The most common cause of abnormal coagulation may be drug therapy. Patients with prosthetic heart valves or histories of MI, CVA, or thromboembolism are frequently placed on anticoagulant therapy using coumarin derivatives such as dicumarol and warfarin.[42,51] These drugs are vitamin K antagonists that decrease production of vitamin K–dependent coagulation factors II, VII, IX, and X. The effectiveness of anticoagulation therapy is monitored by PT laboratory test. The recommended level of therapeutic anticoagulation for most patients is an INR of 2.0 to 3.0, with prosthetic heart valve patients generally in the 2.5 to 3.5 range.[42] Traditional recommendations for periodontal treatment are as follows:

1. Consult the patient's physician to determine the nature of the underlying medical problem and the degree of required anticoagulation.
2. The procedure to be done determines the acceptable INR. Infiltration anesthesia, scaling, and root planing may be done safely in patients with an INR less than 3.0. Block anesthesia, minor periodontal surgery, and simple extractions usually require an INR less than 2.0 to 2.5. Complex surgery or multiple extractions may require an INR less than 1.5 to 2.0.
3. The physician must be consulted about any changes (discontinuing or reducing) in anticoagulant dosage until the desired INR is achieved. The dentist must inform the physician what degree of intraoperative and postoperative bleeding is usually expected with the procedures planned. If the INR is higher than the level at which significant bleeding is likely to accompany a particular procedure, the physician may elect to change anticoagulant therapy. Often, the anticoagulant is discontinued for 2 to 3 days before periodontal treatment (clearance half-life of warfarin is 36 to 42 hours), and the INR is checked on the day of therapy. If the INR is within the acceptable target range, the procedure is done and the anticoagulant resumed immediately after treatment.
4. Careful technique and complete wound closure are paramount. For all procedures, application of pressure can minimize hemorrhage. Use of oxidized cellulose, microfibrillar collagen, topical thrombin, and tranexamic acid should be considered for persistent bleeding.

NOTE: Discontinuing anticoagulant therapy before dental surgery (as previously stated) was common in the past. However, many clinicians may no longer recommend discontinuing anticoagulation for many procedures because this has significant potential risks to patient health.[43,90] Recent evidence related to the risks of altering anticoagulant therapy along with the lack of evidence for bleeding complications suggest that treating patients without reducing or discontinuing medications may be more prudent. See later section on anticoagulant/antiplatelet therapy under section for a detailed explanation of these more recent considerations.

Heparin is generally used for short-term anticoagulation and is given IV (usually in a hospital environment). It is a powerful anticoagulant with a duration of action of 4 to 8 hours. Periodontal treatment is rarely required while a patient is taking heparin.

Antiplatelet Medications. Aspirin interferes with normal platelet aggregation and can result in prolonged bleeding. Because it binds irreversibly to platelets, the effects of aspirin last at least 4 to 7 days. Aspirin is generally used in small doses of 325 mg or less per day, which usually does not alter bleeding time. In general, patients taking low doses of aspirin daily do not need to discontinue aspirin therapy before periodontal procedures.[90] However, higher doses may increase bleeding time and predispose the patient

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to postoperative bleeding.[54] For patients taking more than 325 mg of aspirin per day, aspirin may need to be discontinued 7 to 10 days before surgical therapy that might result in significant bleeding, in consultation with the physician. Nonsteroidal antiinflammatory drugs (NSAIDs), such as ibuprofen, also inhibit platelet function. Because NSAIDs bind reversibly, the effect is transitory, lasting only a short time after the last drug dose. The bleeding time is used when questions arise about the potential effect of aspirin or NSAIDs. Aspirin should not be prescribed for patients who are receiving anticoagulation therapy or who have illnesses related to bleeding tendencies.

Thrombocytopenic Purpuras

Thrombocytopenia is defined as a platelet count of less than 100,000/mm^3. Bleeding caused by thrombocytopenia may be seen with idiopathic thrombocytopenic purpuras, radiation therapy, myelosuppressive drug therapy (e.g., chemotherapy), leukemia, or infections. Purpuras are hemorrhagic diseases characterized by extravasation of blood into the tissues under the skin or mucosa, producing spontaneous petechiae (small red patches) or ecchymoses (bruises).

Periodontal therapy for patients with thrombocytopenia should be directed toward reducing inflammation by removing local irritants to avoid the need for more aggressive therapy.[54,67] Oral hygiene instructions and frequent maintenance visits are paramount. Physician referral is indicated for a definitive diagnosis and to determine any alterations in planned therapy. Scaling and root planing are generally safe unless platelet counts are less than 60,000/mm^3. No surgical procedures should be performed unless the platelet count is greater than 80,000/mm^3. Platelet transfusion may be required before surgery. Surgical technique should be as atraumatic as possible, and local hemostatic measures should be applied.

Nonthrombocytopenic Purpuras

Nonthrombocytopenic purpuras result from either vascular wall fragility or thrombasthenia (impaired platelet aggregation). Vascular wall fragility may result from hypersensitivity reactions, scurvy, infections, chemicals (phenacetin, aspirin), dysproteinemia, and other causes. Thrombasthenia occurs in uremia, Glanzmann's disease, aspirin ingestion, and von Willebrand's disease.[67] Both types of nonthrombocytopenic purpura may result in immediate bleeding after gingival injury. Treatment consists primarily of direct pressure applied for at least 15 minutes. This initial pressure should control the bleeding unless coagulation times are abnormal or reinjury occurs. Surgical therapy should be avoided until the qualitative and quantitative platelet problems are resolved.

Blood Dyscrasias

Numerous disorders of red and white blood cells may affect the course of periodontal therapy. Alterations in wound healing, bleeding, tissue appearance, and susceptibility to infection may occur. Clinicians should be aware of the clinical signs and symptoms of blood dyscrasias, availability of screening laboratory tests, and need for physician referral.

Leukemia

Altered periodontal treatment for patients with leukemia is based on their enhanced susceptibility to infections, bleeding tendency, and the effects of chemotherapy.[54] The treatment plan for leukemia patients is as follows:

1. Refer the patient for medical evaluation and treatment. Close cooperation with the physician is required.
2. Before chemotherapy, a complete periodontal treatment plan should be developed with a physician (see previous discussion).
   - Monitor hematologic laboratory values daily: bleeding time, coagulation time, PT, and platelet count.
   - Administer antibiotic coverage before any periodontal treatment because infection is a major concern.
   - Extract all hopeless, nonmaintainable, or potentially infectious teeth at least 10 days before the initiation of chemotherapy, if systemic conditions allow.
   - Periodontal debridement (scaling and root planing) should be performed and thorough oral hygiene instructions given if the patient's condition allows. Twice-daily rinsing with 0.12% chlorhexidine gluconate is recommended after oral hygiene procedures. Recognize the potential for bleeding caused by thrombocytopenia. Use pressure and topical hemostatic agents as indicated.
3. During the acute phases of leukemia, patients should receive only emergency periodontal care. Any source of potential infection must be eliminated to prevent systemic dissemination. Antibiotic therapy is frequently the treatment of choice, combined with nonsurgical or surgical debridement as indicated.
4. Oral ulcerations and mucositis are treated palliatively with agents such as viscous lidocaine. Systemic antibiotics may be indicated to prevent secondary infection.
5. Oral candidiasis is common in the leukemic patient and can be treated with nystatin suspensions (100,000 U/ml 4 times daily) or clotrimazole vaginal suppositories (10 mg 4 or 5 times daily).[58]
6. For patients with chronic leukemia and those in remission, scaling and root planing can be performed without complication, but periodontal surgery should be avoided if possible.
   - Platelet count and bleeding time should be measured on the day of the procedure. If either is low, postpone the appointment and refer the patient to a physician.

Agranulocytosis

Patients with agranulocytosis (cyclic neutropenia and granulocytopenia) have an increased susceptibility to infection. The total white blood cell count is reduced, and granular leukocytes (neutrophils, eosinophils, basophils) are reduced or disappear. These disorders are often marked by early, severe periodontal destruction.[115] When possible, periodontal treatment should be done during periods of disease remission. At such times, treatment should be as conservative as possible while reducing potential sources of systemic infection. After physician consultation, severely affected teeth should be extracted. Oral hygiene instruction should include use of chlorhexidine rinses twice daily. Scaling and root planing should be performed carefully under antibiotic protection.

For more information on renal, liver, and pulmonary diseases, please visit the companion Expert CONSULT website at www.expertconsult.com.

MEDICATIONS AND CANCER THERAPIES

Some medications prescribed to cure, manage, or prevent diseases have effects on periodontal tissues, wound healing, or the host immune response that require an understanding, appreciation, and in some cases, modification of treatment. Bisphosphonates, anticoagulation medications, antiplatelet medications, steroids, chemotherapy, and radiation therapy are briefly addressed here. Readers

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are encouraged to seek additional information and advice from other sources.

Bisphosphonates

Bisphosphonate medications are primarily used to treat cancer (IV administration) and osteoporosis (oral administration). They act by inhibiting osteoclastic activity, which leads to less bone resorption, less bone remodeling, and less bone turnover.[83a] The use of bisphosphonates in cancer treatment is aimed at preventing the often lethal imbalance of osteoclastic activity. In the treatment of osteoporosis, the goal is simply to harness osteoclastic activity to minimize or prevent bone loss. The major difference in the use of bisphosphonates for cancer versus osteoporosis is the potency and route of administration. Potency is influenced by the chemical properties and pharmacokinetics of these agents with bone. See Chapter 27 for a description of the chemical structure, activity, and role of bisphosphonates in the development of bisphosphonate-related osteonecrosis of the jaw (BRONJ).

Clinically, BRONJ presents as exposed alveolar bone occurring spontaneously or after a dental procedure (see Figures 27-29 and 27-30). Individuals treated with high potency, nitrogen-containing bisphosphonates, especially those administered via IV for cancer treatment (e.g., zoledronate), appear to be at greater risk for BRONJ than individuals taking oral bisphosphonates for prevention and treatment of osteoporosis. The incidence in patients treated for cancer has been reported to range from 2.5% to 5.4%.[114a] Estimating the incidence in patients taking oral bisphosphonates for osteoporosis is more difficult but appears to range from 0.007% to 0.04%.[112a] Even if this is an underestimation of the actual risk for individuals taking oral bisphosphonates, the incidence appears to be low. The risk for individuals treated with oral bisphosphonates for a period of less than 3 years appears to be minimal or zero.[52] Regular use of oral bisphosphonates for a period greater than 3 years suggests a risk profile that increases with time and length of use.[52]

As with many multifactorial diseases and conditions, it is likely that factors in addition to bisphosphonate therapy contribute to the individual risk of BRONJ. Potential risk factors thought to contribute to the development of BRONJ include systemic corticosteroid therapy, smoking, alcohol, poor oral hygiene, chemotherapy, radiotherapy, diabetes, and hematologic disease.[28] Reported factors or conditions leading to BRONJ include extractions, root canal treatment, periodontal infections, periodontal surgery, and dental implant surgery.[52a] Clearly, both periodontal disease and treatment (especially surgery) pose a risk for patients treated with bisphosphonates. The bacterial-induced inflammatory process of periodontitis that causes bone resorption can lead to bone necrosis. Likewise, periodontal treatment, especially surgery, may cause bone necrosis in the presence of bisphosphonates. Caution is warranted for any patient who has been or will be treated with bisphosphonates.

Health care providers need to evaluate patients carefully, communicate with medical health care providers, inform patients, and consider treatment options and risks carefully. A careful intraoral examination is prudent for all patients treated with bisphosphonate therapy (IV or oral) to determine whether bone exposures exist and to assess any local conditions that might predispose them to the development of BRONJ. A thorough medical history should be reviewed, evaluated, and recorded with details about any bisphosphonate treatment, including medication type, dose, route of administration, and duration. Comorbidities, such as previous and current medications, treatments, and existing disease or pathology, should be considered. Radiographs should be carefully evaluated for signs of bisphosphonate toxicity. Finally, Marx has suggested that a laboratory blood test for the serum C-terminal telopeptide fragment of type I collagen (CTX) can be used as a means of assessing an individual's risk of developing BRONJ.[52] Marx reports that lower CTX values are associated with greater risk (Table 37-10). It is important to recognize that these values are based on retrospective evaluation of patients with osteonecrosis of the jaws and that prospective studies to validate these findings have not been done. The CTX laboratory test is a measure of the specific C-terminal fragment of type I collagen cleaved by osteoclasts and serves as a good indicator of bone resorption activity. However, its use as a measure of risk for BRONJ is controversial and not confirmed by prospective studies.

TABLE 37-10 C-Terminal Telopeptide Laboratory Risk Assessment for Bisphosphonate Therapy

C-Terminal Telopeptide (CTX) Value	Risk for BRONJ
300–600 pg/ml (normal)	None
150–299 pg/ml	None or minimal
101–149 pg/ml	Moderate
≤100 pg/ml	High

Data from Marx RE: *Oral and intravenous bisphosphonate-induced osteonecrosis of the jaws,* Hanover Park, MD, 2007, Quintessence Publishing Co, Inc..

Optimal periodontal/oral health should be achieved and maintained for all patients. For individuals treated with IV bisphosphonates, invasive treatment, such as extractions, periodontal surgery, implant surgery, and bone augmentation procedures, should be avoided. Caution and careful consideration of risks must be considered before any treatment for individuals with a history of taking oral bisphosphonates for periods longer than 3 years. This is an area of current research that will undoubtedly continue to evolve as the pathophysiology is better understood. Providers are encouraged to consult other sources for updates on this important topic.

Anticoagulant/Antiplatelet Therapy

Many patients with a variety of conditions are placed on anticoagulant or antiplatelet medications to prevent thrombosis (blood clotting) or thromboembolism. Examples of patients at risk who may be on anticoagulant or antiplatelet therapy include those with heart value replacements, heart rhythm disorders, and congenital heart defects, as well as individuals with a history or risk of myocardial infarction, stroke, or deep vein thrombosis. These medications, although effective in reducing the risk of thrombosis, may increase the risk for bleeding complications, especially in patients undergoing surgical procedures.

As stated earlier, traditional management of patients on anticoagulant or antiplatelet therapy was to discontinue therapy about 3 to 5 (antiplatelet) or 7 to 10 (anticoagulant) days before planned surgical procedures. Recent evidence and new thoughts regarding the management of patients on anticoagulant or antiplatelet therapy suggest that treating them (e.g., periodontal surgery, extractions, etc) without altering their anticoagulant/antiplatelet mediations is safe and does not lead to intraoperative or postoperative bleeding complications. Furthermore, the increased risk of morbidity/mortality in those individuals discontinuing the anticoagulant or antiplatelet therapy may be significant.

Controlled clinical studies have demonstrated that intraoperative bleeding is not likely to be a problem with simple extractions or periodontal surgery if antiplatelet therapy (e.g., aspirin) is continued.[12,50] In these studies, there were no episodes of uncontrolled

bleeding, all bleeding was controlled with local measures, and there were no cases of postoperative bleeding problems. Conversely, the risk of stopping antiplatelet therapy may be serious. In a retrospective evaluation of 52 patients undergoing cataract surgery, 1 in 10 whose antiplatelet therapy was stopped or reduced suffered a stroke.[85]

Similarly, clinical studies of patients on anticoagulant therapy undergoing extractions and other oral surgical procedures have demonstrated minimal bleeding problems when therapy is continued.[17,20,113,114,120] In a review of the literature, Wahl et al[114] reported that only 12 patients (<1.3%, 950 total) receiving continuous anticoagulant therapy required more than local measures to control hemorrhage after minor oral surgical procedures (2400 total). Most of the 950 patients had anticoagulation levels that were well above currently recommended therapeutic levels. Only 3 patients (<0.31%) had anticoagulation levels within or below currently recommended therapeutic levels. In contrast, 5 of 526 (0.95%) patients who experienced 575 interruptions of continuous anticoagulant therapy, suffered serious embolic complications; four of these patients died. In a prospective study of 131 patients undergoing 511 extractions, patients whose oral anticoagulant therapy was reduced 72 hours before surgery to achieve an INR of 1.5 to 2.0 (target 1.8) were found to have postoperative bleeding warranting subsequent local intervention in 10 cases (15.1%).[84] There were only six cases (9.2%) in the group that continued their oral anticoagulant therapy (mean INR = 2.9).

These studies suggest that the risks of serious morbidity associated with discontinuing anticoagulant/antiplatelet therapy should be avoided and that the risk of bleeding while maintaining this therapy is minimal.

Corticosteroids

Approximately 5% of the adults in the US habitually take corticosteroids for the treatment of various conditions, potentially putting them at risk for secondary adrenal insufficiency.[48] Patients who habitually use corticosteroids have an increased likelihood of developing hypertension, osteoporosis, and peptic ulcer disease. Care should be taken to minimize the risk of adverse outcomes in these patients. BP should be monitored, and medications that may exacerbate peptic ulceration (e.g., acetylsalicylic acid [ASA], NSAIDs) should be avoided.

Stressful events, such as trauma, illness, surgery, emotional upset, or athletic events, normally increase circulating endogenous cortisol levels through stimulation of the hypothalamic-pituitary-adrenal (HPA) axis. Pain appears to increase the requirement for cortisol release.[76] There is concern that the normal release of cortisol in response to stressful events, such as a dental procedure, may be impaired in patients exposed to habitual corticosteroid use. Hence the concern for whether patients who are habitually taking corticosteroids require perioperative supplementation for dental procedures. Historically, recommendations were based on the medication type, amount, and duration of corticosteroid use. However, current thinking about the need for perioperative corticosteroid supplementation has been adjusted.

Studies investigating the stress response to minor general and oral surgical procedures conclude that significant increases in cortisol are generally not seen until 1 to 5 hours after surgery and appear to be associated more with postoperative pain and the loss of local anesthesia than with the preoperative and intraoperative stress of the procedure.[13,94,95] In fact, the administration of adequate analgesics in the postoperative period can diminish the release (requirement) of cortisol.[13]

Evidence indicates that a vast majority of individuals with adrenal insufficiency can receive routine dental treatment without the need for supplemental glucocorticosteroids.[18,48] Patients currently taking corticosteroids generally have enough exogenous and endogenous cortisol to handle routine dental procedures if their usual dose is taken within 2 hours of the planned procedure. Thus, for most patients, supplemental corticosteroid administration is not required when uncomplicated minor surgical procedures, including periodontal surgery, are performed with local anesthesia with or without sedation.[48] Topical corticosteroids generally have minimal HPA effect, and steroid supplementation is not required for these patients.

Individuals who may be at risk for adrenal crisis requiring supplementation include those who are undergoing lengthy, major surgical procedures, those expected to have significant blood loss and those who have extremely low adrenal function. Low adrenal function can be identified with an ACTH stimulation test. For these individuals, consultation with the physician and steroid supplementation is indicated.

Immunosuppression and Chemotherapy

Immunosuppressed patients have impaired host defenses as a result of an underlying immunodeficiency or drug administration (primarily related to organ transplantation or cancer chemotherapy).[57,91] Because chemotherapy is often cytotoxic to bone marrow, destruction of platelets and red and white blood cells results in thrombocytopenia, anemia, and leukopenia. Immunosuppressed individuals are at greatly increased risk for infection, and even minor periodontal infections can become life threatening if immunosuppression is severe.[75,78] Intraorally, bacterial, viral, and fungal infections may manifest. Patients receiving bone marrow transplantation require special attention because these patients receive extremely high-dose chemotherapy and are particularly susceptible to dissemination of oral infections.

Treatment in these patients should be directed toward the prevention of oral complications that could be life threatening. The greatest potential for infection occurs during periods of extreme immunosuppression; therefore treatment should be conservative and palliative. It is always preferable to evaluate the patient before initiation of chemotherapy.[57,91] Teeth with a poor prognosis should be extracted, with thorough debridement of remaining teeth to minimize the microbial load. The clinician must teach and emphasize the importance of good oral hygiene. Antimicrobial rinses, such as chlorhexidine, are recommended, especially for patients with chemotherapy-induced mucositis, to prevent secondary infection.

Chemotherapy is usually performed in cycles, with each cycle lasting several days, followed by intervening periods of myelosuppression and recovery. If periodontal therapy is needed during chemotherapy, it is best done the day *before* chemotherapy is given, when white blood cell counts are relatively high. Coordination with the oncologist is critical. Dental treatment should be done when white cell counts are above 2000/mm^3, with an absolute granulocyte count of 1000 to 1500/mm^3.[54]

Radiation Therapy

The use of radiotherapy, alone or in conjunction with surgical resection, is common in the treatment of head and neck tumors. The side effects of ionizing radiation include dramatic perioral changes of significant concern to dental health personnel.[41,58,92] The extent and severity of mucositis, dermatitis, xerostomia, dysphagia, gustatory alteration, radiation caries (Figure 37-3), vascular

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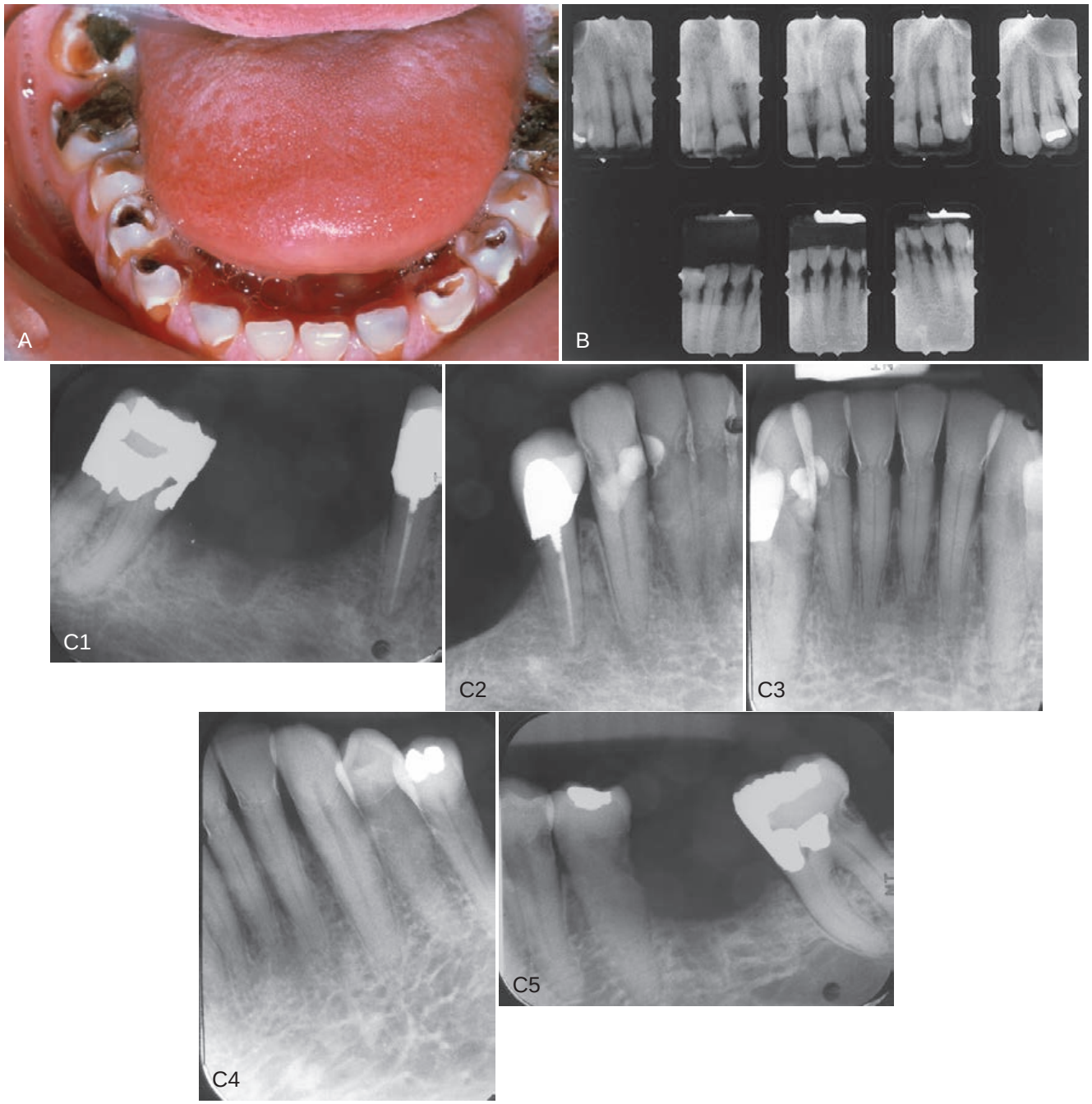

Figure 37-3 **A,** Clinical view of patient with radiation caries. Notice how the caries primarily affects the smooth tooth surfaces and the cusp tips. **B,** Radiographs of anterior teeth of 52-year-old male with postradiation caries. Patient received 6000-cGy radiation treatment to the posterior mandible and base of tongue for squamous cell carcinoma. Radiation caries developed within 1 year after radiation treatment, affecting the cervical areas and incisal edges of the anterior teeth. **C,** Radiographs of the mandibular teeth of 60-year-old male with postradiation caries. Patient received chemotherapy and 65-Gy intensity modulated radiation therapy (IMRT) to the lower head and neck for pharyngeal carcinoma. Radiation caries affecting the cervical areas of most remaining teeth. Notice the radiopaque layer on the outer surface of carious areas attributed to remineralization treatment with fluoride. *(***A** *and* **C** *courtesy Dr. Eric Sung, Hospital Dentistry, University of California Los Angeles.*)

changes, trismus, temporomandibular joint degeneration, and periodontal change depend on the type of radiation used, fields of irradiation, number of ports, types of tissues in the fields, and dosage.

Patients scheduled to receive head and neck radiation therapy require dental consultation *at the earliest possible time* to reduce the morbidity of the known perioral side effects.[89] *Preirradiation treatment* depends on the patient's prognosis, compliance, and residual dentition in addition to the fields, ports, dose, and immediacy of radiotherapy. The initial visit should include panoramic and intraoral radiographs, a clinical dental examination, a periodontal evaluation, and a physician consultation. The physician should be asked about the amount of radiation to be administered, extent and location of the lesion, nature of any surgical procedures already

performed or to be performed, number of radiation ports, exact fields to be irradiated, mode of radiation therapy, and patient's prognosis (i.e., likelihood of metastasis). Preirradiation treatment should commence immediately after the physician consultation. The first decision should involve possible extractions because radiation can cause side effects that interfere with healing.

For head and neck squamous cell carcinomas, the radiation dose is usually 5000 to 7000 cGy (centigray; 1 cGy = 1 rad) delivered in a fractionated method (150-200 cGy/day over a 6- to 7-week course).[16,58] This is considered "full-course" radiation treatment, and the degree of perioral side effects depends on which tissues are irradiated, that is, the radiation fields. If this dose is administered to the salivary gland tissues, xerostomia will ensue. The parotid is the most radiosensitive of the salivary glands; saliva may become extremely viscous or nonexistent, depending on the dose delivered to the particular gland. Xerostomia causes a decrease in the normal salivary cleansing mechanisms, buffering capacity of saliva, and pH of oral fluids.[58] Oral bacterial populations shift to a preponderance of cariogenic forms (e.g., *Streptococcus mutans, Actinomyces* spp., *Lactobacillus* spp.). Radiation-induced caries may progress rapidly and primarily affects smooth tooth surfaces (see Figure 37-3).

High-dose radiation therapy results in hypovascularity of irradiated tissues with a reduction in wound-healing capacity.[65,111,112] Most severe among the resulting oral complications is *osteoradionecrosis* (ORN). Decreased vascularity renders the bone less capable of resolving trauma or infection. Such events may cause severe destruction of bone. The risk of ORN continues for the remainder of the patient's life and does not decrease with time.[53]

Periodontal disease can be a precipitating factor in ORN.[21,34] Tooth extraction after radiation treatment involves a high risk for developing ORN, and surgical flap procedures are generally discouraged after radiation. For these reasons, it is important that the clinician address the patient's periodontal disease *before* radiation begins, whenever possible. Teeth that are nonrestorable or severely periodontally diseased should be extracted, ideally at least 2 weeks before radiation.[58] Extractions should be performed in a manner that allows primary closure. Mucoperiosteal flaps should be gently elevated; teeth should be extracted in segments; alveolectomy should be performed, allowing no rough bony spicules to remain; and primary closure should be provided without tension. It is unnecessary to extract teeth that can be retained with conservative restorative, endodontic, or periodontal therapy. However, prudence dictates extraction of questionable teeth because periodontal treatment after irradiation may be limited to nonsurgical forms of therapy. Flap surgery or extraction of teeth after radiation may lead to ORN. Management of ORN is often difficult and costly, involving progressively more aggressive treatment if bone does not respond to conservative therapy. Costly hyperbaric oxygen therapy is frequently required for complete resolution.

During radiation therapy, patients should receive weekly prophylaxis, oral hygiene instruction, and professionally applied fluoride treatments, unless mucositis prevents such treatment. Patients should be instructed to brush daily with a 0.4% stannous or 1.0% sodium fluoride gel. Custom gel trays allow optimum fluoride application.[111] All remaining teeth should receive thorough debridement (scaling and root planing).

Postirradiation follow-up consists of palliative treatment given as indicated. Viscous lidocaine may be prescribed for painful mucositis, and salivary substitutes may be given for xerostomia. Daily topical fluoride application and oral hygiene are the best means of preventing radiation caries over time. A long-term, 3-month recall interval is ideal.

PROSTHETIC JOINT REPLACEMENT

The main treatment consideration for patients with prosthetic joint replacements relates to the potential need for antibiotic prophylaxis before periodontal therapy. Currently, no scientific evidence indicates that prophylactic antibiotics prevent late prosthetic joint infections, which might occur from transient bacteremia induced by dental treatment.[26,54,93] Furthermore, although dental-induced bacteremia could theoretically cause prosthetic joint infection, scant reports demonstrate dental treatment as a source of joint infection, and none of these actually documents a cause-and-effect relationship.[23,82,108] Thus, in previous reports, the American Dental Association (ADA), American Academy of Orthopedic Surgeons (AAOS), American Academy of Oral Medicine (AAOM), and British Society for Antimicrobial Chemotherapy (BSAC) all agreed that routine antibiotic prophylaxis before dental treatment was not indicated for most patients with prosthetic joint replacements.[1,4,8,31,106] However, antibiotic prophylaxis was indicated for almost all patients within the first 2 years after joint replacement and for so-called high-risk patients, including those with previous prosthetic joint infections, immunosuppression, rheumatoid arthritis, systemic lupus erythematosus, type 1 diabetes, hemophilia, and malnourishment.[4] Importantly, many authors consider patients with severe periodontal disease or other potential dental infections to be "high risk," and antibiotic prophylaxis may be indicated for these patients before dental treatment.[64,106]

In February 2009, the AAOS released an "information statement" regarding antibiotic prophylaxis for bacteremia in patients with joint replacements.[10] Their earlier information statement, developed jointly with the ADA, was published in 1997[4] and revised in 2003.[8] It intended to reduce the risk to patients from unnecessary use of antibiotics. The 2009 information statement, which was not developed collaboratively with the ADA, will greatly increase the number of patients exposed to antibiotics because it states that all patients with prosthetic joint replacements should be given antibiotic prophylaxis prior to invasive procedures. Commentaries that challenge the statement have been published in the *Journal of the American Dental Association* (JADA)[38] and the *Journal of the Canadian Dental Association* (JCDA).[62]

Although the evidence demonstrates no need for antibiotic prophylaxis in most patients, many orthopedic surgeons still prefer

TABLE 37-11 Antibiotic Regimens for Prevention of Prosthetic Joint Infections*

Patient Characteristics	Drug Regimen
Patients not allergic to penicillins	Cephalexin, cephradine, or amoxicillin: 2 g orally 1 hour before dental procedure
Patients allergic to penicillins	Clindamycin: 600 mg orally 1 hour before dental procedure
Patients not allergic to penicillins, but unable to take oral medication	Cefazolin 1 g or ampicillin 2 g intramuscularly or intravenously 1 hour before dental procedure
Patients allergic to penicillins, and unable to take	Clindamycin 600 mg intravenously 1 hour before dental oral medications procedure (must be diluted and injected slowly)

*No second doses were recommended.

Data from American Dental Association and American Academy of Orthopedic Surgeons: *Am Dent Assoc J* 128:1004-1008, 1997.

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such treatment,[98] most likely because of the high morbidity and potential mortality associated with prosthetic joint infections. Consultation with the patient's orthopedic surgeon before periodontal treatment is in the patient's best interest and may help assess the risk for joint infection relative to current dental status and type of periodontal treatment planned. Because individuals with significant periodontal disease are considered "high risk," antibiotic prophylaxis before treatment is common in the periodontal practice (Table 37-11).[4]

Providers are encouraged to monitor the status of these important statements and implied guidelines from other resources.

For more information on periodontal therapy for the pregnant patient and periodontal therapy and infectious diseases, please visit the companion Expert CONSULT website at www.expertconsult.com.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 38

Periodontal Therapy in the Female Patient

Joan Otomo-Corgel

CHAPTER OUTLINE

PUBERTY
Management
MENSES
Periodontal Manifestations
Management
PREGNANCY
Periodontal Manifestations
Role of Pregnancy Hormones
Maternal Immunoresponse
Other Oral Manifestations of Pregnancy
Clinical Management
Treatment
ORAL CONTRACEPTIVES
Management
MENOPAUSE (online only)
Oral Changes
Clinical Management
CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some boxes may be out of numeric order in this printed chapter.

Throughout a woman's life cycle, hormonal influences affect therapeutic decision making in periodontics. Historically, therapies have been gender biased. However, the advent of new research has provided keener appreciation of the unique systemic influences on oral, periodontal, and implant tissues. Oral health care professionals have greater awareness of and better capabilities for dealing with hormonal influences associated with the reproductive process. Periodontal and oral tissue responses may be altered, creating diagnostic and therapeutic dilemmas. Therefore it is imperative that the clinician recognize, customize, and appropriately alter periodontal therapy according to the individual woman's needs based on the stage of her life cycle.

This chapter reviews phases of the female life cycle from puberty through menopause. Periodontal manifestations, systemic effects, and clinical management are presented.

PUBERTY

Puberty occurs between the average ages of 11 to 14 in most women. The production of sex hormones (estrogen and progesterone) increases, then remains relatively constant during the remainder of the reproductive phase. Also, the prevalence of gingivitis increases, without an increase in the amount of plaque. Gram-negative anaerobes, especially *Prevotella intermedia,* have been implicated in association with puberty gingivitis. Kornman and Loesche[56] postulated that this anaerobic organism may use ovarian hormone as a substitute for vitamin K growth factor. Levels of black-pigmented *Bacteroides,* especially *P. intermedia* (formerly known as *Bacteroides intermedius*), are thought to increase with increased levels of gonadotropic hormones in puberty. *Capnocytophaga* species also increase in incidence, as well as in proportion. These organisms have been implicated in the increased bleeding tendency observed during puberty.

Recent studies associated with pubertal gingivitis indicate proportionately elevated motile rods, spirochetes, and *P. intermedia.*[79] Statistically significant increases in gingival inflammation and in the proportions of *P. intermedia* and *P. nigrescens* have been seen in pubertal gingivitis.[83] A recent study of 11- to 17-year-old adolescents found higher levels of *Actinobacillus actinomycetemcomitans* and *Fusobacterium nucleatum,* which were associated with bleeding indices, probing depth, and attachment loss.[69]

During puberty, periodontal tissues may have an exaggerated response to local factors. A hyperplastic reaction of the gingiva may occur in areas where food debris, materia alba, plaque, and calculus are deposited. The inflamed tissues become erythematous, lobulated, and retractable. Bleeding may occur easily with mechanical debridement of the gingival tissues. Histologically, the appearance is consistent with inflammatory hyperplasia.

During the reproductive years, women tend to have a more vigorous immune response, including higher immunoglobulin concentrations, stronger primary and secondary responses, increased resistance to the induction of immunologic tolerance, and a greater

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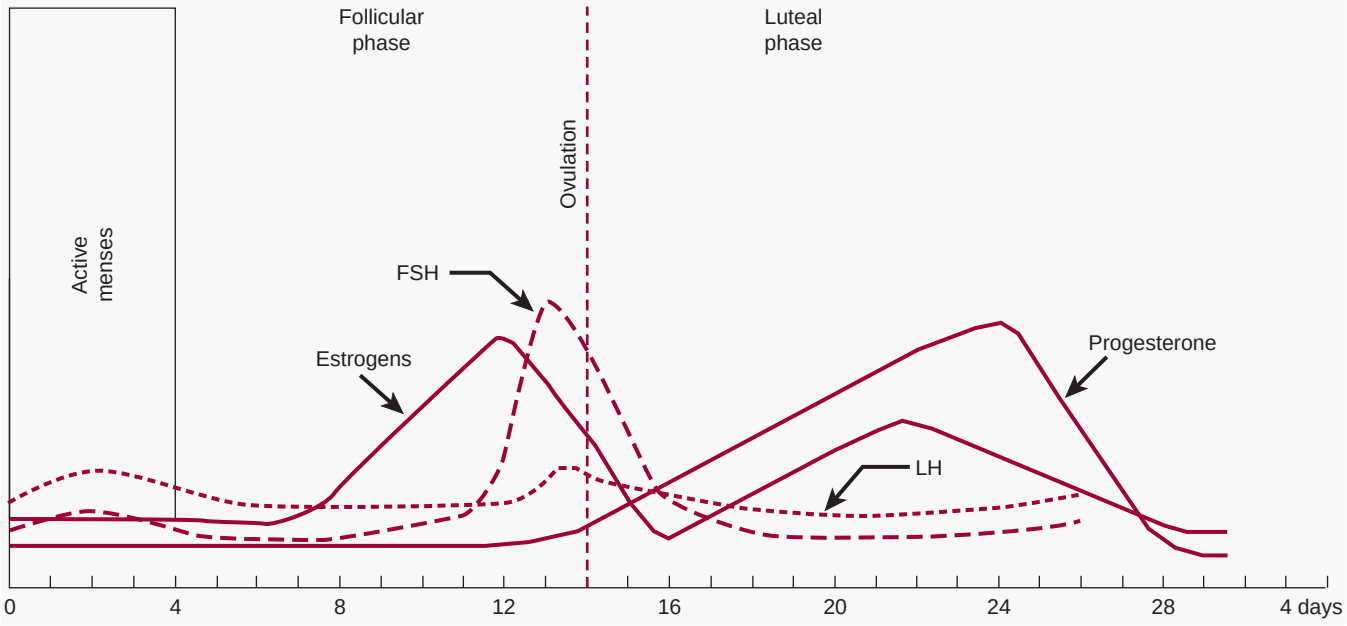

Figure 38-1 Female reproductive cycle. Note the peak levels of progesterone and estrogen compared with follicle-stimulating hormone *(FSH)* and luteinizing hormone *(LH)*.

ability to reject tumors and homografts.[114] Allergy, sensitivity, and asthma occur more often in young men, but after puberty, women become more susceptible than their male counterparts.

Management

During puberty, education of the parent or caregiver is part of successful periodontal therapy. Preventive care, including a vigorous program of oral hygiene, is also vital.[5] Milder gingivitis cases respond well to scaling and root planing, with frequent oral hygiene reinforcement. Severe cases of gingivitis may require microbial culturing, antimicrobial mouthwashes and local site delivery, or antibiotic therapy. Periodontal maintenance appointments may need to be more frequent when periodontal instability is noted.

The clinician should recognize the periodontal manifestations and/or intraoral lesions reflected by systemic diseases (i.e., diabetes).[24,91] Thorough review of the patient's medical history and medical referral should occur when deemed necessary. The clinician should be aware of the effects of chronic regurgitation of gastric contents on intraoral tissues; this age group also is susceptible to eating disorders, namely, bulimia and anorexia nervosa. *Perimolysis* (smooth erosion of enamel and dentin), typically on the lingual surfaces of maxillary anterior teeth, varies with the duration and frequency of the behavior.[16] Also, enlargement of the parotid glands (occasionally sublingual glands) has been estimated to occur in 10% to 50% of patients who "binge and purge."[73] Therefore a diminished salivary flow rate may also be present, which will increase oral mucous membrane sensitivity, gingival erythema, and caries susceptibility.

MENSES

Periodontal Manifestations

During the reproductive years, the ovarian cycle is controlled by the anterior pituitary gland. The gonadotropin follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are produced from the anterior pituitary gland. The secretion of gonadotropins also depends on the hypothalamus. Ongoing changes in the concentration of the gonadotropins and ovarian hormones occur during the monthly menstrual cycle (Figure 38-1). Under the influence of FSH and LH, estrogen and progesterone are steroid hormones produced by the ovaries during the menstrual cycle. During the reproductive cycle, the purpose of estrogen and progesterone is to prepare the uterus for implantation of the egg.

The monthly reproductive cycle has two phases. The first phase is referred to as the *follicular phase.* Levels of FSH are elevated, and estradiol (E2), the major form of estrogen, is synthesized by the developing follicle and peaks approximately 2 days before ovulation. The effect of estrogen stimulates the egg to move down the fallopian tubules (ovulation) and stimulates proliferation of the stroma cells, blood vessels, and glands of the endometrium.

The second phase is called the *luteal phase*. The developing corpus luteum synthesizes both estradiol and progesterone. Estrogen peaks at 0.2 ng/ml and progesterone at 10.0 ng/ml to complete the rebuilding of the endometrium for implantation of the fertilized egg. The corpus luteum involutes, ovarian hormone levels drop, and menstruation ensues. It has been postulated that ovarian hormones may increase inflammation in gingival tissues and exaggerate the response to local irritants. Gingival inflammation seems to be aggravated by an imbalance or increase in sex hormones. Numerous studies have demonstrated in vitro and in vivo that sex hormones affect and modify the actions of cells of the immune system. In addition, evidence suggests that the interaction between estrogen and cells of the immune system can have nonimmune regulatory effects.[7,20] Possible mechanisms have been suggested for the increase in hormonal gingival interaction in the menstrual cycle. Tumor necrosis factor alpha (TNF-α), which fluctuates during the menstrual cycle[13]; elevated prostaglandin E_2 (PGE_2) synthesis[75]; and angiogenetic factors, endothelial growth factors, and receptors may be modulated by progesterone and estrogen, contributing to increases in gingival inflammation during certain stages of the menstrual cycle.

Progesterone has been associated with increased permeability of the microvasculature, altering the rate and pattern of collagen

production in the gingiva,[70] increasing folate metabolism,[95,124] and altering the immune response. During menses, progesterone increases from the second week, peaks at approximately 10 days, and dramatically drops before menstruation. (Note that this is based on a 28-day cycle; individual cycles are variable.) Progesterone plays a role in stimulating the production of prostaglandins that mediate the body's response to inflammation. PGE_2 is one of the major secretory products of monocytes and is increased in inflamed gingiva. Miyagi et al[76] found that the chemotaxis of polymorphonuclear leukocytes (PMNs, neutrophils) was enhanced by progesterone but reduced by estradiol. Testosterone did not have a measurable effect on PMN chemotaxis. The researchers suggested that the altered PMN chemotaxis associated with gingival inflammation may be caused by the effects of sex hormones. Physiologic, experimental, and clinical data confirm differences in immune responses between the two sexes.[133]

Gingival tissues have been reported to be more edematous during menses and erythematous before the onset of menses in some women. A recent study reported higher gingival indices during ovulation and before menstruation despite reported increases in oral symptoms during menses.[71] In addition, an increase of gingival exudate has been observed during the menstrual period and is sometimes associated with a minor increase in tooth mobility.[39] The incidence of postextraction osteitis is also higher during initiation of menses. No significant hematologic laboratory findings accompany this, other than a slightly reduced platelet count and a slight increase in clotting time.

When the progesterone level is highest (during luteal phase of cycle), intraoral recurrent aphthous ulcers,[33] herpes labialis lesions, and candidal infections occur in some women as a cyclic pattern. Because the esophageal sphincter is relaxed by progesterone, women may be more susceptible to *gastroesophageal reflux disease* (GERD) during this time of the cycle as well. Symptoms of GERD include heartburn, regurgitation, and chest pain, and when reflux is severe, some patients develop unexplained coughing, hoarseness, sore throat, gingivitis, and asthma.[108]

Management

During the peak level of progesterone (about 7 to 10 days before menstruation), *premenstrual syndrome* (PMS) may also occur. There appears to be no significant difference in the estrogen and progesterone levels between women with PMS and those without PMS. However, women with PMS seem to have lower levels of certain neurotransmitters such as enkephalins, endorphins, γ-aminobutyric acid (GABA), and serotonin. Depression, irritability, mood swings, and difficulty with memory and concentration may be symptoms of neurotransmitter reduction. Patients are more sensitive and less tolerant of procedures, have a heightened gag reflex, and may have an exaggerated response to pain.

Increased gingival bleeding and tenderness associated with the menstrual cycle require closer periodontal monitoring. Periodontal maintenance should be titrated to the individual patient's need and if problematic, 3- to 4-month intervals should be recommended. An antimicrobial oral rinse before cyclic inflammation may be indicated. Emphasis should be placed on oral hygiene. For the patient with a history of excessive postoperative hemorrhage or menstrual flow, scheduling surgical visits after cyclic menstruation is prudent. Anemia is common, and appropriate consultation with a physician and recent laboratory tests, when indicated, should be maintained.

During PMS, many women exhibit physical symptoms that include fatigue, sweet and salty food cravings, abdominal bloating, swollen hands or feet, headaches, breast tenderness, nausea, and gastrointestinal upset. GERD may make it more uncomfortable for the patient to lay fully supine, especially after a meal, and the woman may have a more sensitive gag reflex. The clinician should be aware that nonsteroidal antiinflammatory drugs (NSAIDs), infection, and acidic foods exacerbate GERD. Patients taking over-the counter antacids, H_2-receptor antagonists (cimetidine, famotidine, nizatidine, ranitidine), prokinetic agents (cisapride, metoclopramide), and proton pump inhibitors (lansoprazole, omeprazole, pantoprazole, or rabeprazole)[109] may be GERD patients. These medications interact with some antibiotics and antifungals, and thus a review of their pharmacology is necessary. Fluoride rinses and trays, frequent periodontal debridement, and avoidance of mouthwashes with high alcohol content may reduce the associated gingival and caries sequelae.

PMS is often treated by antidepressants. *Selective serotonin reuptake inhibitors* (SSRIs) are generally the first-line choice because they have fewer side effects than other antidepressants, do not require blood monitoring, and are safe in overdoses. Women with PMS taking the SSRI fluoxetine were reported to have a 70% response rate. Fluoxetine was ranked the fifth most dispensed prescription (new and refills) in the United States (US) in 1998, but when the patent was lifted, its sales slowed. However, overall SSRIs ranked second in total dollar sales in the 2000s. (Sertraline was ranked twelfth and is the drug of choice for treatment of PMS.[141]) The clinician should be aware that patients taking fluoxetine have increased side effects with highly protein-bound drugs (e.g., aspirin), and the half-life of diazepam and other central nervous system (CNS) depressants is increased. Additional common SSRIs are fluvoxamine, paroxetine, and citalopram. Other antidepressants that may be prescribed are the selective serotonin and norepinephrine reuptake inhibitors (SNRIs), tricyclics, trazodone, mirtazapine, nefazodone, and maprotiline.

The PMS patient may be difficult to treat because of emotional and physiologic sensitivity. The dentist should treat the gingival and oral mucosal tissues gently. Gauze pads or cotton rolls should be moistened with a lubricant, chlorhexidine rinse, or water before placing them in the aphtha-prone patient. Careful retraction of the oral mucosa, cheeks, and lips is necessary in patients prone to aphthous or herpetic lesions. Because the hypoglycemic threshold is elevated, the clinician should advise the patient to have a light snack before her appointment. Of menstruating women, 70% have PMS symptoms, but only 5% meet the strict diagnostic criteria.

PREGNANCY

Periodontal Manifestations

The link between pregnancy and periodontal inflammation has been known for many years. In 1778, Vermeeren discussed "toothpains" in pregnancy. In 1818, Pitcarin[101] described gingival hyperplasia in pregnancy. Despite awareness regarding pregnancy and its effect on periodontal disease, only recently has evidence indicated an inverse relationship to systemic health. Current research implies periodontal disease may alter the systemic health of the patient and adversely affect the well-being of the fetus by elevating the risk for low-birth-weight, preterm infants.

In 1877, Pinard[100] recorded the first case of "pregnancy gingivitis." Only recently has periodontal research begun to focus on causative mechanisms. The occurrence of pregnancy gingivitis is extremely common, occurring in 30% to 100% of all pregnant women.[44,61,65,112] It is characterized by erythema, edema, hyperplasia, and increased bleeding. Histologically, the description is the same as for gingivitis. However, the etiologic factors are different

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despite clinical and histologic similarities. Cases range from mild-to-severe inflammation (Figure 38-2), which can progress to severe hyperplasia, pain, and bleeding (Figures 38-3 and 38-4).

Other growths that resemble pregnancy granulomas must be ruled out, such as central giant cell granulomas or underlying systemic diseases. Periodontal status before pregnancy may influence the progression or severity as the circulating hormones fluctuate. The anterior region of the mouth is affected more often, and interproximal sites tend to be most involved.[25] Increased tissue edema may lead to increased pocket depths and may be associated with transient tooth mobility.[102] Anterior site inflammation may be exacerbated by increased mouth breathing, primarily in the third trimester, from pregnancy rhinitis. The gingiva is the most common site involved (approximately 70% of all cases), followed by the tongue and lips, buccal mucosa, and palate.[112] An increase in attachment loss may represent active periodontal infection accelerated by pregnancy.[60]

Pyogenic granulomas ("pregnancy tumors," pregnancy epulis) occur in 0.2% to 9.6% of pregnancies. They are clinically and histologically indistinguishable from pyogenic granulomas occurring in nonpregnant women or in men. Pyogenic granulomas appear most often during the second or third month of pregnancy. Clinically, they bleed easily and become hyperplastic and nodular. When excised, the lesions usually do not leave a large defect. They may be sessile or pedunculated and ulcerated, ranging in color from purplish red to deep blue, depending on the vascularity of the lesion and degree of venous stasis.[10] The lesion classically occurs in an area of gingivitis and is associated with poor oral hygiene and calculus. Alveolar bone loss is usually not associated with pyogenic granulomas of pregnancy.

Figure 38-2 Moderate form of pregnancy gingivitis.

Figure 38-3 Pyogenic granuloma of pregnancy ("pregnancy tumor").

Role of Pregnancy Hormones

Subgingival Plaque Composition. Epidemiologic studies indicate a relationship between the level of home care and the severity of gingival inflammation. It appears that the association between signs of gingival inflammation and the amount of plaque is greater after parturition than during pregnancy. An alteration in the compositions of subgingival plaque occurs during pregnancy. As mentioned, Kornman and Loesche[57] found that during the second trimester, gingivitis and gingival bleeding increased, without an increase in plaque levels. Bacterial anaerobic/aerobic ratios increased, in addition to proportions of *Bacteroides melaninogenicus* and *P. intermedia* (2.2% to 10.1%). The authors suggested that estradiol or progesterone can substitute for menadione (vitamin K) as an essential growth factor for *P. intermedia* but not *Porphyromonas gingivalis* or *Eikenella corrodens.* There was also an increase in *P. gingivalis* during the 21st through 27th weeks of gestation, but this was not statistically significant. The relative increase in the numbers of *P. intermedia* may be a more sensitive indicator of an altered systemic hormonal situation than clinical parameters of gingivitis.[117] A recent study concluded subgingival levels of bacteria associated with periodontitis did not change. *P. gingivalis* and *Tannerella forsythia* counts were higher and associated with bleeding on probing at week 12.[2]

Periodontal Disease and Preterm, Low-Birth-Weight Infant. Although the greatest majority of studies support a causal relationship regarding the hypothesis that periodontitis during pregnancy poses an increased risk for adverse pregnancy outcomes, there are conflicting results. Variation in study results may be due to confounding factors, effect modifiers, population studied, timing of intervention or evaluation, and severity of periodontal disease caused by varying definitions.

Several systematic reviews indicate periodontal disease[113,128,135,137] may adversely effect pregnancy outcomes. Intervention trials have shown a positive effect with periodontal therapy and reduction of adverse pregnancy outcomes,[49,67,68,90,122] but three large multicenter

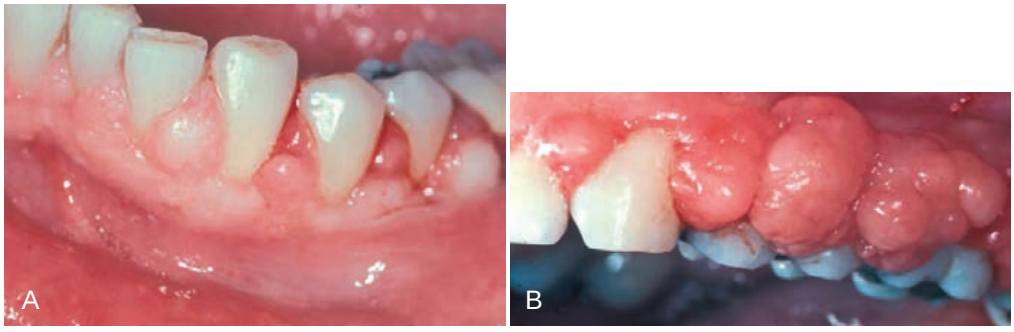

Figure 38-4 Severe pregnancy gingivitis with hyperplasia in patients with poorly controlled non–insulin-dependent diabetes mellitus. **A,** Moderate gingival enlargement. **B,** Severe gingival enlargement.

trials in the US did not support these results.[74,87,118] Studies do indicate that routine nonsurgical periodontal therapy after the first trimester is not associated with adverse pregnancy outcomes.[74]

Initially, Offenbacher et al[89] provided evidence that untreated periodontal disease in pregnant women may be a significant risk factor for preterm (<37 weeks' gestation), low-birth-weight (<2500 g) infants. The relationship between genitourinary tract infection and preterm, low-birth-weight (PLBW) infants is well documented in human and animal studies. Periodontal researchers, suspecting periodontal disease as another source of infection, found that otherwise low-risk mothers of PLBW infants had significantly more periodontal attachment loss than control mothers having normal-weight infants at birth.

The current opinion is that the correlation of periodontal disease to PLBW births may occur as a result of infection and is mediated indirectly, principally by the translocation of bacterial products such as endotoxin (lipopolysaccharide [LPS]) and the action of maternally produced inflammatory mediators.[36] Jared et al[47] noted in utero fetal exposure to oral pathogens increases the risk of neonatal intensive care unit admission and length of stay. Biologically active molecules, such as PGE_2 and TNF-α, which are normally involved in normal parturition, are raised to artificially high levels by the infection process, which may foster premature labor.[3] Gram-negative bacteria in periodontal diseases therefore may permit selective overgrowth or invasion of gram-negative bacteria within the genitourinary tract.

Gingival crevicular fluid (GCF) levels of PGE_2 were positively associated with intraamniotic PGE_2 levels ($p = 0.018$), suggesting that gram-negative periodontal infection may present a systemic challenge sufficient to initiate the onset of premature labor as a source of LPS, or through stimulation of secondary inflammatory mediators such as PGE_2 and interleukin-1 beta (IL-1β).[21] Offenbacher et al[88] suggested a dose-response relationship for increasing GCF PGE_2 as a marker of current periodontal disease activity and decreasing birth weight. Four organisms associated with mature plaque and progressing periodontitis—*T. forsythia, P. gingivalis, A. actinomycetemcomitans,* and *Treponema denticola*—were detected at higher levels in PLBW mothers compared with normal-birth-weight controls (see Chapter 28). Despite research supporting the association of periodontal disease and PLBW,[22,23] more studies with improved methodology are needed to assess the validity of the association.

Preeclampsia. A systematic review of preeclampsia and periodontitis indicated an increased risk during pregnancy. Preeclampsia is a life-threatening condition in late pregnancy characterized by high blood pressure and excess urine protein. High C-reactive protein levels also are associated with preeclampsia in this population.[111,115,127]

Maternal Immunoresponse

The maternal immune system is thought to be suppressed during pregnancy. This response may allow the fetus to survive as an allograft. Documentation of immunosuppressive factors in the sera of pregnant women can be noted by marked increase of monocytes (which in large numbers inhibit in vitro proliferative responses to mitogens, allogenic cells, and soluble antigen),[126] and pregnancy-specific β_1-glycoproteins contribute to diminished lymphocyte responsiveness to mitogens and antigens.[11] In addition, a decrease in the ratio of peripheral T helper cells to T suppressor cells (CD4/CD8) has been reported to occur throughout pregnancy.[104] These changes in maternal immunoresponsiveness suggest an increased susceptibility to developing gingival inflammation. In one study, the gingival index was higher, but percentages of T3, T4, and B cells appeared to decrease in peripheral blood and gingival tissues during pregnancy compared with a control group.[1] Other studies report decreased PMN (neutrophil) chemotaxis, depression of cell-mediated immunity, phagocytosis, and a decreased T-cell response with elevated ovarian hormone levels, especially progesterone.[103] Decreased in vitro responses of peripheral blood lymphocytes to several mitogens and to a preparation of *P. intermedia* have been reported.[12,66,86] Also, evidence suggests a decrease in the absolute numbers of CD4+ cells in peripheral blood during pregnancy compared with the number of these cells postpartum.[78,87] Lapp et al[59] suggest that high levels of progesterone during pregnancy affect the development of localized inflammation by downregulation of IL-6 production, rendering the gingiva less efficient at resisting the inflammatory challenges produced by the bacteria. A recent study indicated live preterm birth is associated with decreased levels of immunoglobulin G (IgG) antibody to periodontal pathogens in women with periodontitis when assessed during the second trimester but not associated with birth outcomes.[27]

Also, ovarian hormone stimulates the production of prostaglandins, in particular PGE_1 and PGE_2, which are potent mediators of the inflammatory response. With the prostaglandin acting as an immunosuppressant, gingival inflammation may increase when the mediator level is high.[30,92] Kinnby et al[54] found that high progesterone levels during pregnancy influenced plasminogen activator inhibitor type 2 (PAI-2) and disturbed the balance of the fibrinolytic system. Because PAI-2 serves as an important inhibitor of tissue proteolysis, this research implies that components of the fibrinolytic system may be involved in the development of pregnancy gingivitis.

During pregnancy, sex hormonal levels rise dramatically (Box 38-1). Progesterone reaches levels of 100 ng/ml, 10 times the peak luteal phase of menses. Estradiol in the plasma may reach 30 times higher levels than during the reproductive cycle. In early pregnancy and during the normal ovarian cycle, the corpus luteum is the major source of estrogen and progesterone. During pregnancy the placenta begins to produce estrogens and progesterone.

Estrogen may regulate cellular proliferation, differentiation, and keratinization, whereas progesterone influences the permeability of the microvasculature,[62,63] alters the rate and pattern of collagen production, and increases the metabolic breakdown of folate (necessary for tissue maintenance).[139] High concentration of sex hormones in gingival tissues, saliva, serum, and GCF also may exaggerate the response.

Regulation of most cellular processes by hormones occurs through the interaction of these products with intracellular receptors. The resulting effects depend on the concentration of unbound hormone diffused through the cell membrane. Vittek et al[130] have demonstrated specific estrogen and progesterone receptors in gingival tissues, providing direct biochemical evidence that this tissue may function as a target organ for sex hormones. Muramatsu and Takaesu[81] found increasing concentration of sex hormones in saliva from the first month and peaking in the ninth month of gestation, along with increasing percentages of *P. intermedia.* Probing depth, number of gingival sites with bleeding, and redness increased until 1 month postpartum. Also, evidence indicates sex hormone concentration in GCF, providing a growth media for periodontal pathogens.

Other Oral Manifestations of Pregnancy

As previously mentioned, perimolysis (acid erosion of teeth) may occur if "morning sickness" or esophageal reflux is severe and involves repeated vomiting of the gastric contents. Severe reflux

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BOX 38-1 Etiology of Gingival Responses to Elevated Estrogen and Progesterone during Pregnancy

Subgingival Plaque Composition

Increase in anaerobic/aerobic ratio
Higher concentrations of *Prevotella intermedia* (substitutes sex hormone for vitamin K–growth factor)
Higher concentrations of *Bacteroides melaninogenicus*
Higher concentrations of *Porphyromonas gingivalis*

Maternal Immunoresponse

Depression of cell-mediated immunity
Decreased neutrophil chemotaxis
Depression of antibody and T-cell responses
Decrease in ratio of peripheral T helper cells to T suppressor-cytotoxic cells (CD4/CD8 ratio)
Cytotoxicity directed against macrophages and B cells may result in diminished immunoresponsiveness.
Decrease in absolute numbers of CD3+, CD4+, and CD19+ cells in peripheral blood during pregnancy versus postpartum
Stimulation of prostaglandin production

Sex Hormone Concentration

Estrogen

Increases cellular proliferation in blood vessels (known in the endometrium).
Decreases keratinization, while increasing epithelial glycogen.
Specific receptors are found in gingival tissues.

Progesterone

Increases vascular dilation, and thus increases permeability (resulting in edema and accumulation of inflammatory cells).
Increases proliferation of newly formed capillaries in gingival tissues (increased bleeding tendency).
Alters rate and pattern of collagen production.
Increased metabolic breakdown of folate (folate deficiency can inhibit tissue repair).
Specific receptors are found in gingival tissues.
Decreases plasminogen activator inhibitor type 2, and thus increases tissue proteolysis.

Estrogen and Progesterone

Affect ground substance of connective tissue by increasing fluidity.
Concentrations increase in saliva and fluid with increased concentrations in serum.

may cause scarring of the esophageal sphincter, and the patient may become a more likely candidate for GERD later in life.

Xerostomia is a frequent complaint among pregnant women. One study found this persistent dryness in 44% of pregnant participants.[28]

A rare finding in pregnancy is *ptyalism*, or *sialorrhea*. This excessive secretion of saliva usually begins at 2 to 3 weeks of gestation and may abate at the end of the first trimester. The etiology of ptyalism has not been identified, but it may result from the inability of nauseated gravid women to swallow normal amounts of saliva, rather than from a true increase in saliva production.[19]

Because pregnancy places the woman in an immunocompromised state, the clinician must be aware of the patient's total health. Gestational diabetes, leukemia, and other medical conditions may appear during pregnancy.

Clinical Management

A thorough medical history is an imperative component of the periodontal examination, especially in the pregnant patient. Because of immunologic alterations, increased blood volume (ruling out mitral valve prolapse and heart murmurs), and fetal interactions, the clinician must diligently and consistently monitor the patient's medical and periodontal stability. Medical history dialog should include pregnancy complications, previous miscarriages, and recent history of cramping, spotting, or pernicious vomiting. The patient's obstetrician should be contacted to discuss her medical status, periodontal or dental needs, and the proposed treatment plan.

Establishing a healthy oral environment and maintaining optimal oral hygiene levels are primary objectives in the pregnant patient. A preventive periodontal program consisting of nutritional counseling and rigorous plaque control measures in the dental office and at home should be reinforced.

Plaque Control. The increased tendency for gingival inflammation during pregnancy should be clearly explained to the patient so that acceptable oral hygiene techniques may be taught, reinforced, and monitored throughout pregnancy. Scaling, polishing, and root planing may be performed whenever necessary during pregnancy. Some practitioners avoid the use of high-alcohol-content antimicrobial rinses in pregnant women and prefer to use non–alcohol-based oral rinses.

Prenatal Fluoride. The prescribing of prenatal fluoride supplements has been an area of controversy. Although two studies have claimed beneficial results,[37,38]others suggest that the clinical efficacy of prenatal fluoride supplements is uncertain and that the mechanism by which prenatal fluorides might impart cariostasis is unclear.[98] The American Dental Association (ADA) does not recommend the use of prenatal fluoride because its efficacy has not been demonstrated. The American Academy of Pediatric Dentistry supports this position as well. The American Academy of Pediatrics has no stated position on prescribing prenatal fluorides.

Treatment

Elective Dental Treatment. Other than good plaque control, it is prudent to avoid elective dental care if possible during the first trimester and the last half of the third trimester. The first trimester is the period of organogenesis, when the fetus is highly susceptible to environmental influences. In the last half of the third trimester, a hazard of premature delivery exists because the uterus is very sensitive to external stimuli. Prolonged chair time may need to be avoided because the woman is most uncomfortable at this time. Further, *supine hypotensive syndrome* may occur. In a semireclining or supine position, the great vessels, particularly the inferior vena cava, are compressed by the gravid uterus. By interfering with venous return, this compression will cause maternal hypotension, decreased cardiac output, and eventual loss of consciousness. Supine hypotensive syndrome can usually be reversed by turning the patient on her left side, thereby removing pressure on the vena cava and allowing blood to return from the lower extremities and pelvic area. A preventive 6-inch soft wedge (rolled towel) should be placed on the patient's right side when she is reclined for clinical treatment.

Early in the second trimester is the safest period for providing routine dental care. The emphasis at this time is on controlling active disease and eliminating potential problems that could arise in late pregnancy. Major elective oral or periodontal surgery may be postponed until after delivery. "Pregnancy tumors" that are painful, interfere with mastication, or continue to bleed or

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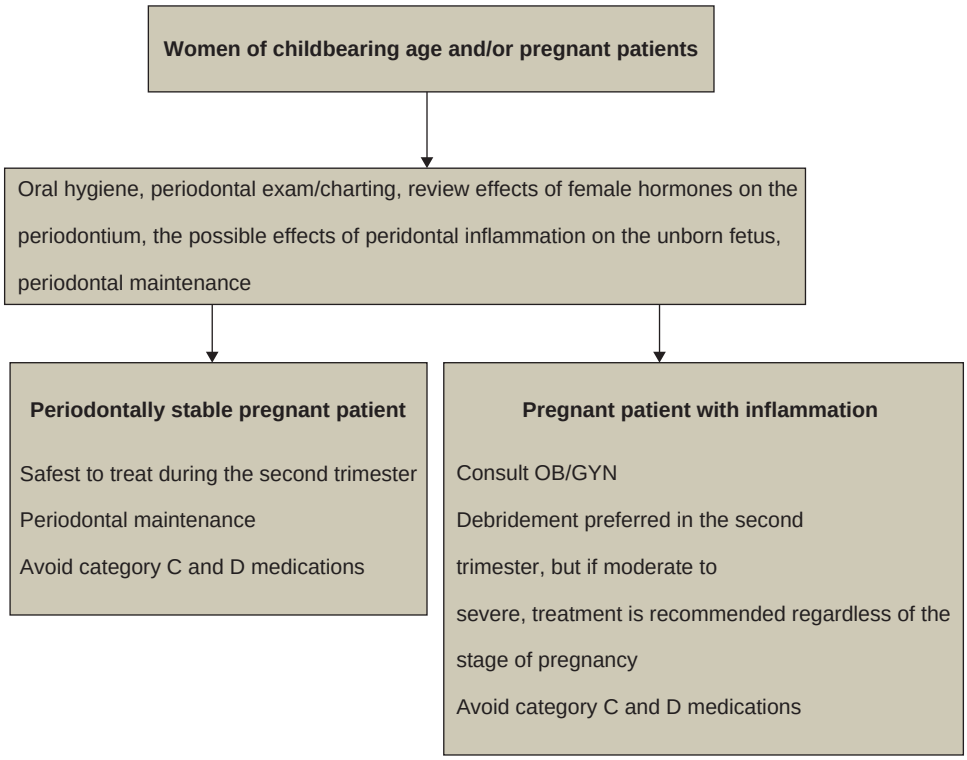

Figure 38-5 Treatment algorithm for the pregnant patient.

suppurate after mechanical debridement may require excision and biopsy before delivery.

The American Academy of Periodontology (www.perio.org) has developed a position statement regarding the need for providing proper periodontal therapy for pregnant patients (Figure 38-5). Because of research indicating possible impact on the fetus, the presence of acute infection, abscesses, or other potential disseminating sources of sepsis may warrant prompt intervention irrespective of the stage of pregnancy.[4]

Dental Radiographs. The safety of dental radiography during pregnancy has been well established, provided features such as high-speed film, filtration, collimation, and lead aprons are used. However, it is most desirable not to have *any* irradiation during pregnancy, especially during the first trimester, because the developing fetus is particularly susceptible to radiation damage.[64] When radiographs are needed for diagnosis, the most important aid for the patient is the protective lead apron. Studies have shown that when an apron is used during contemporary dental radiography, gonadal and fetal radiation is virtually immeasurable.[9]

Even with the obvious safety of dental radiography, x-ray films should be taken selectively during pregnancy and only when necessary and appropriate to aid in diagnosis and treatment. In most cases, only bite-wing, panoramic, or selected periapical films are indicated.

Medications. Drug therapy in the pregnant patient is controversial because drugs can affect the fetus by diffusion across the placenta. Prescriptions should be only the duration absolutely essential for the pregnant patient's well-being and only after careful consideration of potential side effects. The classification system established by the US Food and Drug Administration (FDA) in 1979 to rate fetal risk levels associated with many prescription drugs provides safety guidelines (Box 38-2). The prudent

BOX 38-2 FDA Drug Classification System Based on Potential for Causing Birth Defects

- A Controlled studies in women fail to demonstrate a risk to the fetus in the first trimester (there is no evidence of a risk in later trimesters), and the possibility of fetal harm appears remote.
- B Either animal reproduction studies have not demonstrated a fetal risk but there are no controlled studies in pregnant women, or animal reproduction studies have shown an adverse effect (other than a decrease in fertility) that was not confirmed in controlled studies in women in the first trimester (and there is no evidence of a risk in later trimesters).
- C Either studies in animals have revealed adverse effects on the fetus (teratogenic, embryocidal, or other) and there are no controlled studies in women, or studies in women and animals are not available. Drugs should be given only if the potential benefit justifies the potential risk to the fetus.
- D There is positive evidence of human fetal risk, but the benefits from use in pregnant women may be acceptable despite the risk (e.g., if the drug is needed in a life-threatening situation or for a serious disease for which safer drugs cannot be used or are ineffective).
- X Studies in animals or humans have demonstrated fetal abnormalities, or there is evidence of fetal risk based on human experience, or both, and the risk of using the drug in pregnant women clearly outweighs any possible benefit. The drug is contraindicated in women who are or who may become pregnant.

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practitioner should consult references, such as Briggs et al's *Drugs in Pregnancy and Lactation*[15] and Olin's *Drug Facts and Comparisons*,[93] for information on the FDA pregnancy risk factor associated with prescription drugs. Ideally, no drug should be administered during pregnancy, especially the first trimester.[64] However, it is sometimes impossible to adhere to this rule. Fortunately, most common drugs in dental practice can be given during pregnancy with relative safety, although there are a few important exceptions. Tables 38-1, 38-2, and 38-3 present general guidelines for anesthetic and analgesic, antibiotic, and sedative-hypnotic drugs, respectively.[105,125]

In particular, antibiotics are often needed in periodontal therapy. The effect of a particular medication on the fetus depends on the type of antimicrobial, dosage, trimester, and duration of the course of therapy.[94] Research regarding subgingival irrigation and local site delivery in relation to the developing fetus is inadequate at this date.

Breastfeeding. Usually, there is a risk that the drug can enter breast milk and be transferred to the nursing infant, in whom exposure could have adverse effects (Tables 38-4 and 38-5). Unfortunately, there is little conclusive information about drug dosage and effects through breast milk; however, retrospective clinical studies and empiric observations coupled with known pharmacologic pathways allow recommendations to be made.[64] The amount of drug excreted in breast milk is usually not more than 1% to 2% of the maternal dose; therefore it is highly unlikely that most drugs have any pharmacologic significance for the infant.[136]

The mother should take prescribed drugs just after breastfeeding and then avoid nursing for 4 hours or more, if possible,[64,119] to decrease the drug concentration in breast milk.

ORAL CONTRACEPTIVES

Women may have responses to oral contraceptives (OCs) similar to those seen in pregnant patients. Mullally et al found that current users of OCs had poorer periodontal health.[80] An exaggerated response to local irritants occurs in gingival tissues. Inflammation ranges from mild edema and erythema to severe inflammation with hemorrhagic or hyperplasic gingival tissues. It has been reported

TABLE 38-1 Local Anesthetic and Analgesic Administration during Pregnancy

Drug	FDA Category (Prescription Drug)	During Pregnancy
Local Anesthetics*		
Lidocaine	B	Yes
Mepivacaine	C	Use with caution; consult physician.
Prilocaine	B	Yes
Bupivacaine	C	Use with caution; consult physician.
Etidocaine	B	Yes
Procaine	C	Use with caution; consult physician.
Articaine	B	Yes; no blocks
Analgesics		
Aspirin	C/D, third trimester	Caution; avoid in third trimester.
Acetaminophen	B	Yes
Ibuprofen	B/D, third trimester	Caution; avoid in third trimester.
Codeine†	C	Use with caution; consult physician.
Hydrocodone†	B	Use with caution; consult physician.
Oxycodone†	B	Use with caution; consult physician.
Propoxyphene	C	Use with caution; consult physician.

*Can use vasoconstrictors if necessary.
†Avoid prolonged use.
FDA, US Food and Drug Administration.

TABLE 38-2 Antibiotic Administration during Pregnancy

Drug(s)	FDA Category (Prescription Drug)	During Pregnancy	Risks
Penicillins	B	Yes	Diarrhea
Erythromycin	B	Yes; avoid estolate form	Intrahepatic jaundice in mother
Clindamycin	B	Yes, with caution	Drug concentrated in fetal bone, spleen, lung, and liver
Cephalosporins	B	Yes	Limited information
Tetracycline	D	Avoid	Depression of bone growth, enamel hypoplasia, gray-brown tooth discoloration
Ciprofloxacin	C	Avoid	Possible developing cartilage erosion
Metronidazole	B	Avoid; controversial	Theoretic carcinogenic data in animals
Gentamicin	C	Caution; consult physician	Limited information Ototoxicity
Vancomycin	C	Caution; consult physician	Limited information
Clarithromycin	D	Avoid; use only if potential benefit justifies risk to fetus	Limited information Adverse effects on pregnancy, outcome, and embryo/fetal development in animals

FDA, US Food and Drug Administration.

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TABLE 38-3 Sedative-Hypnotic Drug Administration during Pregnancy

Drug(s)	FDA Category	During Pregnancy
Benzodiazepines	D	Avoid
Barbiturates	D	Avoid
Nitrous oxide	Not assigned	Avoid in first trimester; otherwise use with caution; consult physician.

FDA, US Food and Drug Administration.

TABLE 38-4 Local Anesthetic and Analgesic Administration during Breastfeeding

Drug	During Breastfeeding
Local Anesthetics	
Lidocaine	Yes
Mepivacaine	Yes
Prilocaine	Yes
Bupivacaine	Yes
Etidocaine	Yes
Procaine	Yes
Analgesics	
Aspirin	Avoid
Acetaminophen	Yes
Ibuprofen	Yes
Codeine	Yes
Hydrocodone	No data
Oxycodone	Yes
Propoxyphene	Yes

TABLE 38-5 Antibiotic and Sedative-Hypnotic Administration during Breastfeeding

Drug(s)	During Breastfeeding
Antibiotics*	
Penicillins	Yes
Erythromycin	Yes
Clindamycin	Yes, with caution
Cephalosporins	Yes
Tetracycline	Avoid
Ciprofloxacin	Avoid
Metronidazole	Avoid
Gentamicin	Avoid
Vancomycin	Avoid
Sedative-Hypnotics	
Benzodiazepines	Avoid
Barbiturates	Avoid
Nitrous oxide	Yes

*Antibiotics have the risk of diarrhea and sensitization in the mother and infant.

that more exudate is present in inflamed gingival tissues of OC users than in pregnant women.[116,140] Investigators have suggested several mechanisms for the heightened response in gingival tissues. Kalkwarf[53] reported that the response may be caused by an altered microvasculature, increased gingival permeability, and increasing synthesis of prostaglandin. PGE, a potent mediator of inflammation,[31] appears to rise significantly with increasing levels of sex hormones. Jensen et al[51] found dramatic microbial changes in pregnant and OC groups compared with a nonpregnant group. A sixteenfold increase in *Bacteroides* species was noted in the OC group versus the nonpregnant group, despite no statistically significant clinical differences in gingival index or GCF flow. The authors stated that the increased female sex hormones substituting for the naphthoquinone requirement of certain *Bacteroides* species were most likely responsible for this increase.

The OC-associated gingival inflammation may become chronic (versus the acute inflammation of pregnancy) because of the extended periods that women are exposed to elevated levels of estrogen and progesterone.[55,97] Some have reported that the inflammation increases with prolonged use of OCs. Kalkwarf[53] did not find that duration of use made a significant difference; however, the brand used resulted in different responses. Further studies need to be performed in relation to dosage, duration, and type of OC used in association with the periodontium. The concentration of female sex hormones in current OCs is significantly less than that of the 1970s, with the same level of contraceptive efficacy.

Salivary composition changed notably in patients taking OCs in studies from the 1970s. A decreased concentration of protein, sialic acid, hexosamine fucose, hydrogen ions, and total electrolytes has been reported. Salivary flow rates were increased in one report[72] and decreased in 30% of subjects in another report.[29]

The dental literature reports that women taking OCs experience a twofold to threefold increase in the incidence of localized osteitis after extraction of mandibular third molars.[120] The higher incidence of osteitis is these patients may be attributed to the effects of OCs (estrogens) on clotting factors. However, a number of studies refutes these findings.[18] Evidence thus far is inconclusive on osteitis after third molar extraction and OC use. Also, a spotty melanotic pigmentation of the skin may occur with OC use. This suggests a relationship between the use of OCs and the occurrence of gingival melanosis,[45] especially in fair-skinned individuals.

Management

Medical histories should include OCs under medications, and an oral dialog should include questions regarding OCs in women of childbearing age. The patient should be informed of the oral and periodontal side effects of OCs and the need for meticulous home care and compliance with periodontal maintenance. Treatment of gingival inflammation exaggerated by OCs should include establishing an oral hygiene program and eliminating local predisposing factors. Periodontal surgery may be indicated if resolution after initial therapy (scaling and root planing) is inadequate. It may be advisable to perform extraction of teeth (especially of third molars) on nonestrogenic days (days 23 to 28) of the OC cycle to reduce the risk of a postoperative localized osteitis[34]; however, evidence of this association is inconclusive and warrants further investigation.

Although the results from animal studies have demonstrated antibiotic interference adversely affecting contraceptive sex hormone levels, several human studies have failed to support such an interaction.[8,35,82,85] This issue is controversial, and antibiotics possibly could render OCs ineffective in preventing pregnancies. In 1991, an ADA report stated that all women of childbearing age should be informed of possible reduced efficacy of steroid OCs

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during antibiotic therapy and advised women to use additional forms of contraception during short-term antibiotic therapy.[6] During long-term antibiotic therapy, they should consult their physician about using high-dose OC preparations. Although only research regarding oral manifestations attributed to OCs has been reported in the literature, the same effects presumably could occur with the use of contraceptive implants. Similarly, the remote possibility of reduced efficacy of the contraceptive implant with concurrent antibiotic administration also exists, and women can adhere to the same precautions as with OC use.

For more information on menopause, please visit the companion Expert CONSULT website at www.expertconsult.com.

CONCLUSION

Clinical periodontal therapy includes an understanding of the clinician's role in the total health and well-being of female patients. Dentists do not treat localized infections without affecting other systems (and the fetus or the breastfed infant). Therefore female patients may present with periodontal and systemic considerations that alter conventional therapy.

The cyclic nature of the female sex hormones often is reflected in the gingival tissues as initial signs and symptoms. Medical histories and dialogs should include thoughtful investigation of the individual patient's problems and needs. Questioning should reflect hormonal stability and medications associated with regulation. Patients should be educated regarding the profound effects the sex hormones may play on periodontal and oral tissues, as well as the consistent need for home and office removal of local irritants.

Research regarding female issues and medical/periodontal therapy is in process. In the near future, information regarding specific management and etiology of sex hormone–mediated infections will enhance our ability to provide quality care to our patients.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

Variations in the levels of circulating female hormones have a significant effect on periodontal health. Pregnancy can change inflammatory reactions to dental plaque in the most dramatic fashion with accentuation of vascular proliferation leading to increased inflammation and a hyperplastic gingival response. Puberty can have a similar but usually smaller effect, and changes during menstrual cycles and the use of hormone-based birth control tablets give the smallest changes in gingival health. There is also an increase in the risk of low-birth-weight preterm infants in patients with active periodontal inflammation so all pregnant women should be evaluated periodontally, and appropriate phase I therapy should be performed.

Dentists should consult the Food and Drug Administration (FDA) classification of medications that can or cannot be used in pregnancy and during breastfeeding before beginning periodontal treatment. Consultation with the patient's physician should be part of the management of pregnant and lactating patients.

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CHAPTER 39

Periodontal Treatment for Older Adults

Sue S. Spackman and Janet G. Bauer

CHAPTER OUTLINE

THE AGING PERIODONTIUM
- Intrinsic Changes
- Stochastic Changes
- Physiologic Changes
- Functional Changes
- Clinical Changes

DEMOGRAPHICS (online only)
- Population Distribution
- Health Status
- Functional Status
- Nutritional Status
- Psychosocial Factors
- Dentate Status
- Periodontal Status
- Caries Status
- Dental Visits
- Xerostomia
- Candidiasis

DENTAL AND MEDICAL ASSESSMENTS (online only)
- Review of Dental History
- Review of Medical History
- Review of Medication Use
- Extraoral and Intraoral Examinations
- Assessment of Risk
- Quality of Life

PERIODONTAL DISEASES IN OLDER ADULTS
- Etiology
- Relationship to Systemic Disease

PERIODONTAL TREATMENT PLANNING

PREVENTION OF PERIODONTAL DISEASE AND MAINTENANCE OF PERIODONTAL HEALTH IN OLDER ADULTS
- Chemotherapeutic Agents
- Risk Reduction

CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures, boxes, and tables may be out of numeric order in this printed chapter.

Older adults are expected to compose a larger proportion of the population than in the past. Population growth among long-lived older adults contributes to this increase worldwide. For dentistry, this means that older adults are retaining more of their natural dentition. Currently, almost 70% of older adults in the United States (US) have natural teeth.[12,27] However, retention of teeth may result in more teeth at risk for periodontal disease, and thus the prevalence of periodontal disease may be associated with aging. This association was addressed by Beck[20] at the 1996 World Workshop on Periodontics: "It may be that risk factors do change as people age or at least the relative importance of risk factors change."

This chapter focuses on the interrelationship between aging and oral health, with an emphasis on periodontal health.

THE AGING PERIODONTIUM

Normal aging of the periodontium is a result of cellular aging. In general, cellular aging is the basis for the intrinsic changes seen in oral tissues over time. The aging process does not affect every tissue in the same way. For example, muscle tissue and nerve tissue undergo minimal renewal, whereas epithelial tissue, which is one of the primary components of the periodontium, always renews itself.

Intrinsic Changes

In epithelium, a progenitor population of cells (stem cells), situated in the basal layer, provides new cells. These cells of the basal layer are the least differentiated cells of the oral epithelium. A small

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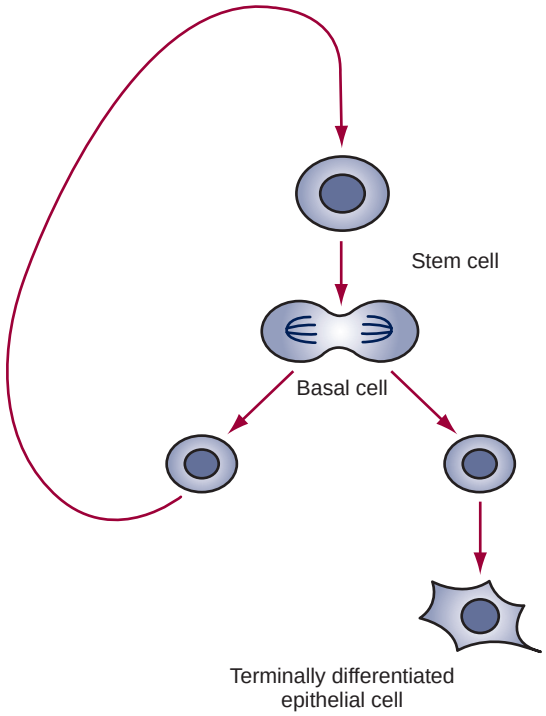

Figure 39-1 Cell renewal cycle in which the basal cell produces the epithelial cell and returns to the progenitor cell population.

subpopulation of these cells produces basal cells and retains proliferative potential of the tissue. A larger subpopulation of these cells (amplifying cells) produces cells available for subsequent maturation. This maturing population of cells continually undergoes a process of differentiation or maturation.

By definition, this differentiated cell, or epithelial cell, can no longer divide. On the other hand, the basal cell remains as part of the progenitor population of cells ready to return to the mitotic cycle and again produce both types of cells. Thus there is a constant source of renewal (Figure 39-1). In the aging process, cell renewal takes place at a slower rate and with fewer cells, so the effect is to slow down the regenerative processes. As the progenitor cells wear out and die, there are fewer and fewer of these cells to renew the dead ones. This effect is characteristic of the age-related and biologic changes that occur with aging.

By the action of gerontogene(s) or replicative senescence (Hayflick's limit and telomere shortening), the number of progenitor cells decreases. Hayflick, an American microbiologist, observed that fetal cells (i.e., fibroblasts) displayed a consistently greater growth potential (approximately 50 cumulative population doublings) than those derived from adult tissues (20 to 30 cumulative population doublings). As a result, the decreased cellular component has a concomitant effect to decrease cellular reserves and protein synthesis. This affects the oral epithelium in that the tissue becomes thin, with reduced keratinization.

Stochastic Changes

Stochastic changes occurring within cells also affect tissue (e.g., glycosylation and cross-linking produce morphologic and physiologic changes). Structures become stiffer, with a loss of elasticity and increased mineralization (fossilization). With a loss of regenerative power, structures become less soluble and more thermally stable. Somatic mutations lead to decreased protein synthesis and structurally altered proteins. Free radicals contribute to the accumulation of waste in the cell.

All these changes produce a decline in the physiologic processes of tissue. Most changes are primarily a result of aging, although some are secondary to physiologic deterioration. For example, loss of elasticity and increased resistance of the tissue may lead to decreased permeability, decreased nutrient flow, and the accumulation of wastes in the cell. Thus vascular peripheral resistance (decreased blood supply) may secondarily decrease cellular function.

Physiologic Changes

In the periodontal ligament, a decrease in the number of collagen fibers leads to a reduction or loss in tissue elasticity. A decrease in vascularity results in decreased production of mucopolysaccharides.

All these types of changes are seen in the alveolar bone. With aging, the alveolar bone shows a decrease in bone density and an increase in bone resorption and a decrease in vascularity also occurs. In contrast, however, cementum shows cemental thickness.

Functional Changes

With aging, the cells of the oral epithelium and periodontal ligament have reduced mitotic activity, and all cells experience a reduction in metabolic rate. These changes also affect the immune system and affect healing in the periodontium. There is a reduction in healing capacity and rate. Inflammation, when present, develops more rapidly and more severely. Individuals are highly susceptible to viral and fungal infections because of abnormalities in T-cell function.

Clinical Changes

Compensatory changes occur as a result of aging or disease. These changes affect the tooth or periodontium that presents the clinical condition. Gingival recession and reductions in bone height are common conditions. Attrition is a compensatory change that acts as a stabilizer between loss of bony support and excessive leveraging from occlusal forces imposed on the teeth.

In addition, a reduction in "overjet" of the teeth is seen, manifesting as an increase in the edge-to-edge contact of the anterior teeth. Typically, this is related to the approximal wear of the posterior teeth. An increase is seen in the food table area, with loss of "sluiceways," and in mesial migration.

Functional changes are associated with reduced efficiency of mastication. Although effectiveness of mastication may remain, efficiency is reduced because of missing teeth, loose teeth, poorly fitting prostheses, or noncompliance of the patient, who may refuse to wear prosthetic appliances.

For more information on demographics and dental and medical assessment, please visit the companion Expert CONSULT website at www.expertconsult.com.

PERIODONTAL DISEASES IN OLDER ADULTS

Etiology

Periodontal disease in older adults is usually referred to as *chronic periodontitis*.[37,60] Because periodontitis is a chronic disease, much of the ravages of the disease detected in older adults results from an accumulation of the disease over time. Research has shown that the advanced stages of periodontitis are less prevalent than the moderate stages in the older adult population.[24] One theory is that many

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sites of advanced periodontal disease have resulted in tooth loss earlier in life, suggesting that older age is not a risk factor for periodontal disease.[37,60]

Evidence is limited on whether the risk factors for periodontal disease differ with age.[20] General health status, immune status, diabetes, nutrition, smoking, genetics, medications, mental health status, salivary flow, functional deficits, and finances may modify the relationship between periodontal disease and age.[20,76]

Some frequently prescribed medications for older adults can alter the gingival tissues. *Steroid-induced gingivitis* has been associated with postmenopausal women receiving steroid therapy. *Gingival overgrowth* can be induced by such medications as cyclosporines, calcium channel blockers, and anticonvulsants (e.g., nifedipine, phenytoin) in the presence of poor oral hygiene. This gingival overgrowth further decreases a person's ability to maintain good oral hygiene.[37]

Relationship to Systemic Disease

In Padilha et al,[52] using data from the Baltimore Longitudinal Study of Aging, the conclusion is reached that "the number of teeth is a significant risk indicator for mortality … and that improving oral health and preventing tooth decay may substantially improve the oral status of the population and increase longevity."

A review of the literature by Loesche and Lopatin[46] indicates that poor oral health has been associated with medical conditions such as aspiration pneumonia and cardiovascular disease. In particular, periodontal disease can be associated with coronary heart disease and cerebrovascular accident (CVA; stroke). In addition, the Surgeon General's Report on Oral Health emphasizes that animal and population-based studies demonstrate an association between periodontal disease and diabetes, cardiovascular disease, and stroke.[66]

Recent investigations confirm these associations. For example, a periodontal examination may assist cardiovascular risk assessment in hypertensive patients. Angeli et al[8] reported an association between periodontal disease and left ventricular mass in untreated patients with essential hypertension.

Pneumonia is a common cause of morbidity and mortality in the older adult. Improvements in oral care have greatly reduced the incidence of pneumonia in elderly nursing home patients. Although the mechanism is currently under investigation, it is thought that the cough reflex can be improved by reducing the oropharyngeal microbial pathogens present.[69] Expanding on these findings, studies have been conducted on the prevention of ventilator-associated pneumonia. Providing oral therapy for intensive care patients to reduce bacterial colonization in the mouth and teeth can reduce mortality and morbidity by 42%.[35]

The presence and extent of periodontal disease may be related to increased risk of weight loss in older, well-functioning adults. This association is independent of smoking and diabetes mellitus. Changes in nutrient intake may be related to periodontal disease and a higher systemic inflammatory burden.[74]

PERIODONTAL TREATMENT PLANNING

Generally, periodontal disease in older adults is not a rapidly progressive disease but often presents as long-standing chronic disease. Because periodontal disease has periods of exacerbation and remission, understanding and documenting periods of active disease versus quiescent periods are essential to the formulation of the treatment plan and prognosis.[60]

Periodontal disease must be diagnosed regardless of age. The goal of periodontal treatment for both young and old patients is to preserve function and eliminate or prevent the progression of inflammatory disease.[72]

The goal of clinically managing periodontal disease in older adults is based on specific, individualized care. The major consideration is improving or maintaining function, with an emphasis on quality-of-life issues. Emphasizing care over cure is the cornerstone of any proposed treatment plan. Prevention, comfort, function, esthetics, and ease of maintenance are the criteria for successful management of an older adult, particularly a frail or functionally dependent older patient.

Several factors must be considered during treatment planning for older individuals.[60] It is important first to remember that periodontal healing and recurrence of disease are *not* influenced by age.[20] Factors to consider in the older patient are medical and mental health status, medications, functional status, and lifestyle behaviors that influence periodontal treatment, outcome, or progression of disease.[72] Periodontal disease severity, ability to perform oral hygiene procedures, and ability to tolerate treatment should be evaluated during treatment planning. The risks and benefits of both surgical and nonsurgical therapy should be considered.[46] The amount of remaining periodontal support or past periodontal destruction, tooth type, number of occlusal contacts, and individual patient preferences are also important.[72] Dental implants are a reliable replacement for missing teeth in older adults[77]; age alone is not a contraindication for implant placement. Risk factors for success of implants has been investigated. While aging is a factor in decision making, the majority of the failures are associated with smoking, diabetes, head and neck radiation, and postmenopausal estrogen therapy.[51]

For older adults, a nonsurgical approach is often the first treatment choice. Depending on the nature and extent of periodontal disease, surgical therapy may be indicated. Surgical technique should minimize the amount of additional root exposure. Individuals responding best to surgical therapy are those who are able to maintain the surgical result. Age alone is not a contraindication to surgery. For individuals who are unable to comply with treatment, who have poor oral hygiene, or who are medically or mentally compromised or functionally impaired, palliative supportive periodontal care instead of surgical periodontal treatment is often the optimal treatment approach.[46,58]

A common goal for all older adults is to decrease bacteria through oral hygiene and mechanical debridement. Clinical trials with older adults show that the development or progression of periodontal disease can be prevented or arrested by the control of plaque. For certain patients, topical antibiotic therapy may complement repeated subgingival instrumentation during supportive care. Oral hygiene maintenance should also focus on root surfaces susceptible to caries.[72]

Decision making for frail and functionally dependent older adults may be challenging to the general dentist. For this reason, dentists, other health care professionals, and other scientists are creating high-quality office-based methods to access evidence-based decision-making programs and accommodating websites to help with complicated oral health care issues. [3,59,70]

PREVENTION OF PERIODONTAL DISEASE AND MAINTENANCE OF PERIODONTAL HEALTH IN OLDER ADULTS

For both younger and older persons, the most important factors determining a successful outcome of periodontal treatment are plaque control and frequency of professional care. Advanced age does not decrease plaque control[9]; however, older adults may have

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difficulty performing adequate oral hygiene because of compromised health, altered mental status, medications, or altered mobility and dexterity.[72] Older adults may change toothbrush habits because of disabilities such as hemiplegia secondary to CVA, visual difficulties, dementia, and arthritis. The newer, lightweight, electric-powered toothbrushes may be more beneficial than a manual toothbrush for older adults with physical and sensory limitations. The proportion of people who floss their teeth decreases after age 40 years.[71] This may be partly caused by impairment of fine motor skills secondary to disease or injury. Interproximal brushes, shaped wooden toothpicks, and mechanical flossing devices often can be used in place of traditional flossing with satisfactory outcomes.

In addition, multidisciplinary strategies are increasingly becoming part of periodontal health care promotion. Assessments of overall health, functional status, and patient education are fundamental to promoting and maintaining optimum periodontal health. Older adults, their families, and caregivers need to be informed and trained by dentists in the appropriate devices, chemotherapeutic agents, and techniques to provide oral self-care and maintain healthy lifestyles. The outcomes are instrumental in achieving overall health, oral and periodontal health, self-esteem, nutrition, and quality of life. Barriers to achieving these benefits are access and costs. For those older adults who are homebound or institutionalized, these barriers inhibit their achieving and maintaining optimum oral and periodontal health.

Chemotherapeutic Agents

Antiplaque Agents. Patients who are unable to remove plaque adequately secondary to disease or disability may benefit from antiplaque agents such as chlorhexidine, sub-antimicrobial tetracycline, and Listerine or its generic counterparts.[50,60,72]

Chlorhexidine is a cationic bisbiguanide that has been used as a broad-spectrum antiseptic in medicine since the 1950s. In Europe, a 0.2% concentration of chlorhexidine has been used for years as a preventive and therapeutic agent.[46,71] Chlorhexidine is either bacteriostatic or bactericidal, depending on the dose. Adverse effects of chlorhexidine include an increase in calculus formation, dysgeusia (altered taste), and permanent staining of teeth.[6] Chlorhexidine is a prescription rinse for short-term use (<6 months); long-term use (>6 months) has not been extensively studied.[71] The American Dental Association (ADA) Council on Dental Therapeutics[4] has approved chlorhexidine to help prevent and reduce supragingival plaque and gingivitis. Although chlorhexidine has not been studied in older adults, outcomes in younger persons, including those with disabilities, suggest that it is also effective in older adults. Chlorhexidine may be particularly useful for older adults who have difficulty with plaque removal and those who take phenytoin, calcium channel blockers, or cyclosporines and who are at risk for gingival hyperplasia.[60,71]

Sub-antimicrobial tetracycline (Periostat) is useful in treating moderate to severe chronic periodontitis. The active ingredient in Periostat is *doxycycline hyclate*. In concert with scaling and root planing, Mohammad et al[50] have shown this treatment to be effective in institutionalized older adults. Periostat is contraindicated for those patients with an allergy to tetracycline.

Listerine antiseptic and its generic counterparts are approved by the ADA Council on Dental Therapeutics[6] to help prevent and reduce supragingival plaque and gingivitis. The active ingredients in Listerine are *methyl salicylate* and three essential oils (eucalyptol, thymol, and menthol). Listerine has been shown to be effective in reducing plaque and gingivitis compared with placebo rinses in young healthy adults. Listerine may exacerbate xerostomia because of its high alcohol content, ranging from 21.6% to 26.9%. Listerine is generally contraindicated in patients under treatment for alcoholism who take Antabuse (disulfiram). Listerine may benefit patients who do not tolerate the taste or staining of chlorhexidine and who prefer OTC medicaments that are less expensive and easier to obtain.[60,71]

The use of inorganic polyphosphate (Poly P) to treat periodontal disease in the aging is being investigated in Japan and points to an alternative method for reduction of bone loss. What is interesting is that the Poly P is considered safe as a food additive and is low risk to harm the patient. Since it is not an antibiotic, bacteria are unlikely to become resistant to it.[80]

Fluoride. Fluoride, "nature's cavity fighter," is the most effective caries-preventive agent currently available. Fluoride's effects are as follows[60,71]:

1. Reduces enamel solubility.
2. Promotes remineralization of early carious lesions.
3. Is bactericidal to bacterial plaque.

Topical fluorides are recommended for the prevention and treatment of dental caries. OTC fluorides include fluoride dentifrices, rinses, and gels that contain concentrations of 230 to 1500 parts per million (ppm) of fluoride ions. Prescription 1.1% neutral sodium fluoride gels are available with a fluoride concentration of 5000 ppm fluoride ion. Professionally applied fluoride gel, foam, or varnish products are between 9050 and 22,600 ppm fluoride ion.[6]

Saliva Substitutes. Saliva substitutes, which are intended to match the chemical and physical traits of saliva, are available to relieve the symptoms of dry mouth. Their composition is varied; however, they usually contain salt ions, a flavoring agent, paraben (preservative), cellulose derivative or animal mucins, and fluoride. The ADA's seal of approval has been granted for some artificial saliva products (e.g., Saliva Substitute, Salivart).[6] Most saliva substitutes can be used as desired by patients and are dispensed in spray bottle, rinse/swish bottle, or oral swab stick.[6,71] In addition, products, such as dry-mouth toothpastes and moisturizing gels, are also available. Biotene products are marketed to relieve the symptoms of xerostomia.

Patients with dry mouth may also benefit by stimulating saliva flow with sugarless candies and sugarless gum. Xylitol chewing gum has been shown to have anticariogenic properties in children. Medicated chewing gum with xylitol and chlorhexidine or xylitol alone has the added benefits of reducing oral plaque scores and gingivitis in elderly persons who live in residential facilities.[7]

Salivary substitutes and stimulants are only effective in the short term. Under investigation is acupuncture-like transcutaneous nerve stimulation (Codetron), a method to treat radiation-induced xerostomia. Unlike traditional acupuncture therapy, Codetron does not use invasive needles to achieve stimulation. This method helps the patient to produce their own saliva and reduce symptoms of xerostomia for several months. Acupuncture therapy has demonstrated improvements lasting up to 3 years.[79]

Risk Reduction

Cessation of tobacco use has been primarily an issue of patient compliance. Most smokers (90%) quit "cold turkey." However, nicotine replacement therapy may help strongly addicted patients. The transdermal nicotine patch or nicotine polacrilex (gum) can reduce the symptoms of nicotine withdrawal. For behavioral modification, the four steps to tobacco cessation are as follows:

1. Ask the patient about tobacco.
2. Advise the patient to stop using tobacco.

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3. Assist the patient in stopping by selecting a quitting date (usually within the next 4 weeks).
4. Arrange patient follow-up services.

Clinical studies show that all four components, used routinely, result in much higher patient cessation rates than if only two or three are used. Both forms of nicotine replacement have been shown to increase cessation rates when used in combination with behavioral interventions.

Nicotine replacement therapy is intended to be used for a few (6 to 12) weeks so that patients can learn psychologic and social coping skills without going through nicotine withdrawal at the same time. It is not recommended for use for more than 6 months.

Nicotine replacement therapy should probably not be considered for the following persons:

- Individuals who have not previously tried to quit.
- Individuals who use only small amounts of tobacco or who are occasional users.
- Individuals who have tried to quit and have not had significant physical symptoms of nicotine withdrawal.
- Individuals who do not receive behavioral support from clinic staff.

Alcoholism and alcohol abuse are diagnosed using the *Diagnostic and Statistical Manual of Mental Disorders,* third edition, revised *(DSM-IIIR),* and fourth edition *(DSM-IV).* The tests suggested to screen for alcohol abuse are the self-administered questionnaire, the Michigan Alcoholism Screening Test (MAST), and the CAGE (from a series of four questions about drinking: *c*ut down, *a*nnoyed, *g*uilty, and *e*ye-opener) questionnaire, which is a four-item screening tool (Figure 39-10). A score of 2 (an affirmative answer to 2 questions) in the elderly patient provides clinical evidence or suspicion of alcohol abuse, with a sensitivity of about 50% and a specificity over 90%. The MAST, with a cutoff score of 5, produces sensitivity ranging from 50% to 70% and a specificity above 90%.

The most common treatment for alcoholism is referral to a substance abuse facility. For the older adults, the most effective treatment is a program that emphasizes older adult–specific groups using nonconfrontational therapy and encouraging reminiscence, as well as discussion of current problems. Discussion of current problems is relevant because of the role that stress plays in recent widowhood, recent retirement, or disruption or removal from one's residence.

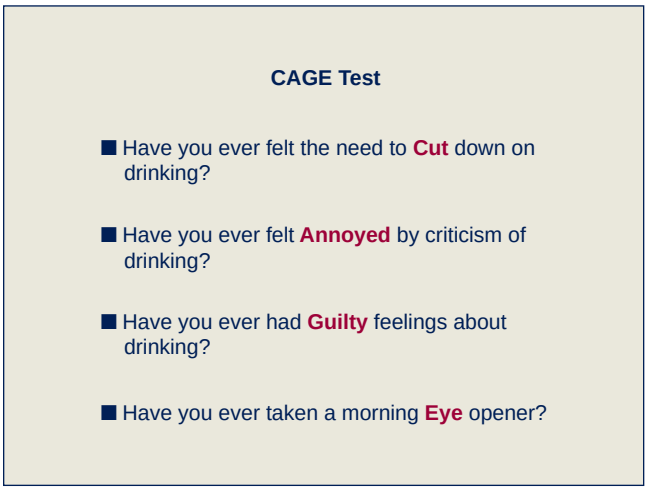

Figure 39-10 Method of determining alcohol abuse using the modified CAGE test. The acronym CAGE is derived from a series of four questions about drinking: *c*ut down; *a*nnoyed; *g*uilty; and *e*ye-opener test. *(From Mallin R:* Am Fam Physician *65:1107, 2002.)*

CONCLUSION

Future oral health care trends will see increased numbers of older adults seeking periodontal therapy. Dental practitioners of the twenty-first century should be comfortable providing comprehensive periodontal care for this segment of the population. Aging dental patients have particular oral and general health conditions that dentists should be familiar with detecting, consulting, and treating. Medical diseases and conditions that occur more often with age may require modification to periodontal preventive tools, as well as for the planning and treatment phases of periodontal care.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

With improvements in life expectancy, dentists can expect to be treating more older patients than in previous years. There are also improvements in dental heath so that more of these patients have surviving teeth. Because of these factors, clinicians need to focus on the special needs of older patients. Many of these patients have reduced salivary flow and so are more susceptible to gingival inflammation and root caries and thus need aggressive preventive treatment, including topical fluorides, antiplaque mouthwashes, focused mechanical oral hygiene techniques, and artificial salivary supplements.

Healthy older patients can be successfully treated with all types of periodontal therapy, including surgical approaches, and dental implants can be successful in this group of patients.

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CHAPTER 40

Treatment of Aggressive and Atypical Forms of Periodontitis

Perry R. Klokkevold

CHAPTER OUTLINE

AGGRESSIVE PERIODONTITIS
Therapeutic Modalities
Treatment Planning and Restorative Considerations
Periodontal Maintenance
PERIODONTITIS REFRACTORY TO TREATMENT (online only)
NECROTIZING ULCERATIVE PERIODONTITIS
CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

The majority of patients with common forms of periodontal disease respond predictably well to conventional therapy, including oral hygiene instruction, nonsurgical debridement, surgery, and supportive periodontal maintenance. However, patients diagnosed with aggressive and some atypical forms of periodontal disease often do not respond as predictably or as favorably to conventional therapy. Fortunately, only a small percentage of patients with periodontal disease are diagnosed with aggressive periodontitis. Patients who are diagnosed with periodontal disease (any type) that is *refractory* to treatment present in small numbers as well. Even fewer patients are diagnosed with necrotizing ulcerative periodontitis. Each of these atypical disease entities poses significant challenges for the clinician not only because they are infrequently encountered, but also because they may not respond favorably to conventional periodontal therapy.[27,45] Furthermore, the severe loss of periodontal support associated with these cases leaves the clinician faced with uncertainty about treatment outcomes and difficulty in making decisions about whether to save compromised teeth or to extract them.

This chapter outlines important considerations for the treatment of patients diagnosed with aggressive periodontitis and atypical forms of periodontal disease, including refractory cases of periodontitis and necrotizing ulcerative periodontitis.

AGGRESSIVE PERIODONTITIS

Aggressive periodontitis, by definition, causes rapid destruction of the periodontal attachment apparatus and the supporting alveolar bone (see Chapter 18). The responsiveness of aggressive periodontitis to conventional periodontal treatment is unpredictable, and the overall prognosis for these patients is poorer than for patients with chronic periodontitis. Because these patients do not respond "normally" to conventional methods and their disease progresses unusually fast, the logical question is whether there are problems associated with an impaired host immune response that may contribute to such a *different* disease and result in a *limited* response to the usual therapeutic measures. Indeed, defects in polymorphonuclear leukocyte (PMN, neutrophil) function have been identified in some patients with aggressive periodontitis.[34,36] Also, in a small number of cases, a systemic disease, such as neutropenia, can be identified that clearly explains the unusual severity of the periodontal disease for that individual.[14,15] In most patients with aggressive periodontitis, however, systemic diseases or disorders cannot be identified. In fact, the irony is that these patients are typically quite healthy. Numerous attempts to examine immunologic profiles in patients with aggressive periodontitis have failed to identify any specific etiologic factors common to all patients.

The prognosis for patients with aggressive periodontitis depends on (1) whether the disease is generalized or localized, (2) the degree of destruction present at the time of diagnosis, and (3) the ability to control future progression. *Generalized* aggressive periodontitis rarely undergoes spontaneous remission, whereas *localized* forms of the disease have been known to arrest spontaneously.[33] This unexplained curtailment of disease progression has sometimes been referred to as a "burnout" of the disease. It appears that cases of localized aggressive periodontitis often have a limited period of rapid periodontal attachment and alveolar bone loss, followed by a slower, more chronic phase of disease progression. Overall, patients with generalized aggressive periodontitis tend to have a poorer prognosis because they typically have more teeth affected by the

disease and because the disease is less likely to go spontaneously into remission compared with patients with localized forms of aggressive periodontitis.

Therapeutic Modalities

Early detection is critically important in the treatment of aggressive periodontitis (generalized or localized) because preventing further destruction is often more predictable than attempting to regenerate lost supporting tissues. Therefore, at the initial diagnosis, it is helpful to obtain any previously taken radiographs to assess the rate of progression of the disease. Together with future radiographs, this documentation will also facilitate the clinician's assessment of treatment success and control of the disease.

Treatment of aggressive periodontitis must be pursued with a logical and regimented approach. Several aspects of treatment must be particularly considered when managing a patient with aggressive periodontitis. One of the most important aspects of treatment success is to educate the patient about the disease, including the causes and the risk factors for disease, and to stress the importance of the patient's role in the success of treatment.[1] Essential therapeutic considerations for the clinician are to control the infection, arrest disease progression, correct anatomic defects, replace missing teeth, and ultimately help the patient maintain periodontal health with frequent periodontal maintenance care. Educating family members is another important factor because aggressive periodontitis is known to have familial aggregation. Thus family members, especially younger siblings, of the patient diagnosed with aggressive periodontitis should be examined for signs of disease, educated about preventive measures, and monitored closely. It cannot be stressed enough that early diagnosis, intervention, and if possible, prevention of disease are more desirable than attempting to reverse the destruction that results from aggressive periodontitis.

Many different treatment approaches have been used to manage patients with aggressive periodontitis, including nonsurgical, surgical, and antimicrobial therapy. Recent advances in our understanding of the role of the host response in disease pathogenesis are leading to new opportunities for treatment. The advantages and limitations of conventional, antimicrobial, and combination therapy for the treatment of aggressive periodontitis, as well as restorative considerations are discussed next.

Conventional Periodontal Therapy. Conventional periodontal therapy for aggressive periodontitis consists of patient education, oral hygiene improvement, scaling and root planing, and regular (frequent) recall maintenance. It may or may not include periodontal flap surgery.[4,64] Unfortunately, the response of aggressive periodontitis to conventional therapy alone has been limited and unpredictable. Patients who are diagnosed with aggressive periodontitis at an early stage and who are able to enter therapy may have a better outcome than those who are diagnosed at an advanced stage of destruction. In general, the earlier the disease is diagnosed, the more conservative the therapy and the more predictable the outcome.

Teeth with moderate to advanced periodontal attachment loss and bone loss often have a poor prognosis and pose the most difficult challenge. Depending on the condition of the remaining dentition, treatment of these teeth may have a limited prospect for improvement and may even diminish the overall treatment success for the patient. Clearly, some of these teeth should be extracted; however, other teeth may be pivotal to the stability of that individual's dentition, and thus it may be desirable to attempt treatment to maintain them. Treatment options for teeth with deep periodontal pockets and bone loss may be nonsurgical or surgical. Surgery may be purely resective, regenerative, or a combination of these approaches.

Surgical Resective Therapy. Resective periodontal surgery can be effective to reduce or eliminate pocket depth in patients with aggressive periodontitis. However, it may be difficult to accomplish if adjacent teeth are unaffected, as often seen in cases of localized aggressive periodontitis. If a significant height discrepancy exists between the periodontal support of the affected tooth and the adjacent unaffected tooth, the gingival transition (following the bone) will often result in deep probing pocket depth around the affected tooth despite surgical efforts. A less-than-ideal outcome must be taken into consideration before deciding to treat increased pocket depth surgically.

It is important to realize the limitations of surgical therapy and to appreciate the possible risk that surgical therapy may further compromise teeth that are mobile because of extensive loss of periodontal support. For example, in a patient with severe

SCIENCE TRANSFER

Treatment of Aggressive and Atypical Forms of Periodontitis

There is a small group of patients that develops aggressive periodontal diseases. In a few of these cases, this is related to specific systemic diseases that affect the immune system, e.g., neutropenia, polymorphonuclear leukocyte dysfunction, and diabetes. However, in most of those patients, it is not possible to identify the reason why such dramatic periodontal disease occurred, and so comprehensive blood tests at present are unlikely to identify the problem. The treatment approach to these patients follows the same rationale of treatment used for regular periodontitis patients, i.e., initial therapy, followed by periodontal surgery and maintenance care. In general, these patients will respond to treatment, particularly those patients that develop aggressive periodontitis as adolescents in whom often the disease activity diminishes once they reach their twenties. All available adjunctive therapy should accompany these patients including systemic antibiotics, localized antimicrobial rinses such as chlorhexidine, and the use of host modulation therapy. The latter of these may in the future provide increased therapeutic success as new protocols and agents become available. These patients can be treated with dental implants without concern for dramatic increases in risk of failure.

Another group of atypical patients is those that are refractory to treatment, which includes patients with high plaque scores, smokers, and poorly controlled diabetics. These patients similarly require the comprehensive use of all available treatment modalities and many can be helped by including systemic antibiotics such as metronidazole and amoxicillin with potassium clavulanate.

All of the patients outlined in this chapter require maintenance programs with strict monitoring of plaque scores and high-frequency recall appointments (at least one every 3 months.)

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horizontal bone loss, surgical resective therapy may result in increased tooth mobility that is difficult to manage, and a nonsurgical approach may be indicated. Therefore careful evaluation of the risks versus the benefits of surgery must be considered on a case-by-case basis.

Regenerative Therapy. The concept and application of periodontal regeneration has been established in patients with chronic forms of periodontal disease (see Chapter 61). The use of regenerative materials, including bone grafts, barrier membranes, and wound-healing agents, are well documented and often used. Intrabony defects, particularly vertical defects with multiple osseous walls, are often amenable to regeneration with these techniques. Most of the success and predictability of periodontal regeneration have been achieved in patients with chronic periodontitis; much less evidence is available about the use of periodontal regeneration for patients with aggressive periodontitis.

Periodontal regenerative procedures have been successfully demonstrated in patients with localized aggressive periodontitis in some clinical case reports. Dodson et al[16] demonstrated the regenerative potential of a severe, localized osseous defect around a mandibular incisor in a healthy, 19-year-old black man diagnosed with localized aggressive periodontitis. The patient presented with severe bone loss localized around one of the mandibular incisors. Using open-flap surgical debridement, root surface conditioning (tetracycline solution), and an allogenic bone graft reconstituted with sterile saline and tetracycline powder, the surgeons reduced the probing pocket depth from 9 to 12 mm down to 1 to 3 mm (3 mm of recession was noted), and significant bone fill of the defect (about 80%) was reported (Figure 40-1). This case illustrates the potential for healing severe defects in patients with localized aggressive periodontitis, especially when local factors are controlled and sound surgical principles are followed. The authors cited several factors that likely contributed to the success of this case, including a probable transition of disease activity from aggressive to chronic, tooth stabilization before surgery, sound surgical management of hard and soft tissues, and good postoperative care.[16]

It is important to note that although the potential for regeneration in patients with aggressive periodontitis appears to be good, expectations are limited for patients with severe bone loss. Depending on the anatomy of the defect and teeth involved, the potential for bone fill and periodontal regeneration may be poor. This is especially true if the bone loss is horizontal and if it has progressed to involve furcations. The usual criteria of case selection and sound

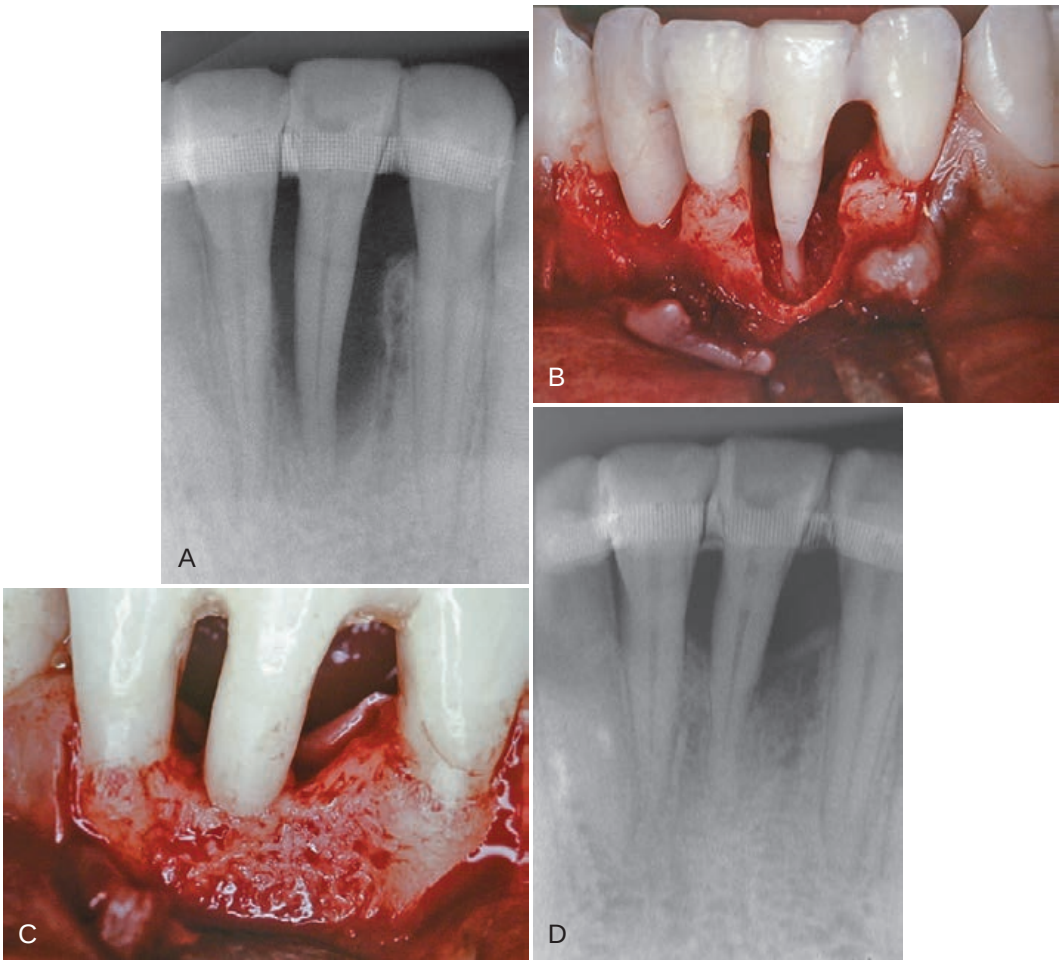

Figure 40-1 Clinical photographs and periapical radiographs demonstrating regenerative success in patient with localized aggressive periodontitis. **A,** Periapical radiograph of the right lateral incisor at the initial diagnosis. Notice the severe, vertical bone loss associated with the right lateral incisor. Tooth has been splinted to adjacent teeth for stability. **B,** Facial view of the circumferential osseous defect around the lower right lateral incisor during open flap surgery. There is complete loss of buccal, lingual, mesial, and distal bone around the lateral incisor, with minimal bone support limited to the apical few millimeters. **C,** Facial view of reentered surgical site 1 year after treatment. Bone fill around all surfaces demonstrates remarkable potential for regeneration of a large osseous defect in a young patient with localized aggressive periodontitis. **D,** Periapical radiograph taken 1 year after regenerative therapy. Note the increased radiopacity and bone fill. *(From Dodson SA, Takei HH, Carranza FA Jr:* Int J Periodont Restor Dent *16:455, 1996.)*

principles of surgical management for regenerative therapy apply equally to cases of aggressive periodontitis. Good clinical judgment must be used to determine whether a particular tooth should be treated with the goal of regeneration.

Recent advances in regenerative therapy have advocated the use of an *enamel matrix protein* to aid in the regeneration of cementum and new attachment in periodontal defects.[67] A systematic review of the literature concluded that treatment with an enamel matrix protein can improve probing attachment level (mean difference, 1.3 mm) and probing pocket depth (mean difference, 1.0 mm) compared with flap debridement alone in patients with chronic periodontitis.[17] However, no evidence has been reported to suggest significant advantages for the use of enamel matrix proteins in patients with aggressive periodontitis. One recent case report described use of the protein in a 15-year-old patient with localized aggressive periodontitis.[8] No comparative sites were treated, so the effect that may be attributed to enamel matrix protein could not be determined. A recent clinical and radiographic study with a split-mouth design included four patients with aggressive periodontitis and four patients with chronic periodontitis. The authors concluded that enamel matrix proteins offered no advantage over surgical debridement alone in these patients.[66] Although this study attempted to use controls to measure the effect of enamel matrix proteins, there were insufficient patients to appreciate a difference in outcome. At this time, it is unknown whether the use of enamel matrix proteins offers significant advantages for the patient with aggressive periodontitis.

Antimicrobial Therapy. The presence of periodontal pathogens, specifically *Actinobacillus actinomycetemcomitans,* has been implicated as the reason that aggressive periodontitis does not respond to conventional therapy alone. These pathogens are known to remain in the tissues after therapy to reinfect the pocket.[11,62] In the late 1970s and early 1980s, the identification of *A. actinomycetemcomitans* as a major culprit and the discovery that this organism penetrates the tissues offered another perspective to the pathogenesis of aggressive periodontitis and offered new hope for therapeutic success, namely, antibiotics.[11] The use of systemic antibiotics was thought to be necessary to eliminate pathogenic bacteria (especially *A. actinomycetemcomitans*) from the tissues. Indeed, several authors have reported success in the treatment of aggressive periodontitis using antibiotics as adjuncts to standard therapy.[38-40,75]

Systemic Administration of Antibiotics. There is compelling evidence that adjunctive antibiotic treatment frequently results in a more favorable clinical response than mechanical therapy alone.[70] In a systematic review, Herrera et al[26] found that systemic antimicrobials in conjunction with scaling and root planing offer benefits over scaling and planing alone in terms of clinical attachment level, probing pocket depth, and reduced risk of additional attachment loss. Patients with deeper, progressive pockets seem to benefit the most from systemic administration of adjunctive antibiotics. Many different antibiotic types and regimens were reviewed. Because of limitations in comparing data from different studies, however, definitive recommendations were not possible.

Figure 40-2 shows the before and after results of an aggressive periodontitis case treated nonsurgically with scaling and root planing and adjunctive antibiotic therapy. The patient, a 34-year-old Asian male, presented with a complaint of loose teeth and bleeding gums. He requested treatment to save his teeth. Treatment consisted of patient education including oral hygiene instructions and nonsurgical therapy with adjunctive antibiotics. All teeth were thoroughly scaled and root planed under local anesthesia over two treatment appointments, and the patient was treated with systemic amoxicillin (500 mg, 3 times a day for 2 weeks) during the period of treatment. All areas responded favorably with probing pocket depths decreasing from 3 to 13 mm down to 2 to 5 mm. Bleeding on probing diminished from generalized to very few isolated areas. He has been on periodontal maintenance with good results for more than 5 years.

Genco et al[21] treated localized aggressive periodontitis patients with scaling and root planing plus systemic administration of tetracycline (250 mg, 4 times daily for 14 days every 8 weeks). Measurements of vertical defects were made at intervals of up to 18 months after the initiation of therapy. Bone loss had stopped, and one-third of the defects demonstrated an increase in bone level, whereas in the control group, bone loss continued.

Liljenberg and Lindhe[38] treated patients with localized aggressive periodontitis with systemic administration of tetracycline (250 mg, 4 times daily for 2 weeks), modified Widman flaps, and periodic recall visits (1 visit every month for 6 months, then 1 visit every 3 months). The lesions healed more rapidly and more completely than similar lesions in control patients. These investigators reevaluated their results after 5 years and found that the treatment group continued to demonstrate resolution of gingival inflammation, gain of clinical attachment, and refill of bone in angular defects.[39] Figures 40-3 and 40-4 show radiographs of a case with similar treatment and results.[5]

Clearly, numerous studies support the use of adjunctive tetracycline along with mechanical debridement for the treatment of *A. actinomycetemcomitans*–associated aggressive periodontitis (Box 40-1). Given the possible emergence of tetracycline-resistant *A. actinomycetemcomitans,* there is concern that tetracycline may not be effective. In these cases the combination of metronidazole and amoxicillin may be advantageous. The combination of these two antibiotics with conventional periodontal therapy[24,25] provides better disease control and better clinical improvement in attachment levels in difficult-to-manage periodontitis cases than similar periodontal therapy without antibiotics. Similar effects were seen for a variety of antibiotic types. However, a lack of sufficient sample sizes among studies makes it difficult to offer specific recommendations about which antibiotics were most effective.[26]

The criteria for selection of antibiotics are not clear. Good clinical and microbiologic responses have been reported with several individual antibiotics and antibiotic combinations (Table 40-1). The optimal antibiotic or combination for any particular infection probably depends on the case. Choices must be made based on patient-related and disease-related factors.

Microbial Testing. Some investigators and clinicians advocate microbial testing to identify the specific periodontal pathogens

BOX 40-1 Systemic Tetracycline in Treatment of Aggressive Periodontitis

Systemic tetracycline (250 mg of tetracycline hydrochloride 4 times daily for at least 1 week) should be given in conjunction with local mechanical therapy. If surgery is indicated, systemic tetracycline should be prescribed and the patient instructed to begin taking the antibiotic approximately 1 hour before surgery. Doxycycline, 100 mg/day, may be used instead of tetracycline. Chlorhexidine rinses should be prescribed and continued for several weeks to enhance plaque control and facilitate healing.

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TABLE 40-1 Antibiotic Therapy for Aggressive Periodontitis

Associated Microflora	Antibiotic of Choice
Gram-positive organisms	Amoxicillin–clavulanate potassium (Augmentin)[12,72]
Gram-negative organisms	Clindamycin[22,23,68,72]
Nonoral gram-negative, facultative rods	Ciprofloxacin[41]
Pseudomonads, staphylococci	
Black-pigmented bacteria and spirochetes	Metronidazole[22,65]
Prevotella intermedia, Porphyromonas gingivalis	Tetracycline[55]
Actinobacillus actinomycetemcomitans	Metronidazole-amoxicillin[22,65] Metronidazole-ciprofloxacin Tetracycline[53]
P. gingivalis	Azithromycin[54]

responsible for disease and to select an appropriate antibiotic based on sensitivity and resistance. There may be specific cases in which bacterial identification and antibiotic-sensitivity testing is invaluable. For example, in localized aggressive periodontitis cases, tetracycline-resistant *Actinobacillus* species have been suspected. If antibiotic susceptibility tests determine that tetracycline-resistant species exist in the lesion, the clinician may be advised to consider another antibiotic or an antibiotic combination, such as amoxicillin and metronidazole.[18,55,66]

In practice, antibiotics are often used empirically without microbial testing. One study evaluated and compared the results of microbial testing offered by two independent laboratories.[46,63] Two microbiologic cultures, sampled simultaneously from the same sites in 20 patients, were submitted separately to each of the two laboratories for bacterial identification and antibiotic-sensitivity testing. The reported presence of bacterial species varied from one laboratory to another, as did their antimicrobial recommendations. Interestingly, the combination of amoxicillin and metronidazole yielded the highest level of agreement (80%). This is likely attributed to the effectiveness of this combination, as well as a clinical predisposition to favor a known regimen. These findings suggest that the usefulness of microbial testing may be limited and led the authors to

Figure 40-2 Intraoral clinical photographs and full mouth radiographs (before and after treatment) of a 34-year-old Asian male with aggressive periodontitis. **A** to **D,** Intraoral clinical photographs of pretreatment periodontal condition. Note the gingival edema, inflammation and bleeding. Probing pocket depths ranged from 3 to 13 mm with generalized bleeding on probing and purulent exudate. **E,** Pretreatment full mouth radiographs demonstrating generalized severe horizontal bone loss. Note the periapical radiolucency at the apex of the upper left premolar indicating pulpal disease.

Figure 40-2, cont'd **F** to **I,** Intraoral clinical photographs of posttreatment (5 years) results. Treatment included nonsurgical scaling and root planing along with adjunctive systemic antibiotics (amoxicillin). Patient improved oral hygiene and continued to be seen for professional maintenance every 3 months. Probing pocket depths have been maintained in the range of 2 to 5 mm with only a few localized areas of bleeding on probing. **J,** Posttreatment (5 years) full mouth radiographs demonstrating no additional bone loss. Note that the endodontically involved maxillary premolar was extracted. The missing tooth has been replaced with a removable partial denture.

conclude that the empiric use of antibiotics, such as a combination of amoxicillin and metronidazole, may be more clinically sound and cost-effective than bacterial identification and antibiotic-sensitivity testing.[46,63]

Nonetheless, the use of microbial testing should be considered whenever a case of aggressive periodontitis is not responding or if the destruction continues despite good therapeutic efforts.

Local Delivery. The use of local delivery to administer antibiotics offers a novel approach to the management of periodontal "localized" infections. The primary advantage of local therapy is that smaller total dosages of topical agents can be delivered inside the pocket, avoiding the side effects of systemic antibacterial agents while increasing the exposure of the target microorganisms to higher concentrations, and therefore more therapeutic levels, of the medication. Local delivery agents have been formulated in many different forms, including solutions, gels, fibers, and chips[19,20,31] (see Chapter 47).

Full-Mouth Disinfection. Another approach to antimicrobial therapy in the control of infection associated with periodontitis is the concept of full-mouth disinfection. The concept, described by Quirynen et al,[56] consists of full-mouth debridement (removal of all plaque and calculus) completed in 2 appointments within a 24-hour period. In addition to scaling and root planing, the tongue is brushed with a chlorhexidine gel (1%) for 1 minute, the mouth is rinsed with a chlorhexidine solution (0.2%) for 2 minutes, and periodontal pockets are irrigated with a chlorhexidine solution (1%).

In a clinical and microbiologic study, 10 patients with advanced chronic periodontitis were randomly assigned to test or control groups. Test patients were treated as just described while control patients received scaling and root planing by quadrant at 2-week intervals along with oral hygiene instructions. At 1 and 2 months after treatment, the test group showed significantly higher reduction in probing pocket depth, especially for pockets that were initially deep (7 to 8 mm). Patients in the test group also had significantly lower pathogenic microorganisms after treatment compared with controls. Several follow-up studies by the same center demonstrated similar results for up to 6 months after therapy.[6,7,67] In another study, the same group included a test group who did *not* use chlorhexidine as part of the one-stage full-mouth

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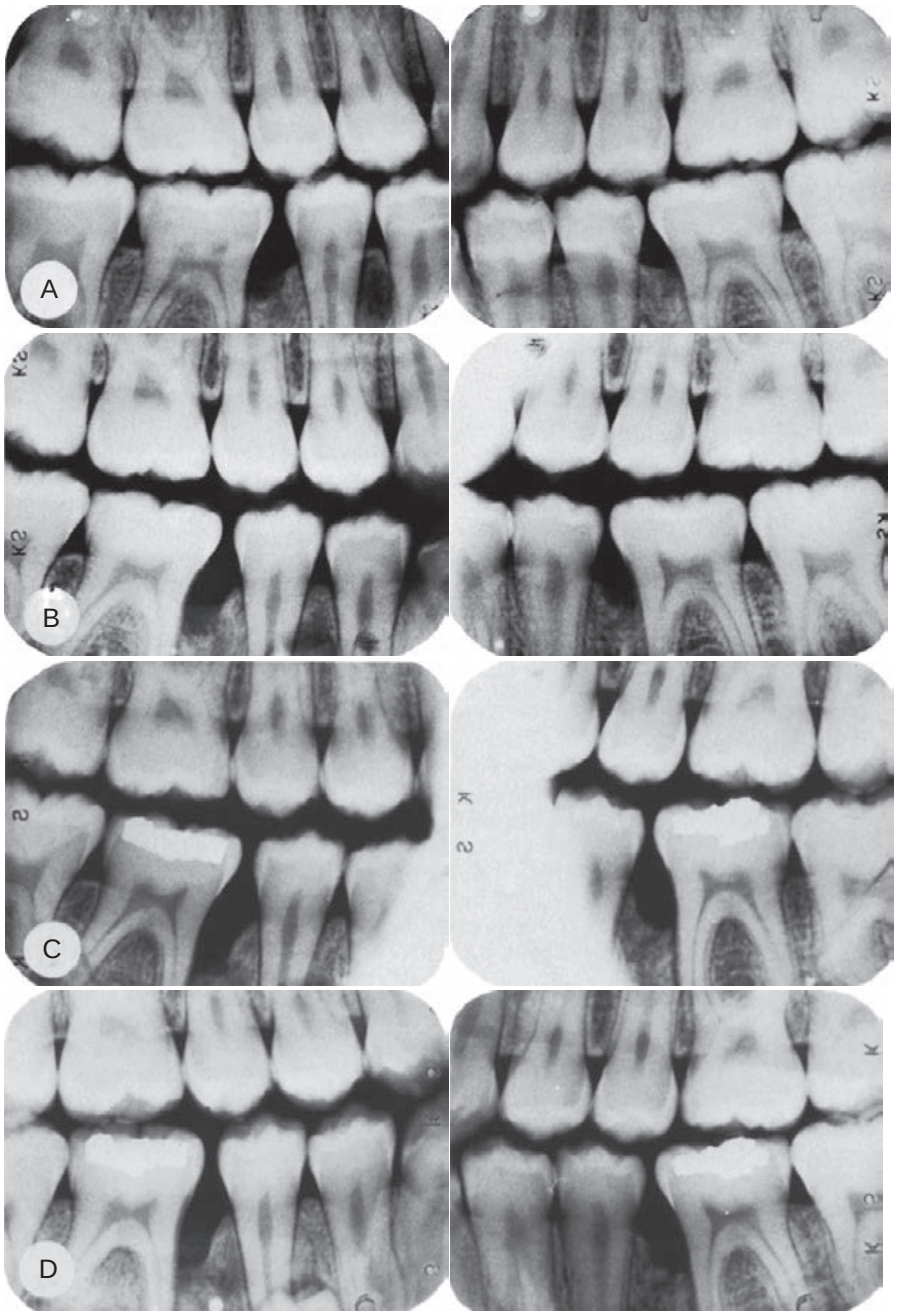

Figure 40-3 Radiographs depicting progression of the osseous lesion in patient with localized aggressive periodontitis (formerly "localized juvenile periodontitis"). **A,** January 29, 1979; **B,** August 16, 1979; **C,** February 22, 1980; **D,** May 15, 1981. Note the progressive deterioration of the osseous level. *(From Barnett ML, Baker RL:* J Periodontol *54:148, 1983.)*

disinfection.[57] Interestingly, the results of both test groups (with and without chlorhexidine) were similar and were significantly better than for controls. The authors concluded that the beneficial effects of one-stage full-mouth disinfection probably result from the full-mouth debridement within 24 hours rather than the adjunctive chlorhexidine treatment.

A few reports have included patients with aggressive (early-onset) periodontitis in their evaluation of the one-stage full-mouth disinfection protocol.[13,50,58] As with the advanced chronic periodontitis group, De Soete et al[13] found a significant reduction in probing pocket depth and gain in clinical attachment in patients with aggressive periodontitis up to 8 months after treatment compared with controls (scaling and root planing by quadrant at 2-week intervals). They also found significant reductions in periodontal pathogens up to 8 months after therapy. *Porphyromonas gingivalis* and *Tannerella forsythia* were reduced to levels below detection.

Host Modulation. A novel approach in the treatment of aggressive periodontitis and difficult-to-control forms of periodontal disease is the administration of agents that modulate the host response. Several agents have been used or evaluated to modify the host response to disease (see Chapter 48).

The use of *sub-antimicrobial dose doxycycline* (SDD) may help to prevent the destruction of the periodontal attachment by controlling the activation of matrix metalloproteinases, primarily

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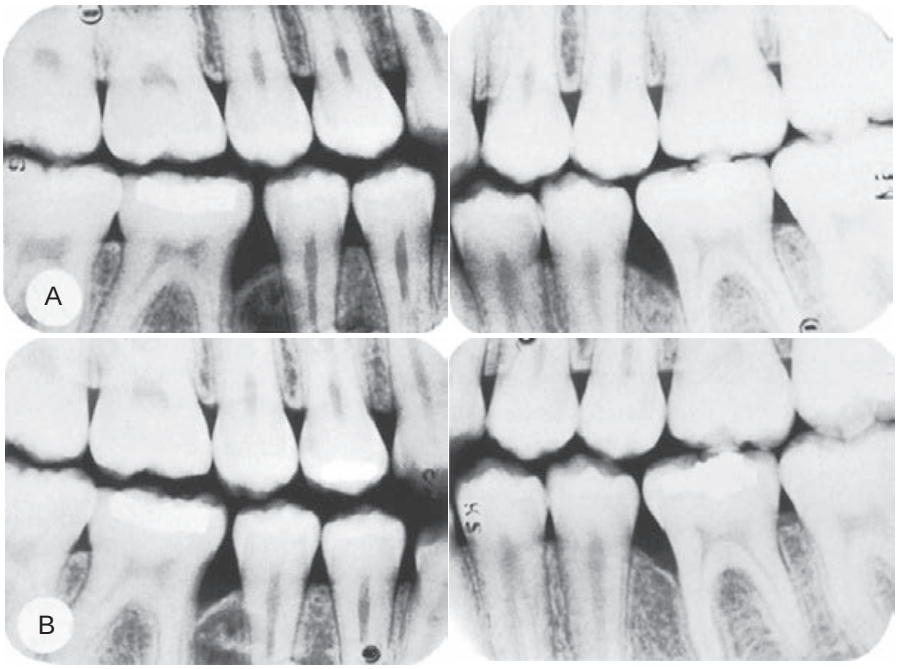

Figure 40-4 Postoperative radiographs of the patient in Figure 40-3. **A,** November 6, 1981; **B,** March 3, 1982. Treatment consisted of oral hygiene instruction, scaling and root planing concurrently with 1 g of tetracycline per day for 2 weeks, and modified Widman flaps. *(From Barnett ML, Baker RL:* J Periodontol *54:148, 1983.)*

collagenase and gelatinase, from both infiltrating cells and resident cells of the periodontium, primarily the neutrophils.[65] SDD as an adjunct to repeated mechanical debridement resulted in clinical improvement in patients with generalized aggressive periodontitis.[35] More than 50% of the patients in this study were smokers. Other agents, such as flurbiprofen, indomethacin, and naproxen, may reduce inflammatory mediator production.[29] Further research needs to be done to substantiate the effects of these agents.

Treatment Planning and Restorative Considerations

Successful management of patients with aggressive periodontitis must include tooth replacement as part of the treatment plan. In some advanced cases of aggressive periodontitis, the overall treatment success for the patient may be enhanced if severely compromised teeth are extracted. The outcome of treatment for these teeth is limited, and more importantly, the retention of severely diseased teeth over time may result in additional bone loss and teeth that are further compromised. The risk of further bone loss is even a greater concern now with the current success and predictability of dental implants and the desire to preserve bone for implant placement. Any additional alveolar bone loss in an area that has already undergone severe bone loss may further compromise residual anatomy and impair the opportunity for tooth replacement with a dental implant. This is especially true for certain areas with poor bone quality or limited bone volume, such as the posterior maxilla. Fortunately, healing of extraction sites is typically uneventful in patients with aggressive periodontitis, and bone augmentation of defect sites is predictable.

In the patient with aggressive periodontitis, the approach to restorative treatment should be made based on a single premise: extract severely compromised teeth early and plan treatment to accommodate future tooth loss. The teeth with the best prognosis should be identified and considered when planning the restorative treatment. The lower cuspids and first premolars are generally more resistant to loss, probably because of the favorable anatomy (single roots, no furcations) and easier access for patient oral hygiene. As a rule, an extensive fixed prosthesis should be avoided, and removable partial dentures should be planned in such a way as to allow for the addition of teeth.

When hopeless teeth are extracted, they need to be replaced. The desire to replace missing teeth in a permanent manner without preparation of adjacent teeth for a fixed partial denture motivated clinicians to attempt transplantation of teeth from one site to another. Transplantation of developing third molars to the sockets of hopeless first molars has been attempted with limited success.[9,37,44] Clearly, the success and predictability of dental implants have obviated the need for attempting to transplant teeth to edentulous sites.

Use of Dental Implants. Initially, the use of dental implants was suggested and implemented with much caution in patients with aggressive periodontitis because of an unfounded fear of bone and implant loss. However, evidence to the contrary appears to support the use of dental implants in patients treated for aggressive periodontal disease.[42,49,51,74] A recent systematic review of implant outcomes in patients treated for aggressive periodontitis suggests a good short-term survival of implants placed in well maintained aggressive periodontitis patients.[2] In a 10-year follow-up study, Mengel et al[47] reported successful implant rehabilitation of partially edentulous patients treated for generalized aggressive periodontitis. However, they reported significantly greater bone and attachment loss around implants in the treated generalized aggressive periodontitis patients as compared to the control group of periodontally healthy patients. Thus it is possible to consider the use of dental implants in the overall treatment plan for patients with aggressive periodontitis.

There is scant evidence to support the use of bone augmentation procedures in preparation for or in combination with implant

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placement in patients treated for aggressive periodontitis. One case report with short-term follow-up suggests it is successful.[28] A prospective study of ten patients treated for generalized aggressive periodontitis undergoing guided bone regeneration followed by implant placement found that the implant survival rate was 100% after 3 years.[48] However, a slightly greater attachment (0.65 mm) and bone (1.78 mm) loss was observed in the aggressive periodontitis group as compared to 10 periodontally healthy controls who received implants without bone augmentation.

Clinical Consideration. It is important to recognize that special consideration must be given to the risk of occlusal overload of implants placed in a periodontally compromised dentition. This is especially true when only one or a few implants are used to replace a limited number of teeth with an implant-supported fixed crown or bridge in a patient with a majority of the remaining dentition being mobile. The immobility of implants in a dentition that does not have a stable vertical occlusal stop may lead to implant overloaded.

Periodontal Maintenance

When patients with aggressive periodontitis are transferred to maintenance care, their periodontal condition must be stable (i.e., no clinical signs of disease and no periodontal pathogens). Each maintenance visit should consist of a medical history review, an inquiry about any recent periodontal problems, assessment risk of factors, a comprehensive periodontal and oral examination, thorough root debridement, and prophylaxis, followed by a review of oral hygiene instructions. If oral hygiene is not good, patients may benefit most from a review of oral hygiene instructions and visualization of plaque in their own mouth before debridement and prophylaxis.

Frequent maintenance visits appear to be one of the most important factors in the control of disease and the success of treatment in patients with aggressive periodontitis.[39,68] In a study of 25 individuals with aggressive (early-onset) periodontitis followed with maintenance every 3 to 6 months for 5 years, it was concluded that patients with aggressive periodontitis could be effectively maintained with clinical and microbiologic improvements after active periodontal therapy.[30] The presence of high bacterial counts (particularly *P. gingivalis* and *Treponema denticola*), number of acute episodes, number of teeth lost, smoking, and stress appear to be significant factors in the small percentage of sites that showed progressive bone loss. In a 5-year follow-up study of 13 patients with aggressive periodontitis, comprehensive mechanical, surgical, and antimicrobial therapy with supportive periodontal maintenance every 3 to 4 months, periodontal disease progression was arrested in 95% of the initially affected lesions. Only 2% to 5% experienced discrete episodes of loss of periodontal support.[10]

A supportive periodontal maintenance program aimed at early detection and treatment of sites that begin to lose attachment should be established. The duration between these recall visits is usually short during the first period after the patient's completion of therapy, generally no longer than 3-month intervals. Acute episodes of gingival inflammation can be detected and managed earlier when the patient is on a frequent monitoring cycle. Monitoring as frequently as every 3 to 4 weeks may be necessary when the disease is thought to be active. If signs of disease activity and progression persist despite therapeutic efforts, frequent visits and good patient compliance, microbial testing may be indicated. The rate of disease progression may be faster in younger individuals, and therefore the clinician should monitor such patients more frequently. Over time the recall maintenance interval can be adjusted (more or less often) to suit the patient's level of oral hygiene and control of disease, as determined by each examination.

Close collaboration between members of the treatment team, including the periodontist, general dentist, dental hygienist, and patient's physician, is required for continuity of care and for patient motivation and encouragement. It is important to monitor and observe the patient's overall physical status as well, because weight loss, depression, and malaise have been reported in patients with generalized aggressive periodontitis. Finally, there is a constant need to reinforce patient education about disease etiology and preventive practices (i.e., oral hygiene and control of risk factors).

For more information on periodontitis that is refractory to treatment, please visit the companion Expert CONSULT website at www.expertconsult.com.

NECROTIZING ULCERATIVE PERIODONTITIS

Necrotizing ulcerative periodontitis (NUP) is a rare disease, especially in developed countries. Often, NUP is diagnosed in individuals with a compromised host immune response (see Chapter 17). The incidence of NUP in specific populations, such as patients who are positive for human immunodeficiency virus (HIV) or who have acquired immunodeficiency syndrome (AIDS), has been reported as 0% to 6%. Most patients diagnosed with NUP apparently have diseases or conditions that impair their host immune response. These patients often have an underlying predisposing systemic factor that renders them susceptible to necrotizing ulcerative periodontal disease. For this reason, patients presenting with NUP should be treated in consultation with their physician.

A comprehensive medical evaluation and diagnosis of any condition that may be contributing to an altered host immune response should be completed. It is also important to rule out any hematologic disease (e.g., leukemia) before initiating treatment of a case that has a similar presentation to NUP (see Chapter 27 and Figures 27-17 and 27-19).

Treatment can be initiated only after a thorough medical history and examination to identify the existence of any systemic diseases, such as leukemia or other hematologic disorders, that might contribute to the oral presentation. Treatment for NUP includes local debridement of lesions with scaling and root planing, lavage, and instructions for good oral hygiene. It may be necessary to use local anesthesia during the debridement because lesions are frequently painful. The use of ultrasonic instrumentation with profuse irrigation may enhance debridement and flushing of the deep lesions. Achieving good oral hygiene may be challenging as well until the lesions and associated pain resolve.

Antimicrobial adjuncts, such as chlorhexidine, added to the oral hygiene regimen may be effective in contributing to the daily reduction of bacterial loads. Patients frequently complain of pain. The use of locally applied topical antimicrobials and systemic antibiotics, as well as systemic analgesics, should be used as indicated by signs and symptoms.

Patients with NUP often harbor bacteria, fungi, viruses, and other nonoral microorganisms, complicating the selection of antimicrobial therapy. Superinfection or overgrowth of fungi and viruses may be propagated by antibiotic therapy. Antifungal and/or antiviral agents can be considered prophylactically against these infections or after they are diagnosed. Because oral hygiene for these patients is complicated by the painful lesions, alternative

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methods should be encouraged. In such patients, irrigation with diluted cleansing and antibacterial agents can be of some benefit.

Ultimately, the successful treatment of NUP may depend on the resolution or treatment of the systemic condition (e.g., immune compromise) that predisposed the individual to the disease. Evaluation and treatment of patients with known systemic conditions, such as HIV infection, should be coordinated with the patient's physician.

CONCLUSION

Aggressive and atypical forms of periodontitis are a challenge for the clinician because they are infrequently encountered and because the predictability of treatment success varies from one patient to another. Clearly, the host immune response plays a significant role in these patients. As a result, these unusual disease entities often do not respond well to conventional therapy. Therapeutic measures must be broad in scope and thorough to ensure that all aspects of the host response are addressed. At a minimum, a frequent professional recall maintenance and personal daily oral hygiene program is necessary. The best treatment for these patients appears to be a combination of conventional treatment with antimicrobial therapy (systemic and/or local delivery) and close follow-up care.

Many questions remain regarding the selection of antibiotic type, dosage, duration, and route of administration. Antibiotic regimens have been successful in selected cases; common practice continues to be somewhat empiric. Bacterial identification may be of value for patients with disease that continues to progress despite diligent efforts by the practitioner and patient. The information can be used to determine antibiotic susceptibility of suspected pathogens. Adjunctive host modulation, although only an emerging area of interest, may prove to be promising in the treatment of patients with aggressive periodontitis, as well as periodontitis that is refractory to treatment.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 41

Treatment of Acute Gingival Disease

Perry R. Klokkevold

CHAPTER OUTLINE

ACUTE NECROTIZING ULCERATIVE GINGIVITIS
- First Visit
- Second Visit
- Third Visit
- Gingival Changes with Healing
- Additional Treatment Considerations
- Persistent or Recurrent Cases

ACUTE PERICORONITIS

ACUTE HERPETIC GINGIVOSTOMATITIS

The treatment of acute gingival disease entails the alleviation of the acute symptoms and elimination of all other periodontal disease, both chronic and acute, throughout the oral cavity. Treatment is not complete if periodontal pathologic changes or factors capable of causing them are still present.

ACUTE NECROTIZING ULCERATIVE GINGIVITIS

Necrotizing ulcerative gingivitis (NUG) results from an impaired host response to a potentially pathogenic microflora. Depending on the degree of immunosuppression, NUG may occur in a mouth essentially free of other gingival involvement or may be superimposed on underlying chronic gingival disease. Treatment should include the alleviation of the acute symptoms and the correction of the underlying chronic gingival disease. The former is the simpler part of the treatment, whereas the latter requires more comprehensive procedures.

The treatment of NUG consists of (1) alleviation of the acute inflammation by reducing the microbial load and removal of necrotic tissue, (2) treatment of chronic disease either underlying the acute involvement or elsewhere in the oral cavity, (3) alleviation of generalized symptoms such as fever and malaise, and (4) correction of systemic conditions or factors that contribute to the initiation or progression of the gingival changes. Chapter 19 provides further information on the management and treatment of NUG in patients with acquired immunodeficiency syndrome (AIDS).

Treatment of NUG should follow an orderly sequence, according to specific steps at three clinical visits.

First Visit

At the first visit, the clinician should do a complete evaluation of the patient, including a comprehensive medical history with special attention to recent illness, living conditions, dietary background, cigarette smoking, type of employment, hours of rest, risk factors for human immunodeficiency virus (HIV), and psychosocial parameters (e.g., stress, depression). The patient is questioned regarding the history of the acute disease and its onset and duration, as follows:

- Is the disease recurrent?
- Are the recurrences associated with specific factors such as menstruation, particular foods, exhaustion, or mental stress?
- Has there been any previous treatment? When and for how long?

The clinician should also inquire as to the type of treatment received and the patient's impression regarding its effect.

The examination of the patient should include general appearance, presence of halitosis, presence of skin lesions, vital signs including temperature, and palpation for the presence of enlarged lymph nodes, especially submaxillary and submental nodes.

The oral cavity is examined for the characteristic lesion of NUG (see Chapters 10 and 17), its distribution, and the possible involvement of the oropharyngeal region. Oral hygiene is evaluated, with special attention to the presence of pericoronal flaps, periodontal pockets, and local factors (e.g., poor restorations, distribution of calculus). Periodontal probing of NUG lesions is likely to be very painful and may need to be deferred until after the acute lesions are resolved.

The goals of initial therapy are to reduce the microbial load and remove necrotic tissue to the degree that repair and regeneration of normal tissue barriers are reestablished. Treatment during this initial visit is confined to the acutely involved areas, which are isolated with cotton rolls and dried. A topical anesthetic is applied, and after 2 or 3 minutes the areas are gently swabbed with a moistened cotton pellet to remove the pseudomembrane and nonattached surface debris. Bleeding may be profuse. Each cotton pellet is used in a small area, then discarded; sweeping motions over large areas with a single pellet are not recommended. After the area is cleansed

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with warm water, the superficial calculus is removed. Ultrasonic scalers are very useful for this purpose because they do not elicit pain, and the water jet and cavitation aid in lavage of the area.

Subgingival scaling and curettage are contraindicated at this time because these procedures may extend the infection into the deeper tissues and may also cause bacteremia. *Unless an emergency exists, procedures, such as extractions or periodontal surgery, are postponed until the patient has been symptom free for 4 weeks, to minimize the likelihood of exacerbating the acute symptoms.*

Patients with moderate or severe NUG and local lymphadenopathy or other systemic signs or symptoms are placed on an antibiotic regimen of amoxicillin, 500 mg orally every 6 hours for 10 days. For amoxicillin-sensitive patients, other antibiotics are prescribed, such as erythromycin (500 mg every 6 hours) or metronidazole (500 mg twice daily for 7 days). Systemic complications should subside in 1 to 3 days. *Antibiotics are not recommended in NUG patients who do not have systemic complications.*

Instructions to the Patient. The patient is discharged with the following instructions:

1. Avoid tobacco, alcohol, and condiments.
2. Rinse with a glassful of an equal mixture of 3% hydrogen peroxide and warm water every 2 hours and/or twice daily with 0.12% chlorhexidine solution.
3. Get adequate rest. Pursue usual activities, but avoid excessive physical exertion or prolonged exposure to the sun, as in golf, tennis, swimming, or sunbathing.
4. Confine toothbrushing to the removal of surface debris with a bland dentifrice and an ultrasoft brush; overzealous brushing and the use of dental floss or interdental cleaners will be painful. Chlorhexidine mouth rinses are also helpful in controlling plaque throughout the mouth.
5. An analgesic, such as a nonsteroidal antiinflammatory drug (NSAID; e.g., ibuprofen), is appropriate for pain relief.
6. Patients who have systemic complications, such as high fever, malaise, anorexia, and general debility and who have been put on an antibiotic treatment are told that bed rest is necessary, as well as copious fluid consumption and administration of analgesics for relief of pain.

Patients are asked to report back to the clinician in 1 to 2 days. The patient should be advised of the extent of total treatment that the condition requires and warned that treatment is not complete when pain stops. The patient should be informed of the presence of chronic gingival or periodontal disease, which must be eliminated to reduce the likelihood of recurrence of the acute symptoms.

A large variety of drugs have been used in the treatment of NUG.[3] Topical drug therapy, however, is only an adjunctive measure; no drug, when used alone, can be considered complete therapy. Systemic antibiotics, when used, also reduce the oral bacterial flora and alleviate the oral symptoms,[11,12] but they are only an adjunct to the complete local treatment that the disease requires. In patients treated by drugs alone or by systemic antibiotics alone the acute painful symptoms often recur after treatment is discontinued.

Second Visit

At the second visit, 1 or 2 days after the first visit, the patient is evaluated for amelioration of signs and symptoms. The patient's condition is usually improved; the pain is diminished or no longer present. The gingival margins of the involved areas are erythematous but without a superficial pseudomembrane.

Scaling is performed if necessary and sensitivity permits. Shrinkage of the gingiva may expose previously covered calculus, which is gently removed. The instructions to the patient are the same as those given previously.

Third Visit

At the next visit, approximately 5 days after the second visit, the patient is evaluated for resolution of symptoms, and a comprehensive plan for the management of the patient's periodontal conditions is formulated. The patient should be essentially symptom free. Some erythema may still be present in the involved areas, and the gingiva may be slightly painful on tactile stimulation (Figure 41-1, *A*). The patient is instructed in plaque control procedures (see Chapter 44), which are essential for the success of the treatment and the maintenance of periodontal health. The patient is further counseled on nutrition, smoking cessation, and other conditions or habits associated with a potential recurrence. The hydrogen peroxide rinses are discontinued, but chlorhexidine rinses can be maintained for 2 or 3 weeks. Scaling and root planing are repeated if necessary. Unfortunately, the patient often discontinues treatment because the acute condition has subsided; however, this is when comprehensive treatment of the patient's chronic periodontal problem should start.

Appointments should be scheduled for the treatment of chronic gingivitis, periodontal pockets, and pericoronal flaps, as well as for the elimination of all forms of local irritation. The patient should be reevaluated at 1 month to determine compliance with oral hygiene, health habits, psychosocial factors, the potential need for reconstructive or esthetic surgery, and the interval of subsequent recall visits.

Gingival Changes with Healing

The characteristic lesion of NUG undergoes the following changes in the course of healing in response to treatment:

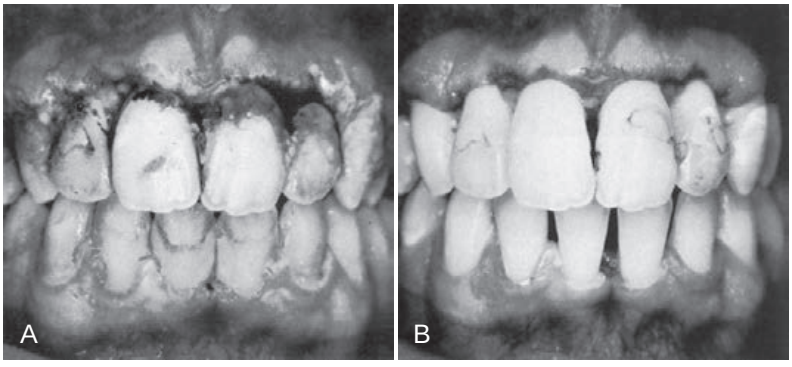

Figure 41-1 Initial response to treatment of patient with acute necrotizing ulcerative gingivitis (NUG). **A,** Severe acute NUG. **B,** Third day after treatment. Patient still has some erythema, but the condition is greatly improved.

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1. Removal of the surface pseudomembrane exposes the underlying red, hemorrhagic, craterlike depressions in the gingiva, indicating inflammation caused by necrosis and microbial infiltration of tissue that has lost the normal barrier function of the epithelium.
2. In the next stage, the bulk and redness of the crater margins are reduced, indicating a reduction in inflammation and re-epithelialization, but the surface remains shiny (Figure 41-1, *B*).
3. This is followed by the early signs of restoration of normal gingival contour and color, indicating reestablishment of the normal barrier function of the epithelium, including keratinization, and further reduction of inflammation.
4. In the final stage, the normal gingival color, consistency, surface texture, and contour may be restored. Portions of the root exposed by the acute disease may be covered by healthy gingiva (Figures 41-2 and 41-3).

Additional Treatment Considerations

Contouring of Gingiva as Adjunctive Procedure. Even in cases of severe gingival necrosis, healing often leads to restoration of the normal gingival contour, although normal architecture of the gingiva may be achieved only after several weeks or months (Figure 41-4). However, if there has been loss of interdental bone, if the teeth are irregularly aligned, or if the entire papilla is lost, healing sometimes results in the formation of a shelf-like gingival margin, which favors retention of plaque and recurrence of gingival inflammation as well as being an esthetic problem. This can be corrected by an attempt to restore lost tissue through periodontal plastic procedures or by reshaping the gingiva

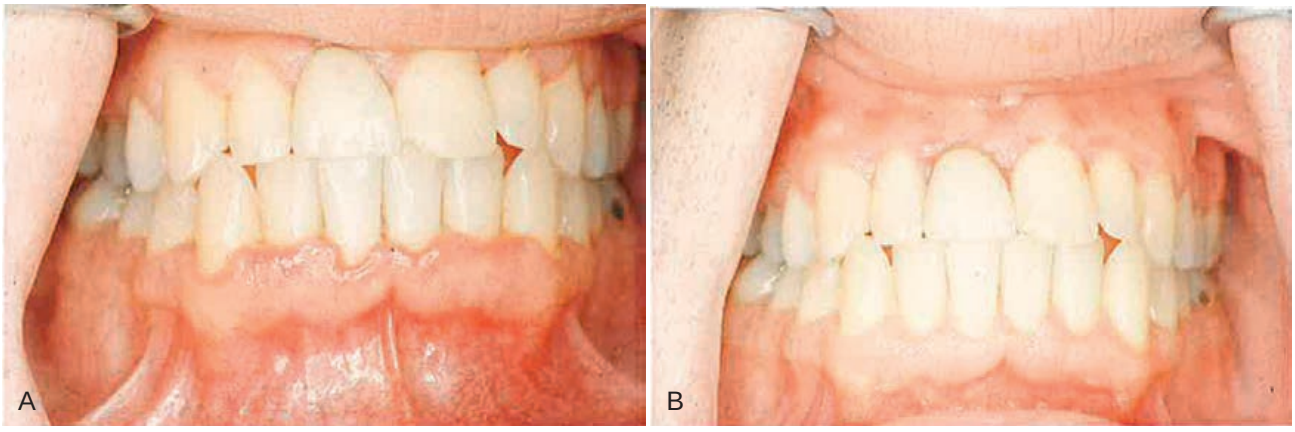

Figure 41-2 Treatment of acute NUG. **A,** Before treatment. Note the characteristic interdental lesions. **B,** After treatment, showing restoration of healthy gingival contour.

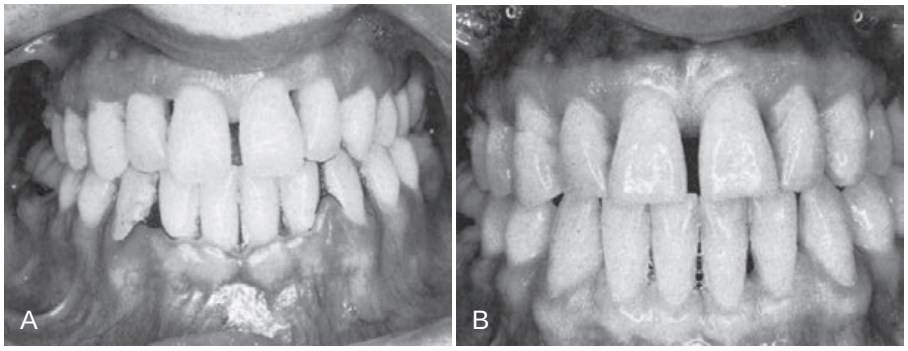

Figure 41-3 Physiologic contour and new attachment of gingiva after treatment of acute NUG. **A,** Acute NUG showing the characteristic punched-out eroded gingival margin with surface pseudomembrane. **B,** After treatment. Note the restoration of physiologic gingival contour and reattachment of the gingiva to the surfaces of the mandibular teeth, which had been exposed by the disease.

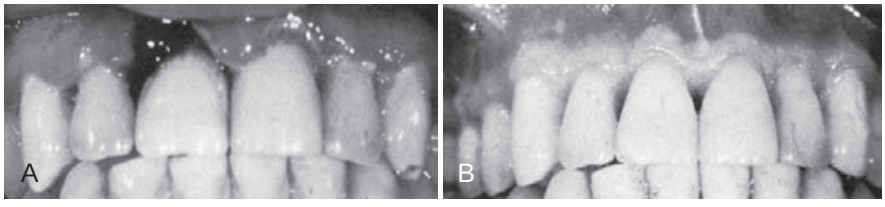

Figure 41-4 Gingival healing after treatment of acute NUG. **A,** Before treatment. Severe acute NUG with crater formation. **B,** After treatment. Note the restored gingival contour.

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Figure 41-5 Reshaping the gingiva in the treatment of acute NUG. **A,** Before treatment, showing bulbous gingiva and interdental necrosis in the mandibular anterior area. **B,** After treatment. Gingival contours still undesirable. **C,** Final result. Physiologic contours obtained by reshaping the gingiva.

surgically (Figure 41-5). Effective plaque control by the patient is particularly important to establish and maintain the normal gingival contour in areas of tooth irregularity.

Role of Drugs. A large variety of drugs have been used for topical treatment of NUG. Topical drug therapy is only an adjunctive measure. *No drug, when used alone, can be considered complete treatment*.

Escharotic drugs, such as phenol, silver nitrate, chromic acid, or potassium bichromate, should not be used. They are necrotizing agents that alleviate pain by destroying the nerve endings of the ulcerated gingiva. However, they also destroy young cells needed for repair and delay healing. Repeated use of these agents results in loss of gingival tissue that is not restored when the disease subsides.[4]

Persistent or Recurrent Cases

Adequate local therapy with optimal home care will resolve most cases of NUG. If a case of NUG persists despite therapy or if it recurs, the patient should be reevaluated, with a focus on the following factors:

1. *Reassessment of differential diagnosis to rule out diseases that resemble NUG.* Several diseases and conditions may initially present with a similar appearance as NUG, such as desquamative gingivitis. A renewed search for skin lesions and other signs or symptoms should be undertaken, with a biopsy if warranted (see Chapters 12 and 17).
2. *Underlying systemic disease causing immunosuppression.* In particular, HIV infection may frequently present with symptoms of NUG or necrotizing ulcerative periodontitis (NUP). The patient should be reassessed for risk factors and may need counseling about testing for HIV or other suspected underlying systemic diseases (e.g., lymphoproliferative disease). The patient will likely need referral to his or her physician for further evaluation.
3. *Inadequate local therapy.* Too often, treatment is discontinued when the symptoms have subsided, without eliminating the chronic gingival disease and periodontal pockets that remain after the superficial acute condition is relieved. Remaining calculus and other local factors that predispose to gingival inflammation may permit recurrence. Recurrent acute involvement in the mandibular anterior area can be associated with persistent pericoronal inflammation arising from partial eruption and pericoronal inflammation of third molars.[8] The anterior involvement is less likely to recur after the third molar situation is corrected.
4. *Inadequate compliance.* Poor plaque control, heavy use of tobacco, ineffective stress management, and continued malnutrition can also contribute to persistence or recurrence of NUG.

The clinician should evaluate the quality and consistency of plaque control. Further assessment and counseling on tobacco use will also determine the role of tobacco in this patient. If the clinician perceives that psychosocial factors are unresolved and are complicating health behaviors and contributing to immunosuppression, the patient should be referred to the appropriate professional. A reassessment of the patient's nutritional state, with potential dietary analysis or nutritional testing, may be required.[5,7]

ACUTE PERICORONITIS

The treatment of pericoronitis depends on the severity of the inflammation, the systemic complications, and the advisability of retaining the involved tooth. All pericoronal flaps should be viewed with suspicion. Persistent symptom-free pericoronal flaps should be removed as a preventive measure against subsequent acute involvement.

The treatment of acute pericoronitis consists of (1) gently flushing the area with warm water to remove debris and exudate and (2) swabbing with antiseptic after elevating the flap gently from the tooth with a scaler. The underlying debris is removed, and the area is flushed with warm water (Figure 41-6). The occlusion is evaluated to determine if an opposing tooth is occluding with the pericoronal flap. It may be necessary to reduce soft tissue surgically and/or to adjust the opposing tooth to alleviate pain. Antibiotics can be prescribed in severe cases and for patients who may have clinical evidence of diffuse microbial infiltration of the tissue. If the gingival flap is swollen and fluctuant, an incision may be necessary to establish drainage and relieve pressure.

Once the acute symptoms have subsided, the prognosis of the tooth can be evaluated. The decision will be governed by the likelihood of whether the tooth will continue eruption into a functional position or if impaction and the factors predisposing to pericoronitis will persist. Bone loss on the distal surface of the second molar is a concern when third molars are impacted along the distal surface.[2] The problem is significantly greater if the third molars are extracted after the roots are formed or when patients are older (i.e., mid-twenties or later). To reduce the risk of bone loss around second molars, partially or completely impacted third molars should be extracted early in their development.

If the decision is made to retain the tooth, the pericoronal flap is surgically reduced (see Figure 41-6). It is necessary to remove

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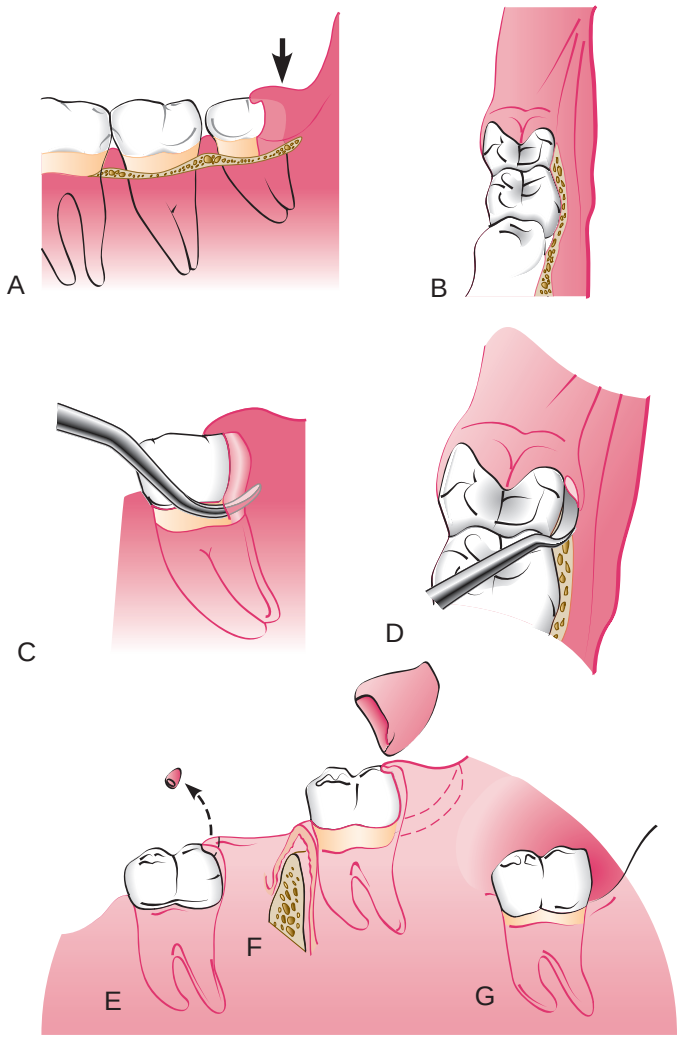

Figure 41-6 Treatment of acute pericoronitis. **A,** Inflamed pericoronal flap *(arrow)* in relation to the mandibular third molar. **B,** Anterior view of third molar and flap. **C,** Lateral view with scaler in position to gently remove debris under flap. **D,** Anterior view of scaler in position. **E,** Incorrect removal of the tip of the flap, permitting the deep pocket to remain distal to the molar. **F,** Removal of section of the gingiva distal to the third molar, after the acute symptoms subsided. The line of incision is indicated by the broken line. **G,** Appearance of the healed area.

the tissue distal to the tooth, as well as the flap on the occlusal surface. Incising only the occlusal portion of the flap leaves a deep distal pocket, which invites recurrence of acute pericoronal involvement. It is critical to leave the patient with a cleansable site. On healing, the patient needs appropriate instruction in long-term maintenance.

ACUTE HERPETIC GINGIVOSTOMATITIS

Primary infection with herpes simplex virus in the oral cavity results in a condition known as acute herpetic gingivostomatitis, which is an oral infection often accompanied by systemic signs and symptoms (see Chapter 10). This infection typically occurs in children, but it can occur in adults as well. It runs a 7- to 10-day course and usually heals without scarring. A recurrent herpetic episode may be precipitated in individuals with a history of herpesvirus infections by dental treatment,[13] respiratory infections, sunlight exposure, fever, trauma, exposure to chemicals, and emotional stress.

Treatment consists of early diagnosis and immediate initiation of antiviral therapy. Until recently, therapy for primary herpetic gingivostomatitis consisted of palliative care. With the development of antiviral therapy, however, the standard of care has changed. in a randomized double-blind placebo-controlled study, Amir et al[1] demonstrated that antiviral therapy with 15 mg/kg of an acyclovir suspension given 5 times daily for 7 days substantially changes the course of the disease without significant side effects. Acyclovir reduced symptoms, including fever, from 3 days to 1 day; decreased new extraoral lesions from 5.5 to 0 days; and reduced difficulty with eating from 7 to 4 days. Furthermore, viral shedding stopped at 1 day for the acyclovir group compared with 5 days for the control group. Overall, oral lesions were present for only 4 days in the acyclovir group but for 10 days in the control group. Although no clear clinical evidence indicates that this regimen will reduce recurrences, research data suggest that a greater number of latent virus copies incorporated into ganglia will increase the severity of recurrences.[1]

In summary, if primary herpetic gingivostomatitis is diagnosed within 3 days of onset, acyclovir suspension should be prescribed: 15 mg/kg 5 times daily for 7 days. If diagnosis occurs after 3 days in an immunocompetent patient, acyclovir therapy may have limited value. All patients, including those presenting more than 3 days after disease onset, may receive palliative care, including removal of plaque and food debris. An NSAID (e.g., ibuprofen) can be given systemically to reduce fever and pain. Patients may use either nutritional supplements or topical anesthetics (e.g.,

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These patients have pain as an important symptom and so need to be treated promptly and need to be contacted day by day to ensure they are comfortable. Necrotizing gingivitis is treated with local root planing, oral hygiene instruction, and oral antimicrobial rinses. Systemic antibiotics are only used if there is evidence of lymphadenopathy or spread beyond the gingiva, except they are always used in immunocompromised patients such as those diagnosed as HIV positive.

It is important to correctly diagnose painful gingiva due to herpetic gingivostomatitis as these patients do not need antimicrobial therapy but instead antiviral medications such as acyclovir are used topically or, in severe cases, systemically. These patients are infectious while they have lesions and so they need to limit close direct contact of their mouth with other people to avoid transmission.

Acute gingival lesions that do not respond to treatment within 2 weeks should be biopsied to secure the correct diagnosis.

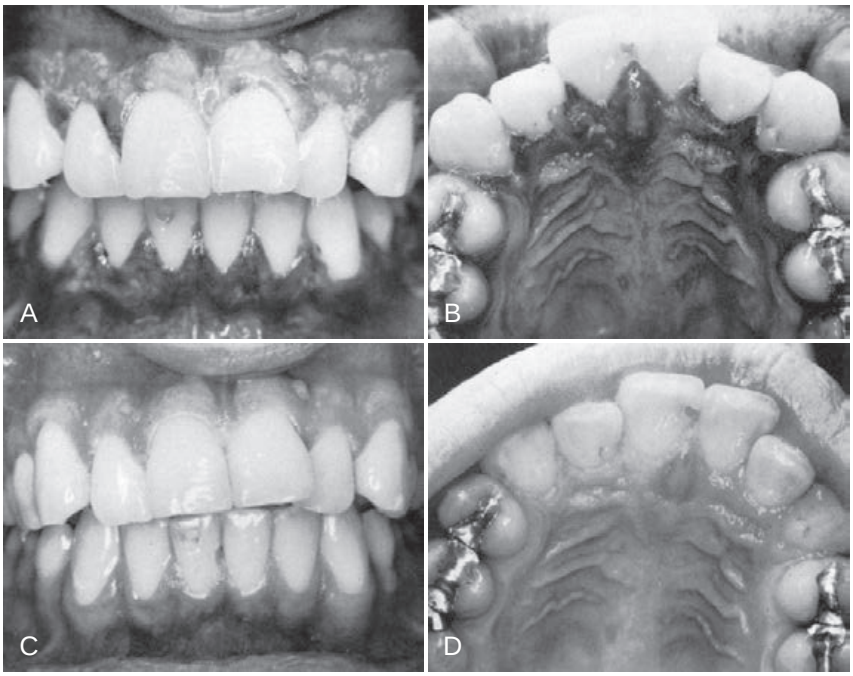

Figure 41-7 Treatment of acute herpetic gingivostomatitis. **A,** Before treatment. Note diffuse erythema and surface vesicles. **B,** Before treatment, palatal view, showing gingival edema and ruptured vesicle on palate. **C,** One month after treatment, showing restoration of normal gingival contour and stippling. **D,** One month after treatment, palatal view.

viscous lidocaine) before eating to aid in proper nutrition during the early phases in acute herpetic gingivostomatitis. Periodontal therapy should be postponed until the acute symptoms subside to avoid the possibility of exacerbation (Figure 41-7).

Local or systemic application of antibiotics is sometimes advised to prevent opportunistic infection of ulcerations, especially in the immunocompromised patient. If the condition does not resolve within 2 weeks, the patient should be referred to a physician for medical consultation.[6] The patient should be informed that herpetic gingivostomatitis is contagious at certain stages such as when vesicles are present (highest viral titer). All individuals exposed to an infected patient should take precautions. Herpetic infection of a clinician's finger, referred to as *herpetic whitlow*, can occur if a seronegative clinician becomes infected with a patient's herpetic lesions.[9,10]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 42

Treatment of Periodontal Abscess

Philip R. Melnick and Henry H. Takei

CHAPTER OUTLINE

CLASSIFICATION OF ABSCESSES
- Periodontal Abscess
- Gingival Abscess
- Pericoronal Abscess
- Acute Versus Chronic Abscess
- Periodontal Versus Pulpal Abscess

SPECIFIC TREATMENT APPROACHES
- Acute Abscess
- Chronic Abscess
- Gingival Abscess
- Pericoronal Abscess

CLASSIFICATION OF ABSCESSES

The periodontal abscess is a localized purulent inflammation of the periodontal tissues.[6] It has been classified into three diagnostic groups: gingival abscess, periodontal abscess, and pericoronal abscess. The *gingival abscess* involves the marginal gingival and interdental tissues. The *periodontal abscess* is an infection located contiguous to the periodontal pocket and may result in destruction of the periodontal ligament and alveolar bone. The *pericoronal abscess* is associated with the crown of a partially erupted tooth.[22]

Periodontal Abscess

The periodontal abscess is typically found in patients with untreated periodontitis and in association with moderate-to-deep periodontal pockets.[5,25] Periodontal abscesses often arise as an acute exacerbation of a preexisting pocket[6] (Figure 42-1). Primarily related to incomplete calculus removal, periodontal abscesses have been linked to a number of clinical situations.[8,15,16,24] They have been identified in patients after periodontal surgery,[12] after preventive maintenance (Figure 42-2),[7,10,17,21] after systemic antibiotic therapy,[26] and as the result of recurrent disease.[15,16] Conditions in which periodontal abscess is not related to inflammatory periodontal disease include tooth perforation or fracture[2,24] (Figure 42-3) and foreign body impaction.[1,23] Poorly controlled diabetes mellitus has been considered a predisposing factor for periodontal abscess formation[22] (Figure 42-4). Formation of periodontal abscess has been reported as a major cause of tooth loss[12-21]; however, with proper treatment followed by consistent preventive periodontal maintenance, teeth with significant bone loss may be retained for many years[7] (see Figure 42-10).

Gingival Abscess

The gingival abscess is a localized, acute inflammatory lesion that may arise from a variety of sources, including microbial plaque infection, trauma, and foreign body impaction.[22] Clinical features include a red, smooth, sometimes painful, often fluctuant swelling (Figure 42-5).

Pericoronal Abscess

The pericoronal abscess results from inflammation of the soft tissue operculum, which covers a partially erupted tooth. This situation is most often observed around the mandibular third molars. As with the gingival abscess, the inflammatory lesion may be caused by the retention of microbial plaque, food impaction, or trauma.

Acute Versus Chronic Abscess

Abscesses are categorized as acute or chronic. The acute abscess is often an exacerbation of a chronic inflammatory periodontal lesion. Influencing factors include increased number and virulence of bacteria present, combined with lowered tissue resistance and lack of spontaneous drainage.[11,25] The drainage may have been prevented by a deep, tortuous pocket morphology, debris, or closely adapted pocket epithelium blocking the pocket orifice. Acute abscesses are characterized by painful, red, edematous, smooth, and ovoid swelling of the gingival tissues.[15,16,25] Exudate may be expressed with gentle pressure; the tooth may be percussion sensitive and feel elevated in the socket (Figure 42-6). Fever and regional lymphadenopathy are occasional findings.[22]

The *chronic abscess* forms after the spreading infection has been controlled by spontaneous drainage, host response, or therapy. Once homeostasis between the host and infection has been reached, the patient may have few or no symptoms.[9] However, dull pain may be associated with the clinical findings of a periodontal pocket, inflammation, and a fistulous tract.[22]

Box 42-1 compares the signs and symptoms of the acute and chronic abscess.

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All patients with abscesses affecting periodontal tissues including gingiva should have a periapical radiograph that tests the vitality of the teeth in the region. This will allow the clinician to clarify if the origin of the abscess is periodontal or pulpal and thus provide the appropriate treatment. All abscesses require drainage, and periodontal abscesses can often be managed by curetting the pocket under local anesthesia to remove plaque or any other etiologic material, such as food debris, resulting in drainage of pus, blood, and edema fluid. If drainage is not obvious, then a gingival incision parallel to the long axis of the teeth may be necessary. Systemic antibiotics are only used if there is evidence of inflammatory spread beyond the gingiva in healthy patients and in immunocompromised patients and diabetics. Occlusal adjustment will often help reduce pain because the affected teeth are often extruded from the inflamed periodontal tissues. Postoperative plaque control is essential.

Appropriate treatment of acute abscesses can in some patients lead to complete resolution of the inflammation and associated bone loss, thus at least 8 weeks should pass before further treatment decisions are made.

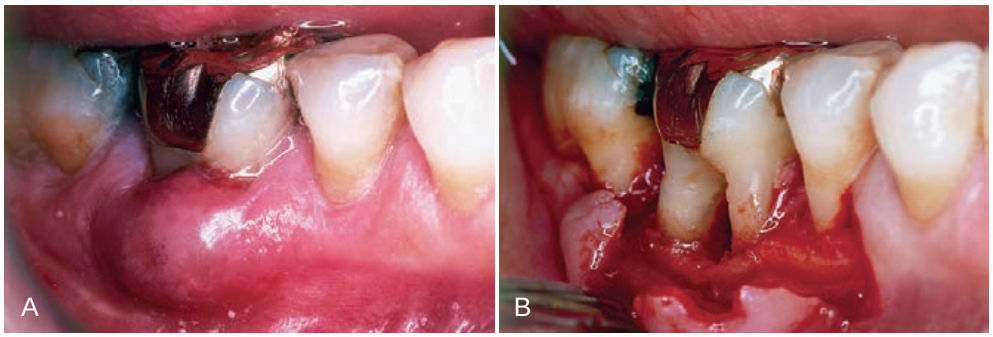

Figure 42-1 **A,** Deep furcation invasions are a common location for the periodontal abscess. **B,** Furcation anatomy often prevents the definitive removal of calculus and microbial plaque.

Figure 42-2 Postprophylaxis periodontal abscess resulting from partial healing of a periodontal pocket over residual calculus.

Periodontal Versus Pulpal Abscess

To determine the cause of an abscess and thus establish a proper treatment plan, it is often necessary to perform a differential diagnosis between a periodontal and pulpal abscess[4] (Box 42-2 and Figures 42-7 and 42-8).

SPECIFIC TREATMENT APPROACHES

Treatment of the periodontal abscess includes two phases: resolving the acute lesion, followed by the management of the resulting chronic condition[24] (Box 42-3).

Acute Abscess

The acute abscess is treated to alleviate symptoms, control the spread of infection, and establish drainage.[19] Before treatment, the patient's medical history, dental history, and systemic condition are reviewed and evaluated to assist in the diagnosis and to determine the need for systemic antibiotics[3] (Boxes 42-4 and 42-5).

BOX 42-1 Signs and Symptoms of Periodontal Abscess

Acute Abscess
Mild to severe discomfort
Localized red, ovoid swelling
Periodontal pocket
Mobility
Tooth elevation in socket
Tenderness to percussion or biting
Exudation
Elevated temperature*
Regional lymphadenopathy*

Chronic Abscess
No pain or dull pain
Localized inflammatory lesion
Slight tooth elevation
Intermittent exudation
Fistulous tract often associated with a deep pocket
Usually without systemic involvement

Data from Dahlen G: *Periodontol 2000* 28:206, 2002; Meng HX: *Ann Periodontol* 4:79, 1999; and Sanz M, Herrera D, van Winkelhoff AJ: The periodontal abscess. In *Clinical periodontology,* Copenhagen, 2000, Munksgaard.
*May indicate the need for systemic antibiotics.

Drainage through Periodontal Pocket. The peripheral area around the abscess is anesthetized with sufficient topical and local anesthetic to ensure comfort. The pocket wall is gently retracted with a periodontal probe or curette in an attempt to initiate drainage through the pocket entrance (see Figure 42-8). Gentle

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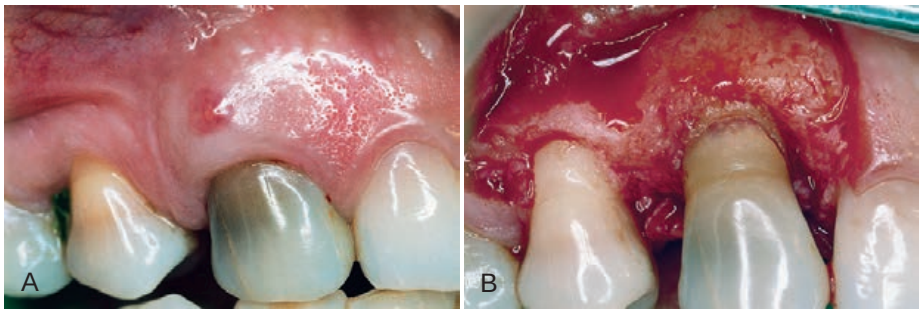

Figure 42-3 A, Fistula is observed in attached gingiva of maxillary right canine. **B,** Elevated flap shows the cause to be a root fracture.

Figure 42-4 Localized periodontal abscess of mandibular right canine of male adult with poorly controlled type 2 diabetes mellitus. For some patients, periodontal abscess formation may be the first sign of the disease.

Figure 42-5 Plaque-associated gingival abscess of mandibular right canine.

Figure 42-6 Patient presenting with acute abscess complained of dull pain and a sensation of tooth elevation in the socket. Signs of tissue distention and exudation are evident.

BOX 42-2 Differential Diagnosis of Periodontal and Pulpal Abscess

Periodontal Abscess

Associated with preexisting periodontal pocket.
Radiographs show periodontal angular bone loss and furcation radiolucency.
Tests show vital pulp.
Swelling usually includes gingival tissue, with occasional fistula.
Pain usually dull and localized.
Sensitivity to percussion may or may not be present.

Pulpal Abscess

Offending tooth may have large restoration.
May have no periodontal pocket, or if present, probes as a narrow defect.
Tests show nonvital pulp.
Swelling often localized to apex, with a fistulous tract.
Pain often severe and difficult to localize.
Sensitivity to percussion.

Modified from Corbet EF: *Periodontol 2000* 34:204, 2004.

BOX 42-3 Treatment Options for Periodontal Abscess

1. Drainage through pocket retraction or incision
2. Scaling and root planing
3. Periodontal surgery
4. Systemic antibiotics
5. Tooth removal

Modified from Sanz M, Herrera D, van Winkelhoff AJ: The periodontal abscess. In *Clinical periodontology,* Copenhagen, 2000, Munksgaard.

BOX 42-4 Indications for Antibiotic Therapy in Patients with Acute Abscess

1. Cellulitis (nonlocalized, spreading infection)
2. Deep, inaccessible pocket
3. Fever
4. Regional lymphadenopathy
5. Immunocompromised patient

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Figure 42-7 **A,** Maxillary right first molar with fistula on the attached gingiva. **B,** Using local anesthesia, periodontal probe is introduced through the fistula and angled toward the root end. **C,** Surgical flap elevation demonstrates failed endodontic therapy and tooth fracture as causing the fistula.

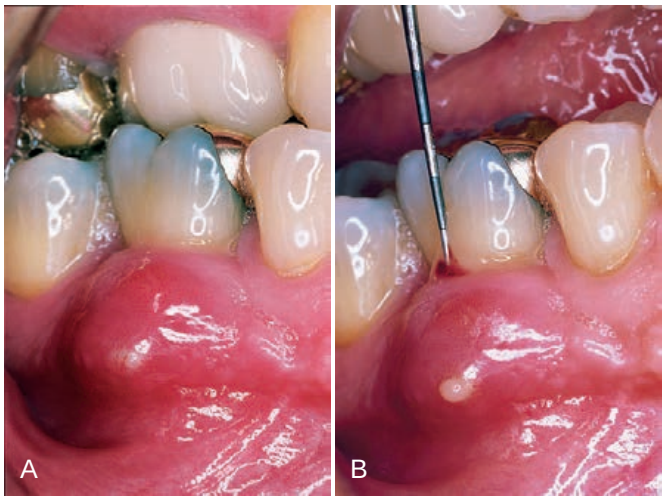

Figure 42-8 **A,** Periodontal abscess of maxillary left first molar. **B,** Periodontal probe is used to retract the pocket wall gently.

Figure 42-9 Gentle digital pressure may be sufficient to express purulent discharge.

digital pressure and irrigation may be used to express exudates and clear the pocket (Figure 42-9). If the lesion is small and access uncomplicated, debridement in the form of scaling and root planing may be undertaken.

If the lesion is large and drainage cannot be established, root debridement by scaling and root planing or surgical access should be delayed until the major clinical signs have abated.[18] In these patients, use of adjunctive systemic antibiotics[13-16] with short-term high-dose regimens is recommended[20] (see Box 42-5). Antibiotic therapy alone without subsequent drainage and subgingival scaling is contraindicated.[14]

BOX 42-5 Antibiotic Options for Periodontal Infections

Antibiotic of Choice

Amoxicillin, 500 mg

- 1.0-g loading dose, then 500 mg 3 times a day for 3 days.
- Reevaluation after 3 days to determine need for continued or adjusted antibiotic therapy.

Penicillin Allergy

Clindamycin

- 600-mg loading dose, then 300 mg 4 times a day for 3 days.
- Azithromycin (or clarithromycin).
- 1.0-g loading dose, then 500 mg 4 times a day for 3 days.

Data from American Academy of Periodontology: *J Periodontol* 67:1553, 2004.

Drainage through External Incision. The abscess is dried and isolated with gauze sponges. Topical anesthetic is applied, followed by local anesthetic injected peripheral to the lesion. A vertical incision through the most fluctuant center of the abscess is made with a #15 surgical blade. The tissue lateral to the incision can be separated with a curette or periosteal elevator. Fluctuant matter is expressed, and the wound edges approximated under light digital pressure with a moist gauze pad.

In abscesses presenting with severe swelling and inflammation, aggressive mechanical instrumentation should be delayed in favor of antibiotic therapy so as to avoid damage to healthy contiguous periodontal tissues.[24]

Once bleeding and suppuration have ceased, the patient may be dismissed. For those who do not need systemic antibiotics, post-treatment instructions include frequent rinsing with warm salt water (1 tbsp/8-oz glass) and periodic application of chlorhexidine gluconate either by rinsing or locally with a cotton-tipped applicator. Reduced exertion and increased fluid intake are often recommended for patients showing systemic involvement. Analgesics may be prescribed for comfort. By the following day, the signs and symptoms have usually subsided. If not, the patient is instructed to continue the previously recommended regimen for an additional 24 hours. This often results in satisfactory healing, and the lesion can be treated as a chronic abscess.[25]

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Figure 42-10 **A,** Chronic periodontal abscess of maxillary right canine. **B,** Using local anesthesia, periodontal probe is inserted to determine severity of the lesion. **C,** Using mesial and distal vertical incisions, a full-thickness flap is elevated, exposing severe bone dehiscence, a subgingival restoration, and root calculus. **D,** Root surface has been planed free of calculus and the restoration smoothed. **E,** Full-thickness flap has been replaced to its original position and sutured with absorbable sutures. **F,** At 3 months, gingival tissues are pink, firm, and well adapted to the tooth, with minimal periodontal probing depth.

Chronic Abscess

As with a periodontal pocket, the chronic abscess is usually treated with scaling and root planing or surgical therapy. Surgical treatment is suggested when deep vertical or furcation defects are encountered that are beyond the therapeutic capabilities of nonsurgical instrumentation (Figure 42-10). The patient should be advised of the possible postoperative sequelae usually associated with periodontal nonsurgical and surgical procedures. As with the acute abscess, antibiotic therapy may be indicated.[25]

Gingival Abscess

Treatment of the gingival abscess is aimed at reversal of the acute phase and when applicable, immediate removal of the cause. To ensure procedural comfort, topical or local anesthesia by infiltration is administered. When possible, scaling and root planing are completed to establish drainage and remove microbial deposits. In more acute situations the fluctuant area is incised with a #15 scalpel blade, and exudate may be expressed by gentle digital pressure. Any foreign material (e.g., dental floss, impression material) is removed. The area is irrigated with warm water and covered with moist gauze under light pressure.

Once bleeding has stopped, the patient is dismissed with instructions to rinse with warm salt water every 2 hours for the remainder of the day. After 24 hours the area is reassessed, and if resolution is sufficient, scaling not previously completed is undertaken. If the residual lesion is large or poorly accessible, surgical access may be required.

Pericoronal Abscess

As with the other abscesses of the periodontium, the treatment of the pericoronal abscess is aimed at management of the acute phase, followed by resolution of the chronic condition. The acute pericoronal abscess is properly anesthetized for comfort, and drainage is established by gently lifting the soft tissue operculum with a periodontal probe or curette. If the underlying debris is easily accessible, it may be removed, followed by gentle irrigation with sterile saline. If there is regional swelling, lymphadenopathy, or systemic signs, systemic antibiotics may be prescribed.

The patient is dismissed with instructions to rinse with warm salt water every 2 hours, and the area is reassessed after 24 hours. If discomfort was one of the original complaints, appropriate analgesics should be employed. Once the acute phase has been controlled, the partially erupted tooth may be definitively treated with either surgical excision of the overlying tissue or removal of the offending tooth.

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CHAPTER 43

Phase I Periodontal Therapy

Dorothy A. Perry and Henry H. Takei

CHAPTER OUTLINE

RATIONALE
TREATMENT SESSIONS
SEQUENCE OF PROCEDURES
RESULTS
HEALING
DECISION TO REFER FOR SPECIALIST TREATMENT

Phase I therapy is the first in the chronologic sequence of procedures that constitute periodontal treatment. The objective is to alter or eliminate the microbial etiology and factors that contribute to gingival and periodontal diseases to the greatest extent possible, therefore halting the progression of disease and returning the dentition to a state of health and comfort.[1] Phase I therapy is referred to by a number of names, including *initial therapy,*[1] *nonsurgical periodontal therapy,*[13] *cause-related therapy,*[9] and the *etiotropic phase of therapy.*[21] All terms refer to the procedures performed to treat gingival and periodontal infections, up to and including tissue reevaluation, which is the point at which the course of ongoing care is determined.

RATIONALE

During phase I, therapy includes the initiation of a comprehensive daily plaque control regimen, thorough removal of calculus and microbial plaque, correction of defective restorations, and treatment of carious lesions.[2,4,8,10,16] These procedures are a required part of periodontal therapy, regardless of the extent of disease present. In many cases, phase I therapy will be the only set of procedures required to restore periodontal health, or they will constitute the preparatory phase for surgical therapy. Figures 43-1 and 43-2 show the results of phase I therapy in two patients with chronic periodontitis.

Phase I therapy is a critical aspect of periodontal treatment. Data from clinical research indicate that the long-term success of periodontal treatment depends predominantly on maintaining the results achieved with phase I therapy and much less on any specific surgical procedures. In addition, phase I therapy provides an opportunity for the dentist to evaluate tissue response and patient motivation about periodontal care, both of which are crucial elements to the overall success of treatment.

Based on the knowledge that microbial plaque is the major etiologic agent in gingival inflammation, one specific aim of phase I therapy for every patient is *effective daily plaque removal* at home. These plaque control procedures can be complex, time consuming, and often require changing long-standing habits. Good oral hygiene is most easily accomplished if the tooth surfaces are free of calculus deposits and other irregularities so that surfaces are easily accessible. Therefore, in addition to teaching periodontal patients adequate home plaque control procedures, management of contributing local factors includes the following therapies, as required:

1. Complete removal of calculus (see Chapters 45 and 46)
2. Correction or replacement of poorly fitting restorations and prosthetic devices (see Chapter 66)
3. Restoration or temporization of carious lesions
4. Orthodontic tooth movement (see Chapter 50)
5. Treatment of food impaction areas
6. Treatment of occlusal trauma (see Chapter 49)
7. Extraction of hopeless teeth
8. Possible use of antimicrobial agents including necessary plaque sampling and sensitivity testing (see Chapter 79)

TREATMENT SESSIONS

After careful analysis and diagnosis of the specific periodontal condition present, the dentist must develop a treatment plan that includes all required procedures and estimates the number of appointments necessary to complete phase I therapy. In most cases, patients require several treatment sessions for the complete debridement of tooth surfaces. Sometimes carious lesions and other contributing factors have to be controlled before an accurate estimate can be made. All of the conditions listed in the following must be considered when determining and developing the phase I treatment plan[13]:

- General health and tolerance of treatment
- Number of teeth present
- Amount of subgingival calculus
- Probing pocket depths and attachment loss
- Furcation involvements
- Alignment of teeth
- Margins of restorations

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Figure 43-1 Results of phase I therapy, severe chronic periodontitis. A, 45-year-old patient with deep probe depths, bone loss, severe swelling, and redness of the gingival tissues. **B,** Results 3 weeks after the completion of phase I therapy. Note that the gingival tissue has returned to a normal contour, with redness and swelling dramatically reduced.

Figure 43-2 Results of phase I therapy, moderate chronic periodontitis. **A,** 52-year-old patient with moderate attachment loss and probe depths in the 4- to 6-mm range. Note that the gingiva appears pink because it is fibrotic. Inflammation is present in the periodontal pockets but disguised by the fibrotic tissue. Bleeding occurs on probing. **B,** Lingual view of the patient with more visible inflammation and heavy calculus deposits. **C** and **D**, 18 months after phase I therapy the same areas show significant improvement in gingival health. The patient returned for regular maintenance visits at 4-month intervals.

- Developmental anomalies
- Physical barriers to access (i.e., limited opening or tendency to gag)
- Patient cooperation and sensitivity (requiring use of anesthesia or analgesia)

SEQUENCE OF PROCEDURES

Step 1: Plaque Control Instruction. Plaque control is the essential component to successful periodontal therapy, and instruction should begin in the first treatment appointment. The patient must learn to correctly brush the teeth, focusing on applying the bristles at the gingival third of the clinical crowns of the teeth, and begin using floss or other aids for interdental cleaning. This is sometimes referred to as "targeted oral hygiene"[18] and emphasizes thorough plaque removal around the periodontal tissues. The multiple appointment approach to phase I therapy permits the dentist to evaluate, reinforce, and improve the patient's oral hygiene skills. (Chapter 44 details plaque control options.)

Step 2. Removal of Supragingival and Subgingival Calculus. Removal of calculus is accomplished using scalers, curettes, ultrasonic instrumentation, or combinations of these devices during one or more appointments. Evidence suggests that the treatment results for chronic periodontitis are similar for all instruments,[6] and some dentists now incorporate laser technology into periodontal therapy, including phase I therapy.[3,15]

Step 3. Recontouring Defective Restorations and Crowns. Corrections for restorative defects, which are plaque traps, may be made by smoothing surfaces and overhangs with burs or hand instruments or by replacing restorations. These procedures can be completed concurrently with other phase I procedures.

Step 4. Management of Carious Lesions. Removal of the carious tissue and placement with either temporary or permanent restorations is indicated in phase I therapy because of the infectious nature of the caries process. Healing of the periodontal tissues will be maximized by removing the reservoir of bacteria in these lesions so that they cannot repopulate the microbial plaque.

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Figure 43-3 Effects of overhanging amalgam margin on interproximal gingiva of maxillary first molar in otherwise healthy mouth. **A,** Clinical appearance of rough, irregular, and overcontoured amalgam. **B,** Gentle probing of interproximal pocket. **C,** Extensive bleeding elicited by gentle probing indicating severe inflammation in the area.

Step 5. Tissue Reevaluation. After scaling, root planing, and other phase I procedures, the periodontal tissues require approximately 4 weeks to heal sufficiently to be probed accurately. Patients also need the opportunity to improve their plaque control skills to both reduce inflammation and adopt new habits. At the reevaluation appointment, periodontal tissues are probed, and all related anatomic conditions are carefully evaluated to determine if further treatment, including periodontal surgery, is indicated. Additional improvement from periodontal surgical procedures can be expected only if phase I therapy resulted in the gingiva being free of overt inflammation and the patient has learned effective daily plaque control.

RESULTS

Scaling and root planing therapy has been studied extensively to evaluate its effects on periodontal disease. Countless studies indicated that this treatment is both effective and reliable. Studies ranging from 1 month to 2 years in length demonstrated up to 80% reduction in bleeding on probing and mean probing depth reductions of 2 to 3 mm. Others demonstrated that the percentage of periodontal pockets of 4-mm or greater depth was reduced more than 50% and up to 80%.[4] Figures 43-1 and 43-2 show examples of the effectiveness of phase I therapy.

The control of infectious organisms during phase I treatment is of critical importance. Recently, there has been considerable interest in providing phase I therapy in one long appointment or two appointments on consecutive days while the patient is receiving an aggressive prescribed regimen of antimicrobial agents rather than staged appointments to treat one quadrant or sextant at a time, usually with 1 week separating appointments. This single-stage treatment sequence has been referred to as "antiinfective" or "disinfection" treatment.[11,14] Recent studies suggested both the one appointment and staged or multiple appointment treatment strategies work well. The modest differences in clinical parameters in comparing healing after one session or multiple sessions were not clinically significant.[7,17] In addition, microbial parameters were not significantly different after 8 months, regardless of treatment modality[17] and the risk of recurrence of periodontal pockets was no greater for either modality.[20] Until evidence indicates otherwise, the sequence and duration of phase I therapy appointments should be determined based on amount of disease present and patient comfort.[5] Staged therapy permits the advantage of evaluating and reinforcing oral hygiene care, and the one or two appointment therapies can be more efficient in reducing the number of office visits the patient is required to attend.

Additional individual treatments, such as caries control and correction of poorly fitting restorations, clearly augment the healing gained through good plaque control and scaling and root planing by making tooth surfaces accessible to cleaning procedures. Figure 43-3 demonstrates the effects of an overhanging amalgam restoration on gingival inflammation in an otherwise healthy periodontium. Maximal healing from phase I treatment is not possible when local conditions retain plaque and provide reservoirs for repopulation of periodontal pathogens.

HEALING

Healing of the gingival epithelium consists of the formation of a long junctional epithelium rather than new connective tissue attachment to the root surfaces. The attachment epithelium reappears 1 to 2 weeks after therapy. Gradual reductions in inflammatory cell population, crevicular fluid flow, and repair of connective tissue result in decreased clinical signs of inflammation, including less redness and swelling. One or two millimeters of recession is often apparent as the result of tissue shrinkage.[4]

Transient root sensitivity frequently accompanies the healing process. Although evidence suggests that relatively few teeth in a few patients become highly sensitive, this development is common and can be disconcerting to patients. The extent of the sensitivity can be diminished through good plaque removal.[19] Warning patients about these potential outcomes, the teeth appearing longer because of shrinkage of periodontal tissues and tooth root sensitivity, at the beginning of the treatment sequence will avoid surprise if these changes occur. Unexpected and possibly uncomfortable consequences to treatment may result in distrust and loss of motivation to continue therapy.

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SCIENCE TRANSFER

The major goal of phase I therapy is to control the factors responsible for the periodontal inflammation; the removal of subgingival bacterial deposits and the subsequent control of plaque levels by patients are particularly significant. Phase I therapy should be comprehensive and include scaling, root planing, and oral hygiene instruction, as well as other therapies such as caries control, replacement of defective restorations, occlusal therapy, orthodontic movement, and smoking cessation. Comprehensive reevaluation after phase I therapy is essential to validate treatment options and to estimate prognosis. Many patients can have their periodontal disease controlled with phase I therapy and not require further surgical intervention. In patients who do need surgical treatment, phase I therapy is advantageous in that it also provides tissue with reduced inflammatory infiltration, thus improving the surgical management of the tissue and improving the healing response.

For patients with 5 mm or more of attachment loss and with pockets present after phase I treatment, surgical treatment should be planned. Advanced cases may be best treated by periodontal specialists.

Those patients who do not demonstrate the ability to have 20% or less of tooth surfaces free of plaque are poor candidates for successful surgical outcomes and should be closely monitored on a recall maintenance program until plaque control is established.

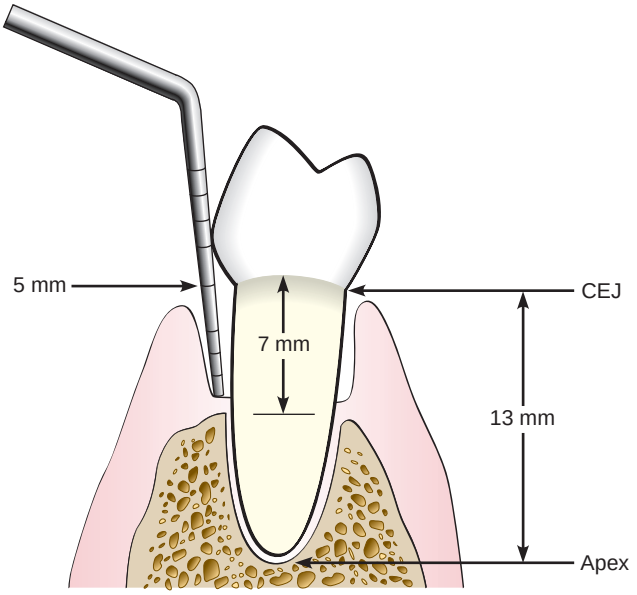

Figure 43-4 The 5-mm standard for referral to a periodontist is based on root length, probing depth, and clinical attachment loss. The standard serves as a reasonable guideline to analyze the case for referral for specialist care. *CEJ,* Cementoenamel junction. *(Redrawn with permission from Armitage G, editor:* Periodontal maintenance therapy, *Berkeley, Calif, 1974, Praxis.)*

DECISION TO REFER FOR SPECIALIST TREATMENT

Often, periodontal conditions heal sufficiently well after phase I therapy that no further treatment is required beyond routine maintenance, making treatment of most periodontal patients the responsibility of the general dentist. However, advanced or complicated cases benefit from specialist care. It is critical to be skilled in determining which patients would benefit from specialist care and should be referred.[12,16]

The *5-mm standard* has been commonly used as a guideline for identifying candidates for referral and relates to the presence of 5 mm or more of clinical attachment loss present at the reevaluation appointment. The rationale behind the 5-mm standard is that the typical root length is about 13 mm and the crest of the alveolar bone is at a level approximately 2 mm apical to the bottom of the pocket. When there is 5 mm of clinical attachment loss the crest of bone is about 7 mm apical to the cementoenamel junction, therefore only about half the bony support for the tooth remains. Specialist care can help preserve teeth in these cases by eliminating deep pockets and regenerating support for the tooth. Figure 43-4 depicts the relationship of clinical attachment loss to tooth support. In terms of probing depths, the treatment of periodontal diseases is generally successful in patients with 6- to 8-mm probe depths. Success rates diminish when probing depths are 9 mm or greater, so early referral of advanced cases is likely to provide the best results.

In addition to the 5-mm standard and evaluation of probe depths, the following factors must also be considered in the decision to refer:

1. *Extent of disease and generalized or localized deep involvement.* Extensive bone loss, even in localized areas suggest the need for specialized reconstructive techniques.
2. *Root length.* Short roots are more seriously jeopardized by 5 mm of clinical attachment loss than long roots.
3. *Hypermobility.* Excessive tooth mobility suggests contributing factors and a more guarded prognosis.
4. *Difficulty of scaling and root planing.* The presence of deep pockets and furcations makes instrumentation much more difficult and results can often be improved with surgical access.
5. *Restorability and importance of particular teeth for reconstruction.* Long-term prognosis of each tooth is important when considering extensive restorative work.
6. *Age of the patient.* Younger patients with extensive attachment loss are more likely to have aggressive forms of disease that require extensive therapy.
7. *Lack of resolution of inflammation after scaling and planing.* If inflammation persists, further therapy is often necessary to gain the most positive results.

Every patient is unique, and the decision process for each patient is complex. The considerations presented in this chapter should provide guidance understanding the significance of phase I therapy and in making referral decisions.

References and Suggested Reading can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 44

Plaque Control for the Periodontal Patient

Dorothy A. Perry

CHAPTER OUTLINE

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SUMMARY

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Plaque control is the regular removal of microbial plaque and the prevention of its accumulation on the teeth and adjacent gingival surfaces. Microbial plaque is the major etiology of periodontal diseases and is related to dental caries; therefore gaining patient cooperation in daily plaque removal is critical to long-term success of all periodontal and dental treatment.[4,5]

Microbial plaque control is an effective way of treating and preventing gingivitis and is a critical part of all the procedures involved in the treatment and prevention of periodontal diseases.[52] In 1965, Löe et al[92] conducted the classic study demonstrating the relationship between microbial plaque accumulation and the development of experimental gingivitis in humans. Subjects in the study stopped brushing and other plaque control procedures, resulting in the development of gingivitis in every person within 7 to 21 days. The composition of the plaque bacteria also shifted so that gram-negative organisms predominated, and these changes were shown to be reversible. Daily removal of microbial plaque led to complete resolution of gingival inflammation for all subjects within 1 week. Good supragingival plaque control has also been shown to affect the growth and composition of subgingival plaque, so that it favors a healthier microflora and reduces calculus formation.[122] Carefully performed daily home plaque control, combined with frequent professionally delivered plaque and calculus removal, reduces the amount of supragingival plaque; decreases the total number of microorganisms in moderately deep pockets, including furcation areas; and greatly reduces the quantity of periodontal pathogens.[32,63]

Microbial plaque growth occurs within hours, and it must be completely removed at least once every 48 hours in the experimental setting with periodontally healthy subjects to prevent inflammation.[121] The American Dental Association (ADA) recommends that individuals brush twice per day and use floss or other interdental cleaners once per day to effectively remove microbial plaque and prevent gingivitis.[9] They recommendation twice daily

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SCIENCE TRANSFER

Plaque control is the most essential element of successful periodontal therapy for all patients. Toothbrushing is the one technique that all patients need. Many patients can get effective plaque control using hand brushes, but powered toothbrushes offer improvements for patients that have reduced dexterity or who need a short-term motivational nudge to upgrade their time spent in oral hygiene. Soft nylon bristled toothbrushes directed at the gingival margin and used without a scrubbing motion are the most effective.

Antimicrobial mouthwashes, such as chlorhexidine, essential oil containing mixtures, and cetylpyridinium, can add to the mechanical plaque removal of brushes. In some patients, the combination of toothbrushing with an antimicrobial mouthwash can be as effective as brushing and flossing in removing interdental plaque. Patients with open embrasures can utilize interdental brushes because these are easier to use and more effective than floss in these cases.

brushing because most individuals do not adequately remove microbial plaque at one brushing and doing it a second time improves the results.

Periodontal lesions are predominantly found in interdental locations, so toothbrushing alone is not sufficient to control gingival and periodontal diseases.[78] It has been demonstrated in healthy subjects that plaque formation begins on the interproximal surfaces where the toothbrush does not reach. Masses of plaque first develop in the molar and premolar areas, followed by the proximal surfaces of the anterior teeth and the facial surfaces of the molars and premolars. Lingual surfaces accumulate the least amount of plaque. Patients consistently leave more plaque on the posterior teeth than the anterior teeth, with interproximal surfaces retaining the highest amounts of plaque, exactly the places in which periodontal infections begin.[121] In addition, periodontal patients have increased susceptibility to disease,[125] complex defects in gingival architecture, and long exposed root surfaces to clean, compounding the difficulty of doing a thorough job.

Chemical inhibitors of plaque and calculus that are incorporated in mouthwashes or dentifrices also play important roles in controling microbial plaque.[8] Also fluorides delivered through toothpastes and mouthrinses are essential for caries control.[10] Many products are available as adjunctive agents to mechanical techniques. These medicaments, as with any drug, should be recommended and prescribed according to the needs of individual patients.

Plaque control is one of the key elements of the practice of dentistry. It permits each patient to assume responsibility for oral health on a daily basis. Without it, optimal oral health through periodontal treatment cannot be attained or preserved.

THE TOOTHBRUSH

Toothbrushes vary in size and design as well as in length, hardness, and arrangement of the bristles[116] (Figure 44-1). Some toothbrush manufacturers claim superiority of design for such factors as minor modifications of bristle placement, length, and stiffness. These claims are primarily based on plaque removal shown to be significantly superior to other toothbrushes in clinical studies. However, the research does not show significant differences in gingivitis scores or bleeding indices, which are the more important measures of improved gingival health. It is questionable whether slight differences in microbial plaque removal are clinically significant because no toothbrush can remove all microbial plaque. In fact, at least one study compared four commercially available toothbrushes for total plaque removal at a single brushing; all four toothbrushes removed plaque equally, and the authors concluded that no one design was superior to others.[28]

When recommending a particular toothbrush, ease of use by the patient and the perception that the brush works well are important considerations. The effectiveness of and potential injury from different types of brushes depend to a great degree on how the brushes are used. Data from in vitro studies of abrasion by different manual toothbrushes suggest that brush designs permitting the bristles to carry more toothpaste while brushing contribute to abrasion more than brush bristles themselves.[35] However, all agree that use of hard toothbrushes, vigorous horizontal brushing, and use of extremely abrasive dentifrices may lead to cervical abrasions of teeth and recession of gingiva.[79]

Toothbrush Design

Toothbrush bristles are grouped in tufts that are usually arranged in three or four rows. Rounded bristle ends cause fewer scratches on the gingiva than flat-cut bristles with sharp ends[31,116] (see Figure 44-1). Two types of bristle material are used in toothbrushes: natural bristles from hogs and artificial filaments made of nylon. Both remove microbial plaque, but nylon bristle brushes vastly predominate in the market.[20] Bristle hardness is proportional to the square of the diameter and inversely proportional to the square of bristle length.[58] Diameters of common bristles range from 0.007 inch (0.2 mm) for soft brushes to 0.012 inch (0.3 mm) for medium brushes and 0.014 inch (0.4 mm) for hard brushes.[65] Soft bristle brushes of the type described by Bass[16] have gained wide acceptance. Preference for handle characteristics is entirely a matter of taste.

Opinions regarding the merits of hard and soft bristles are based on studies that are not comparable, often inconclusive, and contradict one another.[66] Softer bristles are more flexible, clean slightly below the gingival margin when used with a sulcular brushing technique,[17] and reach farther onto the proximal surfaces.[47] Use of hard-bristled toothbrushes is associated with more gingival recession, and frequent brushers who use hard bristles have more recession than those who use soft bristles.[77] However, the manner in which a brush is used and the abrasiveness of the dentifrice affect the abrasion to a greater degree than the bristle hardness itself.[1,96] Bristle hardness does not significantly affect wear on enamel surfaces.[109]

The amount of force used to brush is not critical for effective plaque removal.[127] Vigorous brushing is not necessary and can lead to gingival recession, wedge-shaped defects in the cervical area of root surfaces,[46,110] and painful ulceration of the gingiva.[104]

Toothbrushes must also be replaced periodically, although the amount of visible bristle wear does not appear to affect plaque

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Figure 44-1 Manual toothbrushes. **A,** Toothbrushes from the nineteenth and twentieth centuries, one with an ivory handle from about 1890 *(left),* one with a composition handle from the 1950s *(center),* and one with a sterling silver handle from the early twentieth century *(right).* The ivory-handled brush belonged to a dental student who used the handle to practice cutting preparations and filling the "preps" with amalgam or gold foil. **B,** Various types of toothbrushes are available; note the variation in brush head and handle design. **C,** Brush heads, showing various bristle configurations. *(Antique brushes courtesy Dean John D.B. Featherstone, University of California, San Francisco School of Dentistry Historical Collection, San Francisco.)*

removal for up to 9 weeks.[30] The ADA recommends that toothbrushes be replaced every 3 to 4 months.[9]

Recommendations

- Soft, nylon bristle toothbrushes clean effectively when used properly, remain effective for a reasonable time, and tend not to traumatize the gingiva or root surfaces.
- Toothbrushes need to be replaced about every 3 to 4 months.
- If patients perceive a benefit from a particular brush design, they should use it.

POWERED TOOTHBRUSHES

Electrically powered toothbrushes designed to mimic back-and-forth brushing techniques were invented in 1939. Subsequent models featured circular or elliptical motions, and some had combinations of motions. Currently, powered toothbrushes have oscillating and rotating motions (Figure 44-2), and some brushes use low-frequency acoustic energy to enhance cleaning ability. Powered toothbrushes rely primarily on mechanical contact between the bristles and the tooth to remove plaque. The addition of low-frequency acoustic energy generates dynamic fluid

Figure 44-2 Powered toothbrush designs offer options in head shape and size.

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movement and provides cleaning slightly away from the bristle tips.[45] The vibrations have also been shown to interfere with bacterial adherence to oral surfaces. Neither the sonic vibration nor the mechanical motion of powered toothbrushes has been shown to affect bacterial cell viability.[94] Hydrodynamic shear forces created by these brushes disrupt plaque a short distance from the bristle tips, explaining the additional interproximal plaque removal.[70]

Typically, comparison studies of powered toothbrushes, manual toothbrushes, or other powered devices demonstrate slightly improved plaque removal for the device of interest in a short-term clinical trial.[98,108] A recent Cochrane review reported that only mechanical brushes with oscillating and rotating motions reduced microbial plaque 11% and demonstrated 6% greater reduction in gingival bleeding than manual brushing. These improvements were maintained over 3 months. Although long-term benefits have not been established, this particular style of mechanical brush resulted in better microbial plaque and gingivitis reduction in a number of well-controlled studies.[60]

Patient acceptance of powered toothbrushes is good. One study reported that 88.9% of patients introduced to a powered toothbrush would continue to use it.[130] However, patients have also been reported to quit using powered toothbrushes after 5 or 6 months, presumably when the novelty is gone. Powered toothbrushes have been shown to improve oral health for (1) children and adolescents, (2) children with physical or mental disabilities, (3) hospitalized patients, including older adults who need to have their teeth cleaned by caregivers, and (4) patients with fixed orthodontic appliances. Powered brushes have not been shown to provide benefits routinely for patients with rheumatoid arthritis, children who are well-motivated brushers, or patients with chronic periodontitis.[61]

Recommendations

- Powered toothbrushes with oscillating and rotating motions remove plaque and reduce gingival bleeding slightly better than manual toothbrushes.
- Patients who want to use powered toothbrushes should be encouraged to do so.
- Patients need to be instructed in the proper use of powered devices.
- Patients who are poor brushers, children, and caregivers may particularly benefit from using powered toothbrushes.

DENTIFRICES

Dentifrices aid in cleaning and polishing tooth surfaces. They are used mostly in the form of pastes, although tooth powders and gels are also available. Dentifrices are made up of abrasives (e.g., silicon oxides, aluminum oxides, and granular polyvinyl chlorides), water, humectants, soap or detergent, flavoring and sweetening agents, therapeutic agents (e.g., fluorides, pyrophosphates), coloring agents, and preservatives.[59,97] Composing 20% to 40% of dentifrices, abrasives are insoluble inorganic salts that enhance the abrasive action of toothbrushing as much as 40 times.[96] Tooth powders are much more abrasive than pastes and contain about 95% abrasive materials. The abrasive quality of dentifrices affects enamel only slightly and is a much greater concern for patients with exposed roots. Dentin is abraded 25 times faster and cementum 35 times faster than enamel, so root surfaces are easily worn away, leading to notching and tooth sensitivity.[120] Oral hygiene procedures mainly cause hard tissue damage from abrasive dentifrices, although gingival lesions can also be produced[104,109] (Figure 44-3). Dentifrices are very useful for delivering therapeutic agents to the teeth and gingiva. The pronounced caries-preventive effect of *fluorides* incorporated in dentifrices has been proved beyond question.[119] Fluoride ion must be available in the amount of 1000 to 1100 parts per million (ppm) to achieve caries reduction effects. Toothpaste products that have been tested by the ADA and have fluoride ion available in the appropriate amount carry the ADA seal of approval for caries control and can be relied on to provide caries protection.[10]

Figure 44-3 Vigorous toothbrushing with an abrasive dentifrice can result in trauma to the gingiva and wearing away of the tooth surfaces, especially root surfaces, and can contribute to gingival recession.

"Calculus control toothpastes," also referred to as "tartar control toothpastes," contain *pyrophosphates* and have been shown to reduce the deposition of new calculus on teeth. These ingredients interfere with crystal formation in calculus but do not affect the fluoride ion in the paste or increase tooth sensitivity. Dentifrice with pyrophosphates has been shown to reduce the formation of new supragingival calculus by 30% or more.[73,95,135] Pyrophosphate-containing toothpastes do not affect subgingival calculus formation or gingival inflammation. The inhibitory effects reduce the deposition of new supragingival calculus but do not affect existing calculus deposits. To achieve the greatest effect from calculus control toothpaste, the patient's teeth must be cleaned and completely free of supragingival calculus when starting to use the product daily.

Recommendations

- Dentifrices increase the effectiveness of brushing but should cause a minimum of abrasion to root surfaces.
- Products containing fluorides and antimicrobial agents provide additional benefits for controlling caries and gingivitis.
- Patients who form significant amounts of supragingival calculus benefit from the use of a calculus control dentifrice.

TOOTHBRUSHING METHODS

Many methods for brushing the teeth have been described and promoted as being efficient and effective. These methods can be categorized primarily according to the pattern of motion when brushing and are primarily of historic interest, as follows[71]:

Roll: Roll[3] or modified Stillman technique[67]
Vibratory: Stillman,[118] Charters,[25] and Bass[17] techniques
Circular: Fones technique[44]
Vertical: Leonard technique[85]
Horizontal: Scrub technique[132]

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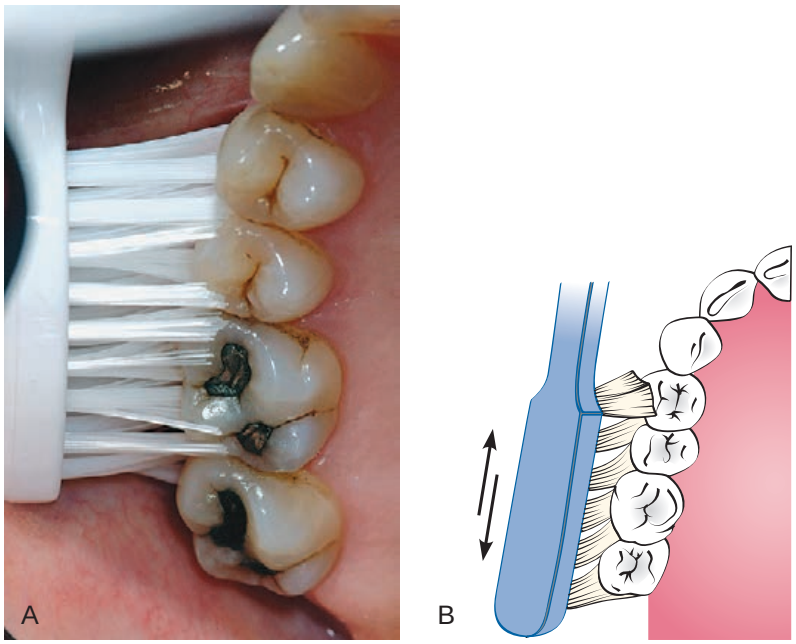

Figure 44-4 Bass method. **A,** Place the toothbrush so that the bristles are angled approximately 45 degrees from the tooth surfaces. **B,** Start at the most distal tooth in the arch, and use a vibrating, back-and-forth motion to brush.

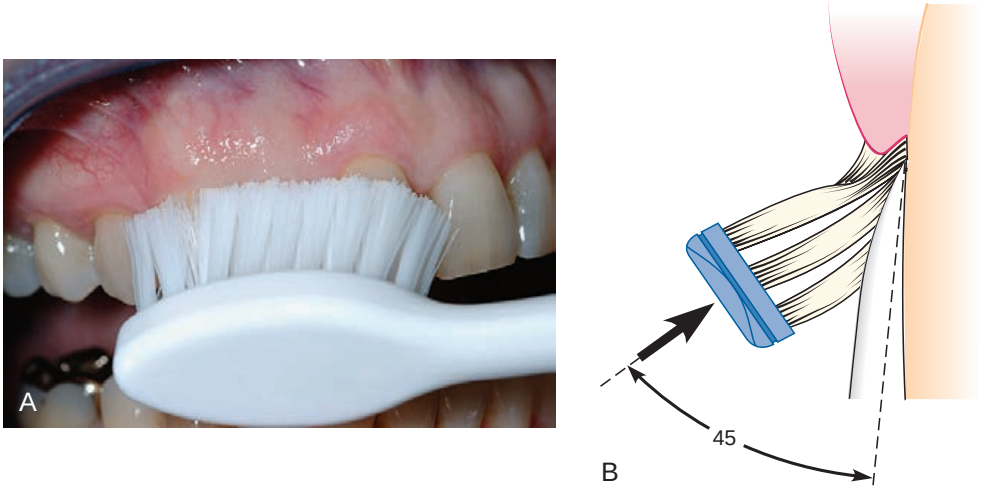

Figure 44-5 Bass method. **A,** Proper position of the brush in the mouth aims the bristle tips toward the gingival margin. **B,** Diagram shows the ideal placement, which permits slight subgingival penetration of the bristle tips.

Patients with periodontal disease are most frequently taught a sulcular brushing technique using a vibratory motion to improve access in the gingival areas. This is referred to as "target hygiene."[123] The method most often recommended is the *Bass technique* because it emphasizes sulcular placement of bristles, adapting the bristle tips to the gingival margin to reach the supragingival plaque and accessing subgingival plaque to the extent possible. A controlled vibrating motion is used to dislodge microbial plaque and avoid trauma. The brush is systematically placed on all the teeth in both arches. Figures 44-4 and 44-5 illustrate this brushing technique.

Bass Technique[17]

1. Place the head of a soft brush parallel with the occlusal plane, with the brush head covering three to four teeth, beginning at the most distal tooth in the arch.
2. Place the bristles at the gingival margin, pointing at a 45-degree angle to the long axis of the teeth.
3. Exert gentle vibratory pressure, using short, back-and-forth motions without dislodging the tips of the bristles. This motion forces the bristle ends into the gingival sulcus area (see Figure 44-4), as well as partly into the interproximal embrasures. The pressure should be firm enough to blanch the gingiva

Brushing with Powered Toothbrushes

The various mechanical motions built into powered toothbrushes do not require special techniques. The patient needs only place the brush head next to the teeth at the gingival margin, using a targeted hygiene approach, and proceed systematically around the dentition.[126] A routine method of brushing all the teeth, similar to the method described for manual brushing, should also be used with powered toothbrushes (Figure 44-6).

Recommendations

- Targeted hygiene[123] focuses brushing efforts on the cervical and interproximal portions of the teeth, where microbial plaque accumulates first.
- Brushing with either a manual or a powered toothbrush requires a systematic routine to clean all the accessible areas.[98]
- Patients will modify any technique to their needs but must achieve the goal of brushing until the teeth are free of plaque.

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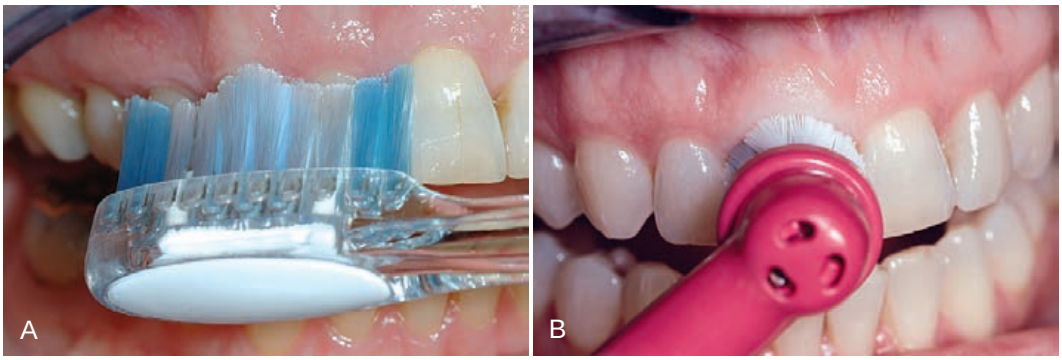

Figure 44-6 Positioning the powered toothbrush head and bristle tips so that they reach the gingival margin is critical to achieving the most effective cleaning results. **A,** Straight-head placement. **B,** Round-head placement.

INTERDENTAL CLEANING AIDS

Any toothbrush, regardless of the brushing method used, does not completely remove interdental plaque. This is true for all brushers, even periodontal patients with wide-open embrasures.[49,112] Daily interdental plaque removal is crucial to augment the effects of toothbrushing because most dental and periodontal diseases originate in interproximal areas.[2]

Tissue destruction associated with periodontal disease often leaves large, open spaces between teeth and long, exposed root surfaces with anatomic concavities and furcations. These areas are both difficult for patients to clean and poorly accessible to the toothbrush.[78] Patients need to understand that the purpose of interdental cleaning is to remove microbial plaque, not just dislodge food wedged between teeth. Many tools are available for interproximal cleaning and they should be recommended based on the size of interdental spaces, presence of furcations, tooth alignment, and presence of orthodontic appliances or fixed prostheses. Also, ease of use and patient cooperation are important considerations. Common aids are dental floss and interdental cleaners such as wooden or plastic tips and interdental brushes.

Dental Floss

Dental floss is the most widely recommended tool for removing plaque from proximal tooth surfaces.[48] Floss is made from nylon filaments or plastic monofilaments, and can be waxed, unwaxed, thick, thin, and even flavored.[7] Some prefer monofilament flosses made of a nonstick material because they are slick and do not fray. Clinical research has demonstrated no significant differences in the ability of the various types of floss to remove dental plaque; they all work equally well.[42,64,75,76] Waxed dental floss was thought to leave a waxy film on proximal surfaces, thus contributing to plaque accumulation and gingivitis. It has been shown, however, that wax is not deposited on tooth surfaces[105] and that improvement in gingival health is unrelated to the type of floss used.[42] Factors influencing the choice of dental floss include the tightness of tooth contacts, roughness of proximal surfaces, and the patient's manual dexterity, not the superiority of any one product. Therefore recommendations about type of floss should be based on ease of use and personal preference.

Technique. The floss must contact the proximal surface from line angle to line angle to clean effectively. It must also clean the entire proximal surface, including accessible subgingival areas, not just be slipped apical into the contact area. The following is a brief description of flossing technique:

Figure 44-7 Dental floss should be held securely in the fingers or tied in a loop.

1. Start with a piece of floss long enough to grasp securely; 12 to 18 inches is usually sufficient. It may be wrapped around the fingers, or the ends may be tied together in a loop.
2. Stretch the floss tightly between the thumb and forefinger (Figure 44-7), or between both forefingers, and pass it gently through each contact area with a firm back-and-forth motion. Do not snap the floss past the contact area because this may injure the interdental gingiva. In fact, zealous snapping of floss through contact areas creates proximal grooves in the gingiva.
3. Once the floss is apical to the contact area between the teeth, wrap the floss around the proximal surface of one tooth, and slip it under the marginal gingiva. Move the floss firmly along the tooth up to the contact area and gently down into the sulcus again, repeating this up-and-down stroke two or three times (Figure 44-8). Then, move the floss across the interdental gingiva, and repeat the procedure on the proximal surface of the adjacent tooth.
4. Continue through the whole dentition, including the distal surface of the last tooth in each quadrant. When the working portion of the floss shreds or becomes dirty, move to a fresh portion of floss.

Flossing can be facilitated by using a *floss holder* (Figure 44-9, *A*). Floss holders are helpful for patients lacking manual dexterity and for caregivers assisting patients in cleaning their teeth. A floss holder should be rigid enough to keep the floss taut when penetrating into tight contact areas, and it should be simple to string with floss. The disadvantage is that floss tools are time-consuming because they must be rethreaded frequently when the floss shreds.

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Disposable, single-use floss holders with prethreaded floss are also available. Short-term clinical studies suggest that plaque reduction and improvement in gingivitis scores are similar for patients using disposable floss devices as for patients who hold the floss with their fingers[23,117] (Figure 44-9, *B*).

Powered flossing devices are also available (Figure 44-10). The devices have been shown to be safe and effective, but no better at plaque removal than holding the floss in the fingers.[29,54]

Establishing a lifelong habit of flossing the teeth is difficult to achieve for both patients and dentists, regardless of whether one uses a tool or flosses with the fingers. In fact, the daily use of floss is universally low. It has been reported that only about 8% of young teenagers in Great Britain floss daily,[93] with similar percentages reported for other countries.[78] No information is available about the establishment of long-term flossing habits comparing the various tools to finger flossing. However, the tools may be useful to help some individuals begin flossing or to make flossing possible if patients have limited dexterity.

Recommendations

- The benefits of interproximal cleaning using dental floss are undisputed.
- Flossing tools work as well as flossing with the traditional method.
- The flossing habit is difficult to establish.

Interdental Cleaning Devices

Concave root surfaces and furcations are often present in periodontal patients who have experienced significant attachment loss and recession, and they are not cleaned well with dental floss. A

Figure 44-8 Dental floss technique. The floss is slipped between the contact area of the teeth (in this case, teeth #7 and #8), is wrapped around the proximal surface, and removes plaque by using several up-and-down strokes. The process must be repeated for the distal surface of tooth #8.

Figure 44-10 Powered flossing devices can be easier for some patients to use than handheld floss. The tip is inserted into the proximal space, and a bristle or wand comes out of the tip and moves in a circular motion when the device is turned on *(left)*. Alternately, the device moves the prestrung floss in short motions to provide interproximal cleaning *(right)*.

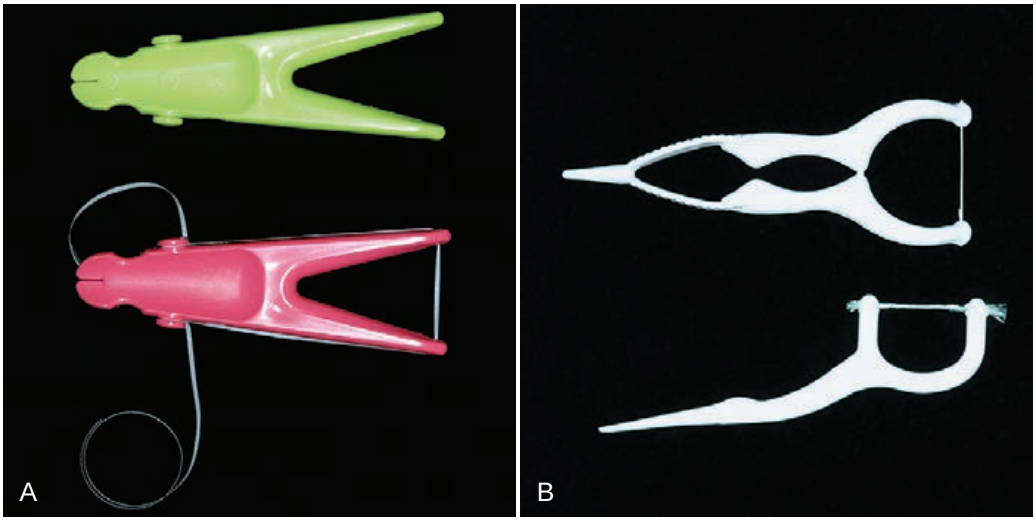

Figure 44-9 Floss holders can simplify the manipulation of dental floss. **A,** Reusable floss tools require stringing the floss around a series of knobs and grooves to secure it. **B,** Disposable floss tools have prestrung floss and are easy to use, but the floss may shred and break, requiring several tools to complete flossing the teeth.

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comparison study of dental floss and interdental brushes used by patients with moderate to severe periodontal disease showed that the interproximal brushes removed slightly more interproximal plaque and that subjects found them easier to use than dental floss. However, no differences were seen in probe depth reductions or bleeding indices.[26] Therefore interproximal cleaning aids, such as interdental brushes, are adequate for proximal cleaning of teeth when interdental spaces permit access.[78]

Embrasure spaces vary greatly in size and shape. Figure 44-11 provides a representation of the size and anatomy of three types of embrasures and the type of interdental cleaner often recommended for each. As a general rule, the larger the space, the larger the device used to clean it should be.

Interdental Brushes. A wide variety of interdental cleaning devices are available for removing microbial plaque from between the teeth (Figure 44-12). The most common types are conical or cylindrical brushes, tapered wooden toothpicks that are round or triangular in cross section, and single-tufted brushes. Many interdental devices have handles for convenient manipulation around the teeth and in posterior areas. Clinical research has shown that the devices are effective on lingual and facial tooth surfaces as well as on proximal surfaces.[78,112,128]

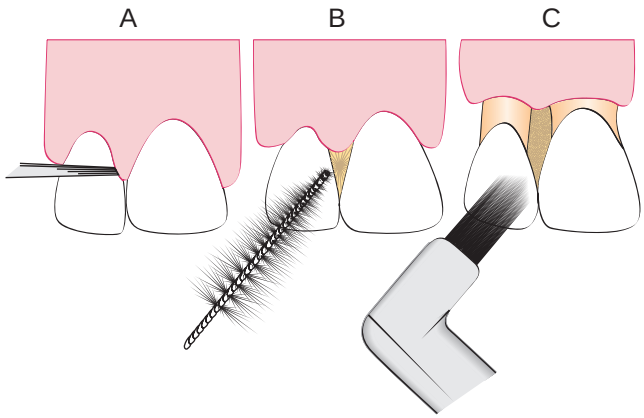

Figure 44-11 Interproximal embrasure spaces vary greatly in patients with periodontal disease. In general, **A,** embrasures with no gingival recession are adequately cleaned using dental floss; **B,** larger spaces with exposed root surfaces require the use of an interproximal brush; and **C,** single-tufted brushes clean efficiently in interproximal spaces with no papillae.

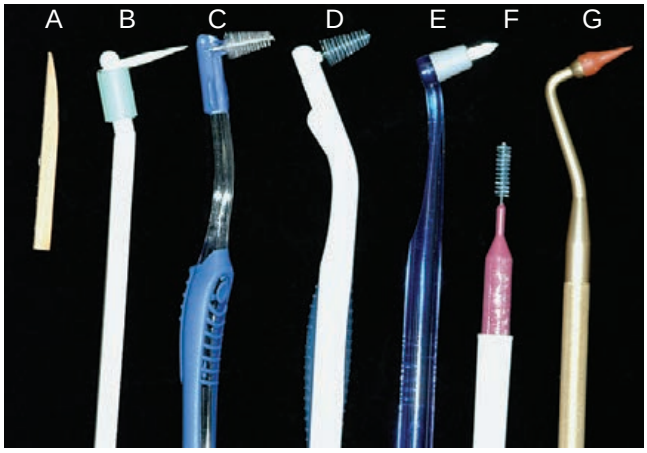

Figure 44-12 Interproximal cleaning devices include wooden tips (**A** and **B**), interproximal brushes (**C** to **F**), and rubber tip stimulators **(G).**

Technique. Interdental brushes of any style are inserted through interproximal spaces and moved back and forth between the teeth with short strokes. For the most efficient cleaning, the diameter of brush should be slightly larger than the gingival embrasures to be cleaned. This size permits bristles to exert pressure on both proximal tooth surfaces, working their way into concavities on the roots.

Single-tufted brushes provide access to furcation areas, or isolated areas of deep recession, and work well on the lingual surfaces of mandibular molars and premolars. These areas are often missed when using a toothbrush.

Wooden or Rubber Tips. Wooden toothpicks are used either with or without a handle (see Figure 44-12, *A* and *B*). Access is easier from the buccal surfaces for tips without handles but is limited primarily to the anterior and bicuspid areas. Wooden toothpicks used on handles improve access to all areas and have been shown to be as effective as dental floss in reducing plaque and bleeding scores in subjects with gingivitis.[87] Triangular wooden tips are also available; this design is most useful in the anterior areas when used from the buccal surfaces of the teeth. Rubber tips are conical and are mounted on handles or the ends of toothbrushes; they can be easily adapted to all proximal surfaces in the mouth. Plastic tips that resemble wooden or rubber tips are also available and are used in the same way. Both rubber and plastic tips can be rinsed and reused and are easily carried in a pocket or purse.

Technique. Toothpicks are common devices and readily available in most homes. They can be used around all surfaces of the teeth when attached to commercially available handles (see Figure 44-12, *B*). Once mounted on the handle, the toothpick is broken off so that it is only 5- or 6-mm long. The tip of the toothpick is used to trace along the gingival margin and into the proximal areas from both the facial and the lingual surface of each tooth (Figure 44-13). Toothpicks mounted on handles are efficient for cleaning along the gingival margin,[48] can penetrate into periodontal pockets and furcations, and permit patients to target their hygiene efforts at the gingival margin.[123]

Soft, triangular wooden picks or plastic alternatives are placed in the interdental space with the base of the triangle resting on the gingiva and the sides in contact with the proximal tooth surfaces (Figure 44-14). The pick is then repeatedly moved in and out of the embrasure to remove plaque. The disadvantage of triangular wooden or plastic tips is that they do not reach well into the posterior areas or on the lingual surfaces.

Rubber tips should be placed into the embrasure space, resting on the gingiva, and used in a circular motion. They can be applied to interproximal spaces and other defects throughout the mouth and are easily adaptable to lingual surfaces.

Recommendations

- Often a toothbrush and dental floss are not sufficient to clean interdental spaces adequately, so it is extremely important to find an interdental device that the patient likes and will use.
- Many interdental cleaning aids are available for patients. The patient might need to try several devices before finding one that is acceptable and cleans adequately.
- In general, the largest brush or device that fits into a space will clean most efficiently (Figure 44-15).

GINGIVAL MASSAGE

Massaging the gingiva with a toothbrush or an interdental cleaning devices produces epithelial thickening, increased keratinization,

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Figure 44-13 Wooden toothpick. **A,** The tip is a common wooden toothpick held in a handle and broken off. It is used to clean subgingivally and reach into periodontal pockets. **B,** The tip can also be used to clean along the gingival margins of the teeth and reach under the gingiva.

Figure 44-14 Triangular wooden tips are also popular with patients. The tip is inserted between the teeth, with the triangular portion resting on the gingival papilla. The tip is moved in and out to remove plaque; however, it is very difficult to use on posterior teeth and from the lingual aspect of all teeth.

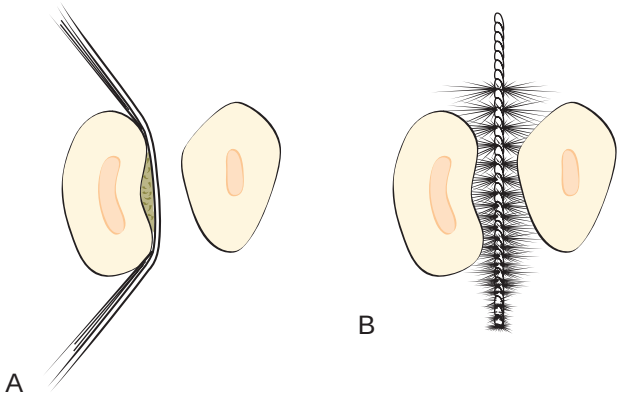

Figure 44-15 Cleaning of concave or irregular proximal tooth surfaces. Dental floss **(A)** may be less effective than an interdental brush **(B)** on long root surfaces with concavities.

and increased mitotic activity in the epithelium and connective tissue.[21,24,51] The increased keratinization occurs only on the oral gingiva and not on the areas more vulnerable to microbial attack: the sulcular epithelium and the interdental areas where the gingival col is present. Epithelial thickening, increased keratinization, and increased blood circulation have not been shown to be beneficial for restoring gingival health.[50] Improved gingival health associated with interdental stimulation is much more likely the result of microbial plaque removal than gingival massage.

For more information on oral irrigation, caries control, chemical plaque control with oral rinses, disclosing agents, frequency of plaque removal, and patient motivation and education and to view Figures 44-16 to 44-18, please visit the companion Expert CONSULT website at www.expertconsult.com.

SUMMARY

- All patients require the regular use of a toothbrush, either manual or electric, at least once per day. The brushing method should emphasize access to the gingival margins of all accessible tooth surfaces, referred to as *targeted hygiene,* and extension as far onto the proximal surfaces as possible.
- Dental floss should be used in all interdental spaces that are filled with gingiva.
- Interdental aids such as interproximal brushes, wooden tips, rubber tips, or toothpicks should be used in all areas where the toothbrush and floss techniques cannot adequately remove the plaque. This includes large embrasure spaces and furcation areas.
- Daily at-home subgingival irrigation is useful for reduction of inflammation and maintenance for patients with residual deep pockets and those who struggle with mechanical interproximal cleaning devices. The effectiveness of irrigation is enhanced by the addition of a chlorhexidine or essential oil rinse to the irrigation water.
- Caries control requires the daily use of a dentifrice with low-concentration fluoride. Topical oral rinses and gels with higher concentrations of fluoride should be used if the patient demonstrates caries risk.
- Chemical antimicrobial agents, such as chlorhexidine and essential oils, can be used to disinfect the patient's mouth and control infection. These oral rinses may be continued indefinitely. Staining of teeth and taste alteration are side effects that may limit the use of these products.
- Reinforcement of daily plaque control practices and routine visits to the dental office for maintenance care are essential to successful microbial plaque control and long-term success of therapy.

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 45

Scaling and Root Planing

Anna M. Pattison and Gordon L. Pattison

CHAPTER OUTLINE

CLASSIFICATION OF PERIODONTAL INSTRUMENTS
- Periodontal Probes
- Explorers
- Scaling and Curettage Instruments
- Cleansing and Polishing Instruments

GENERAL PRINCIPLES OF INSTRUMENTATION (online only)
- Accessibility: Positioning of Patient and Operator
- Visibility, Illumination, and Retraction
- Condition and Sharpness of Instruments
- Maintaining a Clean Field
- Instrument Stabilization
- Instrument Activation
- Instruments for Scaling and Root Planing

PRINCIPLES OF SCALING AND ROOT PLANING (online only)
- Definitions and Rationale
- Detection Skills
- Supragingival Scaling Technique
- Subgingival Scaling and Root-Planing Technique
- Ultrasonic and Sonic Scaling
- Ultrasonic Instruments
- Evaluation

INSTRUMENT SHARPENING (online only)
- Evaluation of Sharpness
- Objective of Sharpening
- Sharpening Stones
- Principles of Sharpening
- Sharpening Individual Instruments

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some text and figures may be out of numeric order in this printed chapter.

Periodontal instruments are designed for specific purposes such as removing calculus, planing root surfaces, curetting the gingiva, and removing diseased tissue. On first investigation, the variety of instruments available for similar purposes appears confusing. With experience, however, clinicians select a relatively small set that fulfills all requirements.

CLASSIFICATION OF PERIODONTAL INSTRUMENTS

Periodontal instruments are classified according to the purposes they serve, as follows:

1. *Periodontal probes* are used to locate, measure, and mark pockets, as well as determine their course on individual tooth surfaces.
2. *Explorers* are used to locate calculus deposits and caries.
3. *Scaling, root-planing, and curettage instruments* are used for removal of biofilm and calcified deposits from the crown and root of a tooth, removal of altered cementum from the subgingival root surface, and debridement of the soft tissue lining the pocket. Scaling and curettage instruments are classified as follows:
   - *Sickle scalers* are heavy instruments used to remove supragingival calculus.
   - *Curettes* are fine instruments used for subgingival scaling, root planing, and removal of the soft tissue lining the pocket.
   - *Hoe, chisel, and file scalers* are used to remove tenacious subgingival calculus and altered cementum. Their use is limited compared with that of curettes.
   - *Ultrasonic and sonic instruments* are used for scaling and cleansing tooth surfaces and curetting the soft tissue wall of the periodontal pocket.[39,40,58]
4. The *periodontal endoscope* is used to visualize deeply into subgingival pockets and furcations, allowing the detection of deposits.
5. *Cleansing and polishing instruments*, such as rubber cups, brushes, and dental tape, are used to clean and polish tooth surfaces. Also available are air-powder abrasive systems for tooth polishing.

The wearing and cutting qualities of some types of steel used in periodontal instruments have been tested,[80,81,142] but specifications vary among manufacturers.[142] Stainless steel is used most often in instrument manufacture. High-carbon-content steel instruments are also available and are considered by some clinicians to be superior.

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Each group of instruments has characteristic features; individual therapists often develop variations with which they operate most effectively. Small instruments are recommended to fit into periodontal pockets without injuring the soft tissues.[105,107,108,159]

The parts of each instrument are referred to as the working end, shank, and handle (Figure 45-1).

Periodontal Probes

Periodontal probes are used to measure the depth of pockets and to determine their configuration. The typical probe is a tapered, rodlike instrument calibrated in millimeters, with a blunt, rounded tip (Figure 45-2). There are several other designs with various millimeter calibrations (Figure 45-3). The World Health Organization (WHO) probe has millimeter markings and a small, round ball at the tip (Figure 45-3, *E*). Ideally, these probes are thin, and the shank is angled to allow easy insertion into the pocket. Furcation areas can best be evaluated with the curved, blunt Nabers probe (Figure 45-4).

When measuring a pocket, the probe is inserted with a firm, gentle pressure to the bottom of the pocket. The shank should be aligned with the long axis of the tooth surface to be probed. Several measurements are made to determine the level of attachment along the surface of the tooth.

Explorers

Explorers are used to locate subgingival deposits and carious areas and to check the smoothness of the root surfaces after root planing. Explorers are designed with different shapes and angles, with various uses (Figure 45-5), as well as limitations (Figure 45-6). The periodontal probe can also be useful in the detection of subgingival deposits (Figure 45-6, *D*).

Scaling and Curettage Instruments

Scaling and curettage instruments are illustrated in Figure 45-7.

Sickle Scalers. Sickle scalers have a flat surface and two cutting edges that converge in a sharply pointed tip. The shape of the instrument makes the tip strong so that it will not break off during use (Figure 45-8). The sickle scaler is used primarily to remove supragingival calculus (Figure 45-9). Because of the design of this instrument, it is difficult to insert a large sickle blade under the gingiva without damaging the surrounding gingival tissues

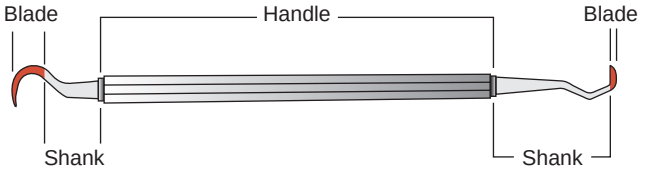

Figure 45-1 Parts of a typical periodontal instrument.

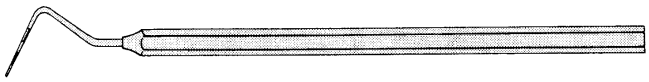

Figure 45-2 Periodontal probe is composed of the handle, shank, and calibrated working end.

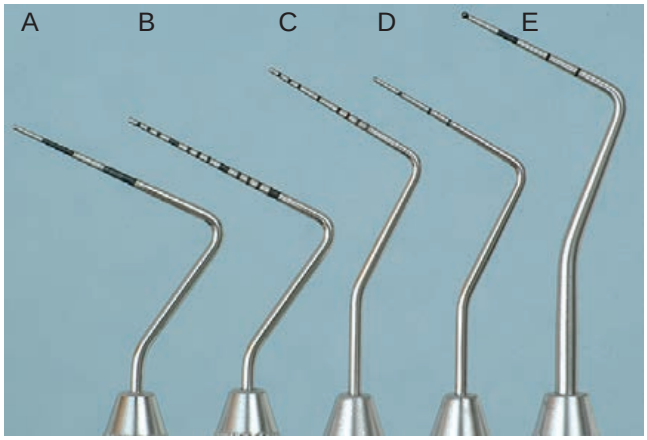

Figure 45-3 Types of periodontal probes. **A,** Marquis color-coded probe. Calibrations are in 3-mm sections. **B,** UNC-15 probe, a 15-mm long probe with millimeter markings at each millimeter and color coding at the fifth, tenth, and fifteenth millimeters. **C,** University of Michigan "O" probe, with Williams markings (at 1, 2, 3, 5, 7, 8, 9, and 10 mm). **D,** Michigan "O" probe with markings at 3, 6, and 8 mm. **E,** World Health Organization (WHO) probe, which has a 0.5-mm ball at the tip and millimeter markings at 3.5, 8.5, and 11.5 mm and color coding from 3.5 to 5.5 mm.

Figure 45-4 Curved #2 Nabers probe for detection of furcation areas, with color-coded markings at 3, 6, 9, and 12 mm.

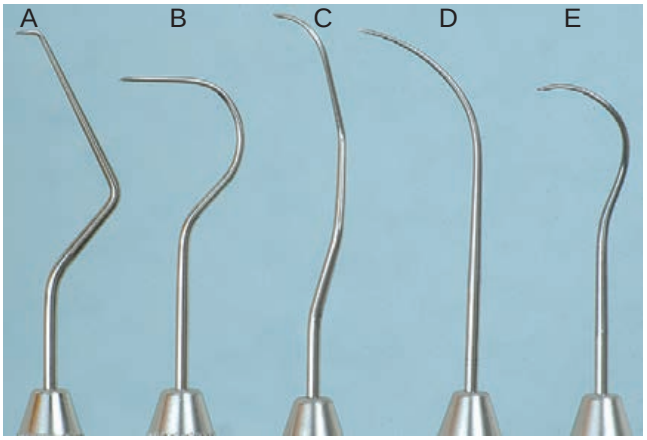

Figure 45-5 Five typical explorers. **A,** #17; **B,** #23; **C,** EXD 11-12; **D,** #3; **E,** #3CH Pigtail.

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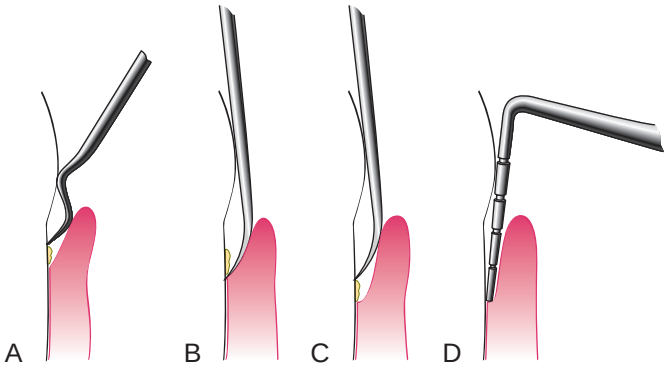

Figure 45-6 Insertion of two types of explorers and a periodontal probe in a pocket for calculus detection. **A,** The limitations of the pigtail explorer in a deep pocket. **B,** Insertion of the #3 explorer. **C,** Limitations of the #3 explorer. **D,** Insertion of the periodontal probe.

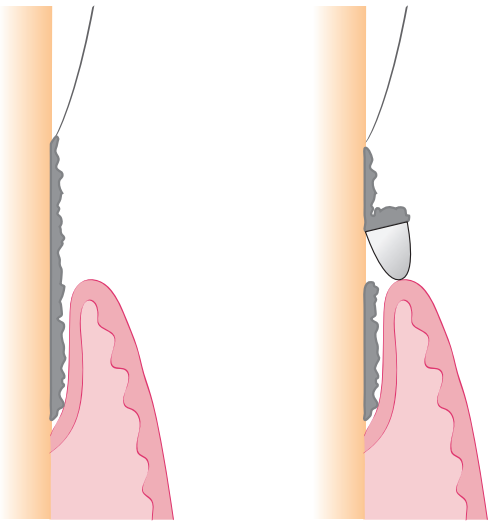

Figure 45-9 Use of a sickle scaler for removal of supragingival calculus.

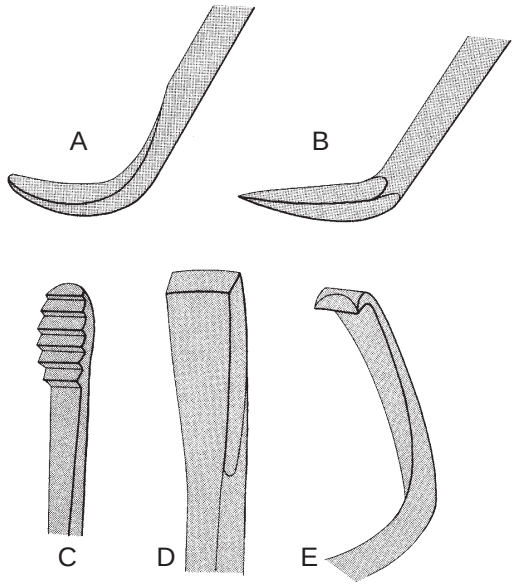

Figure 45-7 The five basic scaling instruments. **A,** Curette; **B,** sickle; **C,** file; **D,** chisel; **E,** hoe.

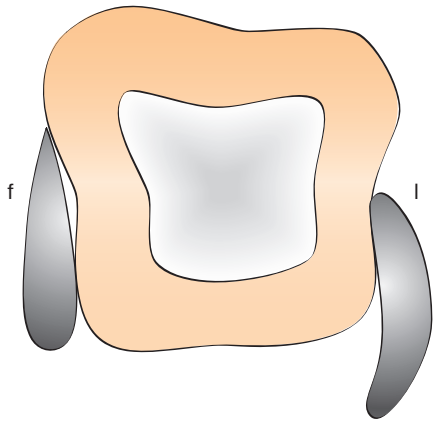

Figure 45-10 Subgingival adaptation around the root is better with the curette than with the sickle; f, facial; l, lingual.

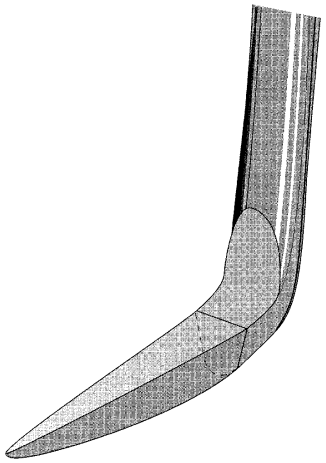

Figure 45-8 Basic characteristics of a sickle scaler: triangular shape, double-cutting edge, and pointed tip.

(Figure 45-10). Small, curved sickle scaler blades such as the 204SD can be inserted under ledges of calculus several millimeters below the gingiva. Sickle scalers are used with a pull stroke.

It is important to note that sickle scalers with the same basic design can be obtained with different blade sizes and shank types to adapt to specific uses. The U15/30 (Figure 45-11), Ball, and Indiana University sickle scalers are large. The Jaquette sickle scalers #1, 2, and 3 have medium-size blades. The curved 204 posterior sickle scalers are available with large, medium, or small blades (Figure 45-12). The Montana Jack sickle scaler and the Nevi 2, Nevi 3, and Nevi 4 curved posterior sickle scalers are all thin enough to be inserted several millimeters subgingivally for removal of light to moderate ledges of calculus. The selection of these instruments should be based on the area to be scaled. Sickle scalers with straight shanks are designed for use on anterior teeth and premolars. Sickle scalers with contra-angled shanks adapt to posterior teeth.

Curettes. The curette is the instrument of choice for removing deep subgingival calculus, root planing altered cementum, and removing the soft tissue lining the periodontal pocket (Figure 45-13). Each working end has a cutting edge on both sides of the

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Figure 45-11 Both ends of a U15/30 scaler.

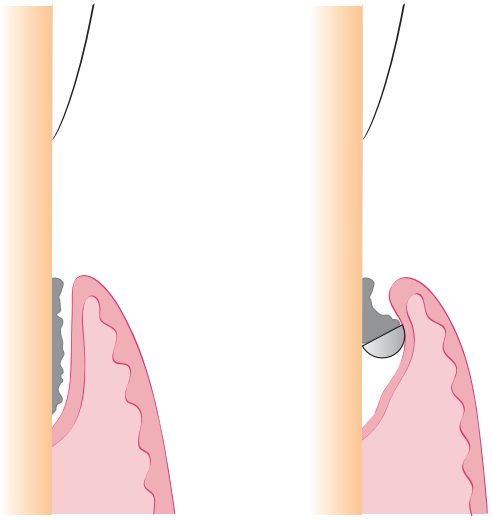

Figure 45-13 The curette is the instrument of choice for subgingival scaling and root planing.

Figure 45-12 Three different sizes of 204 sickle scalers.

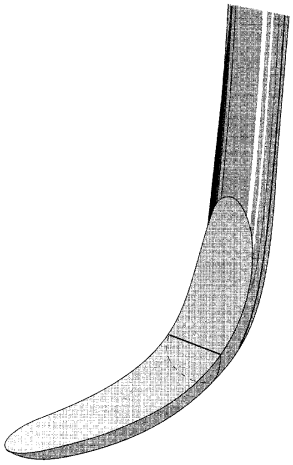

Figure 45-14 Basic characteristics of a curette: spoon-shaped blade and rounded tip.

blade and a rounded toe. The curette is finer than the sickle scalers and does not have any sharp points or corners other than the cutting edges of the blade (Figure 45-14). Therefore curettes can be adapted and provide good access to deep pockets, with minimal soft tissue trauma (see Figure 45-10). In cross-section the blade appears semicircular with a convex base. The lateral border of the convex base forms a cutting edge with the face of the semicircular blade. There are cutting edges on both sides of the blade. Both single- and double-end curettes may be obtained, depending on the preference of the operator.

As shown in Figure 45-10, the curved blade and rounded toe of the curette allow the blade to adapt better to the root surface, unlike the straight design and pointed end of a sickle scaler, which can cause tissue laceration and trauma. There are two basic types of curettes: universal and area specific.

Universal Curettes. Universal curettes have cutting edges that may be inserted in most areas of the dentition by altering and adapting the finger rest, fulcrum, and hand position of the operator. The blade size and the angle and length of the shank may vary, but the face of the blade of every universal curette is at a 90-degree angle (perpendicular) to the lower shank when seen in cross-section from the tip (Figure 45-15, *A*). The blade of the universal curette is curved in one direction from the head of the blade to the toe. The Barnhart curettes #1-2 and 5-6 and the Columbia curettes #13-14, 2R-2L, and 4R-4L (Figures 45-16 and 45-17, *A*) are examples of universal curettes. Other popular universal curettes are the Younger-Good #7-8, the McCall's #17-18, and the Indiana University #17-18 (Figure 45-17, *B*).

Area-Specific Curettes

Gracey Curettes. Gracey curettes are representative of the area-specific curettes, a set of several instruments designed and angled to adapt to specific anatomic areas of the dentition (Figure 45-18). These curettes and their modifications are probably the best

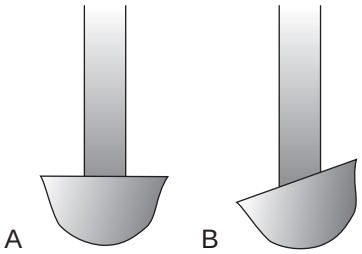

Figure 45-15 Principal types of curettes as seen from the toe of the instrument. **A,** Universal curette. **B,** Gracey curette. Note the offset blade angulation of the Gracey curette.

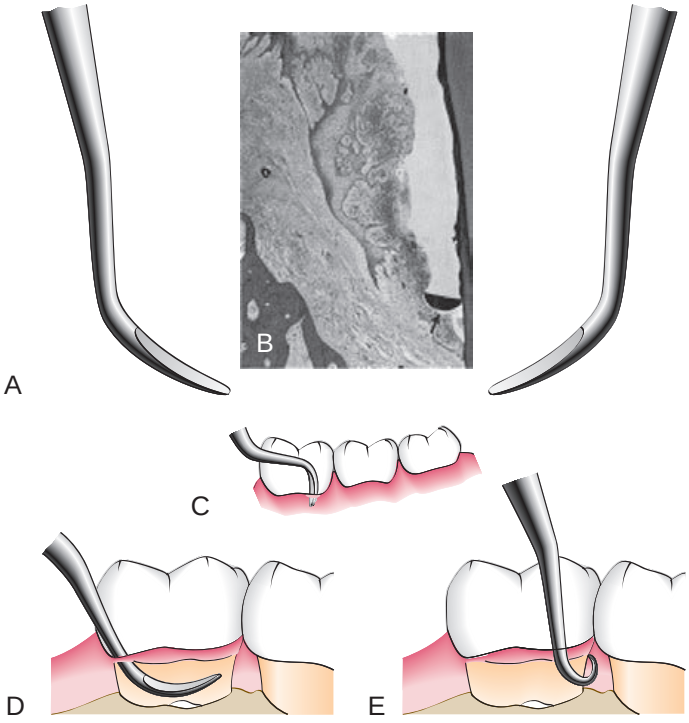

Figure 45-16 **A,** Double-ended curette for the removal of subgingival calculus. **B,** Cross-section of the curette blade *(arrow)* against the cemental wall of a deep periodontal pocket. **C,** Curette in position at the base of a periodontal pocket on the facial surface of a mandibular molar. **D,** Curette inserted in a pocket with the tip directed apically. **E,** Curette in position at the base of a pocket on the distal surface of the mandibular molar.

Figure 45-17 **A,** Columbia #4R-4L universal curette. **B,** Younger-Good #7-8, McCall's #17-18, and Indiana University #17-18 universal curettes.

Figure 45-18 Reduced set of Gracey curettes. *Left to right,* #5-6, #7-8, #11-12, and #13-14.

instruments for subgingival scaling and root planing because they provide the best adaptation to complex root anatomy.

Double-ended Gracey curettes are paired in the following manner:

Gracey #1-2 and 3-4: Anterior teeth
Gracey #5-6: Anterior teeth and premolars
Gracey #7-8 and 9-10: Posterior teeth: facial and lingual
Gracey #11-12: Posterior teeth: mesial (Figure 45-19)
Gracey #13-14: Posterior teeth: distal (Figure 45-20)

Single-ended Gracey curettes can also be obtained; a set of these curettes comprises 14 instruments. Although these curettes are designed to be used in specific areas, an experienced operator can adapt each instrument for use in several different areas by altering the position of his or her hand and the position of the patient.

The Gracey curettes also differ from the universal curettes in that the blade is not at a 90-degree angle to the lower shank. The term *offset blade* is used to describe Gracey curettes because they are angled approximately 60 to 70 degrees from the lower shank (see Figure 45-15, *B*). This unique angulation allows the blade to be inserted in the precise position necessary for subgingival scaling and root planing, provided that the lower shank is parallel with the long axis of the tooth surface being scaled.

Area-specific curettes also have a curved blade. Whereas the blade of the universal curette is curved in one direction (Figure 45-21, *A*), the Gracey blade is curved from head to toe and also along the side of the cutting edge (Figure 45-21, *B*). Thus only a pull stroke can be used. Table 45-1 lists some of the major differences between Gracey (area-specific) curettes and universal curettes.

Gracey curettes are available with either a "rigid" or a "finishing" type of shank. The rigid Gracey has a larger, stronger, and less flexible shank and blade than the standard finishing Gracey. The rigid shank allows the removal of moderate to heavy calculus without

Figure 45-19 Gracey #11-12 curette. Note the double turn of the shank.

Figure 45-20 Gracey #13-14 curette. Note the acute turn of the blade.

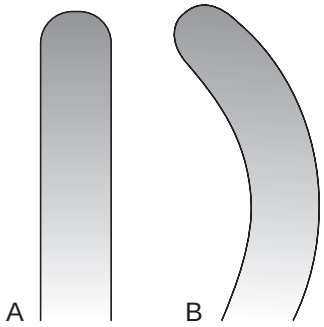

Figure 45-21 **A,** Universal curette as seen from the blade. Note that the blade is straight. **B,** Gracey curette as seen from the blade. The blade is curved; only the convex cutting edge is used.

Figure 45-22 Gracey #15-16. New Gracey curette, designed for mesio-posterior surfaces, combines a Gracey #11-12 blade with a Gracey #13-14 shank.

TABLE 45-1 Comparison of Area-Specific (Gracey) and Universal Curettes

	Gracey Curette	Universal Curette
Area of use	Set of many curettes designed for specific areas and surfaces.	One curette designed for all areas and surfaces.
Cutting Edge		
Use	One cutting edge used; work with outer edge only.	Both cutting edges used; work with either outer or inner edge.
Curvature	Curved in two planes; blade curves up and to the side.	Curved in one plane; blade curves up, not to the side.
Blade angle	Offset blade; face of blade beveled at 60 degrees to shank.	Blade not offset; face of blade beveled at 90 degrees to shank.

Modified from Pattison G, Pattison A: *Periodontal instrumentation*, ed 2, Norwalk, CT, 1992, Appleton & Lange.

using a separate set of heavy scalers, such as sickles and hoes. Although some clinicians prefer the enhanced tactile sensitivity that the flexible shank of the finishing Gracey provides, both types of Gracey curettes are suitable for root planing.

Recent additions to the Gracey curette set have been the Gracey #15-16 and 17-18. The Gracey #15-16 is a modification of the standard #11-12 and is designed for the mesial surfaces of posterior teeth (Figure 45-22). It consists of a Gracey #11-12 blade combined with the more acutely angled #13-14 shank. When the clinician is using an intraoral finger rest, it is often difficult to position the lower shank of the Gracey #11-12 so that it is parallel with the mesial surfaces of the posterior teeth, especially on the mandibular molars. The new shank angulation of the Gracey #15-16 allows better adaptation to posterior mesial surfaces from a front position with intraoral rests. If alternative fulcrums, such as extraoral or opposite-arch rests, are used, the Gracey #11-12 works well and

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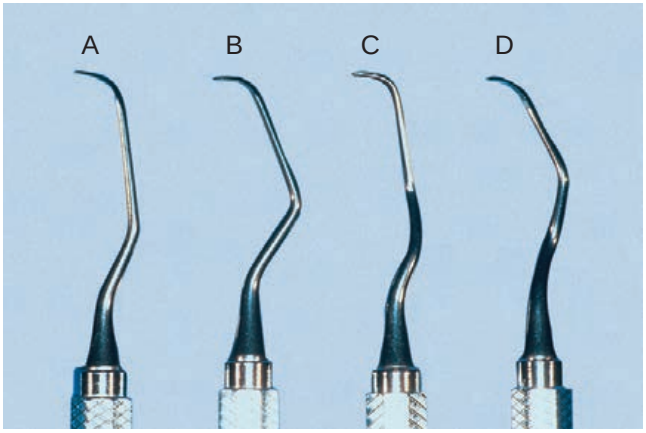

Figure 45-23 After Five curette. Note the extra 3 mm in the terminal shank of the After Five curette compared with the standard Gracey curette. **A,** #5-6; **B,** #7-8; **C,** #11-12; **D,** #13-14.

Figure 45-24 Comparison of After Five curette with standard Gracey curette. Rigid Gracey #13-14 adapted to the distal surface of the first molar and rigid After Five #13-14 adapted to the distal surface of the second molar. Notice the extralong shank of the After Five curette, which allows deeper insertion and better access.

the new #15-16 is not essential. The Gracey #17-18 is a modification of the #13-14. It has a terminal shank elongated by 3 mm and a more accentuated angulation of the shank to provide complete occlusal clearance and better access to all posterior distal surfaces. The horizontal handle position minimizes interference from opposing arches and allows a more relaxed hand position when scaling distal surfaces. In addition, the blade is 1 mm shorter to allow better adaptation of the blade to distal tooth surfaces.

Extended-Shank Curettes. Extended-shank curettes, such as the *After Five* curettes (Hu-Friedy, Chicago), are modifications of the standard Gracey curette design. The terminal shank is 3 mm longer, allowing extension into deeper periodontal pockets of 5 mm or more (Figures 45-23 and 45-24). Other features of the After Five curette include a thinned blade for smoother subgingival insertion and reduced tissue distention and a large-diameter, tapered shank. All standard Gracey numbers except for the #9-10 (i.e., #1-2, 3-4, 5-6, 7-8, 11-12, or 13-14) are available in the After Five series. The After Five curettes are available in finishing or rigid designs. For heavy or tenacious calculus removal, rigid After Five curettes should be used. For light scaling or deplaquing in a periodontal maintenance patient, the thinner, finishing After Five curettes will insert subgingivally more easily.

Mini-Bladed Curettes. Mini-bladed curettes, such as the Hu-Friedy *Mini Five* curettes, are modifications of the After Five

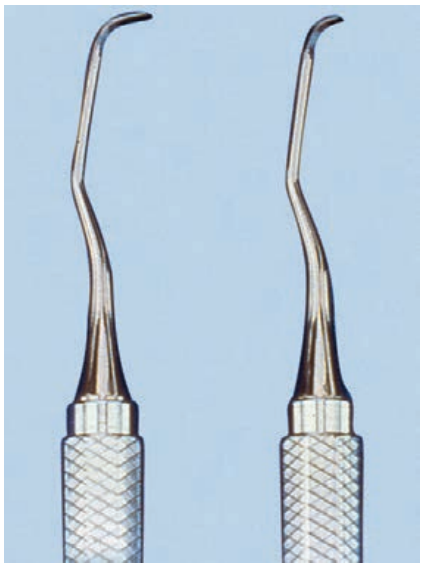

Figure 45-25 Comparison of After Five curette and Mini Five curette. The shorter Mini Five blade (half the length) allows increased access and reduced tissue trauma.

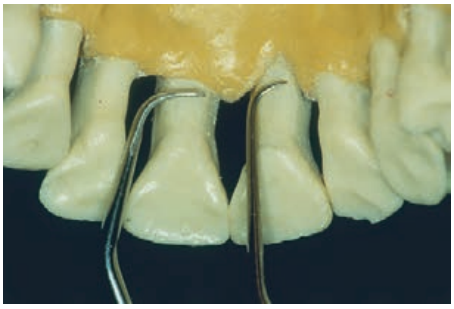

Figure 45-26 Comparison of standard rigid Gracey #5-6 with rigid Mini Five #5-6 on the palatal surfaces of the maxillary central incisors. Mini Five curette can be inserted to the base of these tight anterior pockets and used with a straight vertical stroke. Standard Gracey or After Five curette usually cannot be inserted vertically in this area because the blade is too long.

curettes. The Mini Five curettes feature blades that are half the length of the After Five or standard Gracey curettes (Figure 45-25). The shorter blade allows easier insertion and adaptation in deep, narrow pockets; furcations; developmental grooves; line angles; and deep, tight, facial, lingual, or palatal pockets. In any area in which root morphology or tight tissue prevents full insertion of the standard Gracey or After Five blade, the Mini Five curettes can be used with vertical strokes, with reduced tissue distention, and without tissue trauma (Figure 45-26).

In the past the only solution in most of these areas of difficult access was to use the Gracey curettes with a toe-down horizontal stroke. The Mini Five curettes, along with other short-bladed instruments relatively recently introduced, open a new chapter in the history of root instrumentation by allowing access to areas that previously were extremely difficult or impossible to reach with standard instruments. The Mini Five curettes are available in both finishing and rigid designs. Rigid Mini Five curettes are recommended for calculus removal. The more flexible, shanked, finishing Mini Five curettes are appropriate for light scaling and deplaquing in periodontal maintenance patients with tight pockets. As with the After Five series, the Mini Five curettes are available in all standard Gracey numbers, except the #9-10.

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The recently introduced *Micro Mini Five* Gracey curettes (Hu-Friedy, Chicago) have blades that are 20% thinner and smaller than the Mini Five curettes (Figures 45-27 and 45-28) These are the smallest of all curettes, and they provide exceptional access and adaptation to tight, deep, or narrow pockets; narrow furcations; developmental depressions; line angles; and deep pockets on facial, lingual, or palatal surfaces. In areas in which root morphology or tight, thin tissue prevents easy insertion of other mini-bladed curettes, the Micro Mini Five curettes can be used with vertical strokes without causing tissue distension or tissue trauma.

The *Gracey curvettes* are another set of four mini-bladed curettes; the Sub-0 and the #1-2 are used for anterior teeth and premolars, the #11-12 is used for posterior mesial surfaces, and the #13-14 for posterior distal surfaces. The blade length of these instruments is 50% shorter than that of the conventional Gracey curette, and the blade has been curved slightly upward (Figure 45-29). This curvature allows the Gracey curvettes to adapt more closely to the tooth surface than any other curettes, especially on the anterior teeth and on line angles (Figure 45-30). However, this curvature also carries the risk of gouging or "grooving" into the root surfaces on the proximal surfaces of the posterior teeth when the Gracey curvette #11-12 or 13-14 is used. Additional features that represent improvements on the standard Gracey curettes are a precision-balanced blade tip in direct alignment with the handle, a blade tip perpendicular to the handle, and a shank closer to parallel with the handle.

For many years, the *Morse scaler,* a miniature sickle, was the only mini-bladed instrument available. However, the mini-bladed curettes have largely replaced this instrument (Figure 45-31).

Langer and Mini-Langer Curettes. The Langer and Mini Langer curettes are a set of three curettes combining the shank design of the standard Gracey #5-6, 11-12, and 13-14 curettes with a universal blade honed at 90 degrees rather than the offset blade of the Gracey curette. This marriage of the Gracey and universal curette designs allows the advantages of the area-specific shank to be combined with the versatility of the universal curette blade. The Langer #5-6 curette adapts to the mesial and distal surfaces of anterior teeth; the Langer #1-2 curette (Gracey #11-12 shank) adapts to the mesial and distal surfaces of mandibular posterior teeth; and the Langer #3-4 curette (Gracey #13-14 shank) adapts to the mesial and distal surfaces of maxillary posterior teeth (Figure 45-32). These instruments can be adapted to both mesial and distal tooth surfaces without changing instruments. The standard Langer curette shanks are heavier than a finishing Gracey but less rigid than the rigid Gracey. Langer curettes are also available with either rigid or finishing shanks and can be obtained in the extended-shank (After Five) and mini-bladed (Mini Five) versions.

Schwartz Periotrievers. The Schwartz Periotrievers are a set of two double-ended, highly magnetized instruments designed for the retrieval of broken instrument tips from the periodontal pocket (Figures 45-33 and 45-34). They are indispensable when the clinician has broken a curette tip in a furcation or deep pocket.[134]

Plastic and Titanium Instruments for Implants. Several different companies are manufacturing plastic and titanium instruments for use on titanium and other implant abutment materials. It is important that plastic or titanium instruments be used to avoid scarring and permanent damage to the implants* (Figures 45-35, 45-36, and 45-37).

Hoe Scalers. Hoe scalers are used for scaling of ledges or rings of calculus (Figure 45-38). The blade is bent at a 99-degree angle; the cutting edge is formed by the junction of the flattened terminal surface with the inner aspect of the blade. The cutting edge is

Figure 45-27 Micro Mini Five Gracey curettes. *Left to right,* #1-2, #7-8, #11-12, #13-14.

*References 21, 36, 40, 43, 52, 88, and 128.

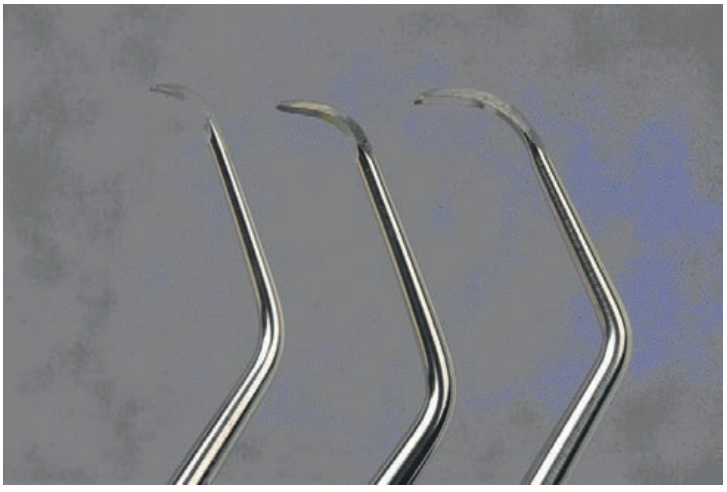

Figure 45-28 Comparison of Gracey curette blades. *Left to right,* Micro Mini Five #7-8, Mini Five #7-8, Standard #7-8.

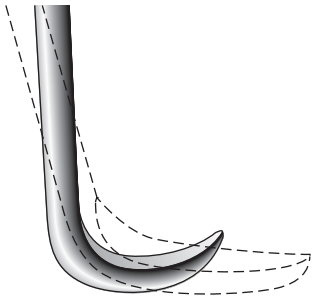

Figure 45-29 Gracey Curvette blade. This diagram shows the 50% shorter blade of the Gracey Curvette superimposed on the standard Gracey curette blade *(dotted lines)*. Notice the upward curvature of the Curvette blade and blade tip. *(Redrawn from Pattison G, Pattison A:* Periodontal instrumentation, *ed 2, Norwalk, Conn, 1992, Appleton & Lange.)*

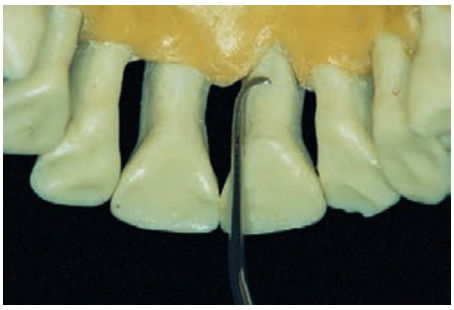

Figure 45-30 Gracey Curvette Sub-0 on the palatal surface of a maxillary central incisor. The long shank and short, curved, and blunted tip make this a superior instrument for deep anterior pockets. This curette provides excellent blade adaptation to the narrow root curvatures of the maxillary and mandibular anterior teeth.

Figure 45-31 Comparison of three different mini-bladed instruments designed for use on the maxillary and mandibular anterior teeth. **A,** Hu-Friedy Mini Five #5-6; **B,** Hu-Friedy Curvette Sub-0; **C,** Hartzell Sub-0.

Figure 45-32 Langer curettes combine Gracey-type shanks with universal curette blades. *Left to right,* #5-6, #1-2, and #3-4.

Figure 45-33 Schwartz Periotriever tip designs. The long blade is for general use in pockets, and the contra-angled tip is for use in furcations. *(From Pattison G, Pattison A:* Periodontal instrumentation, *ed 2, Norwalk, CT, 1992, Appleton & Lange.)*

Figure 45-34 Broken instrument tip attached to the magnetic tip of the Schwartz Periotriever. *(From Pattison G, Pattison A:* Periodontal instrumentation, *ed 2, Norwalk, CT, 1992, Appleton & Lange.)*

Figure 45-35 Plastic probe: Colorvue (Hu-Friedy, Chicago).

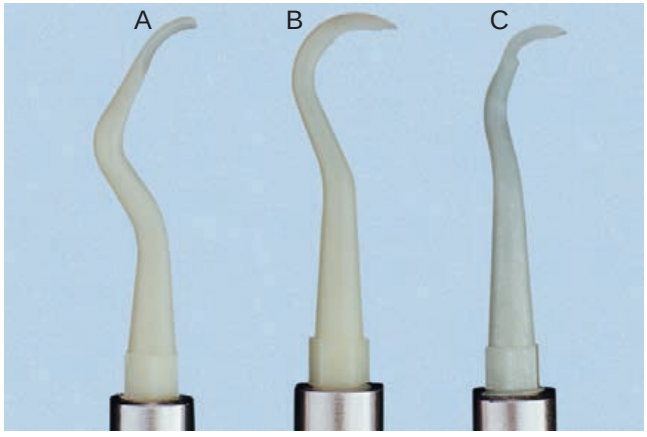

Figure 45-36 Implacare implant instruments (Hu-Friedy, Chicago). These implant instruments have autoclavable stainless steel handles and three different cone-socket plastic tip designs. **A,** Columbia 4R-4L curette tip; **B,** H6-H7 sickle scaler tip; **C,** 204S sickle scaler tip.

Figure 45-37 Titanium Implant Curettes (Paradise Dental Technologies, Missoula, MT). *Left to right,* Barnhart 5-6, Langer 1-2, and NEB 128B-L5 Mini.

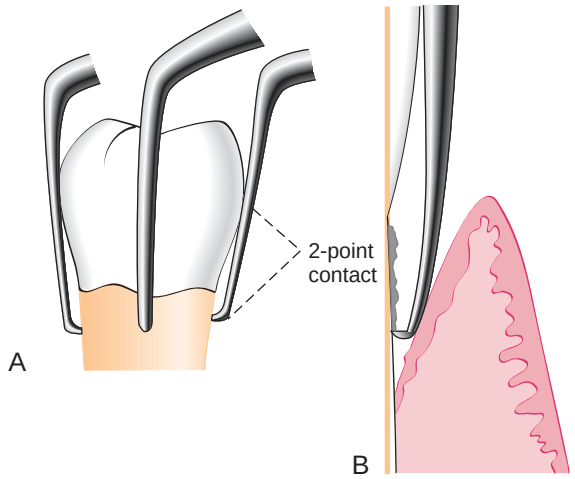

Figure 45-38 A, Hoe scalers designed for different tooth surfaces, showing "two-point" contact. **B,** Hoe scaler in a periodontal pocket. The back of the blade is rounded for easier access. The instrument contacts the tooth at two points for stability.

Figure 45-39 Chisel scaler **(A)** and file scaler **(B)**.

beveled at 45 degrees. The blade is slightly bowed so that it can maintain contact at two points on a convex surface. The back of the blade is rounded, and the blade has been reduced to minimal thickness to permit access to the roots without interference from the adjacent tissues.

Hoe scalers are used in the following manner:

1. The blade is inserted to the base of the periodontal pocket so that it makes two-point contact with the tooth (see Figure 45-38). This stabilizes the instrument and prevents nicking of the root.
2. The instrument is activated with a firm pull stroke toward the crown, with every effort being made to preserve the two-point contact with the tooth.

McCall's #3, 4, 5, 6, 7, and 8 are a set of six hoe scalers designed to provide access to all tooth surfaces. Each instrument has a different angle between the shank and handle.

Files. Files have a series of blades on a base (Figure 45-39). Their primary function is to fracture or crush large deposits of tenacious

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calculus or burnished sheets of calculus. Files can easily gouge and roughen root surfaces when used improperly. Therefore they are not suitable for fine scaling and root planing. Mini-bladed curettes are currently preferred for fine scaling in areas where files were once used. Files are sometimes used for removing overhanging margins of dental restorations.

Chisel Scalers. The chisel scaler, designed for the proximal surfaces of teeth too closely spaced to permit the use of other scalers, is usually used in the anterior part of the mouth. It is a double-ended instrument with a curved shank at one end and a straight shank at the other (see Figure 45-39); the blades are slightly curved and have a straight cutting edge beveled at 45 degrees.

The chisel is inserted from the facial surface. The slight curve of the blade makes it possible to stabilize it against the proximal surface, whereas the cutting edge engages the calculus without nicking the tooth. The instrument is activated with a push motion while the side of the blade is held firmly against the root.

Quétin Furcation Curettes. The Quétin furcation curettes are actually hoes with a shallow, half-moon radius that fits into the roof or floor of the furcation. The curvature of the tip also fits into developmental depressions on the inner aspects of the roots. The shanks are slightly curved for better access, and the tips are available in two widths (Figure 45-40). The BL1 *(buccal-lingual)* and MD1 *(mesial-distal)* instruments are small and fine, with a 0.9-mm blade width. The BL2 and MD2 instruments are larger and wider, with a 1.3-mm blade width.

These instruments remove burnished calculus from recessed areas of the furcation where curettes, even the mini-bladed curettes, are often too large to gain access. Using mini-bladed Gracey curettes and Gracey Curvettes in the roof or floor of the furcation may unintentionally create gouges and grooves. The Quétin instruments, however, are well suited for this area and lessen the likelihood of root damage.

Diamond-Coated Files. Diamond-coated files are unique instruments used for final finishing of root surfaces. These files do not have cutting edges; instead, they are coated with very-fine-grit diamond (Figure 45-41). The most useful diamond files are the buccal-lingual instruments, which are used in furcations and also adapt well to many other root surfaces.

New diamond files are sharply abrasive and should be used with light, even pressure against the root surface to avoid gouging or grooving. When viewing the root surface with the dental endoscope after all tactilely detectable deposits are gone, small embedded remnants of calculus in the root surface can be observed. Diamond files are used similar to an emery board to remove these minute remnants of calculus from the root, creating a surface that is free of all visible accretions. Diamond files can produce a smooth, even, clean, and highly polished root surface.

Diamond files must be used carefully because they can cause overinstrumentation of the root surface. They will remove too much root structure if they are used with excessive force, are poorly adapted to root morphology, or used too long in one place.

Diamond files are particularly effective when used with the dental endoscope, which reveals residual deposits and directs the clinician to the exact area for instrumentation.

Ultrasonic and Sonic Instruments. Ultrasonic instruments may be used for removing plaque, scaling, curetting, and removing stain (see Chapter 46).

Dental Endoscope. A dental endoscope has been introduced for use subgingivally in the diagnosis and treatment of periodontal

Figure 45-40 Quétin furcation curettes: BL2 (larger) and BL1 (smaller). (Hu-Friedy, Chicago).

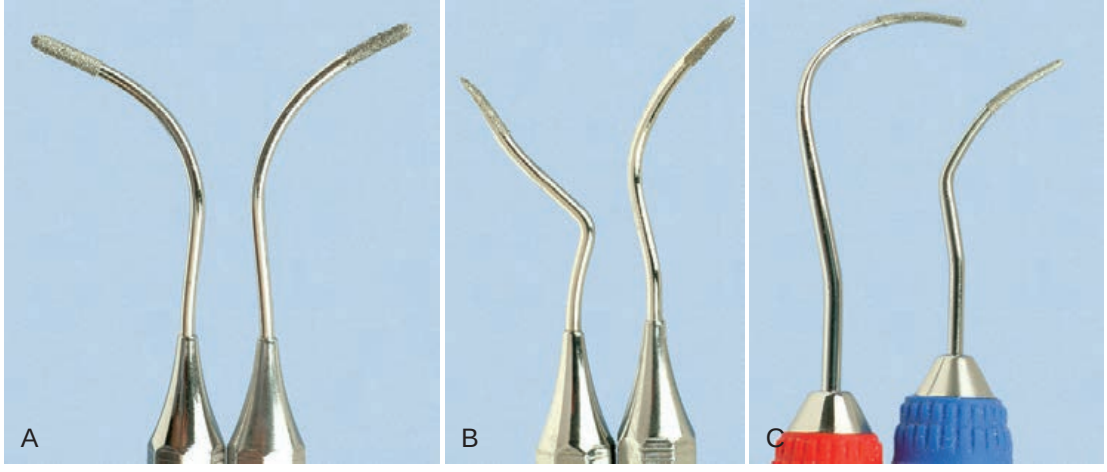

Figure 45-41 Diamond files. **A,** #1,2 (Brasseler, Savannah, GA); **B,** #3,4 (Brasseler). **C,** SDCN 7, SDCM/D 7 (Hu-Friedy, Chicago).

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Figure 45-42 Perioscopy system, dental endoscope. *(Courtesy Perioscopy Incorporated, Oakland, CA.)*

Figure 45-43 Viewing periodontal explorers (left/right/full viewing) for the Perioscopy system. *(Courtesy Perioscopy Incorporated, Oakland, CA.)*

disease (Figure 45-42). The *Perioscopy* system (Perioscopy, Inc, Oakland, CA) consists of a 0.99-mm-diameter, reusable fiberoptic endoscope over which is fitted a disposable, sterile sheath. The fiberoptic endoscope fits onto periodontal probes and ultrasonic instruments that have been designed to accept it (Figure 45-43). The sheath delivers water irrigation that flushes the pocket while the endoscope is being used, keeping the field clear. The fiberoptic endoscope attaches to a medical-grade charged-coupled device (CCD) video camera and light source that produces an image on a flat-panel monitor for viewing during subgingival exploration and instrumentation. This device allows clear visualization deeply into subgingival pockets and furcations (Figure 45-44). It permits operators to detect the presence and location of subgingival deposits and guides them in the thorough removal of these deposits. Magnification ranges from 24X to 48X, enabling visualization of even minute deposits of plaque and calculus. Using this device, operators can achieve levels of root debridement and cleanliness that are much more difficult or impossible to produce without it.[145,146,163,164] The Perioscopy system can also be used to evaluate subgingival areas for caries, defective restorations, root fractures, and resorption.

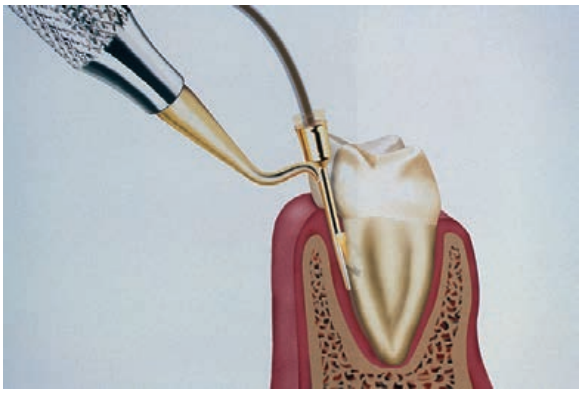

Figure 45-44 Perioscopic instrumentation permits deep subgingival visualization in pockets and furcations. *(Courtesy Perioscopy Incorporated, Oakland, CA.)*

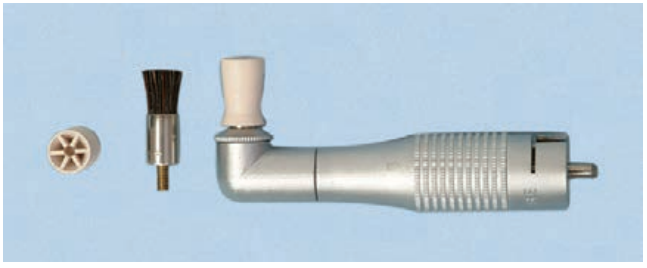

Figure 45-45 Metal prophylaxis angle with rubber cup and brush.

Cleansing and Polishing Instruments

Rubber Cups. Rubber cups consist of a rubber shell with or without webbed configurations in the hollow interior (Figure 45-45). They are used in the handpiece with a special prophylaxis angle. The handpiece, prophylaxis angle, and rubber cup must be sterilized after each patient use, or a disposable plastic prophylaxis angle and rubber cup may be used and then discarded (Figure 45-46). A good cleansing and polishing paste that contains fluoride should be used and kept moist to minimize frictional heat as the cup revolves. Polishing pastes are available in fine, medium, or coarse grits and are packaged in small, convenient, single-use containers. Aggressive use of the rubber cup with any abrasive may remove the layer of cementum, which is thin in the cervical area.

Bristle Brushes. Bristle brushes are available in wheel and cup shapes (see Figure 45-45). The brush is used in the prophylaxis angle with a polishing paste. Because the bristles are stiff, use of the brush should be confined to the crown to avoid injuring the cementum and the gingiva.

Dental Tape. Dental tape with polishing paste is used for polishing proximal surfaces that are inaccessible to other polishing

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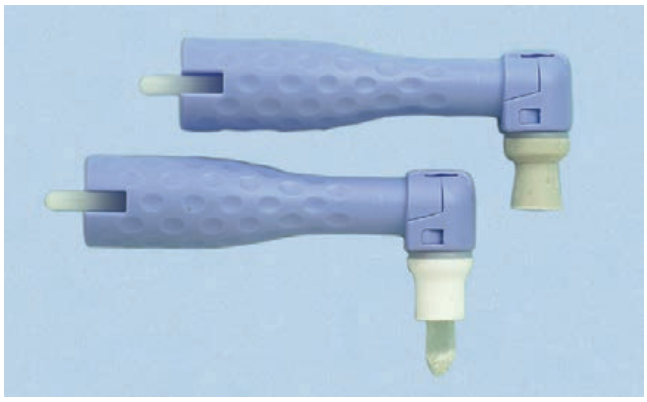

Figure 45-46 Disposable plastic prophylaxis angle with rubber cup and with brush.

Figure 45-47 Prophy-Jet air-powder polishing device. *(Courtesy Dentsply International, York, PA.)*

instruments. The tape is passed interproximally while being kept at a right angle to the long axis of the tooth and is activated with a firm labiolingual motion. Particular care is taken to avoid injury to the gingiva. The area should be cleansed with warm water to remove all remnants of paste.

Air-Powder Polishing. The first specially designed handpiece to deliver an air-powered slurry of warm water and sodium bicarbonate for polishing was introduced in the early 1980s. This device, called the *Prophy-Jet* (Dentsply International, York, PA) is very effective for the removal of extrinsic stains and soft deposits (Figure 45-47). The slurry removes stains rapidly and efficiently by mechanical abrasion and provides warm water for rinsing and lavage. The flow rate of abrasive cleansing power can be adjusted to increase the amount of powder for heavier stain removal. Currently, many manufacturers produce air-powder polishing systems that use various powder formulas.

The results of studies on the abrasive effect of the air-powder polishing devices using sodium bicarbonate and aluminum trihydroxide on cementum and dentin show that significant tooth substance can be lost.[2,19,106,114] Damage to gingival tissue is transient and insignificant clinically, but amalgam restorations, composite resins, cements, and other nonmetallic materials can be roughened.[13,41,64,86,157] Polishing powders containing glycine rather than sodium bicarbonate recently have been introduced for subgingival biofilm removal from root surfaces.[93a,113] Air-powder polishing can be used safely on titanium implant surfaces.[71,88,122]

Patients with medical histories of respiratory illnesses and hemodialysis are not candidates for the use of the air-powder polishing device.[141,161] Powders containing sodium bicarbonate should not be used on patients with histories of hypertension, sodium-restricted diets, or medications affecting the electrolyte balance.[121] Patients with infectious diseases should not be treated with this device because of the large quantity of aerosol created. A preprocedural rinse with 0.12% chlorhexidine gluconate should be used to minimize the microbial content of the aerosol.[17] High-speed evacuation should also be used to eliminate as much of the aerosol as possible.[54a]

For more information on the general principles of instrumentation, principles of scaling and root planing, and instrument sharpening, please visit the companion Expert CONSULT website at www.expertconsult.com.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

Scaling and root planing is the foundation of periodontal treatment. The thorough removal of subgingival plaque and calculus is essential for successful periodontal therapy. Because the removal of subgingival calculus embedded in the root is such a demanding clinical skill, it takes years of experience and a desire for perfection for clinicians to become highly competent. In many cases, such as furcations, it is impossible to root plane all surfaces and remove all calculus during phase I therapy and frequently there will be residual calculus visually present when the root surfaces are exposed during flap surgery. Therefore root planing should also be an important part of all flap surgeries, and in some difficult cases, it will be necessary to use mechanical instruments, such as ultrasonic scalers and rotary instruments, to achieve total removal of all embedded calculus.

Clinicians should educate patients so that they appreciate the time and high level of skill necessary for successful root planing. In most cases, local infiltration anesthesia is needed so that patients are comfortable and the clinician can focus on obtaining a smooth glass-like root surface free of calculus. The presence of residual calculus after treatment will compromise the ability to obtain healthy gingiva and pocket depth reduction. The persistence of bleeding on probing after treatment can be the result of root roughness associated with incompletely removed subgingival calculus and persistence of unhealthy bacterial biofilms on the root surface.

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CHAPTER 46

Sonic and Ultrasonic Instrumentation and Irrigation

Carol A. Jahn and David Jolkovsky

CHAPTER OUTLINE

MECHANISM OF ACTION OF POWER SCALERS
TYPE AND BENEFIT OF POWER INSTRUMENTS
Efficiency
Tip Designs
CLINICAL OUTCOMES OF POWER-DRIVEN INSTRUMENTS
SPECIAL CONSIDERATIONS
Aerosol Production
Cardiac Pacemakers
PRINCIPLES OF INSTRUMENTATION
HOME/SELF-APPLIED IRRIGATION
MECHANISM OF ACTION OF IRRIGATION
Safety
CLINICAL OUTCOMES OF IRRIGATION
INDIVIDUALS WITH SPECIAL CONSIDERATIONS
SUMMARY

For many years, practitioners have sought ways to enhance and prolong the outcomes from scaling and root planning. In the office setting, technologic advances and new designs of ultrasonic and sonic power scalers have transformed the role of power-driven oscillating instruments in periodontal therapy. At home, the pulsating dental water has been clinically proven to help patients maintain periodontal health by removing biofilm supragingivally and subgingivally and reducing inflammation.

MECHANISM OF ACTION OF POWER SCALERS

Various physical factors play a role in the mechanism of action of power scalers. These factors include frequency, stroke, and water flow. In addition to rate of flow, the physiologic effects of water may play a contributing role in the efficacy of power instruments.

Water contributes to three physiologic effects that play a role in the efficacy. These are acoustic streaming, acoustic turbulence, and cavitation. Acoustic steaming is the unidirectional fluid flow caused by ultrasound waves. Acoustic turbulence is created when the movement of the tip causes the coolant to accelerate, producing an intensified swirling effect. This turbulence continues until cavitation occurs. Cavitation is the formation of bubbles in water caused by the high turbulence. The bubbles implode and produce shock waves in the liquid, creating further shock waves throughout the water. In vitro, the combination of acoustic streaming, acoustic turbulence, and cavitation has been shown to disrupt microflora.[49,79–81]

TYPE AND BENEFIT OF POWER INSTRUMENTS

Sonic units work at a frequency of 2000 to 6500 cycles per second and use a high or low speed air source from the dental unit. Water is delivered via the same tubing used to deliver water to a dental handpiece. Sonic scaler tips are large in diameter and universal in design. A sonic scaler tip travels in an elliptical or orbital stroke pattern. This stroke pattern allows the instrument to be adapted to all tooth surfaces.

Magnetostrictive ultrasonic devices work in a frequency range of 18,000 to 50,000 cycles per second. Metal stacks that change dimension when electrical energy is applied power magnetostrictive technology. Vibrations travel from the metal stack to a connecting body that causes the vibration of the working tip. Tips move in an elliptical or orbital stroke pattern. This allows the tip four active working surfaces (Figure 46-1).

Piezoelectric ultrasonic units work in a frequency range of 18,000 to 50,000 cycles per second. They have ceramic discs that are located in the handpiece power piezoelectric technology. They change in dimension as electrical energy is applied to the tip. Piezoelectric tips move in a linear pattern, giving the tip two active surfaces. A variety of insert tip designs and shapes are available for use (Figure 46-2).

Efficiency

Power instrumentation has the potential to make scaling less demanding, more time efficient, and more ergonomically friendly.

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Figure 46-1 A magnetostrictive ultrasonic device. *(Courtesy Dentsply International, York, PA.)*

Figure 46-2 A Piezoelectric ultrasonic device. *(Courtesy Hu-Friedy, Chicago.)*

Figure 46-3 An ultrasonic insert with universal design. *(Courtesy Dentsply International, York, PA.)*

Modified tip designs allow for improved access in many areas, including furcations. Newer, slimmer designs operate effectively at lower power settings, thus improving patient comfort. Sonic and ultrasonic tips can reduce the time needed for scaling.

Tip Designs

There are tips designed to remove heavy supragingival calculus or to definitively debride periodontal pockets. Large diameter tips are created in a universal design and are indicated for the removal of large, tenacious deposits. A high power setting is generally recommended. Thinner diameter tips may be site specific in design. The straight tip design is ideal for use in treating patients with gingivitis and deplaquing maintenance patients (Figures 46-3 and 46-4). The right and left contra-angled instruments allow for greater access and adaptation to root morphology. These inserts are designed to work in a low power setting. They can be used for exploration. The amount of water delivered for lavage can be controlled through the selection of either traditional flow or a focused tip delivery flow. Contra-angled designs and larger, ergonomic grips enhance comfort and ergonomics (Figure 46-5).

Figure 46-4 An ultrasonic insert with universal design. *(Courtesy Hu-Friedy, Chicago.)*

Figure 46-5 Site-specific designed inserts with comfort grip. *(Courtesy Dentsply International, York, PA.)*

CLINICAL OUTCOMES OF POWER-DRIVEN INSTRUMENTS

Numerous clinical outcomes have been evaluated from the use of power-driven instruments. A systematic review of the literature on the treatment of chronic periodontitis found no difference in the efficacy of subgingival debridement using ultrasonic/sonic scalers versus hand instruments in the treatment of single-rooted teeth. A benefit for multirooted teeth could not be determined because of a lack of clinical data.[78] Likewise, a review by the American Academy of Periodontology found no differences in outcomes between sonic, magnetostrictive, and piezoelectric scalers.[27]

It is well established that power driven instruments remove biofilms, bacteria, and calculus through mechanical action. With the advent of new designs and thinner tips, deplaquing of root surfaces may be effectively accomplished by power-scalers.* Both sonic and ultrasonic instruments have been shown to be effective

*References 2, 4–6, 10, 14, 17, 22, 24, 26, 36, 37, 45, 48, 50, 51, 55, 57, 58, 64, 65, 67, 76, 77, 79, and 85.

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in removing calculus similar to hand instrumentation.* Ultrasonic instruments through high-speed action produce a cavitational activity and acoustic microstreaming that some believe may help enhance the disruption of the bacteria in subgingival biofilms.

The primary expected clinical outcomes from scaling and root planing are a reduction in bleeding and probing depth and a gain in clinical attachment. In comparing power scalers to hand instruments, both types demonstrate similar outcomes for reductions in bleeding on probing and probing depth and gains in clinical attachment (see Table 46-1).†

SPECIAL CONSIDERATIONS

Furcations present one of the greatest scaling challenges. In many instances, the opening of the furcation is narrower than the conventional hand instrument. For this reason, power scalers may be recommended as a means to improve access when scaling furcations (see Table 46-2).

Aerosol Production

Universal infection control procedures can help minimize the amount of aerosol produced. Harrel and Molinari[42] recommend three levels of defense in the reduction of dental aerosols. The first recommended layer of defense is personal protective barriers such as a mask, gloves, and safety glasses. The second layer is routine use of an antiseptic preprocedural rinse. The final layer is the use of a high-speed evacuation device either by a dental assistant or attached to the instrument being used.[41] In all cases and with all patients, the Centers for Disease Control and Prevention (CDC) guidelines for infection control should be adhered to.

Cardiac Pacemakers

Newer models of cardiac pacemakers often have bipolar titanium insulation that shields the units from the effects of sonic-type devices making magnetorestrictive, piezoelectric, and sonic instruments generally safe for use on people with pacemakers.[70] However, a 1998 study by Miller et al[60] found atrial and ventricular pacing was inhibited by electromagnetic interference produced by a magnetostrictive ultrasonic scaler. A sonic scaler was also tested but did not produce the same effect.[61] In light of this, as a precaution, some practitioners have advocated using only hand instruments on individuals with pacemakers.[35] There are no FDA warnings or precautions on the devices that prevent them from being used on patients with pacemaker. If in doubt, consult with the physician regarding any precautions or warnings from the manufacturer of the product.

PRINCIPLES OF INSTRUMENTATION

Ultrasonic technique is different from instrumentation with hand scalers. A modified pen grasp is used with an ultrasonic scaler along with an extraoral fulcrum (Figure 46-6). The purpose of the extraoral fulcrum is that it allows the operator to maintain a light grasp and easier access physically and visually to the oral cavity (Figures 46-7 and 46-8). Alternate fulcrums of cross arch or opposite arch fulcrums are acceptable alternatives.

Light pressure is needed with a power instrument. The tip is traveling at a set frequency in a set stroke pattern. Increased clinician pressure on the tip causes decreased clinical efficacy.

Sonic/ultrasonic instrumentation requires removal from the coronal to the apical portion of the deposit. This stroke pattern allows the insert to work at its optimal stroke pattern and frequency

Figure 46-6 Modified pen grasp with tip. *(Courtesy Hu-Friedy, Chicago.)*

Figure 46-7 Intraoral placement of the ultrasonic tip. *(Courtesy Hu-Friedy, Chicago.)*

Figure 46-8 Intraoral placement of the ultrasonic tip. *(Courtesy Hu-Friedy, Chicago.)*

for quick effective deposit removal. A deplaquing stroke should be used when the focus is removal of biofilm and soft debris for the resolution of gingival inflammation. This stroke entails accessing every square millimeter of the tooth surface during ultrasonic deplaquing as a result of the limited lateral dispersion of the lavage subgingivally.

HOME/SELF-APPLIED IRRIGATION

The body of evidence on the dental water jet consistently has been shown to significantly reduce gingivitis, bleeding on probing, and periodontal pathogens.* These oral health improvements have been

*References 14, 17, 26, 36, 45, 48, 51, 52, 55, 67, 68, 71, and 74.
†References 4, 5, 10, 24, 51, 52, 57, 58, 64, 65, 71, and 77.

*References 1, 3, 7, 12, 15, 16, 18–20, 23, 25, 28, 30, 32, 33, 44, 46, 47, 54, 56, 61, 63, 69, 73, 82, and 83.

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TABLE 46-1 Increased Understanding of the Benefits of a Dental Water Jet

Understanding of Periodontal Diseases (Approximate Years)	Significant Developments and Research in Dental Water Jet (DWJ) Technology
Nonspecific Plaque Era (1965)	
Plaque was considered to be a homogenous, toxic substance that caused gingivitis. Gingivitis could progress slowly to periodontitis. Amount of plaque and length of time plaque attached to tooth were paramount factors in disease. All plaque was considered essentially the same, and all plaque was considered bad.[59]	DWJ initially introduced to dental community in early 1960s. Clinical and laboratory studies showed the following: • DWJ is a significant aid to oral hygiene procedures.[8] • DWJ can decrease plaque formation.[39] • Use of DWJ significantly reduces gingivitis[19] and calculus formation as adjunct to tooth brushing.[56] • These devices do not produce bacteremia.[37,75]
Specific Plaque Hypothesis Era (1975)	
Supragingival and subgingival plaque shown to be different. Supragingival plaque begins first, then with time can grow apically and evolve into subgingival plaque, which can cause bone destruction around a tooth.[66] Elevation of specific bacterial species associated with specific periodontal diseases. Periodontal pathogens are located subgingivally.[62]	Clinical studies in the 1970s through 1990s continued to show significant clinical reductions in bleeding, inflammation, and plaque formation using DWJ and marginal irrigation with water, chlorhexidine gluconate, and essential oils.* Studies show that DWJ irrigation with a jet tip can reach subgingival areas inhabited by periodontal pathogens.[11,23,28,29]
Host Bacteria Interrelationship Era (1985) and Era of Periodontal Medicine (Present)	
Diabetes and other systemic diseases shown to make patient more susceptible to periodontal diseases. Additionally, control of periodontal disease shown to reduce need for insulin.[38]	There is an improvement in clinical parameters in diabetic patients using DWJ and reduced inflammatory mediators over traditional routine oral hygiene regimens.[1] Periodontal disease found to be an important risk factor for ALL forms of cerebrovascular disease, especially nonhemorrhagic stroke.[34,85] Moderate-to-severe periodontitis increases systemic inflammation, which is associated with cardiovascular disease and gram-negative bacteria in periodontal pockets (biofilms) have been associated with arthromas.[34] In 2009, at a consensus meeting, the *American Journal of Cardiology* and *Journal of Periodontology* issued guidelines for patients with atherosclerotic cardiovascular disease who have untreated or uncontrolled periodontitis that sated these patients should be treated with a focus on *reducing and controlling the bacterial accumulations and eliminating inflammation.*[34] Clinical benefits of oral irrigation found to be related to reduction of proinflammatory mediators in serum (i.e., reduces systemic inflammation).[26] Efficiency of DWJ irrigation at removing biofilm ex vivo and in vivo shown in scanning electron microscopy studies.[39] DWJ irrigation (along with toothbrushing) is an effective alternative to dental floss for the reduction of bleeding, gingivitis, and plaque and in some cases, may provide superior results for reducing bleeding and gingivitis.[7]

* References 3, 12, 15, 16, 20, 25, 32, 36, 46, 47, 82, 83.

demonstrated with the use of either water or an antimicrobial agent. Emerging evidence indicates the dental water jet effectively removes biofilm[40] and is as effective as dental floss when added to toothbrushing.[7,74] Table 46-1 compares the understanding of periodontal disease to significant developments in dental water jet technology.

MECHANISM OF ACTION OF IRRIGATION

The mechanism of action of irrigation occurs through the direct application of a pulsed stream of water or other solution. Studies have found pulsation and pressure to be critical components of an irrigation device.[8,9,72] Pulsating devices have been shown to be three times as effective as a continuous-stream irrigating syringe.[72] Pulsation provides for a compression and decompression phase that may account for expedient clearing of bacteria from the pocket.[8] A pulsating device allows for control of the pressure rate. The majority of studies on home irrigation that have demonstrated clinical efficacy have been done using a dental water jet with 1200 pulsations per minute set on a medium to a high pressure setting (50 psi to 90 psi)[8,9,73] (Figures 46-9 and 46-10). Dental water jets with varying pulsation and pressure are available, but like other self-care products, research from one product brand should not be extrapolated to other brands since they may have used a different pressure setting and pulsation rate.

A pulsation rate of 1200 per minute has been shown to create two zones of hydrokinetic activity[23]: the impact zone, in which the solution initially contacts, and the flushing zone, in which the solution reaches into the subgingival sulcus[40] (Figure 46-11). The outcome of hydrokinetic activity is subgingival penetration.

Animation 46-1

Pulsating action of a jet tip.

(Animation courtesy of Water Pik, Inc, Fort Collins, CO.*)*

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Figure 46-9 A dental water jet with 1200 ppm and a pressure setting that ranges from 20 psi to 90 psi. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

Figure 46-10 A cordless dental water jet, which also has 1200 ppm. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

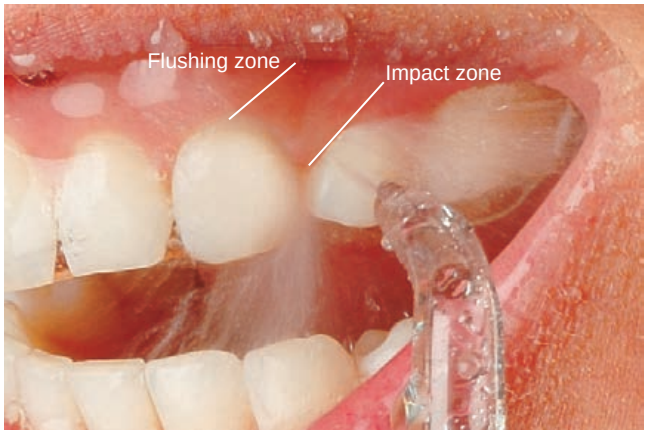

Figure 46-11 Pulsation creates two zones of hydrokinetic activity: the impact zone and the flushing zone. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

Figure 46-12 Jet tip. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

Figure 46-13 Site-specific tip. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

Home irrigation has been demonstrated to penetrate subgingivally with both a jet tip[29] (Figure 46-12) and a soft, site-specific, subgingival tip[13] (Pik Pocket subgingival irrigation tip, Water Pik, Inc, Fort Collins, CO) (Figure 46-13).

Supragingival irrigation is irrigation with a jet tip placed above the gingival margin resulting in penetration of a solution into the subgingival sulcus to approximately 50%.[11,29]

Animation 46-2

Depth of penetration with a dental water jet.

(Animation courtesy of Water Pik, Inc, Fort Collins, CO.)

The jet tip is generally used for full-mouth irrigation. Recently, other types of supragingival tips enhanced with soft, tapered bristles (Figure 46-14) have been introduced and shown to be effective in increasing the removal of plaque.[39,73] Irrigation with the soft, site-specific tip is called *subgingival irrigation.* This refers to the placement of the tip, which is placed slightly below the gingival margin (Figure 46-15). The subgingival tip is used for the localized irrigation of a specific site such as a deep pocket, furcation, implant, or crown and bridge. Studies with this site-specific, subgingival tip show that it can deliver a solution into a pocket of 6 mm or less

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Figure 46-14 Tip with soft tapered bristles. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

Figure 46-15 The Pik Pocket tip is gently placed slightly subgingivally. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

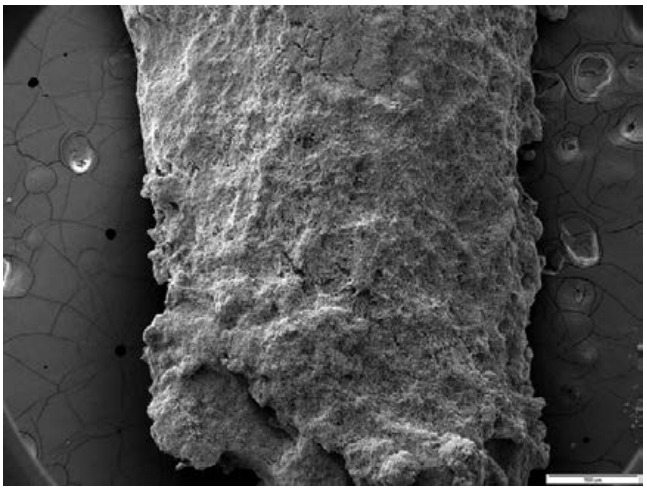

Figure 46-16 The use of the Pik Pocket tip around an implant. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

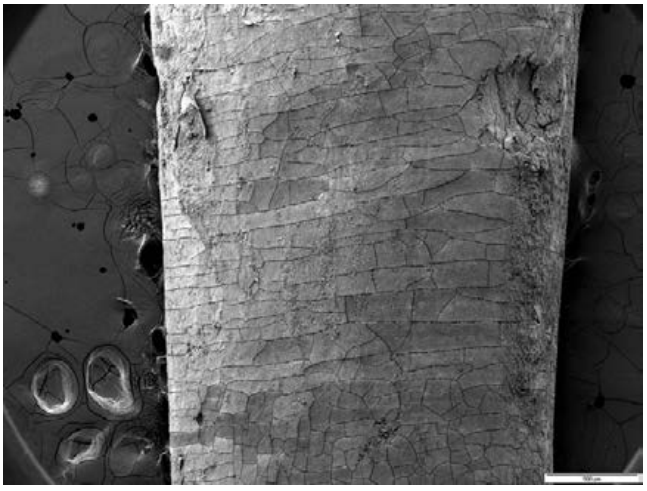

Figure 46-17 Control tooth with no irrigation. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

up to 90% of its depth.[13] In pockets over 6 mm, the depth of penetration has been shown to be 64%.[13]

Safety

Concerns regarding the safety of oral irrigation with regard to soft tissue damage and penetration of bacteria into the pocket have been voiced; although no scientific evidence exists to support these claims. Several investigators have evaluated the soft tissue and found no trauma or adverse reaction from using a pulsating oral irrigator. Researchers have found that irrigation reduces the amount of bacteria in the gingival crevice or periodontal pocket.* Specifically, Cobb et al and Drisko et al found that bacteria was reduced up to 6 mm.[23,28]

CLINICAL OUTCOMES OF IRRIGATION

Table 46-2 highlights the body of evidence on the dental water jet. Evaluated outcomes include removal of plaque biofilm and reductions in calculus, gingivitis, bleeding on probing, probing depth, periodontal pathogens, and inflammatory mediators.* Home irrigation has been studied and found safe and effective for those with gingivitis,† implants,[30] crown and bridge,[53] orthodontics,[16,46,73] intermaxillary fixation,[69] and diabetes[1]; patients who are noncompliant with dental floss[7]; and in periodontal maintenance.‡

Early studies found that irrigation with water leads to a reduction in calculus[43,56]: up to as much as 50% over brushing alone.[56] Recently, a study conducted with the scanning electron microscope found that a 3-second pulsating lavage at a 60 psi with either a jet tip or small brush tip removed 99% of plaque biofilm[39] (Figures 46-16 and 46-17).

As early as the 1960s, it was shown that oral irrigation with water added to toothbrushing reduced gingivitis by 52% compared to a 30% reduction for toothbrushing alone.[57] Throughout the years, other researchers have found concurring results with the use of plain water.§ The use of an antimicrobial agent, such as diluted

*References 15, 19, 20, 23, 28, 33, 46, 47, and 63.

*References 1, 3, 7, 12, 15, 16, 19, 20, 25, 30–33, 43, 44, 47, 54, 56, 61, 69, 73, 82, and 83.
†References 15, 19, 20, 23, 28, and 63.
‡References 25, 31, 32, 47, 61, and 73.
§References 1, 7, 19, 25, 33, 43, 44, 56, 61, and 73.

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TABLE 46-2 Reduction of Inflammation and Plaque Biofilm

Study	Duration	N	Agent Used	% Bleeding Reduction	% Gingivitis Reduction	% Plaque Biofilm Reduction
Al-Mubarak et al[1]	3 months	50	Water	43.8	66.9	64.9
Barnes et al[7]	4 weeks	105	Water	36.2–59.2	10.8–15.1	8.8–17.3
Brownstein et al[15]	8 weeks	44	CHX (0.06%)	52–59	25.4–31.1*	14.3–19*
			Water	NR		NR
Burch et al[16]	2 months	47	Water	57.1–76.6	NR	52–55.7
Chaves et al[19]	6 months	105	CHX (0.04%)	54	26	35
			Water	50	26	16
Ciancio et al[20]	6 weeks	61	Essential oils†	27.6	54–55.7	23–24
			Water and alcohol 5%	13.6–31.2	59.8–61.9	9.6–13.3
Cutler et al[25]	2 weeks	52	Water	56	50	40
Flemmig et al[33]	6 months	175	CHX (0.06%)	35.4	42.5	53.2
			Water	24	23.1	0.1
Flemmig et al[32]	6 months	60	Acetylsalicylic acid 3%		8.9	55.6
			Water	50	29.2	0
Felo et al[30]	3 months	24	CHX (0.06%)	62	45	29
Fine et al[31]	6 weeks	50	Essential oils†	14.8–21.7	NR	36.8–37.7
			Water	7.5–10.6	NR	15.5–18.4
Jolkovsky et al[47]	3 months	58	CHX (0.4%)	NR	33.1	51.6
			Water	NR	18.6	25.6
Lobene et al[56]	5 months	155	Water	NR	52.9	7.9
Newman et al[61]	6 months	155	Water	22.8	17.8	6.1
			Water and zinc sulfate (0.57%)	8.8	6.5	9.2
Sharma et al[73]	4 weeks	128	Water	84.5	NR	38.9
Walsh et al[82]	8 weeks	8	CHX (0.2%)	NR	45	77
			Quinine salt	NR	14	0

Data from Cianco SG: *Compendium Contin Educ Dent* 30: 7–14, 2009.
CHX, Chlorhexidine; *NR,* not reported.
*Percentages were reported for differences between CHX and water irrigation groups.
†Reported the range for prophy and nonprophy groups.

chlorhexidine or an essential oil, generally enhances reductions in gingivitis and bleeding.*

Emerging evidence indicates that home irrigation may play a role in modulating the host response, particularly the inflammatory mediators associated with clinical attachment loss and alveolar bone loss.[1,25] This evidence lends support to the safety of irrigation, as well as its potential for helping periodontal maintenance patients maintain stability.

INDIVIDUALS WITH SPECIAL CONSIDERATIONS

Some clinical trials have focused on groups with special oral or medical health needs. Both children and adults undergoing orthodontic therapy have shown significant benefits from using a dental water jet.[16,73] A new, small brush tip that cleans and irrigates simultaneously has been shown to remove 3.76 times more plaque than brushing and flossing with a floss threader.[73] For individuals with implants, a soft, site-specific subgingival tip used with 0.06% chlorhexidine improved oral health better than rinsing with 0.12% chlorhexidine[30] (Figure 46-18). For those with special medical considerations, home irrigation has been studied and shown safe and effective in a group of individuals with either type 1 or type 2 diabetes.[1]

*References 3, 15, 19, 20, 32–34, 47, and 82.

TABLE 46-3 Chlorhexidine Dilutions (Based on 0.12% Concentration) Shown Effective in Clinical Trials

Concentrations	Amount of Water	Amount of Chlorhexidine
0.02%[3,82]	5 parts	1 part
0.04%[20,47]	3 parts	1 part
0.06%[15,30,33,63]	1 part	1 part

Various solutions can be used in a dental water jet. Plain water,* diluted chlorhexidine† (Table 46-3), and full-strength essential oil[20,31] all have evidence to support their use.

SUMMARY

Power scalers have emerged from adjuncts for removing heavy supragingival calculus to a tool that may be used for all aspects of scaling: deplaquing, supragingival scaling, and subgingival scaling. The clinical outcomes achieved are similar to those for hand instrumentation. The advantages gained from using power instruments are potentially greater access subgingivally and in furcation areas and increased efficiency in time needed for scaling.

*References 1, 3, 7, 16, 18, 19, 23, 32, 33, 43, 46, 54, 56, 61, 69, 71, and 82.
†References 3, 15, 19, 30, 33, 47, 63, and 82.

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SCIENCE TRANSFER

Sonic and ultrasonic instruments are as effective as hand instruments used for root planing. They offer advantages in that they are efficient and the new smaller tips have better access to hard-to-reach subgingival areas such as narrow furcations and root concavities. Some of the older implanted cardiac pacemakers could be inhibited by magnetostrictive ultrasonic instruments, thus these instruments should not be used for these patients.

Sonic and ultrasonic instruments are primarily used during initial therapy, but they also are valuable during periodontal surgery to remove residual calculus from previously inaccessible locations such as furcations, grooves concavities, and distal surfaces of posterior teeth.

Powered irrigation oral hygiene devices may add to the effectiveness of the patient's other oral hygiene procedures when used with water or with antimicrobial solutions such as chlorhexidine. Pulsed jets are more effective than constant stream devices and can give reductions in gingivitis, bleeding on probing, and periodontal pathogenic bacteria similar to that seen with flossing. These devices are particularly useful in patients with fixed orthodontic appliances, complex prosthetic treatment, and intermaxillary fixation, as well as in patients noncompliant with floss.

BOX 46-1 Advantages and Disadvantages of Mechanized Instruments as Compared with Manual Instruments

Advantages	Disadvantages
Increased efficiency	More precautions and limitations
Multiple surfaces of tip are capable of removing deposits	Client comfort (water spraying)
No need to sharpen	Aerosol production
Less chance for repetitive stress injuries	Temporary hearing shifts
Handpiece size large	Noise
Reduced lateral pressure	Less tactile sensation
Less tissue distention	Reduced visibility
Water	
Lavage	
Irrigation	
Acoustic microstreaming	

From Darby ML, Walsh MM: *Dental hygiene*, 3 ed, Saunders, St Louis, 2010.

Figure 46-18 Tooth after a 3-second pulsating lavage with a jet tip at medium pressure. *(Courtesy Water Pik, Inc, Fort Collins, CO.)*

Home irrigation is safe and effective for a wide variety of patients, including those in periodontal maintenance; those with calculus buildup, gingivitis, orthodontic appliances, maxillary fixation, crown and bridge, implants, and diabetes; and those who are noncompliant with floss. Clinical outcomes include the reduction of plaque, calculus, gingivitis, bleeding on probing, probing depth, periodontal pathogens, and inflammatory mediators.

BOX 46-2 Indications, Precautions, and Contraindications for Use of Mechanized Instruments

Indications
- Supragingival debridement of dental calculus and extrinsic stain
- Subgingival debridement of calculus, oral biofilm, root surface constituents, and periodontal pathogens
- Removal of orthodontic cement
- Gingival and periodontal conditions and diseases
- Surgical interventions
- Margination (reduces amalgam overhangs)

Precautions
- Unshielded pacemakers
- Infectious diseases: human immunodeficiency virus, hepatitis, tuberculosis (active stages)
- Demineralized tooth surface
- Exposed dentin (especially associated with sensitivity)
- Restorative materials (porcelain, amalgam, gold, composite)
- Titanium implant abutments unless using special insert, e.g., Quixonic SofTip Prophy Tips
- Children (primary teeth)
- Immunosuppression from disease or chemotherapy
- Uncontrolled diabetes mellitus

Contraindications
- Chronic pulmonary disease: asthma, emphysema, cystic fibrosis, pneumonia
- Cardiovascular disease with secondary pulmonary disease
- Swallowing difficulty (dysphagia)

From Darby ML, Walsh MM: *Dental hygiene*, 3 ed, Saunders, St Louis, 2010.

To view video 46-1: the pulsating action of a jet tip, please visit the companion Expert CONSULT website at www.expertconsult.com

To view video 46-2: the depth of penetration with a dental water jet, please visit the companion Expert CONSULT website at www.expertconsult.com

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 47

Antiinfective Therapy

Sebastian Ciancio and Angelo Mariotti

CHAPTER OUTLINE

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Comparative Studies
LOCAL DELIVERY OF ANTIMICROBIAL AGENTS AND PERIIMPLANT MUCOSITIS/IMPLANTITIS
CONCLUSION

It is well established that the various periodontal diseases are caused by bacterial infection. Bacteria begin reattaching to the crowns of teeth soon after the teeth have been cleaned and begin to form a biofilm. Over time, this supragingival plaque biofilm becomes more complex, leading to a succession of bacteria that are more pathogenic. Bacteria grow in an apical direction and become subgingival, and eventually, as bone is destroyed, a periodontal pocket is formed. In a periodontal pocket the bacteria form a highly structured and complex *biofilm*. As this process continues, the bacterial biofilm extends so far subgingivally that the patient cannot reach it during oral hygiene efforts. Additionally, this complex biofilm now may offer some protection from the host's immunologic mechanisms in the periodontal pocket, as well as from antibiotics used for treatment. It has been suggested that an antibiotic strength 500 times greater than the usual therapeutic dose may be needed to be effective against bacteria arranged in biofilms.[40]

It is therefore logical to treat periodontal pockets by mechanical removal of local factors (including calculus that harbors bacteria) and also by disruption of the subgingival plaque biofilm itself. Mechanical removal includes manual instrumentation (e.g., scaling and root planing) and machine-driven instrumentation (e.g., ultrasonic scalers), and these procedures can be considered "antiinfective therapy." Many chemotherapeutic agents are now available to clinicians treating periodontal diseases. *Systemic* antiinfective therapy (oral antibiotics) and *local* antiinfective therapy (placing antiinfective agents directly into the periodontal pocket) can reduce the bacterial challenge to the periodontium. It is also possible that systemically administered nonsteroidal antiinflammatory agents (NSAIDs) may play a role in future adjunctive therapy.[68,93]

Bacteria and their toxic products cause loss of attachment and loss of bone. Ultimately, however, the host's own immunologic response to this bacterial infection can cause even more bone destruction ("indirect bone loss") than that caused by pathogenic bacteria and their by-products. This immunologic response can be influenced by environmental (e.g., tobacco use), acquired (e.g., systemic disease), and genetic risk factors.[77] Chemotherapeutic agents can modulate the host's immune response to bacteria and reduce the host's self-destructive immunologic response to bacterial pathogens and thus reduce bone loss.[71,72,75] It is also incumbent on health care providers to counsel patients concerning the detrimental effects of systemic factors, including medications, stress, and tobacco use.[40]

This chapter reviews the indications and protocols for optimizing the use of antiinfective agents in the treatment of periodontal diseases.

It is important to note that significant work has been performed in a systematic *evidence-based approach* to evaluate the various antiinfective and host modulation therapies[99] (see Chapters 78 and 79). Metaanalysis of similar research studies has given power to statistical analysis for evaluating antiinfective chemotherapeutic agents in the treatment of periodontal diseases. Unfortunately, a standardized research protocol has not yet been implemented. Therefore some studies, although relevant, have not been used in the evidence-based approach because of their study design. Further evidence-based and similar research is needed to define protocols more

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precisely for the use of antiinfective agents in treating periodontal diseases.

DEFINITIONS

An antiinfective agent is a *chemotherapeutic agent* that acts by reducing the number of bacteria present. An antibiotic is a naturally occurring, semisynthetic, or synthetic type of antiinfective agent that destroys or inhibits the growth of selective microorganisms, generally at low concentrations. An antiseptic is a chemical antimicrobial agent applied topically or subgingivally to mucous membranes, wounds, or intact dermal surfaces to destroy microorganisms and inhibit their reproduction or metabolism. In dentistry, antiseptics are widely used as the active ingredient in antiplaque and antigingivitis oral rinses and dentifrices. Disinfectants, a subcategory of antiseptics, are antimicrobial agents that are generally applied to inanimate surfaces to destroy microorganisms.[21]

Antiinfective agents can be administered *locally* or *orally*. When administered orally, many of these agents can be found in gingival crevicular fluid (GCF). With either approach, their purpose is to reduce the number of bacteria present in the diseased periodontal pocket. *Systemic administration* of antibiotics may be a necessary adjunct in controlling bacterial infection because bacteria can invade periodontal tissues, making mechanical therapy alone sometimes ineffective.[4,18,20,31,76] *Local administration* of antiinfective agents, generally directly in the pocket, has the potential to provide greater concentrations directly to the infected area and reduce possible systemic side effects.

Additionally, a single chemotherapeutic agent can have a dual mechanism of action. For example, tetracyclines (especially doxycycline) are chemotherapeutic agents that can reduce collagen and bone destruction through their ability to inhibit the enzyme collagenase. As antibiotic agents, they also can reduce periodontal pathogens in periodontal tissues.[20]

SYSTEMIC ADMINISTRATION OF ANTIBIOTICS

Background and Rationale

The treatment of periodontal diseases is based on the infectious nature of these diseases (Table 47-1). Ideally, the causative microorganism(s) should be identified and the most effective agent should be selected using antibiotic-sensitivity tests. Although this appears simple, the difficulty lies primarily in identifying specific etiologic microorganism(s) rather than microorganisms simply associated with various periodontal disorders.[19,20]

An ideal antibiotic for use in prevention and treatment of periodontal diseases should be specific for periodontal pathogens, allogenic and nontoxic, substantive, not in general use for treatment of other diseases, and inexpensive.[32] Currently, an ideal antibiotic for the treatment of periodontal diseases does not exist.[49] Although oral bacteria are susceptible to many antibiotics, no single antibiotic at concentrations achieved in body fluids inhibits all putative periodontal pathogens.[97] Indeed, a combination of antibiotics may be necessary to eliminate all putative pathogens from some periodontal pockets[70] (Table 47-2).

As always, the clinician in concert with the patient must make the final decision on any treatment. Thus the treatment of the individual patient must be based on the patient's clinical status, nature of the colonizing bacteria, ability of the agent to reach the site of infection, and the risks and benefits associated with the proposed treatment plan. The clinician is responsible for choosing the correct antimicrobial agent. Some adverse reactions include allergic/anaphylactic reactions, superinfections of opportunistic bacteria, development of resistant bacteria, interactions with other medications, upset stomach, nausea, and vomiting.[6] Most adverse reactions take the form of gastrointestinal upset.[49] Other concerns include the cost of the medication and the patient's willingness and ability to comply with the proposed therapy.

No consensus exists concerning the magnitude of the risk for developing bacterial resistance. Common and indiscriminate use of antibiotics worldwide has contributed to increasing numbers of resistant bacterial strains over the last 15 to 20 years, and this trend is likely to continue given the widespread use of antibiotics.[96] The overuse, misuse, and widespread prophylactic application of antiinfective drugs are some of the factors that have led to the emergence of resistant microorganisms. Increasing levels of resistance of subgingival microflora to antibiotics has been correlated with the increased use of antibiotics in individual countries.[91] However, researchers have noted that the subgingival microflora tends to revert to similar proportions of antibiotic-resistant isolates 3 months after therapy.[29,43]

TABLE 47-1 Antibiotics Used to Treat Periodontal Diseases

Category	Agent	Major Features
Penicillin*	Amoxicillin	Extended spectrum of antimicrobial activity; excellent oral absorption; used systemically.
	Augmentin†	Effective against penicillinase-producing microorganisms; used systemically.
Tetracyclines	Minocycline	Effective against broad spectrum of microorganisms; used systemically and applied locally (subgingivally).
	Doxycycline	
	Tetracycline	Effective against broad spectrum of microorganisms; used systemically and applied locally (subgingivally).
		Chemotherapeutically used in sub-antimicrobial dose for host modulation (Periostat).
		Effective against broad spectrum of microorganisms.
Quinolone	Ciprofloxacin	Effective against gram-negative rods; promotes health-associated microflora.
Macrolide	Azithromycin	Concentrates at sites of inflammation; used systemically.
Lincomycin derivative	Clindamycin	Used in penicillin-allergic patients; effective against anaerobic bacteria; used systemically.
Nitroimidazole‡	Metronidazole	Effective against anaerobic bacteria; used systemically and applied locally (subgingivally) as gel.

**Indications*: localized aggressive periodontitis (LAP), generalized aggressive periodontitis (GAP), medically related periodontitis (MRP), refractory periodontitis (RP).
†Amoxicillin and clavulanate potassium.
‡*Indications*: LAP, GAP, MRP, RP, necrotizing ulcerative periodontitis.

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TABLE 47-2 Common Antibiotic Regimens Used to Treat Periodontal Diseases*

	Regimen	Dosage/Duration
Single Agent		
Amoxicillin	500 mg	Three times daily for 8 days
Azithromycin	500 mg	Once daily for 4–7 days
Ciprofloxacin	500 mg	Twice daily for 8 days
Clindamycin	300 mg	Three times daily 10 days
Doxycycline or minocycline	100–200 mg	Once daily for 21 days
Metronidazole	500 mg	Three times daily for 8 days
Combination Therapy		
Metronidazole + amoxicillin	250 mg of each	Three times daily for 8 days
Metronidazole + ciprofloxacin	500 mg of each	Twice daily for 8 days

Data from Jorgensen MG, Slots J: *Compend Contin Educ Dent* 21:111, 2000.
*These regimens are prescribed with a review of the patient's medical history, periodontal diagnosis, and antimicrobial testing. Clinicians must consult pharmacology references such as *Mosby's GenRx*[67] or manufacturer's guidelines for warnings, contraindications, and precautions.

Tetracyclines

Tetracyclines have been widely used in the treatment of periodontal diseases. They have been frequently used in treating refractory periodontitis, including *localized aggressive periodontitis* (LAP)[100] (see Table 47-1). Tetracyclines have the ability to concentrate in the periodontal tissues and inhibit the growth of *Aggregatibacter actinomycetemcomitans.* In addition, tetracyclines exert an anticollagenase effect that can inhibit tissue destruction and may aid bone regeneration.[17,59,95]

Pharmacology. The tetracyclines are a group of antibiotics produced naturally from certain species of *Streptomyces* or derived semisynthetically. These antibiotics are bacteriostatic and are effective against rapidly multiplying bacteria. They generally are more effective against gram-positive bacteria than gram-negative bacteria. Tetracyclines are effective in treating periodontal diseases in part because their concentration in the gingival crevice is 2 to 10 times that in serum.[2,7,37] This allows a high drug concentration to be delivered into periodontal pockets. In addition, several studies have demonstrated that tetracyclines at a low GCF concentration (2 to 4 μg/ml) are very effective against many periodontal pathogens.[8,9]

Clinical Use. Tetracyclines have been investigated as adjuncts in the treatment of LAP.[82] *A. actinomycetemcomitans* is a frequent microorganism associated with LAP and is tissue invasive. Therefore mechanical removal of calculus and plaque from root surfaces may not eliminate this bacterium from the periodontal tissues. Systemic tetracycline can eliminate tissue bacteria and has been shown to arrest bone loss and suppress *A. actinomycetemcomitans* levels in conjunction with scaling and root planing.[81] This combination therapy allows mechanical removal of root surface deposits and elimination of pathogenic bacteria from within the tissues. Increased posttreatment bone levels have been noted using this method.[85] Because of increased resistance to tetracyclines, metronidazole or amoxicillin with metronidazole has been found more effective in treating aggressive periodontitis in children. Some investigators believe metronidazole combined with amoxicillin–clavulanic acid is the preferable antibiotic.[94]

Long-term use of low antibacterial doses of tetracyclines has been advocated in the past. One long-term study of patients taking low doses of tetracycline (250 mg/day for 2 to 7 years) showed persistence of deep pockets that did not bleed on probing. These sites contained high proportions of tetracycline-resistant, gram-negative rods *(Fusobacterium nucleatum).* After the antibiotic was discontinued, the flora was characteristic of sites with disease.[49] Therefore it is not advisable to prescribe long-term regimens of tetracyclines because of the possible development of resistant bacterial strains.[55] Although often used in the past as antiinfective agents, especially for LAP and other types of aggressive periodontitis, tetracyclines now tend to be replaced by more effective combination antibiotics.[49]

Specific Agents. Tetracycline, minocycline, and doxycycline are semisynthetic members of the tetracycline group that have been used in periodontal therapy.

Tetracycline. Tetracycline HCl requires administration of 250 mg, 4 times daily (qid). It is inexpensive, but compliance may be reduced by having to take four capsules per day. Side effects are gastrointestinal (GI) disturbances, photosensitivity, hypersensitivity, increased blood urea nitrogen (BUN), blood dyscrasias, dizziness, and headache. Also, tooth discoloration occurs when administered to children up to age 12 years.

Minocycline. Minocycline is effective against a broad spectrum of microorganisms. In patients with adult periodontitis, it suppresses spirochetes and motile rods as effectively as scaling and root planing, with suppression evident up to 3 months after therapy. Minocycline can be given twice daily (bid), thus facilitating compliance compared with tetracycline. Although associated with less phototoxicity and renal toxicity than tetracycline, minocycline may cause reversible vertigo. Minocycline administered 200 mg/day for 1 week results in a reduction in total bacterial counts, complete elimination of spirochetes for up to 2 months, and improvement in all clinical parameters.[21,24]

Side effects are similar to those of tetracycline; however, there is an increased incidence of vertigo. It is the only tetracycline that can discolor permanently erupted teeth and gingival tissue when administered orally.

Doxycycline. Doxycycline has the same spectrum of activity as minocycline and may be equally as effective.[20] Because doxycycline can be given only once daily (qd), patients may be more compliant. Compliance is also favored because its absorption from the GI tract is only slightly altered by calcium, metal ions, or antacids, as is absorption of other tetracyclines. Side effects are similar to tetracycline HCl; however, it is the most photosensitizing agent in the tetracycline category.

The recommended dosage when used as an antiinfective agent is 100 mg bid the first day, then 100 mg qd. To reduce GI upset, 50 mg can be taken bid. When used in a sub-antimicrobial dose (to inhibit collagenase), doxycycline is recommended in a 20-mg dose twice daily.[17,26] Periostat (CollaGenex Pharmaceuticals Inc, Newtown, PA) and generic forms are currently available in a dose of 20 mg of doxycycline.

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Metronidazole

Pharmacology. Metronidazole is a nitroimidazole compound developed in France to treat protozoal infections. It is bactericidal to anaerobic organisms and is believed to disrupt bacterial deoxyribonucleic acid (DNA) synthesis in conditions with a low reduction potential. Metronidazole is not the drug of choice for treating *A. actinomycetemcomitans* infections. However, metronidazole is effective against *A. actinomycetemcomitans* when used in combination with other antibiotics.[69,70] Metronidazole is also effective against anaerobes such as *Porphyromonas gingivalis* and *Prevotella intermedia*.[39]

Clinical Use. Metronidazole has been used clinically to treat gingivitis, acute necrotizing ulcerative gingivitis (NUG), chronic periodontitis, and aggressive periodontitis. It has been used as monotherapy and also in combination with both root planing and surgery or with other antibiotics. Metronidazole has been used successfully to treat NUG.[61]

Studies in humans have demonstrated the efficacy of metronidazole in the treatment of gingivitis and periodontitis.[60] A single dose of metronidazole (250 mg orally) appears in both serum and GCF in sufficient quantities to inhibit a wide range of suspected periodontal pathogens. Administered systemically (750 to 1000 mg/day for 2 weeks), metronidazole reduces the growth of anaerobic flora, including spirochetes, and decreases the clinical and histopathologic signs of periodontitis.[60] The most common regimen is 250 mg 3 times daily (tid) for 7 days.[61] Currently, the critical level of spirochetes needed to diagnose an anaerobic infection, the appropriate time to give metronidazole, and the ideal dosage or duration of therapy are unknown.[39] As monotherapy (no concurrent root planing), metronidazole is inferior and at best only equivalent to root planing. Therefore, if used, metronidazole should not be administered as monotherapy.

Soder et al[83] showed that metronidazole was more effective than placebo in the management of sites unresponsive to root planing. Nevertheless, many patients still had sites that bled on probing despite metronidazole therapy. The existence of refractory periodontitis as a diagnostic consideration indicates that some patients do not respond to conventional therapy, including root planing, surgery, or both.

Studies have suggested that when combined with amoxicillin or amoxicillin–clavulanate potassium (Augmentin), metronidazole may be of value in the management of patients with LAP or refractory periodontitis (see later discussion).

Side Effects. Metronidazole has an Antabuse effect when alcohol is ingested. The response is generally proportional to the amount ingested and can result in severe cramps, nausea, and vomiting. Products containing alcohol should be avoided during therapy and for at least 1 day after therapy is discontinued. Metronidazole also inhibits warfarin metabolism. Patients undergoing anticoagulant therapy should avoid metronidazole because it prolongs prothrombin time.[61] It also should be avoided in patients who are taking lithium. It also produces a metallic taste in the mouth, which may affect compliance.

Penicillins

Pharmacology. Penicillins are the drugs of choice for the treatment of many serious infections in humans and are the most widely used antibiotics. Penicillins are natural and semisynthetic derivatives of broth cultures of the *Penicillium* mold. They inhibit bacterial cell wall production and therefore are bactericidal.

Clinical Use. Penicillins other than amoxicillin and amoxicillin–clavulanate potassium (Augmentin) have not been shown to increase periodontal attachment levels, and their use in periodontal therapy does not appear to be justified.

Side Effects. Penicillins may induce allergic reactions and bacterial resistance; up to 10% of patients may be allergic to penicillin.

Amoxicillin. Amoxicillin is a semisynthetic penicillin with an extended antiinfective spectrum that includes gram-positive and gram-negative bacteria. It demonstrates excellent absorption after oral administration. Amoxicillin is susceptible to penicillinase, a β-lactamase produced by certain bacteria that breaks the penicillin ring structure and thus renders penicillins ineffective.

Amoxicillin may be useful in the management of patients with aggressive periodontitis, in both localized and generalized forms. Recommended dosage is 500 mg tid for 8 days.[49,50]

Amoxicillin–Clavulanate Potassium. The combination of amoxicillin with clavulanate potassium makes this antiinfective agent resistant to penicillinase enzymes produced by some bacteria. Amoxicillin with clavulanate (Augmentin) may be useful in the management of patients with LAP or refractory periodontitis.[68] Bueno et al[13] reported that Augmentin arrested alveolar bone loss in patients with periodontal disease that was refractory to treatment with other antibiotics, including tetracycline, metronidazole, and clindamycin.

Cephalosporins

Pharmacology. The family of β-lactams known as cephalosporins is similar in action and structure to penicillins. They are frequently used in medicine and are resistant to a number of β-lactamases normally active against penicillin.

Clinical Use. Cephalosporins are generally not used to treat dental-related infections. The penicillins are superior to cephalosporins in their range of action against periodontal pathogenic bacteria.

Side Effects. Patients allergic to penicillins must be considered allergic to all β-lactam products. Rashes, urticaria, fever, and GI upset have been associated with cephalosporins.[96]

Clindamycin

Pharmacology. Clindamycin is effective against anaerobic bacteria and has a strong affinity for osseous tissue.[89] It is effective in situations in which the patient is allergic to penicillin.

Clinical Use. Clindamycin has shown efficacy in patients with periodontitis refractory to tetracycline therapy. Walker et al[96] showed that clindamycin assisted in stabilizing refractory patients; dosage was 150 mg qid for 10 days. Jorgensen and Slots[50] recommend a regimen of 300 mg bid for 8 days.

Side Effects. Clindamycin has been associated with pseudomembranous colitis, but the incidence is higher with cephalosporins and ampicillin. When needed, however, clindamycin can be used with caution, but it is not indicated in patients with a history of colitis. Diarrhea or cramping that develops during clindamycin therapy may be indicative of colitis, and clindamycin should be discontinued. If symptoms persist, the patient should be referred to an internist.

Ciprofloxacin

Pharmacology. Ciprofloxacin is a quinolone active against gram-negative rods, including all facultative and some anaerobic putative periodontal pathogens.[80]

Clinical Use. Because it demonstrates minimal effect on *Streptococcus* species, which are associated with periodontal health, ciprofloxacin therapy may facilitate the establishment of a microflora associated with periodontal health. At present, ciprofloxacin is the only antibiotic in periodontal therapy to which all strains of *A. actinomycetemcomitans* are susceptible. It also has been used in combination with metronidazole.[70]

Side Effects. Nausea, headache, metallic taste in the mouth, and abdominal discomfort have been associated with ciprofloxacin. Quinolones inhibit the metabolism of theophylline, and caffeine and concurrent administration can produce toxicity. Quinolones have also been reported to enhance the effect of warfarin and other anticoagulants.[96]

Macrolides

Pharmacology. Macrolide antibiotics contain a many-membered lactone ring to which one or more deoxy sugars are attached. They inhibit protein synthesis by binding to the 50S ribosomal subunits of sensitive microorganisms. Macrolides can be bacteriostatic or bactericidal, depending on the concentration of the drug and the nature of the microorganism. The macrolide antibiotics used for periodontal treatment include erythromycin, spiramycin, and azithromycin.

Clinical Use. Erythromycin does not concentrate in GCF, and it is not effective against most putative periodontal pathogens. For these reasons, erythromycin is not recommended as an adjunct to periodontal therapy.

Spiramycin is active against gram-positive organisms; it is excreted in high concentrations in saliva. It is used as an adjunct to periodontal treatment in Canada and Europe but is not available in the United States (US). Spiramycin has minimal effect on increasing attachment levels.

Azithromycin is a member of the azalide class of macrolides. It is effective against anaerobes and gram-negative bacilli. After an oral dosage of 500 mg qd for 3 days, significant levels of azithromycin can be detected in most tissues for 7 to 10 days.[10,47] The concentration of azithromycin in tissue specimens from periodontal lesions is significantly higher than that of normal gingiva.[62] It has been proposed that azithromycin penetrates fibroblasts and phagocytes in concentrations 100 to 200 times greater than that of the extracellular compartment. The azithromycin is actively transported to sites of inflammation by phagocytes, then released directly into the sites of inflammation as the phagocytes rupture during phagocytosis.[33] Therapeutic use requires a single dose of 250 mg/day for 5 days after an initial loading dose of 500 mg.[96]

Recent data have suggested that azithromycin can be an effective adjunctive therapy for increasing attachment levels in patients with aggressive periodontitis,[41] as well as in reducing the degree of gingival enlargement.[25] At this time, these data must be carefully considered since the outcomes are derived from small subject populations. To ascertain the efficacy of azithromycin in the management of periodontal diseases, future studies will need to increase the number of subjects, improve diagnostic methods and tools, and determine appropriate dose, duration, and frequency of azithromycin therapy.

SERIAL AND COMBINATION ANTIBIOTIC THERAPY

Rationale

Because periodontal infections may contain a wide diversity of bacteria, no single antibiotic is effective against all putative pathogens. Indeed, differences exist in the microbial flora associated with the various periodontal disease syndromes.[100] These "mixed" infections can include a variety of aerobic, microaerophilic, and anaerobic bacteria, both gram-negative and gram-positive. In these cases, it may be necessary to use more than one antibiotic, either serially or in combination.[69] Before combinations of antibiotics are used, however, the periodontal pathogen(s) being treated must be identified and antibiotic-susceptibility testing performed.[98]

Clinical Use

Antibiotics that are bacteriostatic (e.g., tetracycline) generally require rapidly dividing microorganisms to be effective. They do not function well if a bactericidal antibiotic (e.g., amoxicillin) is given concurrently. *When both types of drugs are required, they are best given serially, not in combination.*

Rams and Slots[69] reviewed combination therapy using systemic metronidazole along with amoxicillin, amoxicillin-clavulanate (Augmentin), or ciprofloxacin. The metronidazole-amoxicillin and metronidazole-Augmentin combinations provided excellent elimination of many organisms in adult and LAP that had been treated unsuccessfully with tetracyclines and mechanical debridement. These drugs have an additive effect regarding suppression of *A. actinomycetemcomitans*. Tinoco et al[87] found metronidazole and amoxicillin to be clinically effective in treating LAP, although 50% of patients harbored *A. actinomycetemcomitans* 1 year later. Metronidazole-ciprofloxacin combination is effective against *A. actinomycetemcomitans*; metronidazole targets obligate anaerobes, and ciprofloxacin targets facultative anaerobes. This is a powerful combination against mixed infections. Studies of this drug combination in the treatment of refractory periodontitis have documented marked clinical improvement. This combination may provide a therapeutic benefit by reducing or eliminating pathogenic organisms and a prophylactic benefit by giving rise to a predominantly streptococcal microflora.[70]

Systemic antibiotic therapy combined with mechanical therapy appears valuable in the treatment of recalcitrant periodontal infections and LAP infections involving *A. actinomycetemcomitans*. Antibiotic treatment should be reserved for specific subsets of periodontal patients who do not respond to conventional therapy. Selection of specific agents should be guided by the results of cultures and sensitivity tests for subgingival plaque microorganisms.

Pharmacologic Implications

Principles of antibiotic therapy for the proper selection of an antibiotic minimally require identification of the causative organism, determination of the antibiotic sensitivity, and an effective method of administration.[45] The use of antibiotics to treat gingival diseases is contraindicated since it is a local infection that can be easily treated with scaling and appropriate home care by the patient.[86] In regard to destructive periodontal diseases, there are limited data to support the use of systemic antibiotics in the treatment of these diseases. Although bacterial infections of the periodontium are considered to be important in the initiation of the disease, currently, there is no one microbe or group of microbes that have been demonstrated as the cause of these diseases. It is therefore not surprising

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SCIENCE TRANSFER

Antiinfective agents are used either locally in delayed release products or systemically with administration by mouth. In patients with periodontitis, local antiinfective agents, such as chlorhexidine (PerioChip), doxycycline (Atridox), or minocycline (Arestin), used with scaling and root planing, have all been shown to give modest changes in average pocket depth reduction when compared to control areas in which scaling and root planing was used alone. Periimplantitis also can be treated with locally released antimicrobial agents such as doxycycline and minocycline.

Systemic antibiotics have demonstrated similar results to local agents when used in conjunction with sealing and root planing. The most effective of these is metronidazole used alone or in conjunction with amoxicillin or Augmentin on a daily basis for 1 to 2 weeks.

These antiinfective approaches are not as dramatic in their effects on advanced periodontitis as periodontal regenerative surgical techniques. For this reason, they are best used in those patients in whom periodontal surgery is contraindicated or as a precursor to surgery during initial therapy.

that systemic antibiotics have had only a modest effect in the management of periodontal diseases. At this time, systemic antibiotics for the treatment of periodontal diseases have been indicated primarily for adjunctive use in the treatment of aggressive periodontal diseases.[40,43]

Guidelines for the use of antibiotics in periodontal therapy include the following:

1. The clinical diagnosis and situation dictate the need for possible antibiotic therapy as an adjunct in controlling active periodontal disease (Figure 47-1). The patient's diagnosis can change over time. For example, a patient who presents with generalized, mild chronic periodontitis can return to a diagnosis of periodontal health after initial therapy. However, if this patient has been treated and continues to have active disease, the diagnosis may change to generalized severe chronic periodontitis.
2. Disease activity, as measured by continuing attachment loss, purulent exudate, and bleeding on probing,[56,57] may be an indication for periodontal intervention and possible microbial analysis through plaque sampling.
3. When used to treat periodontal disease, antibiotics are selected based on the patient's medical and dental status, current medications,[49] and results of microbial analysis, if performed.
4. Microbiologic plaque sampling may be performed according to the instructions of the reference laboratory. The samples usually are taken at the beginning of an appointment before instrumentation of the pocket. Supragingival plaque is removed, and an endodontic paper point is inserted subgingivally into the deepest pocket(s) present to absorb bacteria in the loosely associated plaque. This endodontic point is placed in reduced transfer fluid or a sterile transfer tube and sent overnight to the laboratory. The laboratory will then send the referring dentist a report that includes the pathogens present and any appropriate antibiotic regimen. At this time, there are scant data to suggest that microbial identification from a plaque sample can be used to clinically improve the periodontal condition of the patient.
5. Metaanalysis of randomized clinical trials and quasi-experimental studies have shown systemic antibiotics can improve attachment levels when used as adjuncts to scaling and root planing. The same benefits could not be shown when antibiotics were used as a stand-alone therapy.[43]
6. When systemic antibiotics were used as adjuncts to scaling and root planing, improvements were observed in the attachment levels of patients with chronic and aggressive periodontitis, although patients with aggressive periodontitis experienced greater benefits.[43] The mean attachment level change depended on the antibiotic used and ranged from 0.09 to 1.10 mm.[43]
7. Identification of which antibiotics were most effective for the treatment of destructive periodontal diseases was limited because of the insufficient size of sample found in randomized clinical trials of a systematic review.[43] Using a meta-analysis to evaluate eight different antibiotics or antibiotic combinations, only tetracycline and metronidazole were shown to significantly improve attachment levels when used as adjuncts to scaling and root planing for patients with destructive periodontal diseases.[43]
8. Debridement of root surfaces, optimal oral hygiene, and frequent periodontal maintenance therapy are important parts of comprehensive periodontal therapy. As mentioned earlier, an antibiotic strength 500 times greater than the systemic therapeutic dose may be required to be effective against the bacteria arranged in biofilms. It therefore is important to disrupt this biofilm physically so that the antibiotic agents can have access to the periodontal pathogens.[40]
9. Although there are adequate data to suggest that systemic antibiotics can be of benefit in the treatment of destructive periodontal diseases, there are limited data available to identify which antibiotics are suitable for which infection, the optimum dosage, frequency and duration of antibiotic therapy, when the regimen with antibiotics should be introduced in the treatment schedule, the long-term outcomes of antibiotic use, potential hazards of these agents (e.g., antibiotic resistance, changes in oral microflora, etc)[43] and the economic ramifications of this type of pharmacologic intervention

The selection of an antibiotic must be made based on factors other than the empirical decisions by the clinician. Unfortunately, there is no one best choice of antibiotic at present (i.e., no "silver bullet"). Therefore the clinician must integrate history of the patient's disease, clinical signs and symptoms, and results of radiographic examinations and possibly microbiologic sampling to determine the course of periodontal therapy. The clinician must obtain a thorough medical history, including current medications and possible adverse effects of combining these medicines, before prescribing any antibiotic therapy. The clinician must make the final decision with the patient. Risks and benefits concerning antibiotics as adjuncts to periodontal therapy should be discussed with the patient before antibiotics are used.

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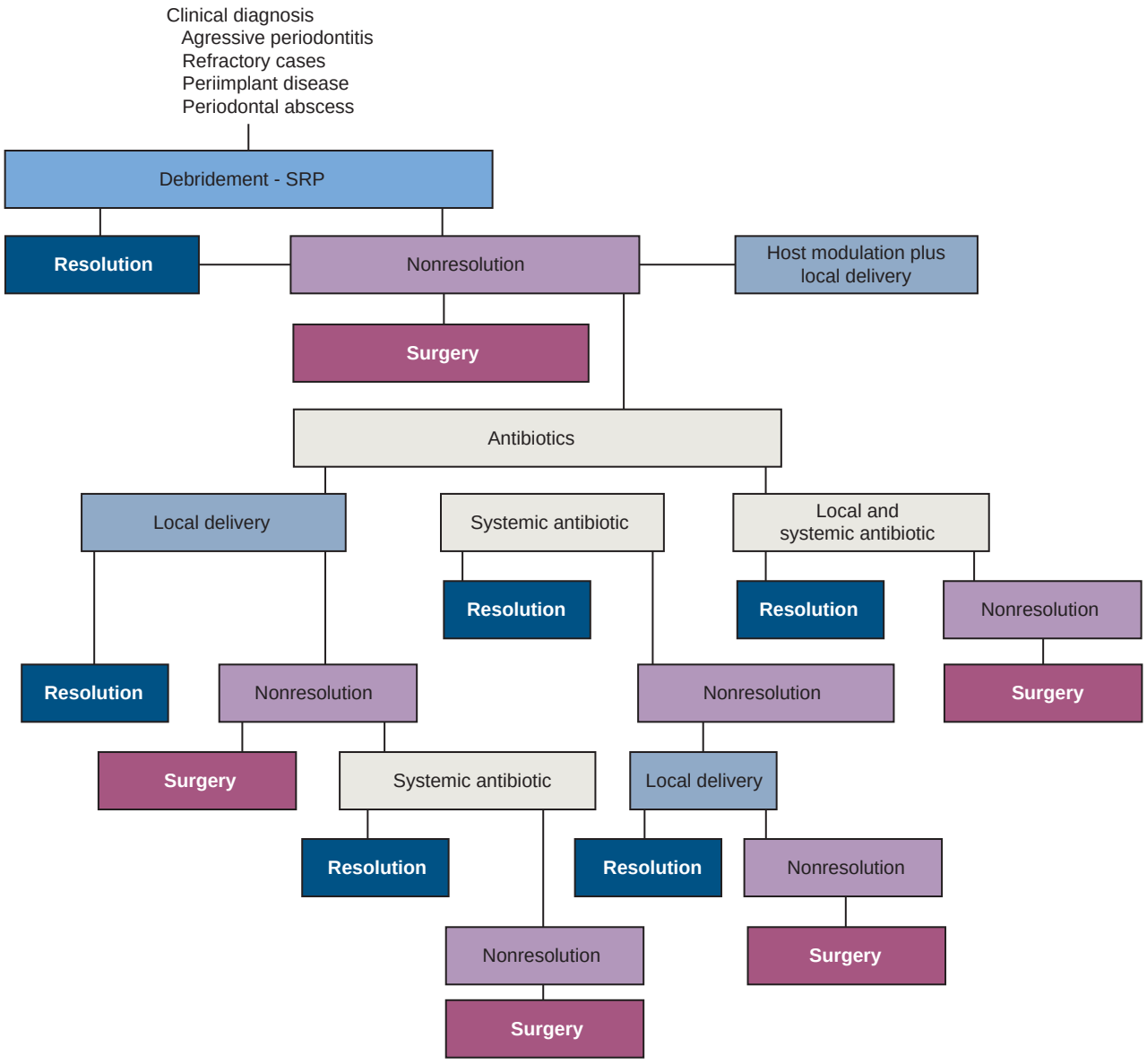

Figure 47-1 A decision tree for selection of antibiotic therapy.

LOCAL DELIVERY AGENTS

Locally delivered antimicrobial agents are available as adjuncts to scaling and root planing and as aids in the control of growth of bacteria on barrier membranes.[67] Although not discussed in this chapter, locally delivered NSAIDs are also being evaluated as adjunctive agents in the treatment of periodontal disease.[44,92] When placed into periodontal pockets, they reduce the subgingival microflora, probing depths, and clinical signs of inflammation. A recent systematic review concluded "adjunctive local therapy generally reduced probing depth levels. Differences between treatment and SRP-only groups in the baseline to followup period typically favored treatment groups but usually only modestly (e.g., from about 0.1 mm to nearly 0.5 mm). Effects for clinical attachment level gains were smaller and statistical significance less common."[11] A report on locally delivered agents prepared by the American Academy of Periodontology stated "the clinician's decision to use locally delivered agents (LDA) should be based upon a consideration of clinical findings, the patient's dental and medical history, scientific evidence, patient preferences and advantages and disadvantages of alternative therapies." The report also stated that use of LDAs may be of value when probing depths greater than 5 mm with inflammation are still present after conventional therapy. However if multiple sites are present in the same quadrant, therapy other than LDAs should be considered.[5]

Subgingival Chlorhexidine

A resorbable delivery system has been tested for the subgingival placement of chlorhexidine gluconate with positive clinical results (Figure 47-2). This product is available in the US and a number of other countries. PerioChip is a small chip (4.0 × 5.0 × 0.35 mm) composed of a biodegradable hydrolyzed gelatin matrix, cross-linked with glutaraldehyde and also containing glycerin and water, into which 2.5 mg of chlorhexidine gluconate has been incorporated per chip. This delivery system releases chlorhexidine and maintains drug concentrations in the GCF greater than 100 µg/ml for at least 7 days,[84] concentrations well above the tolerance of most oral bacteria.[12] Because the chip biodegrades in 7 to 10 days, a second appointment for removal is not needed.

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Figure 47-2 Placement of chlorhexidine gluconate chip (PerioChip).

Figure 47-3 Placement of 10% doxycycline (Atridox) gel.

Two multicenter, randomized, double-blind, parallel-group, controlled clinical trials of this chip were conducted in the US with a total of 447 patients in 10 centers.[48] In these studies, patients received a supragingival prophylaxis for up to 1 hour, followed by scaling and root planing for 1 hour. Chips were placed in target sites with probing depth of 5 to 8 mm at baseline that bled on probing and again at 3 and 6 months if probing depth remained at 5 mm or greater. Sites in control subjects received either a placebo chip (inactive) with scaling and root planing or scaling and root planing alone. Sites in test subjects received either a chlorhexidine chip (active) with scaling and root planing or scaling and root planing alone. Examinations were performed at baseline and again at 3, 6, and 9 months. At 9 months, significant decreases in probing depth from baseline favoring the active chip compared with controls were observed: chlorhexidine chip with scaling and root planing, –0.95 ± 0.05 mm; placebo chip with scaling and root planing, –0.69 ± 0.05 mm ($p = 0.001$); scaling and root planing alone, –0.65 ± 0.05 mm ($p = 0.00001$). Although statistically significant, the net clinical changes were limited. The proportion of pocket sites with a probing depth reduction of 2 mm or more was increased in the chlorhexidine chip group (30%) compared with scaling and root planing alone (16%), a statistically significant difference on a per-patient basis ($p < 0.0001$).

No signs of staining were noted in any of the previous three studies as a result of the chlorhexidine chip treatment, as measured by a stain index. Adverse effects were minimal, with a few patients who complained of slight pain and swelling in the first 24 hours after chip placement.

Tetracycline-Containing Fibers

The first local delivery product available in the US, an ethylene/vinyl acetate copolymer fiber (diameter, 0.5 mm) containing tetracycline, 12.7 mg per 9 inches, was extensively studied. When packed into a periodontal pocket, it was well tolerated by oral tissues, and for 10 days it sustained tetracycline concentrations exceeding 1300 µg/ml, well beyond the 32 to 64 µg/ml required to inhibit the growth of pathogens isolated from periodontal pockets.* In contrast, GCF concentrations of only 4 to 8 µg/ml were reported after systemic tetracycline administration, 250 mg qid for 10 days (total oral dose, 10 g).[36,37]

Studies demonstrated that tetracycline fibers applied with or without scaling and root planing reduced probing depth, bleeding on probing, and periodontal pathogens and provided gains in clinical attachment level. Such effects were significantly better than those attained with scaling and root planing alone or with placebo fibers. No change in antibiotic resistance to tetracycline was found after tetracycline fiber therapy among the tested putative periodontal pathogens. However, these fibers are no longer commercially available.

Subgingival Doxycycline

A gel system using a syringe with 10% doxycycline (Atridox) (Figure 47-3) is available. It was the only local delivery system accepted by the American Dental Association (ADA) when the ADA had an acceptance program for professional products, although that program was terminated in 2008. This product is available in the US and a number of other countries.

In a 9-month multicenter study of 180 patients, treatment with 10% doxycycline gel alone was more effective than the other treatments at all time periods, with the exception of the 3-month clinical attachment level value. For the 10% doxycycline gel group, the reduction in clinical attachment level at 9 months showed a gain of 0.4 mm compared with vehicle control; the reduction in probing depth was 0.6 mm greater than vehicle control; and the reduction of bleeding on probing was 0.2 units greater than vehicle control. The differences were clinically small but statistically significant. Although resistance was not evaluated in this study, the local application of doxycycline has previously been reported to show transient increases in resistance in oral microbes and no overgrowth of foreign pathogens.[58]

Two multicenter clinical trials each studied 411 patients with moderate-to-severe periodontitis.[30] At baseline, patients were randomized to one of four treatment groups: 10% doxycycline gel, vehicle control, oral hygiene only, and scaling and root planing. Sites with probing depth of 5 mm or greater that bled on probing were treated at baseline and then again with the same treatment at 4 months. Clinical assessments were made for 9 months, measuring clinical attachment level, probing depth, and bleeding on probing. All treatment groups in both studies showed clinical improvements from baseline over the 9-month period. The results for all parameters measured were significantly better in the 10% doxycycline gel group compared with vehicle control and oral hygiene only. Compared with scaling and root planing, the effects of 10% doxycycline gel as monotherapy on clinical attachment level gain and probing depth reduction were equivalent (see Chapter 48). Most recently, a 6-month, randomized, multicenter, placebo-controlled examiner-masked study[66] in 171 subjects evaluated the combined use of systemically delivered doxycycline hyclate (20 mg, bid) plus locally delivered doxycycline hyclate gel (10%) in combination with scaling and root planing versus scaling and root planing plus placebo. The

*References 34, 35, 64, 65, 88, and 99.

Figure 47-4 Placement of minocycline microspheres (Arestin).

results of the study showed that the combination therapy provided statistically significantly greater clinical benefits than control therapy for all clinical measures at both 3 and 6 months. The clinical parameters measured were probing depth, clinical attachment level, bleeding on probing, and gingival health.[66]

Subgingival Minocycline

A locally delivered, sustained-release form of minocycline microspheres (Arestin) is available for subgingival placement as an adjunct to scaling and root planing (Figure 47-4). The 2% minocycline is encapsulated into bioresorbable microspheres in a gel carrier. This product is available in the US and a number of other countries. When compared to controls (scaling and root planing with nonactive vehicle as subgingival irrigant), there was a statistically significant increase in clinical attachment levels in patients who presented with pockets of 6 mm or greater probing depth.[22]

In a four-center, double-blind, randomized trial, patients with periodontal pockets at least 5 mm deep were selected, and either 2% minocycline gel or vehicle were applied once every 2 weeks for 4 applications after initial scaling and root planing.[90] A total of 343 teeth (976 sites) were included in the minocycline group, with 299 teeth (810 sites) in the control group. Reductions in *P. gingivalis* and *P. intermedia* at weeks 2, 4, 6, and 12 and at weeks 6 and 12 for *A. actinomycetemcomitans* were statistically significant. These results demonstrated the advantages of supplementing standard subgingival debridement with minocycline gel application. The three primary clinical efficacy variables in this study were probing depth, clinical attachment level, and bleeding index. There was a trend toward clinical improvement in both treatment groups for all three measures, and the reduction in probing depth was significantly greater with minocycline gel. When sites with probing depth of at least 7 mm and significant bleeding at baseline were considered, the improvements were greater than with 5-mm pockets. The improvements with minocycline were statistically significantly better than the control group.

Applications of 2% minocycline were also evaluated in a 3-month study in 30 patients.[38] Active or placebo gel was placed subgingivally at planed sites in each subject according to a double-blind protocol, immediately after scaling and root planing and 2 and 4 weeks later. Differences between groups in mean probing depth did not reach statistical significance at any visit, but mean clinical attachment levels favored the minocycline group ($p < 0.05$) at both reassessments. The number of sites that bled after deep probing at 12 weeks also favored the minocycline group ($p < 0.05$).

Subgingival Metronidazole

A topical medication containing an oil-based metronidazole 25% dental gel (glyceryl mono-oleate and sesame oil) has been tested in a number of studies.[1] This product is not available in the US. It is applied in viscous consistency to the pocket, where it is liquidized by the body heat and then hardens again, forming crystals in contact with water. As a precursor, the preparation contains metronidazole-benzoate, which is converted into the active substance by esterases in GCF. Two 25% gel applications at a 1-week interval have been used.[54]

Studies have shown that metronidazole gel is equivalent to scaling and root planing but have not shown adjunctive benefits with scaling and root planing. In a 6-month study of 30 patients, treatment consisted of 2 applications of the dental gel in 2 randomly selected quadrants at 1-week intervals, as well as simultaneous subgingival scaling of the remaining quadrants.[85] Oral hygiene instructions were given on day 21. Statistical analyses showed that both treatments were effective in reducing probing depth and bleeding on probing over the 6-month period. At the end of the follow-up period, the mean reduction in probing depth was 1.3 mm after gel treatment and 1.5 mm after subgingival scaling. Bleeding on probing was reduced by 35% and 42%, respectively. No significant differences between the two treatments were detected. Darkfield microscopy showed a shift toward a seemingly healthier microflora for both treatment modalities; this effect persisted throughout the 6-month period.

A large, multicenter study of 206 subjects investigated 2 applications of metronidazole gel in 2 randomly selected quadrants versus 2 quadrants of scaling.[1] Probing depths were reduced by 1.2 mm in the gel and 1.5 mm in the scaling group. At 6 months, the differences between treatments were statistically but not clinically significant. Also, bleeding on probing was reduced by 88% for both treatment groups.

Comparative Studies

Few researchers have attempted to compare these devices side-by-side, but in one study, doxycycline polymer, metronidazole gel, and PerioChip were compared in 47 periodontal patients. The study found that all controlled release polymer devices increased gingival attachment levels but that there was a slightly greater improvement in patients treated with the doxycycline polymer.[78]

LOCAL DELIVERY OF ANTIMICROBIAL AGENTS AND PERIIMPLANT MUCOSITIS/IMPLANTITIS

Since there are a number of similarities between the pathogens involved in periodontitis periimplant mucositis and periimplantitis, it is logical that similar clinical approaches should be implemented for periimplant mucositis and periimplantitis. An early study by Mombelli et al,[63] utilizing tetracycline-containing fibers that are no longer available, demonstrated in 25 patients with periimplant mucositis that there was a significant reduction of four periopathogenic species, but other species, such as *P. gingivalis* and *A. actinomycetemcomitans,* had very low baseline and were not significantly affected. There was a slight but not significant improvement in the probing depth at 12 months. Mombelli's study showed that the local delivery of tetracycline from polymeric fibers may be used to improve the clinical and microbiologic parameters around infected implants when compared with scaling and root planing alone.

In another study,[14] investigators applied the controlled release of doxycycline (Atridox) into periimplant pockets and noted

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differences from scaling and root planing alone. This study also showed the efficacy of such devices in treating periimplant mucositis. Patients who received the doxycycline treatment showed significantly greater probing attachment levels and lesser pocket probing depth and bleeding index ($p < 0.05$) than those who received scaling/root planing alone.[14]

A study by Persson et al[79] evaluated 31 implants with periimplantitis in 25 patients and monitored clinical and radiographic changes over 12 months after adjunctive placement of minocycline microspheres (Arestin). Minocycline microspheres (Arestin) were locally delivered to each implant site with bone loss and a probing pocket depth (PPD) equal to or greater than 5 mm. Rescue therapy with Arestin was allowed at days 180 and 270 at any site exhibiting an increase in PPD equal or greater than 2 mm from the previous visit. Six implants in six subjects were either rescued or exited because of persisting active periimplantitis. Successful implants showed a statistically significant reduction in both PPD and percentage of sites with bleeding on probing between baseline and day 360 ($p < 0.05$). At mesial implant sites, the mean PPD reduction amounted to 1.6 mm (95% CI: 0.9 to 2.2 mm, $p < 0.001$) and was accompanied by a statistically significant reduction of the bleeding on probing value ($p < 0.001$).

Although studies evaluating the use of the PerioChip (chlorhexidine) for periimplant mucositis were not identified, one 12-month study compared the clinical and microbiologic results after application of minocycline microspheres as an adjunct to mechanical treatment of incipient periimplant infections compared with an adjunctive treatment using application of 1 ml of a 1% chlorhexidine gel.[73] Sixteen patients in the minocycline group and 14 in the chlorhexidine group completed the study. The findings of the study were that the adjunctive use of minocycline microspheres resulted in improvements of probing depths and bleeding scores, whereas the adjunctive use of chlorhexidine only resulted in limited reduction of bleeding scores. For the deepest sites of the treated implants in the minocycline group, the mean probing depth was reduced from 5.0 to 4.4 mm at 12 months. This study could not show any significant difference in the levels of bacterial species or groups at any time point between the two antimicrobial agents tested.

CONCLUSION

Scaling and root planing alone are effective in reducing pocket depths, gaining increases in periodontal attachment levels, and decreasing inflammation levels (bleeding on probing). When scaling and root planing are combined with the subgingival placement of sustained-release vehicles, however, additional clinical benefits are possible, including further reduction in pocket depths, additional gain in clinical attachment level, (e.g., 0.39 mm for minocycline gel), and further decrease in inflammation. Improvement in clinical attachment levels also occurs with the chlorhexidine chip (0.16 mm) and doxycycline gel (0.34 mm).[42] When systemic antibiotics are used as adjuncts to scaling and root planing, the evidence indicates that some systemic antibiotics (e.g., metronidazole and tetracycline) provide additional improvement in attachment levels (0.35 mm for metronidazole; 0.40 mm for tetracycline) when used as adjuncts to scaling and root planing.[43] Use of antiinfective chemotherapeutic treatment adjuncts does not result in significant patient-centered adverse effects.

The decision as to when to use local or systemic antimicrobials should be based on the clinicians consideration of the clinical findings, the patient's medical and dental history,[27,28] patient preferences, and potential benefits of adjunctive therapy with these agents.

There are extensive reviews of the local delivery agents available for periodontitis.[42,46,52,53,74]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 48

Host Modulation

Maria Emanuel Ryan and Ying Gu

CHAPTER OUTLINE

INTRODUCTION (online only)
SYSTEMICALLY ADMINISTERED AGENTS
- Nonsteroidal Antiinflammatory Drugs
- Bisphosphonates
- Sub-Antimicrobial-Dose Doxycycline

LOCALLY ADMINISTERED AGENTS
- Nonsteroidal Antiinflammatory Drugs
- Enamel Matrix Proteins, Growth Factors, and Bone Morphogenetic Proteins

HOST MODULATION AND COMPREHENSIVE PERIODONTAL MANAGEMENT
SUB-ANTIMICROBIAL-DOSE DOXYCYCLINE
- Mechanisms of Action
- Clinical Research Data on Distinct Patient Populations
- Suggested Uses and Other Considerations

EMERGING HOST MODULATORY THERAPIES (online only)
HOST MODULATION FACTORS IN SYSTEMIC DISORDERS (online only)
SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Some text, figures, and boxes may be out of numeric order in this printed chapter.

For more introductory information, please visit the companion Expert CONSULT website at www.expertconsult.com.

Intervention in periodontal disease can now include host modulatory therapy (HMT) as one of the available adjunctive treatment options. The term *adjunctive* is meant to imply "in addition to conventional therapies" or "in addition to other established therapies." For the management of periodontal diseases, conventional approaches were initially mechanical in nature, that is, surgery, as well as scaling and root planing (SRP). Initially, adjunctive therapies were solely antimicrobial such as the use of antiseptics and antibiotics (local and/or systemic). New adjunctive approaches involve modulation of the host response. Researchers are also investigating HMTs, which aim to modify or reduce destructive aspects of the host response so that the immune-inflammatory response to plaque is less damaging to the periodontal tissues. Removal of plaque by SRP targets one aspect of the pathogenic process by reducing the bacterial burden and therefore the antigenic challenge that drives the inflammatory response in the host tissues. However, the bacterial challenge is never completely eliminated after SRP, and recolonization by bacterial species occurs. HMTs offer the potential for downregulating destructive aspects and upregulating protective aspects of the host response so that, in combination with conventional treatments to reduce the bacterial burden, the balance between health (resolution of inflammation and wound healing) and disease progression (continued proinflammatory events) is tipped in the direction of a healing response.

HMT is a *means of treating the host side of the host–bacteria interaction*. The host response is responsible for most of the tissue breakdown that occurs, leading to the clinical signs of periodontitis. HMTs offer the opportunity for modulating or reducing this destruction by treating aspects of the chronic inflammatory response. HMTs do not "switch off" normal defense mechanisms or inflammation; instead, they ameliorate excessive or pathologically elevated inflammatory processes to enhance the opportunities for wound healing and periodontal stability.

HMT can be used to reduce excessive levels of enzymes, cytokines, and prostanoids and should not reduce levels below constitutive levels. HMTs can also modulate osteoclast and osteoblast function (Figure 48-3) but should not impact normal tissue turnover. HMT is key to addressing many of the risk factors that have adverse effects on the host response, which are either not easily managed (e.g., smoking, diabetes) or cannot be changed (e.g., genetic susceptibility). In addition, host modulatory agents might be used to increase the levels of a person's own protective or anti-inflammatory mediators. Use of systemic HMTs for treatment of a patient's periodontal condition may also provide benefits for other

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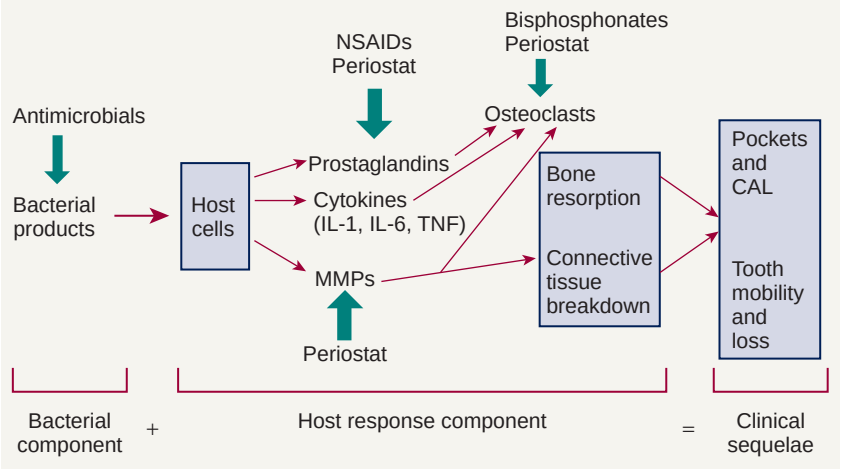

Figure 48-3 Potential adjunctive therapeutic approaches. Possible adjunctive therapies and points of intervention in the treatment of periodontitis are presented related to the pathologic cascade of events. *CAL,* Clinical attachment loss.

inflammatory disorders such as arthritis, cardiovascular disease, dermatologic conditions, diabetes, rheumatoid arthritis, and osteoporosis. Also, patients who are currently taking host modulatory agents, such as nonsteroidal antiinflammatory drugs (NSAIDs), bisphosphonates, or tetracyclines, as well as newer agents targeting specific cytokines for the management of medical conditions, may be experiencing periodontal benefits from these systemic medications prescribed for the management of other chronic inflammatory conditions.

A variety of different drug classes have been evaluated as host modulation agents, including the NSAIDs, bisphosphonates, tetracyclines, enamel matrix proteins, growth factors, and bone morphogenetic proteins. This section discusses chemotherapeutics developed as HMTs researched as adjuncts to treat periodontitis to date, use of HMTs in clinical practice, and what the future might hold for HMTs.

SYSTEMICALLY ADMINISTERED AGENTS

Nonsteroidal Antiinflammatory Drugs

NSAIDs inhibit the formation of prostaglandins, including PGE_2, which is produced by neutrophils, macrophages, fibroblasts, and gingival epithelial cells in response to the presence of LPS, a component of the cell wall of gram-negative bacteria. PGE_2 has been extensively studied in periodontal disease because it upregulates bone resorption by osteoclasts.[33,43,64] Levels of PGE_2 have been shown to be elevated in patients with periodontal disease compared with healthy patients.[35,64] PGE_2 also inhibits fibroblast function and has inhibitory and modulatory effects on the immune response.[36]

NSAIDs inhibit prostaglandins and therefore reduce tissue inflammation. They are used to treat pain, acute inflammation, and a variety of chronic inflammatory conditions. NSAIDs include the salicylates (e.g., aspirin), indomethacin, and the propionic acid derivatives (e.g., ibuprofen, flurbiprofen, and naproxen). The ability of NSAIDs to block PGE_2 production, thereby reducing inflammation and inhibiting osteoclast activity in the periodontal tissues, has been investigated in patients with periodontitis. Studies have shown that systemic NSAIDs, such as indomethacin,[97] flurbiprofen,[96] and naproxen[42] administered daily for up to 3 years, significantly slowed the rate of alveolar bone loss compared with placebo. However, NSAIDs have some serious disadvantages when considered for use as a HMT for periodontitis. Daily administration for extended periods is necessary for periodontal benefits to become apparent, and NSAIDs are associated with significant side effects, including gastrointestinal problems, hemorrhage (from decreased platelet aggregation), and renal and hepatic impairment. Furthermore, research shows that the periodontal benefits of taking long-term NSAIDs are lost when patients stop taking the drugs, with a return to or even an acceleration of the rate of bone loss seen before NSAID therapy, often referred to as a "rebound effect."[98] For these reasons, the long-term use of NSAIDs as an adjunctive treatment for periodontitis has never really developed beyond research studies.

It was previously anticipated that the selective cyclooxygenase-2 (COX-2) inhibitors may offer promise as adjunctive treatments in the management of periodontitis. The enzyme cyclooxygenase, which converts arachidonic acid to prostaglandins, exists in two functionally distinct isoforms, COX-1 and COX-2. COX-1 is constitutively expressed and has antithrombogenic and cytoprotective functions. Therefore, inhibition of COX-1 by nonselective NSAIDs causes side effects such as gastrointestinal ulceration and impaired hemostasis. COX-2 is induced after stimulation by various cytokines, growth factors, and LPS and results in the production of elevated quantities of prostaglandins. Inhibition of COX-2 by selective COX-2 inhibitors results in reduction of inflammation. Researchers considered that the use of selective COX-2 inhibitors offered the prospect for reducing periodontal inflammation without the side effects typically observed after long-term (nonselective) NSAID therapy, and preliminary studies identified that selective COX-2 inhibitors slowed alveolar bone loss in animal models[5,41] and modified prostaglandin production in human periodontal tissues.[89] However, the selective COX-2 inhibitors were later identified to be associated with significant and life-threatening adverse effects, resulting in some drugs being withdrawn from the market. In summary, NSAIDs (including the selective COX-2 specific inhibitors) are presently not indicated as adjunctive HMTs in the treatment of periodontal disease.

Bisphosphonates

The bisphosphonates are bone-seeking agents that inhibit bone resorption by disrupting osteoclast activity. Their precise mechanism of action is unclear, but research has shown that bisphosphonates interfere with osteoblast metabolism and secretion of lysosomal enzymes.[94] More recent evidence has suggested that bisphosphonates also possess anticollagenase properties.[59] The ability of bisphosphonates to modulate osteoclast activity clearly may be useful in the treatment of periodontitis. Research has demonstrated that in naturally occurring periodontitis in beagles, treatment with the bisphosphonate alendronate significantly increased

bone density compared with placebo.[74] In animal models of experimentally induced periodontitis, bisphosphonates reduced alveolar bone resorption.[82,94] In human studies, these agents resulted in enhanced alveolar bone status and density.[16,76]

Some bisphosphonates have the unwanted effects of inhibiting bone calcification and inducing changes in white blood cell counts. Also, there have been recent reports of avascular necrosis of the jaws following bisphosphonate therapy, with the resultant risk of bone necrosis following dental extractions.[11] The recent reports of bisphosphonate-related osteonecrosis of the jaw, although primarily associated with intravenous administration of bisphosphonates rather than oral administration, has impeded the development of bisphosphonates as an HMT to manage periodontitis. As with NSAIDs, at present there are no bisphosphonate drugs that are approved and indicated for treatment of periodontal diseases.

Sub-Antimicrobial-Dose Doxycycline

Sub-antimicrobial-dose doxycycline (SDD) is a 20-mg dose of doxycycline (Periostat) that is approved and indicated as an adjunct to SRP in the treatment of chronic periodontitis. It is taken twice daily for 3 months, up to a maximum of 9 months of continuous dosing. The 20-mg dose exerts its therapeutic effect by enzyme, cytokine, and osteoclast inhibition rather than by any antibiotic effect. Research studies have found no detectable antimicrobial effect on the oral flora or the bacterial flora in other regions of the body and have identified clinical benefit when used as an adjunct to SRP. At present, SDD (Periostat) is the only systemically administered HMT specifically indicated for the treatment of chronic periodontitis that is approved by the US Food and Drug Administration (FDA) and accepted by the American Dental Association (ADA). In addition, a modified-release SDD was recently approved by the FDA (Oracea) for the treatment of the common skin disorder rosacea and is routinely prescribed within the dermatology community. Studies conducted by Preshaw et al,[73] utilizing this same modified-release SDD versus placebo in 266 subjects with periodontitis as an adjunct to SRP resulted in significantly greater clinical benefits than SRP alone in the treatment of periodontitis. It will be interesting to see what the long-term benefits to oral health will be in rosacea patients being prescribed this new drug. There has also been considerable off-label use of this new modified-release SDD for the treatment of periodontal diseases based on the understanding that once-a-day administration can increase the level of compliance as compared to twice-a-day oral administration.

LOCALLY ADMINISTERED AGENTS

Nonsteroidal Antiinflammatory Drugs

Topical NSAIDs have shown benefit in the treatment of periodontitis. One study of 55 patients with chronic periodontitis who received topical ketorolac mouthrinse reported that gingival crevicular fluid (GCF) levels of PGE_2 were reduced by approximately half over 6 months and that bone loss was halted.[45] In addition, locally administered ketoprofen has been investigated. To date, topically administered NSAIDs have not been approved as local HMTs for the management of periodontitis.

Enamel Matrix Proteins, Growth Factors, and Bone Morphogenetic Proteins

A number of local HMTs have been investigated for potential use as adjuncts to surgical procedures, not only to improve wound healing but also to stimulate regeneration of lost bone, periodontal ligament, and cementum, restoring the complete periodontal attachment apparatus. These have included enamel matrix proteins bone morphogenetic proteins (BMP-2, BMP-7), growth factors (platelet-derived growth factor, insulinlike growth factor), and tetracyclines. The locally applied HMTs currently approved by the FDA for adjunctive use during surgery are enamel matrix proteins (Emdogain), recombinant human platelet-derived growth factor-BB (GEM 21S), and BMP-2 (INFUSE), which are covered in much greater detail in other chapters. The initial local host modulatory agent approved by the FDA for adjunctive use during surgery to assist with clinical attachment gain and wound healing was Emdogain; this has been followed by platelet-derived growth factor combined with a resorbable synthetic bone matrix (GEM 21S) to assist in regenerative procedures approved recently by the FDA, as well as rhBMP-2 (INFUSE) soaked on to an absorbable collagen sponge to assist with ridge and sinus augmentation. The technology behind GEM 21 has already been marketed for use in wound healing, particularly in patients with diabetes, and INFUSE has been used for quite some time for the healing of fractures by the orthopedic community.

The remainder of this chapter focuses on the clinical utility of host modulation for nonsurgical procedures in clinical practice and the use of SDD (Periostat) in clinical practice.

HOST MODULATION AND COMPREHENSIVE PERIODONTAL MANAGEMENT

The term *periodontal management* suggests a much broader concept of periodontal care than the term *periodontal treatment.* This concept is extremely important considering the chronic nature of the disease. Management includes thorough medical and dental history and examination (clinical charting and radiographs), assessment of risk factors, diagnosis, development of a treatment strategy, initial and definitive treatment planning, review of treatment outcomes and reevaluation, long-term supportive periodontal therapy (maintenance care), and assessment of prognosis. In addition, as new data continue to emerge regarding biochemical assessments of disease activity (measuring levels of proinflammatory mediators, bone and connective tissue breakdown products in the GCF, saliva, and tissues of the oral cavity), new diagnostic and prognostic tests may become part of our established protocols for comprehensive periodontal disease management in the future. However, controlling the bacteria that cause periodontal infections remains a central focus of effective periodontal treatment. Understanding the importance of the host response and the impact of risk factors now allows clinicians to provide complementary treatment strategies simultaneously for their patients (Figure 48-4).

Those patients most likely requiring the use of HMT include those with risk factors that are either nonmodifiable or not easily modified. If the decision is made to use HMT, this must be discussed with the patient and the rationale for treatment thoroughly explained. This takes time at the chairside, but it is time well spent; patients become increasingly interested in their periodontal status and are more likely to develop ownership of their management, thereby enhancing compliance with all aspects of care, including plaque control, risk reduction, and treatment protocols. Compliance with HMT is greatly facilitated if the rationale for prescribing is clearly explained. The need for compliance with the prescribed drug regimen is important because with SDD, for example, a tablet must be taken twice daily (once in the morning and once in the evening) and should not be taken with calcium supplements. Compliance may be improved by administration of a modified-release SDD

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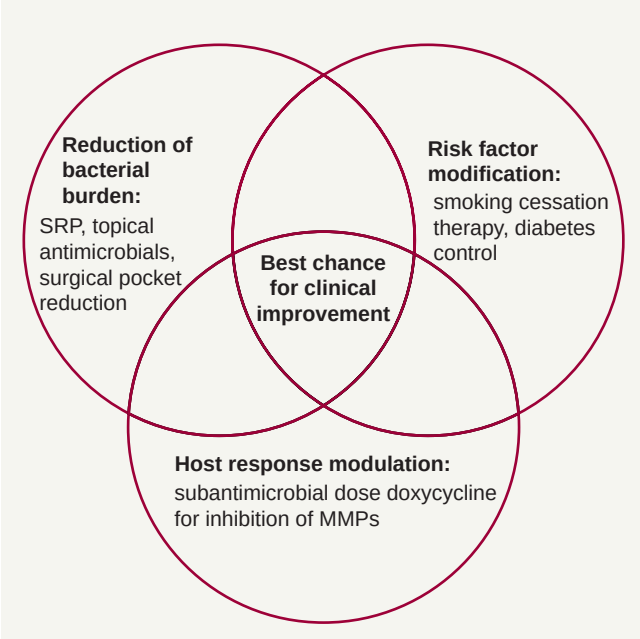

Figure 48-4 Complementary treatment strategies in periodontitis. The best chance for clinical improvement may come from implementing complementary treatment strategies that target different aspects of the periodontal balance. Reduction of the bacterial burden by scaling and root planing *(SRP)* is the cornerstone of treatment and can be augmented by the use of topical antimicrobials and surgical pocket therapy. In addition to this antibacterial treatment approach, the host response can be treated by the use of host modulatory therapy, such as sub-antimicrobial-dose doxycycline, for the inhibition of matrix metalloproteinases *(MMPs)*. Risk factor assessment and modification must form a key part of any periodontal treatment strategy, including smoking cessation counseling. These different but complementary treatment strategies can be used together as part of a comprehensive management approach.

capsule taken only once a day. It should be emphasized to the patient that the use of HMT is not a substitute for excellent plaque control (just as it is not a substitute for excellent debridement and root surface instrumentation by the treating clinician). To achieve the best results, patients must be interested and well informed about their condition so that compliance is maximized. Furthermore, patients must also be convinced that comprehensive and frequent recall appointments are absolutely necessary in the maintenance phase of this chronic and often progressive disease, which can be very well controlled with adequate follow-up.

In addition to patient motivation, oral hygiene instruction, and SRP to reduce the bacterial challenge, a key treatment strategy when managing periodontitis patients is *risk factor modification*. The harmful effects of smoking on the periodontal tissues are well documented,[46] and successful smoking cessation therapy will likely be a major benefit to patients with periodontitis. Smoking cessation counseling can be undertaken in the dental office (if staff are appropriately trained) or through collaboration with the patient's physician or specialized clinics. Given the evidence that smokers have worse periodontal disease than nonsmokers[83,99] and that the magnitude and predictability of clinical improvements after treatment are significantly reduced in smokers,[1,69] smoking cessation counseling should form a major part of treatment for smokers with periodontitis. Patients with poorly controlled diabetes are also at increased risk for periodontitis,[54] and periodontal therapy may have an impact on diabetic control.[36] Collaboration with medical colleagues when treating diabetic patients with periodontitis is warranted to ascertain the degree of diabetic control.[21] Other possible risks for periodontitis include nonmodifiable factors such as genetics, gender, and race. As the relevance of different risk factors is established through epidemiologic research, clinicians must remain aware of their responsibilities for informing and changing patients' behaviors in relation to modifiable risks.

The management of patients with periodontitis can therefore involve the following complementary treatment strategies:

- Patient education and motivation, including oral hygiene instruction; use of antiseptics in rinses, toothpastes, and irrigation; and explanation of the rationale for any adjunctive treatments.
- Reduction of the bacterial burden by high-quality SRP.
- Site-specific antibacterial treatment with local delivery systems or systemic antimicrobial therapy in select cases.
- Host response modulation by HMT.
- Risk factor modification and risk reduction strategies.
- Periodontal surgery with or without HMT.

It is the responsibility of the dentist to customize the treatment plan for each individual patient by selecting and providing appropriate treatments, following discussion and informed decision making by the patient. Good communication and showing an interest in the patient's condition are essential to maximize compliance and modify risk factors. The best chance for clinical improvement may come from a combination of targeted treatment approaches for each patient (see Figure 48-4). In fact, Novak et al published very striking improvements in probing depth reductions and clinical attachment level gains in a 6-month, randomized, multicenter, examiner-blinded, placebo-controlled study, which showed that combination therapy of HMT (SDD) plus a locally delivered antimicrobial (doxycycline hyclate gel) and SRP provided optimal improvements in clinical parameters when compared to SRP alone in the treatment of moderate-to-severe periodontitis.[61]

SUB-ANTIMICROBIAL-DOSE DOXYCYCLINE (SDD)

As previously discussed, SDD is currently the only FDA-approved, systemically administered HMT indicated specifically in the treatment of periodontitis. SDD is used as an adjunct to SRP and must not be used as a stand-alone therapy (monotherapy). Because SDD, previously called "low-dose doxycycline" (LDD) and currently marketed as Periostat, is based on a sub-antimicrobial dosage of doxycycline, a member of the tetracycline family of compounds, the use of tetracyclines for the management of periodontal diseases must be put in perspective.

In regard to incorporation of a medical pharmacologic approach into the management of a disease in the dental practice setting, no class of drugs has made more of an impact on periodontal therapy than the tetracyclines. They have been used in conjunction with SRP, the "gold standard" of nonsurgical therapy, as well as with both resective and regenerative surgical procedures. The tetracyclines have been used locally and systemically as antimicrobial agents and more recently, systemically as a host modulation agent (SDD). The tetracyclines have been prescribed not only to address chronic periodontitis but also to manage specific and often more aggressive types of periodontitis. Most recently, the tetracyclines have been advocated for the management of patients with systemic diseases such as diabetes and rosacea (Oracea); doxycycline has led to improvements in both the periodontal health of compromised

diabetic patients and long-term markers of glycemic control (e.g., glycated hemoglobin).[37] As an adjunct to mechanical therapies, the goal of tetracycline therapy has been to enhance reattachment or even to stimulate new attachment of the supporting apparatus and osseous formation.

This section concentrates on the use of these pleiotropic compounds for modulation of the host response in the treatment of periodontitis.

Mechanisms of Action

In addition to its antibiotic properties, doxycycline (as well as the other members of the tetracycline family) has the ability to downregulate MMPs, a family of zinc-dependent enzymes that are capable of degrading extracellular matrix molecules, including collagen.[6,78] MMPs are secreted by the major cell types in the periodontal tissues (fibroblasts, keratinocytes, macrophages, PMNs, endothelial cells) and play a key role in periodontitis. Excessive quantities of MMPs are released in inflamed periodontal tissues, resulting in breakdown of the connective tissue matrix. The predominant MMPs in periodontitis, particularly MMP-8 and MMP-9, derive from PMNs[30] and are extremely effective in degrading type I collagen, the most abundant collagen type in gingiva and periodontal ligament.[52] Levels of PMN-type MMPs have been shown to increase with severity of periodontal disease and decrease after therapy.[24,30] The release of large quantities of MMPs in the periodontium leads to significant anatomic disruption and breakdown of the connective tissues, contributing to the clinical signs of periodontitis.

The rationale for using SDD as a HMT in the treatment of periodontitis is that doxycycline downregulates the activity of MMPs by a variety of synergistic mechanisms, including reductions in cytokine levels, and stimulates osteoblastic activity and new bone formation by upregulating collagen production (Figure 48-5).

Clinical Research Data On Distinct Patient Populations

Tetracyclines work well as host modulation agents because of their pleiotropic effects on multiple components of the host response (see Figure 48-3). The only enzyme (MMP) inhibitors that have been approved for clinical use and tested for the treatment of periodontitis are members of the tetracycline family of compounds. In early studies investigating the use of different commercially available tetracyclines, Golub et al[31] reported that the semisynthetic compound (e.g., doxycycline) was more effective than the parent compound tetracycline in reducing excessive collagenase activity in the GCF of chronic periodontitis patients. Because doxycycline was found to be a more effective inhibitor of collagenase than either minocycline or tetracycline[9,25] and because of its safety profile, pharmacokinetic properties, and ready systemic absorption, recent clinical trials have focused on this compound. In an effort to eliminate the side effects of long-term tetracycline therapy, especially the emergence of tetracycline-resistant organisms, SDD capsules were prepared and tested.[30] Each capsule contained 20 mg of doxycycline versus the commercially available 50 mg and 100 mg, antimicrobially effective, capsules or tablets. Multiple clinical studies using sub-antimicrobial doses of doxycycline have shown no difference in the composition or resistance level of the oral flora.[85,93] More recent studies also demonstrate no appreciable differences in either fecal or vaginal microflora samples.[91] In addition, these studies have shown no overgrowth of opportunistic pathogens, such as *Candida,* in the oral cavity, gastrointestinal system, or genitourinary system.

With regard to MMP inhibition, Golub et al[24] reported that a 2-week regimen of SDD reduced collagenase in GCF and in the adjacent gingival tissues surgically excised for therapeutic purposes. Subsequent studies using SDD therapy adjunctive to routine scaling and prophylaxis indicated continued reductions in the

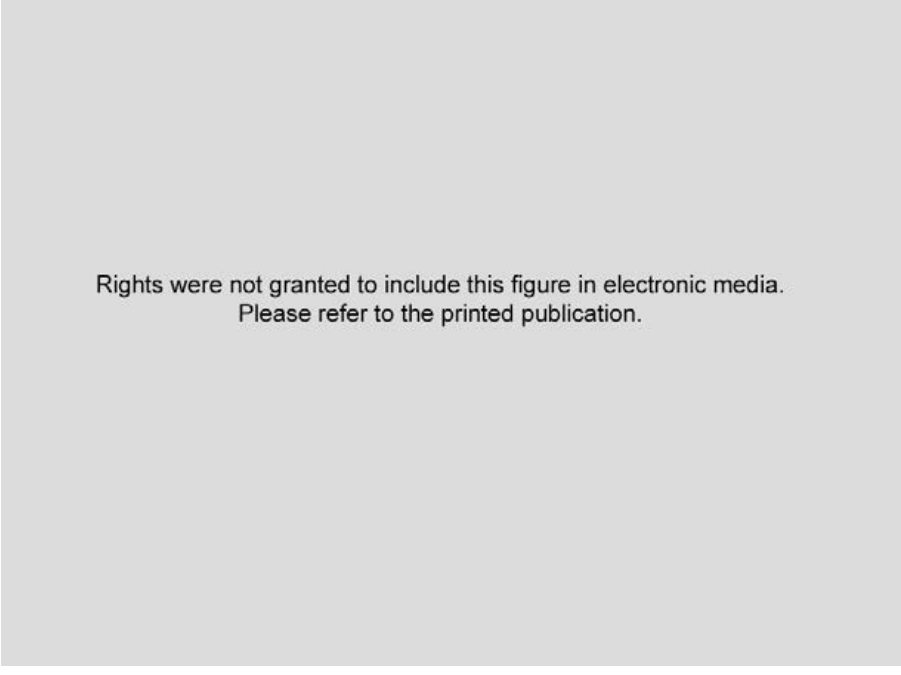

Figure 48-5 Schematic of periodontal pocket indicating the pleiotropic mechanisms by which doxycycline inhibits connective tissue breakdown. Downregulation of destructive events occurring in the periodontal tissues by doxycycline results from modulation of a variety of different proinflammatory pathways. *(From Golub LM, Lee HM, Ryan ME, et al:* Adv Dent Res *12:12, 1998.)*

excessive levels of collagenase in the GCF after 1 month of treatment. After cessation of SDD administration, however, there was a rapid rebound of collagenase activity to placebo levels, suggesting that a 1-month treatment regimen with this host modulation agent was insufficient to produce a long-term benefit.[3] In contrast, during the same study, a 3-month regimen produced a prolonged drug effect without a rebound in collagenase levels to baseline during the no-treatment phase of the study. The mean levels of GCF collagenase were significantly reduced (47.3% from baseline levels) in the SDD group versus the placebo group, who received scaling and prophylaxis alone (29.1% reduction from baseline levels). Accompanying these reductions in collagenase levels were gains in the relative attachment levels in the SDD group.[3,29] Continuous drug therapy over several months appears to be necessary for maintaining collagenase levels near normal over prolonged periods. However, it is reasonable to speculate that levels of these MMPs will eventually increase again in the more susceptible patients, and those individuals having the most risk factors and the greatest microbial challenge will require more frequent HMT than other patients.

General Patient Populations. Data from clinical trials of SDD are summarized in Table 48-1. In addition, a series of double-blind, placebo-controlled studies of 3, 6, and 9 months' duration all showed clinical efficacy based on reductions in probing depth and gains in clinical attachment as well as biochemical efficacy, based on the inhibition of collagenase activity and protection of serum α_1-antitrypsin (a naturally occurring protective mediator) from collagenase attack in the periodontal pocket.[14,25,47] Golub et al[27] showed that a 2-month regimen of SDD significantly decreased both the level of bone-type collagen breakdown products (ICTP; carboxy terminal peptide, a pyridinoline-containing cross-linked peptide of type I collagen) and MMP-8 and MMP-13 enzyme levels (neutrophil and bone-type collagenase) in chronic periodontitis subjects (Figure 48-6).

For more information on general patient populations, please visit the companion Expert CONSULT website at www.expertconsult.com.

High-Risk Patients: Smokers. The harmful effects of cigarette smoking and the reduced response to periodontal treatment in smokers compared with nonsmokers are well established.[46] A recent metaanalysis of randomized clinical trials of SDD used as an adjunct to SRP revealed a benefit when using SDD in smokers with periodontitis[60,70] (see Table 48-1). A hierarchic treatment response was observed such that nonsmokers who received SDD demonstrated the best clinical improvements and smokers who received placebo had the poorest treatment response. The responses of the smokers who received SDD and the nonsmokers who received placebo were intermediate to the two extremes and were broadly identical. This suggests that even patients traditionally considered resistant to periodontal treatment (i.e., smokers) can benefit from SDD, with a treatment response similar to that expected when treating a nonsmoker by scaling and root planing alone.

Special Patient Populations. More recent Phase IV (i.e., post-licensing) clinical studies have revealed success using SDD in particular populations of susceptible individuals. Much interest has focused on genetic susceptibility to periodontal disease, particularly whether a specific variation in the genes that regulate the cytokine IL-1 confers increased susceptibility to disease. This polymorphism is known as the *periodontitis-associated genotype* (PAG), the presence of which can be characterized using a commercially available screening test, the PST genetic susceptibility test. Investigation of patients who possess this gene polymorphism has been driven by the assumption that local phenotypic differences exist in chronic periodontitis associated with this genotype (e.g., that PAG-positive patients produce more IL-1 cytokines for a given bacterial challenge, resulting in increased tissue damage and more extensive periodontal disease). However, few studies exist of the impact of cytokine gene polymorphism on tissue IL-1 cytokine levels in periodontal disease to support this, although IL-1β levels in shallow periodontal pockets have been reported to be higher in patients with the genotype than those without.[19] The studies that have investigated associations between PAG and periodontal disease status have thus far generated conflicting data (as reviewed by Taylor et al).[84] A reasonable assumption currently is that there

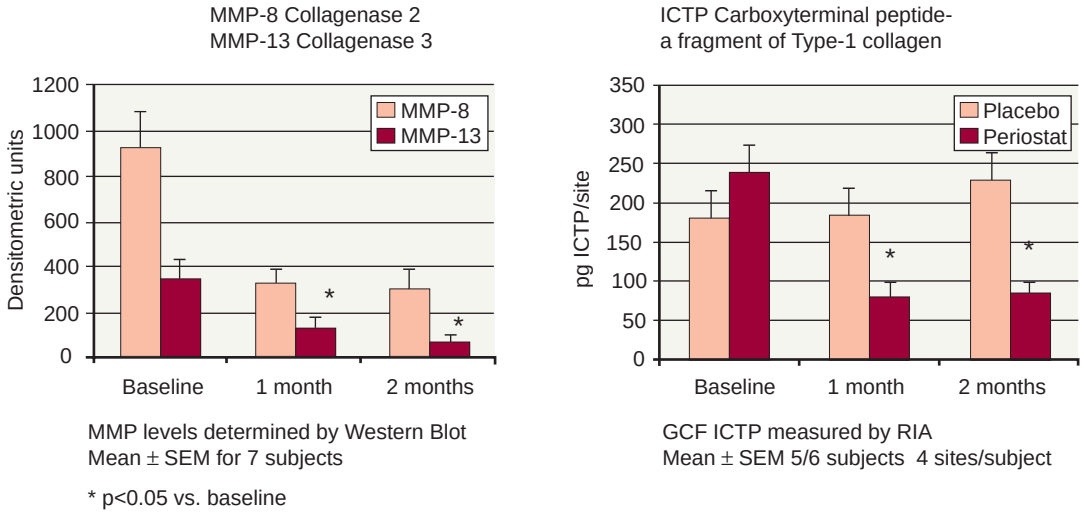

Figure 48-6 Effect of sub-antimicrobial-dose doxycycline (SDD) on gingival crevicular fluid (GCF) collagenase (MMP-8, MMP-13) and ICTP. A 2-month regimen of SDD significantly decreased levels of matrix metalloproteinases (MMP-8 and MMP-13, neutrophil and bone-type collagenases, respectively) and ICTP compared with placebo in GCF samples of adult periodontitis patients. Decreased levels of GCF bone-type collagen breakdown products (ICTP, pyridinoline-containing cross-linked peptide of type I collagen) in the SDD group versus the placebo group provides biochemical evidence of a reduction in bone resorption. *RIA,* Radioimmunoassay. *(From Golub LM, Lee HM, Greenwald RA, et al:* Inflamm Res *46:310, 1997.)*

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TABLE 48-1 Summary of Data Reported in Clinical Trials of Sub-Antimicrobial-Dose Doxycycline (SDD)[72]

			MEAN CAL CHANGE (mm)		MEAN PD REDUCTION (mm)		% SITES WITH CAL GAIN‡		% SITES WITH PD REDUCTION†	
Study (Year)	**Duration**	**Study Groups (*N*)**	**4–6 mm Pockets**	**7+ mm Pockets**	**4–6 mm pockets**	**7+ mm pockets**	**≥2 mm**	**≥3 mm**	**≥2 mm**	**≥3 mm**
Caton et al (2000)[12]	Study: 9 months	SRP + SDD (90)	1.03*	1.55*	0.95†	1.68†	46	22	47*	22*
	(drug: 9 months)	SRP + placebo (93)	0.86	1.17	0.69	1.20	38	16	35	13
Novak et al (2002)[62]	Study:9 months	SRP + SDD (10)	1.00	1.78	1.20	3.02	29	15	48	26
	(drug:6 months)	SRP + placebo (10)	0.56	1.24	0.97	1.42	21	11	21	6
Emingil et al (2004)[17]	Study: 12 months	SRP + SDD (10)	0.21 (all sites)	1.59* (all sites)						
	(drug: 3 months)	SRP + placebo(10)	0.05 (all sites)	1.32 (all sites)						
Preshaw et al (2004)[71]	Study: 9 months	SRP + SDD (107)	1.27†	2.09*	1.29†	2.31†	58*	33†	62†	37†
	(drug: 9 months)	SRP + placebo (102)	0.94	1.60	0.96	1.77	44	20	45	21
Lee et al (2004)[48]	Study: 9 months	SRP + SDD (240)	1.56*(all sites)	1.63* (all sites)						
	(drug: 9 months	SRP + placebo (17)	0.80(all sites)	1.19 (all sites)						
Choi et al (2004)[13]	Study: 4 months	SRP + SDD (15)	2.2*(test sites)	1.6* (test sites)						
	(drug: 4 months)	SRP + placebo (17)	0.6(test sites)	1.1 (test sites)						
Gurkan et al (2005)[39]	Study: 6 months	SRP + SDD (13)	1.12	2.15	1.80	3.38				
	(drug: 3 months)	SRP + placebo (13)	0.78	1.76	1.46	2.57				
Preshaw et al	Study: 9 months	SRP + SDD# (116)	1.23*	1.89†	1.22†	2.16*	59†	33†	63§	37
(2005)¶[70]	(drug:9 months)	SRP + placebo# (135)	0.96	1.43	0.88	1.53	43	19	44	18
		SRP + SDD## (81)	1.03	1.71	1.01	1.80	44	21	45†	20
		SRP + placebo## (60)	0.85	1.58	0.80	1.62	37	15	31	13
Mohammad et al	Study: 9 months	SRP + SDD (12)	2.14§	3.18*	1.57§	3.22§				
(2005)[58]	(drug: 9 months)	SRP + placebo (12)	0.02	0.25	0.63	0.98				
Górska and Nedzi-	Study: 3 months	SRP + SDD (33)	0.33* (all sites)	0.29* (all sites)						
Góra (2006)[34]	(drug: 3 months)	SRP + placebo (33)	0.04 (all sites)	0.08 (all sites)						
Needleman et al	Study: 6 months	SRP + SDD## (18)	0.65 (all sites)	1.40 (all sites)						
(2007)[60]	(drug: 3 months)	SRP + placebo## (16)	0.40 (all sites)	0.98 (all sites)						

*$p < 0.05$ compared with placebo.
†$p < 0.01$ compared with placebo.
‡Percentage of sites with CAL gain and PD reduction ≥2 mm and ≥3 mm calculated for all sites that had 6+ mm probing depths at baseline.
§$p < 0.001$ compared with placebo.
¶Same study population as Preshaw et al (2004),[71] stratified by smoking status: *#*, nonsmokers, *##*, smokers.
N, Number of subjects; *SRP*, scaling and root planing; *CAL*, clinical attachment level, CAL change can be positive (CAL gain) or negative (CAL loss); *PD*, probing depth.

are genetic associations between polymorphisms in the IL-1 gene cluster and periodontal disease but that unambiguous results are not yet apparent because of the heterogeneity of the disease and/or the variable design of the reported studies. Cullinan et al concluded that IL-1 genotype is a contributory but nonessential risk factor for periodontal disease progression.[15]

A 5-month preliminary investigation by Ryan et al[79] was designed to evaluate the impact of treatment on IL-1 and MMP levels in PST-positive patients who presented with elevated levels of these biochemical markers in their GCF. These patients were initially treated with SRP, resulting in no change in the levels of these biochemical markers after 1 month. Al-Shammari et al[2] reported similar findings, with no changes in GCF levels of IL-1β and ICTP before and after SRP in patients who had not been genotyped. When the genotype-positive patients received SDD and these biochemical markers were monitored at 2 and 4 months, a significant decrease (50% to 61%) in the IL-1β and MMP-9 levels was noted after treatment with SDD. Correspondingly, gains in clinical attachment and reduced probing depths were also observed. The study concluded that a sub-antimicrobial dose of doxycycline may provide PST-positive patients with a therapeutic strategy that specifically addresses their exaggerated host response.

Another recent study was conducted in susceptible patients with severe generalized periodontitis using host modulation (SDD) as an adjunct to repeated subgingival debridement.[62] Seventy percent of the patients who completed this 9-month double-blind, placebo-controlled study were smokers. SDD as an adjunct to mechanical therapy versus mechanical therapy alone resulted in significantly greater mean probing depth reductions in pockets of 7 mm or greater at baseline as early as 1 month after therapy (2.52 mm versus 1.25 mm, respectively). These improvements in the SDD group compared with the group receiving mechanical therapy only were maintained during the 5.25 months of therapy (2.85 mm versus 1.48 mm, respectively) and even at 3 months after stopping drug therapy (3.02 mm versus 1.41 mm), demonstrating that no rebound effect occurred. Because of the beneficial effects of HMT in susceptible patients, multicenter studies are using SDD in other susceptible populations, including diabetic, osteoporotic, and institutionalized geriatric patients, as well as smokers.

For more information on special patient populations, please visit the companion Expert CONSULT website at www.expertconsult.com.

Suggested Uses and Other Considerations

Until relatively recently, treatment options for periodontal disease have focused solely on reducing the bacterial challenge by nonsurgical therapy, surgery, and systemic or local antimicrobial therapy. The development of SDD as an HMT, driven by research into the pathogenesis of periodontal disease, is a great example of how translational research can lead to new treatments. By better understanding the biochemical processes that are important in periodontal disease, a pharmacologic principle (doxycycline downregulates MMP activity) has been used in the development of a new drug treatment. Data presented from research studies show the clinical benefits of adjunctive SDD, and the science behind SDD has been transferred into clinical practice. In other words, dentists now have the opportunity to use SDD for patient care, with the aim being to enhance the treatment response to conventional therapy.

Candidate Patients. When deciding whether to use SDD as an adjunct to SRP, first consider the patient's motivation toward periodontal care, the medical history, and the patient's willingness to take a systemic drug treatment. SDD is contraindicated in any patient with a history of allergy or hypersensitivity to tetracyclines. It should not be given to pregnant or lactating women or children less than 12 years old (because of the potential for discoloration of the developing dentition). Doxycycline may reduce the efficacy of oral contraceptives, and therefore alternative forms of birth control should be discussed, if necessary. There is a risk of increased sensitivity to sunlight (manifested by an exaggerated sunburn) seen with higher doses of doxycycline, although this has not been reported in the clinical trials using the sub-antimicrobial dose.

The rationale for using SDD must be clearly explained to the patient. By discussing the etiology of periodontal disease, the available treatment options, and the anticipated outcomes, patients become more interested in their periodontal management, are more likely to comply with treatment, and take more responsibility for managing their disease. Therefore the anticipated compliance and likely commitment to treatment must also be gauged when considering SDD therapy. Patients who show little enthusiasm for complying with the treatment plan or with oral hygiene practices are less likely to be good candidates for systemic drug therapy.

Treatable Periodontal Conditions. SDD is indicated in the management of chronic periodontitis, and studies to date have focused on chronic and aggressive forms of periodontitis.[12,17,62,71] SDD should not be used in conditions such as gingivitis and periodontal abscess or when an antibiotic is indicated. SDD can be used in patients with aggressive periodontitis who are being treated nonsurgically. Furthermore, emerging studies have supported efficacy of SDD as an adjunct to periodontal surgery.[20] SDD may also be of benefit in cases that are refractory to treatment, as well as in patients with risk factors such as smoking, diabetes, osteoporosis/osteopenia, genetic susceptibility, and in whom the treatment response might be limited.

Side Effects. Doxycycline at antibiotic doses (≥100 mg) is associated with adverse effects, including photosensitivity, hypersensitivity reactions, nausea, vomiting, and esophageal irritation. However, in the clinical trials of SDD (20-mg dose), it was reported that the drug was well tolerated, and the profile of unwanted effects was virtually identical in the SDD and placebo groups.[12,17,62,71] The types of adverse events did not differ significantly between treatment groups, and the typical side effects of the tetracycline class were not observed indicating that the appearance of adverse events is dose related.[12,71] Furthermore, there was no evidence of adverse events that could be attributed to antimicrobial effects of treatment and no evidence of developing antibiotic resistance of the microflora after 2 years of continuous use.[12,85,86,92,93] Therefore the drug appears to be well tolerated, with a very low incidence of adverse effects.

Sequencing Prescription with Periodontal Treatment. SDD is indicated as an adjunct to mechanical periodontal therapy and should not be used as a stand-alone or monotherapy. SDD should be prescribed to coincide with the first round of SRP and is prescribed for 3 months, up to a maximum of 9 to 24 months of continuous dosing depending on the patient risk. Modification of any risk factors, such as smoking, nutrition, stress, contributing medications, faulty restorations, poor oral hygiene, and poor diabetic control, should also be addressed at this time. A patient's refusal or inability to modify contributing risk factors is an important consideration for treatment planning and evaluation of therapeutic responses.

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For more information on sequencing prescription with periodontal treatment, please visit the companion Expert CONSULT website at www.expertconsult.com.

Combining with Periodontal Surgery or Local Delivery Systems. Most clinical research to date has focused on using SDD as an adjunct to nonsurgical periodontal treatment. However, emerging data in which SDD was used as an adjunct to access flap surgery in 24 patients revealed better probing depth reductions in surgically treated sites greater than 6 mm compared with surgically treated sites in patients given placebo.[20] Furthermore, the SDD group demonstrated greater reductions in ICTP (carboxy-terminal peptide, a breakdown product of collagen) than the placebo group, indicating that collagenolytic activity was reduced in the patients taking SDD.

SDD treatment can also be combined with the local delivery of antibiotics into the periodontal pocket through sustained-delivery systems. The two treatment approaches target different aspects of the pathogenic process: local delivery systems deliver antimicrobial concentrations of an antibacterial agent directly into the site of the pocket, whereas SDD is a systemic host response modulator. Thus combining these two complementary treatment strategies is another example of how antibacterial therapy (SRP + local antibiotics) can be combined with HMT (SDD) to maximize the clinical benefit for patients. Preliminary results from a 6-month, 180-patient clinical trial designed to evaluate the safety and efficacy of SDD combined with a locally applied antimicrobial (Atridox) and SRP versus SRP alone demonstrated that patients receiving the combination of treatments experienced more than a 2-mm improvement in mean attachment gains and probing depth reductions ($p < 0.0001$) compared with SRP alone.[61]

Monitoring Benefits of Therapy. To improve the ability of dentists to make appropriate treatment decisions for patients undergoing periodontal therapy, it would be extremely useful if they had access to the types of diagnostic tests available to their medical colleagues. Such tests might be used, for example, to distinguish between active and inactive lesions. Studies have shown that SRP alone, although effective for improving clinical parameters such as probing depths, may not be sufficient to reduce excessive levels of many underlying destructive mediators, particularly in more susceptible patients. It would be valuable therefore if it were possible to monitor the levels of such inflammatory mediators as treatment progresses. SDD results in downregulation of MMP activity in inflamed periodontal tissues.[24,27] Therefore, in theory, MMP levels could be monitored before, during, and after SRP plus SDD treatment. Published data support a concomitant reduction in MMP levels in GCF[13,17] and improvements in clinical parameters when combining SDD and SRP.[17] Although chairside tests for MMPs have been developed,[51] they are not in widespread use because of concerns about their specificity or sensitivity.

In the absence of new chairside tests or a centralized diagnostic facility for monitoring the inflammatory status of the tissues, dentists must rely on clinical periodontal monitoring to assess the outcomes of treatment. In addition to the reductions in probing depths and gains in attachment that may be observed after SRP plus SDD, the quality of the periodontal tissues also tends to improve after treatment with SDD. More sensitive radiographic techniques, assessments of bone density, and bone height changes used solely in clinical trials in the past may be possible in clinical practice in the future. Until such diagnostic techniques are made widely available, however, clinicians must rely on clinical judgment to determine the most appropriate course of therapy.

For more information on emerging host modulatory therapies and host modulation factors in systemic disorders, please visit the companion Expert CONSULT website at www.expertconsult.com.

SUMMARY

Periodontal pathogens and destructive host responses are involved in the initiation and progression of periodontitis. Therefore the successful long-term management of this disease may require a treatment strategy that integrates therapies that address both etiologic components. Evidence for the role of MMPs, cytokines, and other mediators in the pathogenesis of periodontal disease distinguishes them as viable targets for a chemotherapeutic approach. The introduction of novel, adjunctive therapies such as host modulation to enhance the efficacy of existing mechanical procedures can contribute favorably to an integrated approach for the long-term, clinical management of periodontitis.

HMTs are an emerging treatment concept in the management of periodontitis. The use of HMT as an adjunct may be particularly useful in susceptible, high-risk patients in whom a prolonged and excessive host response to the presence of bacteria promotes the activity of MMPs and osteoclasts. SDD is the only systemically administered HMT currently approved and indicated as an adjunct to SRP for treating periodontitis. Clinical trials have demonstrated a clear treatment benefit when using SDD versus SRP alone. SDD should be used as part of a comprehensive treatment strategy that includes antibacterial treatments (SRP, plaque control, oral hygiene instruction, local antimicrobials, and periodontal surgery), host response modulation (SDD), and assessment and management of periodontal risk factors. In the future, a range of HMTs targeting different aspects of the destructive cascade of breakdown events in the periodontal tissues are likely to be developed as adjunctive treatments for periodontitis. The further development of these agents will permit dentists to treat specific aspects of the underlying biochemical basis for periodontal disease. The goal is to maximize the treatment response by reducing inflammation and inhibiting destructive processes in the tissues, which will result in enhanced periodontal stability after conventional periodontal treatments such as SRP and surgery. The dentist is now in the exciting position to be able to combine established treatment strategies with new systemic and local drug treatments for this common, chronic disease.

The findings discussed with regard to the use of HMT to better manage chronic periodontal disease may have applications to other chronic systemic diseases such as arthritis, diabetes, osteoporosis, and cardiovascular disease. In addition, studies utilizing locally applied antimicrobials as part of an intensive periodontal therapy (IPT) regimen have shown very promising results. Future studies may demonstrate that in addition to our current standard therapies, IPT with adjunctive antibiotics and/or host modulation for the management of periodontal disease may have profound positive effects on the overall health status of high risk patients. The proper management of local infection and inflammation (periodontitis) will have a significant impact on general overall health of the population.

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At the present time, the only FDA-approved agent for host modulation treatment of periodontal disease is sub-antimicrobial dose doxycycline (SDD) 20 mg per day. This can be used in conjunction with initial therapy and needs to be taken over a prolonged period of up to 9 months. Patients with a history of allergy to the tetracyclines and doxycycline are not able to use this therapy. Also, it is contraindicated in pregnant and lactating women and children younger than 12 years of age. Patients taking oral contraceptives may have reduced protection from pregnancy with this therapy. Most patients will have a small (i.e., less than 1 mm) additive effect on pocket depth reduction when this systemic approach is added to scaling and root planing.

The downregulation of the destructive elements of the host immune response has the potential to add to the spectrum of treatment choices in the future; however, other agents, such as systemic nonsteroidal antiinflammatory drugs (NSAIDs) such as ibuprofen, can cause significant side effects with long-term use and are not advised. Antiosteoporotic agents, such as the bisphosphonates, have minimal effect on periodontal bone loss and carry other risks such as localized bone necrosis.

Modulation of host response may be valuable in enhancing the effects of locally released antimicrobial agents, such as doxycycline and minocycline, and in the nonsurgical treatment of periodontitis in smokers.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 49

Occlusal Evaluation and Therapy

Michael J. McDevitt

CHAPTER OUTLINE

PATHOGENESIS
EVIDENCE-BASED DECISION MAKING
TERMINOLOGY
OCCLUSAL FUNCTION AND DYSFUNCTION
PARAFUNCTION
CLINICAL EXAMINATION (online only)
TMD Screening Evaluation
Testing for Mobility of Teeth
Centric Relation Assessment
Evaluation of Excursions
Articulated Diagnostic Casts
OCCLUSAL THERAPY (online only)
Occlusal Appliance Therapy
Occlusal Adjustment
Occlusal Stability for Restorative Dentistry
SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Among the numerous local and systemic factors with the potential to influence the progression of periodontitis, the patient's occlusion remains a variable requiring exacting diagnosis. All the disciplines of dentistry include comprehensive analysis of occlusal relationships as essential to the determination of appropriate care. The functional demands of the occlusion may fall well within or may exceed the tolerances and the adaptability of a patient's periodontium and their entire masticatory system. The full range of knowledge and skill to analyze all aspects of occlusal anatomy and function is beyond the scope of this text. Rather, this chapter presents practical guidelines for the assessment and management of the occlusion specific to a patient's unique susceptibility to periodontitis.

PATHOGENESIS

Our understanding of the host-driven inflammatory response of each patient to a pathogenic bacterial biofilm is growing exponentially.[32] Each individual's susceptibility appears to be so specific that they are actually their only reference in the interpretation of possible contributing factors to progressive loss of supporting bone. Additionally, we observe that destructive events may be episodic and are clearly site specific. Our diagnostic responsibility includes careful measuring of periodontal structures in the entire circumference of each tooth, accurate documentation, and timely reassessment. Periodontal deterioration that occurs rapidly or is excessive for a person's age should prompt the clinician to investigate any variable that might amplify that patient's periodontitis. If a local factor, such as an occlusal relationship, can influence the course of the disease, then its analysis must be as precise as any other aspect of the periodontal examination.

EVIDENCE-BASED DECISION MAKING

Ideally, all our diagnostic and therapeutic decisions would reflect evidence from multiple prospective clinical trials subject to systematic review. Prospective human investigations of occlusal trauma are considered unethical so periodontics has conscientiously struggled to reach discipline-wide consensus about the interaction of patient's occlusion and their periodontal status.[11] To be clinically applicable, the investigation's methodology must parallel the clinical diagnosis and treatment of an individual patient with periodontitis.[13] Historically, data management and statistical credibility and methodology within retrospective studies limited the ability of dedicated researchers to interpret the role of occlusion in an individual's periodontitis experience.[14,31,34] Grouping of data points, especially in large study populations, departs from the site specificity that periodontal diagnosis requires. If occlusal trauma is affecting a tooth, the effect on the periodontium is site specific for only that tooth. Treatment for a patient with periodontitis would never be based on an average of diagnostic references but rather on their unique susceptibility, anatomy, occlusion, and history.

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Nunn and Harrell[28] reported in 2001 the retrospective findings of a group of patients with periodontitis in which the analysis was based on the measurement of loss of attachment of each individual tooth and the presence or absence of occlusal interferences. This study and a similar investigation[4] confirmed that trauma from occlusion amplified loss of attachment. Harrell and Nunn[12] also reported that eliminating occlusal interferences had a positive influence on the outcome of treatment when trauma from occlusion was found to be a contributing local factor. The positive influence of occlusal adjustment on the outcome of both surgical and nonsurgical periodontal therapies had also been reported by Burgett.[5] Evidence now appears to support the possibility that trauma from occlusion has the capability to amplify damage to an inflamed periodontium.

Interest in the occlusion within the discipline of periodontics appears to be increasing, especially with the rapid growth of implant replacement of missing teeth. Despite some conflicting evidence in the literature, common ground for consensus exists. There is general agreement that occlusal force has an effect on the periodontium, as described in Chapter 15, and that susceptibility to periodontitis is unique to each patient. Occlusal forces present themselves across a broad spectrum. No or minimal occlusal contact on a tooth results in disuse atrophy of the periodontium, which may result in instability of that tooth. Harmonious occlusal force on a tooth stimulates physiologic arrangement of its periodontal attachment fibers and osseous architecture and encourages its stability. Forces that exceed the tolerance of the periodontium result in resorption of the bone and disruption of the attachment.[16,25,27] In the healthy person, the periodontium around teeth subject to excessive occlusal force experiences adaptation and repair or remodeling with no loss of attachment, as often occurs with orthodontics. For the patient who is losing bone as the result of periodontitis, coupling the ongoing inflammatory disease with excessive occlusal force may result in the amplification of destruction and damage to the periodontium of affected teeth.[28] If this conclusion is valid, then the clinician has the responsibility to correlate the precise analysis of the periodontal status of each tooth with its occlusal responsibilities and its possible occlusal excesses.

TERMINOLOGY

The following is a list of key descriptive terms used in this chapter, along with common synonyms:

Maximum intercuspation: The position of the mandible when there is maximal interdigitation and occlusal contacts between maxillary and mandibular teeth; also called *centric occlusion* and *intercuspal position.*

Centric relation: The position of the mandible when both condyle-disc assemblies are in their most superior position in their respective glenoid fossa and against the slope of the articular eminence of each respective temporal bone.

Initial contact in centric relation: The first occlusal contact in the centric relation closure arc.

Excursive movement: Any movement of the mandible away from maximum intercuspation.

Lateral excursion: Movement of the mandible laterally to the right or to the left from maximum intercuspation.

Working side: The side of either dental arch corresponding to the side of mandible moving away from the midline during a lateral excursion.

Nonworking side: The side of either arch corresponding to the side of the mandible moving toward the midline during a lateral excursion; also called balancing side.

Protrusion: Movement of the mandible anteriorly from maximum intercuspation.

Retrusion: Movement of the mandible posteriorly relative to a more anterior position.

Guidance: The pattern of opposing tooth contact during excursive movements of the mandible.

Disclusion: The separation of certain teeth caused by guidance provided by other teeth during an excursion. When anterior guidance provides for separation of posterior teeth during an excursion, posterior disclusion is achieved.

Interference: Any occlusal contact in the centric relation closure arc or in any excursion that prevents the remaining occlusal surfaces from achieving stable contact or from functioning harmoniously and/or that encourages masticatory system disharmony; also called an *occlusal discrepancy.*

OCCLUSAL FUNCTION AND DYSFUNCTION

Excellent sources of comprehensive understanding of dental anatomy and function include texts by Wheeler,[3] McNeill,[23] and Dawson.[8,9] The determination if trauma from occlusion is occurring is based on the conclusion that the composite of all occlusal forces on a specific tooth exceeds the tolerance or adaptability of its periodontium. The identification of masticatory system disharmonies begins with appreciation of physiologic norms allowing the clinician to recognize dysfunctional relationships, which might influence the accuracy of diagnosis.[9,27,29]

Centric relation is a term used to describe the position of both condyles when they are fully seated in the fossa of their respective temporomandibular joints (TMJs). Rotation of the mandible around an axis through both condyles is called the *centric relation closure arc* (see Chapter 20). This is strictly a skeletal relationship until tooth contact occurs. Maximum intercuspation occurs when opposing teeth make contact, with optimal interdigitation, at the most stable endpoint of mandibular closure. Stability is enhanced by simultaneous bilateral contact of multiple posterior teeth with occlusal forces in the long axis of most posterior teeth. If the initial tooth contact in the centric relation arc of closure occurs simultaneously with maximum intercuspation, then the teeth do not displace condyles. Conversely, if the teeth are firm and any contact occurs before maximum intercuspation, then incline relationships of opposing occlusal surfaces will guide the mandible into intercuspal position, requiring one or both condyles to become dislocated from their fossa.[9,28] If teeth are mobile and contact first in the centric relation closure arc, then *they may move away from opposing teeth rather than cause condylar displacement.*

Cusp-fossa or cusp-marginal ridge relationships of posterior teeth provide resistance to vertical loading and functional stability to a patient's dentition. When occlusal forces load teeth in their long axis, the periodontium is the most resistant and supportive.[8,9] The anterior teeth can be stable with little occlusal loading in centric occlusion if favorably influenced by oral musculature. If the anterior teeth are in contact in maximum intercuspation, they are coupled. Movement of the mandible from centric occlusion is called an *excursion.* Movement forward is called a *protrusive excursion,* and movement to either side is called a *lateral excursion.* If the mandible can move posteriorly, it is called *retrusion.* There is evidence that contact of posterior teeth in excursions can overload those teeth with negative dental, periodontal, muscular, and TMJ consequences.* The ideal relationship might be light coupling of

*References 1, 6, 9, 29, 37, 38, and 39.

anterior teeth in centric occlusion with immediate separation, called *disclusion,* of all posterior teeth in all excursions.[38] During a lateral excursion, posterior teeth that contact on the same side as the direction of mandibular movement are described as having a working contact. Posterior teeth that contact on the side opposite the direction of the lateral excursion are described as having a nonworking contact. Although nonworking contacts are classically associated with more potential negative consequences,[39] analysis of working contacts and the function of anterior teeth is critically important. Contacts disruptive to mandibular movement or stressful to individual teeth are called *occlusal interferences* or *discrepancies.* Our ability to analyze the occlusion to identify contacts that may amplify a patient's periodontitis affecting certain teeth is strategic to correct diagnosis.

Inflammation disrupts the integrity of the attachment apparatus, resulting in less resistance to force from opposing teeth. When bone loss has occurred, there is less root surface area supported[2,15] and fewer sensory fibers in the periodontal ligament, limiting protective muscle modulation of occlusal forces.[33] The clinician must differentiate between inflammation-caused intolerance to occlusal forces, normal forces on teeth with reduced periodontal support, excessive occlusal forces, and well-tolerated forces on teeth affected by periodontitis.

PARAFUNCTION

Bruxism may cause occlusal forces on teeth susceptible to periodontitis to be increased in intensity and/or frequency, magnifying potential amplification of damage.[7,18,19,35] Daytime occlusal parafunction is commonly limited to clenching of the teeth during incidents requiring a person's focused effort or mental concentration. Nighttime bruxing of the teeth can take the form of grinding the teeth in various excursions and/or clenching of the teeth. Sleep bruxism is probably an extension of the rhythmic masticatory muscle activity that is also observed in nonbruxers. Why nuclei in the brainstem allow bruxing to occur with some individuals while others are spared is unclear.[7,18,19] Bruxing is associated with greater frequency and persistence of TMJ dysfunction, orofacial pain, and possibly periodontal attachment loss.[35] Sensory input of teeth subject to bruxism is probably dampened, which may interfere with both diagnosis and treatment.[30] There seems to be limited influence on bruxing tendencies from occlusal interferences.[20] Selective serotonin reuptake inhibitors, such as Prozac, have been reported to encourage bruxism.[10]

For more information on the clinical examination and occlusal therapy, please visit the companion Expert CONSULT website at www.expertconsult.com.

SUMMARY

Confirmation of appropriateness of occlusal therapy is the product of thorough and complete evaluation of both the patient's occlusion and their masticatory system. The sequence of occlusal treatment begins with antiinflammatory therapy and progresses through reversible appliance therapy before considering any of the irreversible options. This provides the clinician with the most careful approach to assessing and to treating the occlusion of a patient with periodontitis.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 50

Adjunctive Role of Orthodontic Therapy

Vincent G. Kokich

CHAPTER OUTLINE

BENEFITS OF ORTHODONTIC THERAPY
PREORTHODONTIC OSSEOUS SURGERY (online only)
Osseous Craters
Three-Wall Intrabony Defects
ORTHODONTIC TREATMENT OF OSSEOUS DEFECTS (online only)
Hemiseptal Defects
Advanced Horizontal Bone Loss
Furcation Defects
Root Proximity
Fractured Teeth and Forced Eruption
Hopeless Teeth Maintained for Orthodontic Anchorage
ORTHODONTIC TREATMENT OF GINGIVAL DISCREPANCIES (online only)
Uneven Gingival Margins
Significant Abrasion and Overeruption
Open Gingival Embrasures
SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Orthodontic tooth movement may be a substantial benefit to the adult periorestorative patient. Many adults who seek routine restorative dentistry have problems with tooth malposition that compromise their ability to clean and maintain their dentitions. If these individuals also are susceptible to periodontal disease, tooth malposition may be an exacerbating factor that could cause premature loss of specific teeth.

Orthodontic appliances have become smaller, less noticeable, and easier to maintain during orthodontic therapy. Many adults are taking advantage of the opportunity to have their teeth aligned to improve the esthetics of their smiles. If these individuals also have underlying gingival or osseous periodontal defects, these defects often can be improved during orthodontic therapy if the orthodontist is aware of the situation and designs the appropriate tooth movement. In addition, implants have become a major part of the treatment plan for many adults with missing teeth. If adjacent teeth have drifted into edentulous spaces, orthodontic therapy is often helpful to provide the ideal amount of space for implants and subsequent restorations.

This chapter shows the ways in which adjunctive orthodontic therapy can enhance the periodontal health and restorability of teeth.

BENEFITS OF ORTHODONTIC THERAPY

Orthodontic therapy can provide several benefits to the adult periodontal patient. The following six factors should be considered:

1. Aligning crowded or malposed maxillary or mandibular anterior teeth permits the adult patient better access to clean all surfaces of their teeth adequately. This could be a tremendous advantage for patients who are susceptible to periodontal bone loss or do not have the dexterity to maintain their oral hygiene.
2. Vertical orthodontic tooth repositioning can improve certain types of osseous defects in periodontal patients. Often, the tooth movement eliminates the need for resective osseous surgery.
3. Orthodontic treatment can improve the esthetic relationship of the maxillary gingival margin levels before restorative dentistry. Aligning the gingival margins orthodontically avoids gingival recontouring, which could require bone removal and exposure of the roots of the teeth.
4. Orthodontic therapy also benefits the patient with a severe fracture of a maxillary anterior tooth that requires forced eruption to permit adequate restoration of the root. Erupting the root allows the crown preparation to have sufficient resistance form and retention for the final restoration.
5. Orthodontic treatment allows open gingival embrasures to be corrected to regain lost papilla. If these open gingival embrasures are located in the maxillary anterior region, they can be unesthetic. In most patients, these areas can be corrected with a combination of orthodontic root movement, tooth reshaping, and restoration.

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During orthodontic movement in patients, it is essential that all calculus is removed and plaque is minimized. In most patients, this can be accomplished with initial therapy and detailed oral hygiene instructions accompanied by the use of antibacterial agents such as chlorhexidine. Failure to control these factors can lead to accentuation of gingivitis and even bone loss. The control of periodontal inflammation is so important that in a few patients (e.g., those with furcation involvement), it may be necessary to use periodontal flap surgery to get access to the root surfaces so that root planing is completed before moving the teeth.

Orthodontic extrusion may be used to move teeth coronally in selected patients needing crown lengthening. In some cases, this can also be done with hopeless teeth as a prelude to implant placement so that bone levels are more coronal with improved bone dimension and for enhanced support of interproximal bone for maintenance of an esthetic papillary form to avoid black triangles.

Intrabony defects are sometimes improved by orthodontic movement, but this is not a reliable way to treat all advanced cases of periodontitis.

6. Orthodontic treatment could improve adjacent tooth position before implant placement or tooth replacement. This is especially true for the patient who has been missing teeth for several years and has drifting and tipping of the adjacent dentition.

For more information on preorthodontic osseous surgery, orthodontic treatment of osseous defects, and orthodontic treatment of gingival discrepancies, please visit the companion Expert CONSULT website at www.expertconsult.com.

SUMMARY

There are many benefits to integrating orthodontics and periodontics in the management of adult patients with underlying periodontal defects. The key to treating these patients is communication and proper diagnosis before orthodontic therapy, as well as continued dialog during orthodontic treatment. Not all periodontal problems are treated in the same way. This chapter provides a framework for the integration of orthodontics to solve periodontal problems.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 51

Diagnosis and Management of Endodontic-Periodontic Lesions

Kenneth C. Trabert and Mo K. Kang

CHAPTER OUTLINE

BIOLOGIC EFFECTS OF PULPAL INFECTION ON PERIODONTAL TISSUES
BIOLOGIC EFFECTS OF PERIODONTAL INFECTION ON DENTAL PULP
DIFFERENTIAL DIAGNOSIS OF PULPAL AND PERIODONTAL INFECTION
TREATMENT CONSIDERATIONS OF ENDODONTIC-PERIODONTIC LESIONS

Refer to the Expert CONSULT companion website at www.expertconsult.com for a supplement on this topic. All figures are online.

The simultaneous existence of pulpal problems and inflammatory periodontal disease can complicate diagnosis and treatment planning and affect the sequence of care to be performed. This chapter will analyze these problems.

Persistent infection in the pulp tissue may lead to secondary infection and breakdown of tissues in the periodontium. Continued periradicular bone loss and extension of the lesion to the external mucosal surface may result in the formation of a sinus tract (Figure 51-1 online). Conversely, severe periodontal disease may initiate or exacerbate inflammatory changes in the pulp tissue. This mutuality of infection between pulp and periodontium is mediated through physical routes, allowing for communication between the two structures. The main and obvious route of communication is the apical foramina. Advanced pulpitis will lead to pulp necrosis, which often is accompanied by inflammatory bone resorption at the root apex, as found in cases of *chronic periradicular periodontitis* (CPP) or chronic radicular abscess (CPA) (Figure 51-2 online). This is also known as *retrograde periodontitis* because it represents the periodontal tissue breakdown from an apical to cervical direction and is the opposite of *orthograde periodontitis* that results from a sulcular infection. This is typically identified as a periapical radiolucency (Figure 51-3 online). By far, the retrograde periodontitis is the most common example of pulpal diseases leading to secondary periodontal breakdown. Apical foramina can also lead to pulpal inflammatory changes secondary to severe periodontitis in cases in which the periodontal defect reaches the apical foramina.

Alternatively, lateral or accessory canals may be the route of such communications. Prevalence of accessory root canals in various human teeth and their contribution to the complexity of the root canal system have been well established. Accessory canals are found along the length of the root canals, albeit to varying frequencies, depending on their location. Earlier studies, using the "clearing technique" for transparent root canal visualization, showed that 59.5% of maxillary second premolars possess lateral canals; 78.2% of those are found in the apical regions.[156] Notably, accessory canals were also found in midroot and cervical regions, albeit with reduced frequencies at 16.2% and 4.0%, respectively. A subsequent study showed that 28.4% of permanent molars exhibit patent accessory canals in furcation regions,[51] suggesting that these accessory canals allow the pulpal and periodontal continuum to exist. Root canal therapies fail frequently in maxillary molars because of unidentified second mesial canals, which are found in a surprisingly high percentage (80.8%) of teeth.[67] Clearly, accessory canals can lead to CPP resulting from chronic pulpal diseases. This can be readily detected in periapical radiographs (Figure 51-4 online), and the periodontal lesions heal after successful completion of endodontic therapy. Questions arise as to whether pulpal disease can develop from periodontal infections through accessory root canals. Kirkham[76] reported that out of 100 permanent human teeth extracted as the result of severe periodontal disease, only 2 teeth possessed accessory canals within the periodontal pockets. Thus the likelihood of primary periodontal infection reaching the dental pulp through accessory canals is rare.

The third route is dentinal tubules. Dentinal tubules maintain a tapered structure along the length from the pulpodentin complex (PDC) to the dentinoenamel junction (DEJ) with the diameter of 2.5 μm at PDC and 0.9 μm at the DEJ.[149] It is therefore conceivable that dentin is a permeable structure and that the permeability changes at different locations differ according to the size and density of the dentinal tubules. Clearly, the tubular diameter is large

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enough to allow bacterial penetration and colonization inside the tubule. Because of the tapered structure, permeability would increase when the carious lesion extends deeper into the dentin. Bacterial colonization in the tubules from infected root canals has been well documented.[132] Also, bacterial invasion into dentinal tubules from the periodontal pocket has been demonstrated,[50] suggesting that dentinal tubules may allow pulpal irritation from chronic periodontal infections.

BIOLOGIC EFFECTS OF PULPAL INFECTION ON PERIODONTAL TISSUES

The effects of pulpal disease on the surrounding periodontal tissues is widely accepted.

For more information on the biologic effects of pulpal infection on periodontal tissues, please visit the companion Expert CONSULT website at www.expertconsult.com.

When the endodontic infection is left untreated, it becomes one of the risk factors for the progression of periodontal disease.[130] Untreated and unresolved infections of endodontic origin can lead to additional periodontal pocket formation, increased bone loss, and impaired wound healing and can aggravate the development and progression of the periodontal disease after periodontal treatment has been completed.

BIOLOGIC EFFECTS OF PERIODONTAL INFECTION ON DENTAL PULP

The effects of periodontal disease on the dental pulp appear to be more controversial compared to the effects of pulpal disease on the periodontium.[11,131] Not all researchers agree about the effect of periodontal disease on the pulp. Even though inflammation and localized pulpal necrosis have been observed next to lateral canals exposed by periodontal disease,[126,130,131] other studies have not confirmed a correlation between periodontal disease and changes within the pulp.[25,97,148]

For more information on the biologic effects of periodontal infection on dental pulp, please visit the companion Expert CONSULT website at www.expertconsult.com.

DIFFERENTIAL DIAGNOSIS OF PULPAL AND PERIODONTAL INFECTION

Acute infection of both the periodontium and the pulp must be differentiated from one another for clinicians to be able to establish a correct diagnosis with reasonable certainty and to initiate appropriate therapy.

For more information on the differential diagnosis of pulpal and periodontal infection, please visit the companion Expert CONSULT website at www.expertconsult.com.

Making the accurate differentiation between pulpal and periodontal lesions can be challenging. If the lesion originates from a pulpal infection and yet was treated by extensive periodontal therapy, it will not resolve. Conversely, performing endodontic therapy on a tooth that has an extensive periodontal defect and a vital pulp will result in the persistence of the periodontal infection. Thus, identifying the primary cause of infection is a critical determinant of treatment outcome. Perhaps the most important consideration when making such a distinction is to base the diagnosis on multiple findings. Those include patients' symptoms, coronal integrity, shape and size of the radiographic lesions, periodontal probing, and tooth vitality.

Patients' Subjective Symptoms. Patients suffering from the acute phase of pulpal infection present with symptoms that are generally absent in chronic periodontal infection. During the initial stage of pulpitis, patients may complain of sensitivity and pain exacerbated by certain stimuli, including temperature changes, pressure, and/or biting. If the symptoms result from a reversible pulpitis, they will usually resolve spontaneously with time as the result of various mechanisms such as closure of dentinal tubules, clearance of microbial irritants and toxins, and reparative dentin formation. Persistent inflammation leads to irreversible pulpitis, which is often associated with sharp and untriggered pain, although asymptomatic pulpitis may also occur as described previously. The acute pain to thermal stimuli may subside after several days as the pulp becomes necrotic, and the bacteria and their by-products migrate apically in the complex canal system. As the infection extends to and then past the apical foramen or a lateral periradicular canal, the tooth becomes particularly sensitive to bite pressure and percussion. After several days, the necrotic tooth may develop a periradicular abscess that results in elevation of the tooth from the dentoalveolar complex. Patients frequently report that the tooth feels "high" on occlusion. However, patients with irreversible pulpitis or chronic pulpal infection (i.e., necrosis) may be completely asymptomatic. Thus, the diagnosis of primary pulpal infection must be made with objective findings, such as the patients' responses to percussion, palpation, biting, periodontal probing, and vitality testing, as well as a thorough evaluation of the patient's addition to the subjective symptoms. Use of transillumination is also extremely important to rule out the possibility of root fracture.

In periradicular and periodontal abscesses, the extent of pain may vary. Generally, an acute periradicular abscess causes extreme pain to pressure, bite, percussion, and, at times, to palpation if the infection has penetrated the cortical bony plate. Periodontal abscesses are thought to cause less pain because there is little or no elevation of the periosteum. Edema and swelling are characteristics that may be shared by both conditions. The swelling and edema with a periodontal abscess is generally confined to the cervical portion of the tooth. A periradicular abscess is usually more sensitive to palpation around the tooth apex if the infection has penetrated through the bony cortical plate. Redness and a smooth appearance of the marginal gingival tissues are more common with abscesses of periodontal origin, whereas redness can be detected more apically if a pulpal abscess has started to swell and elevate the surrounding tissues.

Objective findings in periodontal abscesses include bleeding on probing, suppuration, increased pocket depth, increased tooth mobility, and occasionally, lymphadenopathy.[57] Abscesses of endodontic origin usually probe normally but may also display increased mobility, depending on the amount of bone loss. Patients may describe the tooth as feeling longer or higher than the adjacent teeth.[48]

Suppuration and drainage from periapical and periodontal abscesses differ greatly. Periodontal abscesses are associated with severe periodontal destruction. In a study investigating the incidence of anaerobes in periodontal abscesses, approximately 66% were determined to have a suppurative exudate that was evident during probing. In the same study, 100% of the patients had

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bleeding during probing; more than 75% had severe edema, redness, and swelling; and 78% of the patients had some degree of mobility. Only a few of the patients suffered from lymphadenopathy. Over 60% of the patients had not received previous periodontal therapy and nearly 70% of the teeth were molars. The average measured pocket depth was 7.28 mm.[57] In another study that cultured facultative anaerobic bacteria, the teeth most affected were maxillary and mandibular anteriors and mandibular molars. One of the typical features in this study, as in the previous one, was the fact that most of the teeth had been previously untreated.[96] Other aspects of periodontal therapy that correlate highly with the development of acute periodontal abscess formation are (1) patients undergoing current periodontal treatment,[135] (2) the incomplete removal of calculus,[29] (3) a history of a previous abscess at the same site,[99] and (4) the previous use of antibiotics for dental or nondental reasons.[56]

The type of drainage in an acute periradicular abscess usually comes from one of two sites. The most prevalent site is a sinus tract that develops when the area of swelling breaks through the mucoperiosteum and exits the mucosal tissue, either near or at some distance from the site of the infection. Additionally, the path of least resistance may be along the periodontal ligament and the infection may dissect the ligament along the surface of the root and exit the tooth at the height of the epithelial attachment. This results in a periodontal defect that probes along a narrow path to the apex of the root. Both the sinus tract and narrow sulcular lesions can usually be traced to the infected tooth or offending root using a gutta percha point.

Coronal Integrity. Periodontal infection without pulpal involvement may present with intact crown structure and absence of coronal defects (see Figure 51-6, *A,* online). On the other hand, endodontic infection is almost always associated with loss of coronal integrity, such as occurs with caries, failing restorations, extensive restorations and the existence of cracks or fractures that extend to the pulpal tissues (see Figures 51-3 and 51-4 online). However, this does not mean that all periodontal infections are devoid of coronal defects nor that all endodontic lesions exhibit loss of coronal integrity. When pulp tissue is severed by laxation or avulsion, for example, one would expect to find a necrotic pulp in the absence of coronal defects. If left untreated, such lesions originating from primary pulpal infection will lead to the breakdown of the periodontium as in CPP or CPA. Of course, primary periodontal lesions can develop in teeth with coronal defects. Furthermore, true combined lesions (so-called "endo-perio" lesions) would present with periodontal infection with extensive coronal destruction. However, careful examination of the coronal status either during intraoral or radiographic examination and careful attention to the patients' subjective symptoms can provide supportive information in deciding whether the lesion originates from an endodontic or periodontal infection.

Radiographic Appearance. Periapical radiographs can provide distinguishing information whether the lesion is of pulpal or periodontal origin. Although radiographic findings are objective data, interpretation of radiographs can be highly subjective, depending on who is reading the radiograph. Thus it is important to understand to focus on specific entities on a radiograph, including the coronal status, crestal bone height and shape, presence of periapical or lateral radiolucency, bony trabeculation, integrity of lamina dura, and careful evaluation of the obturation status of the root canal space if endodontic therapy has been previously attempted. Coronal status as revealed on a radiograph can help the differential diagnosis as described previously. Also, periradicular lesions originating from primary pulpal infection will lead to a retrograde periodontitis, which migrates from the root apex in a cervical direction (see Figure 51-3 online). On the contrary, periodontal infections will lead to the loss of crestal bone from the cervical apical direction (see Figures 51-6 and 51-7 online). Thus, radiographic lesions appear different in endodontically treated teeth compared to periodontal lesions, and the shape of the bony lesions can help distinguish between the two. For example, radiographic lesions representing orthograde severe periodontitis will appear wider at the cervical end than the apical portion of the lesion. Periapical or lateral radiolucencies may also result from differences in trabeculation patterns not associated with pulpal infection. For this reason, it is critical to consider the integrity of the lamina dura, which almost always is violated in the periapical or lateral radiolucency representing a chronic or acute pulpal infection. If the tooth had been endodontically treated, assessment of the previous obturation qualities (i.e., voids, short or over fill, missed canals, etc.) is also important. These radiographic features will provide very useful and often distinguishing information in making the differential diagnosis between pulpal and periodontal lesions more accurate.

Vitality. The testing of tooth vitality often becomes one of the most important tests that can differentiate a periodontal and a periradicular infection. Teeth with a periodontal infection usually test vital to thermal testing unless the acute condition is a true combined lesion in which both endodontic and periodontal compartments have become diseased. Teeth with both a periradicular infection and a periodontal abscess usually test nonvital. Exceptions to this are either extremely calcified canals, extensively restored teeth, or multirooted teeth in which some canals may be necrotic as the result of either pulpal or periodontal disease. Other canals may still retain vital tissue that respond to thermal or electric pulp testing. Thermal testing is usually the most reliable way of determining pulpal health or disease. Patients with an irreversible pulpitis often report a lingering painful response to a thermal stimulus. In later stages of pulpitis, heat exacerbates the symptom more than the cold, and the application of cold may even cause short-term pain relief.[70] Although thermal testing can be informative as to the status of the pulp, a patient's response to thermal stimuli may be confused with hypersensitivity resulting from exposed dentin and patent dentinal tubules without pulpitis.[3] Therefore, thermal testing must be combined with other diagnostic criteria, as discussed previously, to distinguish between the lesions originating from pulpal or periodontal infection.

TREATMENT CONSIDERATIONS OF ENDODONTIC-PERIODONTIC LESIONS

In managing the lesions of pulpal or periodontal origin, making an accurate diagnosis as to the source of infection is a critical determinant of the treatment outcome. Primary pulpal lesions combined with secondary periodontal defects would completely resolve by conventional root canal therapy (RCT) alone. Figure 51-4 (online) shows a chronic pulpal infection associated with pulp necrosis that led to a periodontal defect via the endodontic-periodontic continuum through a lateral canal. This is a classic example of primary pulpal infection causing a secondary periodontal defect. In this case, RCT alone led to complete resolution of the periradicular periodontitis involving the furcal defect. This type of lesion and the healing pattern are observed in almost all periodontal defects resulting from primary pulpal infection and rapid bone loss in the periodontal tissues.

SCIENCE TRANSFER

Bacterial-induced inflammation of the pulpal and periodontal tissues can often be seen together. Endodontic lesions are more likely to spread to surround periodontal tissues with subsequent bone destruction than periodontal infections spread to the pulpal tissues to cause retrograde pulpitis.

The diagnosis of the etiology of dental abscesses is based on patient history in which pulpal pathology causes significant pain exacerbated by hot and cold temperature changes; radiographic evaluation may show a circumscribed periapical lesion when the origin is pulpal. Vitality testing can detect changes of sensation caused by pulpal inflammation and necrosis. If there is evidence of pulpal disease and the possibility of associated periodontal bone loss, the endodontic treatment should be completed first and then the patient should be reevaluated. In many cases, apparent periodontal pathology, including bone loss, suppuration, and pocket depth resolves if there has been a pulpal lesion that has been successfully treated.

Residual periodontal problems can be treated after completion of successful endodontic treatment, and in many cases, successful regeneration of periodontal defects is possible in endodontically treated teeth.

Lesions originating from pulpal infections require endodontic therapy but would not resolve lesions resulting from primary periodontal infections. This is exemplified in Figure 51-7 (online). In Case 1, a preoperative radiograph of "independent" endodontic-periodontic lesions shows a periapical radiolucency spanning the entire length of the distal root of mandibular first molar and moderate bone loss at the mesial aspect. On completion of endodontic therapy, the periodontal breakdown around the distal root completely resolved, whereas the mesial bony defect remained unchanged. Case 2 shows a "combined" endodontic-periodontic lesion involving extensive periradicular defects around the apices of the mandibular first molar and a periodontal defect in the bifurcation. After successful endodontic therapy alone, the periradicular lesions and the furcation defect have resolved, but incomplete healing is noted in the coronal aspect of the furcation. This finding presumes that the unhealed defect originates from a primary periodontal infection.

Therefore, endodontic-periodontic lesions require both endodontic and periodontal therapies for complete healing to occur. This is true whether the endodontic-periodontic lesions are independent or combined. One important consideration is the sequence of therapies: Which of the two should be performed first? As discussed earlier, endodontic lesions are often associated with more pronounced symptoms than periodontal lesions.

More importantly, in combined endodontic-periodontic lesions, some periodontal defects will resolve on completion of the endodontic treatment, whereas the opposite would not be the case. After resolution of the secondary periodontal defect stemming from a primary pulpal infection, the residual periodontal disease may be more accurately and predictably managed. These considerations indicate that combined endodontic-periodontic lesions are best treated by first performing the necessary endodontic care followed by periodontal therapy.

In cases that present with an abscess, the periodontal and periradicular abscesses are managed differently. Previous studies have suggested that the treatment of the patient with an acute periodontal abscess should be performed in two stages. In the first stage, the management of the acute lesion is performed. In the second stage, a more comprehensive treatment of the original and any residual lesions are accomplished.

The management of the acute periodontal abscess involves establishing drainage via the periodontal pocket and subgingival scaling and root planing. Curettage of the epithelium lining, the pocket, and surrounding connective tissue is then accomplished, followed by compression of the pocket wall. If the swelling is large and fluctuant, flap surgery or incision and drainage may be necessary to relieve the pressure. In cases where the bone loss is too extensive and the prognosis for the tooth is hopeless, extraction may be required.[153]

Rationale use of antibiotics should be considered in acute periodontal abscesses.[63] The use of systemic antibiotics may be indicated when patients have elevated temperatures, cellulitis, and systemic disease and are immunocompromised.[153] In a study by Jaramillo,[63] some periodontal pathogens showed resistance to tetracycline, metronidazole, and amoxicillin but not azithromycin. In the management of acute periradicular abscesses, the abscess should first be drained by performing a pulpectomy or incision and drainage. The choice of which technique to use may be based on time constraints. The incision and drainage procedure requires less time to accomplish and yet relieves the pressure that has accumulated under the subperiosteal tissues that results in a fluctuant, swollen, and painful lesion. The pulpectomy removes the infected tissues and microorganisms from within the canal system, and calcium hydroxide is placed into each of the canals. Calcium hydroxide provides both biocompatibility and helps to block the canals from coronal leakage. Its use has proved to be a suitable intracanal medicament because of its stability and bactericidal effect in a limited space.[69]

If adequate drainage is achieved via incision and drainage, debridement, and medication of the canal system, antibiotics generally have no additional benefit. In the event of systemic complications, such as fever, lymphadenopathy, or cellulitis or in an immunocompromised patient, antibiotics may be prescribed in addition to drainage of the tooth.[95]

Penicillin V or amoxicillin are still the antibiotics of choice against the majority of bacteria isolated from acute endodontic infections. If penicillin V therapy is ineffective, the combination of penicillin V with metronidazole or amoxicillin–clavulanate potassium is recommended. The use of clindamycin is another excellent alternative.[72] The management of pain associated with acute apical periodontitis is well controlled by prescribing systemic nonsteroidal antiinflammatory drugs (NSAIDs) with or without the additional use of Tylenol. Narcotics are usually unnecessary unless an acute flare-up occurs.[139] The correct diagnosis of periodontal and periapical abscesses is the most critical step in the resolution of the disease process. Failure to properly diagnose and treat these often encountered conditions result in the progression of the disease, continued loss of bone and periodontal attachment apparatus, a poor prognosis, and possible loss of the tooth.

Reference can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 52

Phase II Periodontal Therapy

Fermin A. Carranza and Henry H. Takei

CHAPTER OUTLINE

OBJECTIVES OF THE SURGICAL PHASE
Surgical Pocket Therapy
Results of Pocket Therapy
POCKET ELIMINATION VERSUS POCKET MAINTENANCE
REEVALUATION AFTER PHASE I THERAPY
CRITICAL ZONES IN POCKET SURGERY
Zone 1: Soft Tissue Pocket Wall
Zone 2: Tooth Surface
Zone 3: Underlying Bone
Zone 4: Attached Gingiva
INDICATIONS FOR PERIODONTAL SURGERY
METHODS OF POCKET THERAPY
Criteria for Method Selection
Approaches to Specific Pocket Problems

Although in a strict sense, all instrumental therapy can be considered surgical, this chapter refers only to those techniques that include the intentional severing or incising of gingival tissue* with the following purposes:

- Controlling or eliminating periodontal disease.
- Correcting anatomic conditions that may favor periodontal disease, impair esthetics, or impede placement of the correct prosthetic appliances.
- Placing implants to replace lost teeth and improving the environment for their placement and function.

OBJECTIVES OF THE SURGICAL PHASE

The surgical phase of periodontal therapy has the following main objectives:

1. Improvement of the prognosis of teeth and their replacements.
2. Improvement of esthetics.

The surgical phase consists of techniques performed for pocket therapy and for the correction of related morphologic problems, namely, mucogingival defects. In many cases, procedures are combined so that one surgical intervention fulfills both objectives.

The purpose of surgical pocket therapy is to eliminate the pathologic changes in the pocket walls; to create a stable, easily maintainable state; and if possible, to promote periodontal regeneration. To fulfill these objectives, surgical techniques (1) increase accessibility to the root surface, making it possible to remove all irritants; (2) reduce or eliminate pocket depth, making it possible for the patient to maintain the root surfaces free of plaque; and (3) reshape soft and hard tissues to attain a harmonious topography. Pocket reduction surgery seeks to reduce pocket depth by either resective or regenerative means or often by a combination of both methods (Box 52-1). Chapters 59 to 61 describe the different techniques used for these purposes.

The second objective of the surgical phase of periodontal therapy is the correction of anatomic morphologic defects that may favor plaque accumulation and pocket recurrence or impair esthetics. It is important to understand that these procedures are not directed to treat disease but aim to alter the gingival and mucosal tissues to correct defects that may predispose to disease. They are performed on noninflamed tissues and in the absence of periodontal pockets. Three types of techniques fall into this category, as follows (see Box 52-1):

- *Plastic surgery techniques* are used to create or widen the attached gingiva by placing grafts of various types.
- *Esthetic surgery techniques* are used to cover denuded roots and to recreate lost papillae.
- *Preprosthetic techniques* are used to adapt the periodontal and neighboring tissues to receive prosthetic replacements; these include crown lengthening, ridge augmentation, and vestibular deepening.

The plastic and esthetic surgery techniques are presented in Chapter 63 and the preprosthetic techniques in Chapter 65.

In addition, periodontal surgical techniques for the placement of dental implants are available. These involve not only the implant placement techniques but also a variety of surgical procedures to adapt the neighboring tissues, such as the sinus floor or the mandibular nerve canal, for subsequent placement of the implant (see Box 52-1). These methods are discussed in Chapters 71 and 72.

*Scaling and root planing are not included because these procedures are not used intentionally to act on the gingival tissue.

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BOX 52-1 Periodontal Surgery

Pocket Reduction Surgery

Resective (gingivectomy, apically displaced flap, and undisplaced flap with or without osseous resection)

Regenerative (flaps with grafts, membranes, etc)

Correction of Anatomic/Morphologic Defects

Plastic surgery techniques to widen attached gingiva (free gingival grafts, other techniques, etc)

Esthetic surgery (root coverage, recreation of gingival papillae)

Preprosthetic techniques (crown lengthening, ridge augmentation, and vestibular deepening)

Placement of dental implants, including techniques for site development for implants (guided bone regeneration, sinus grafts)

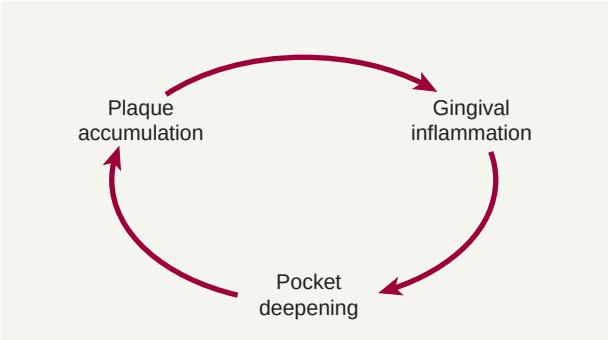

Figure 52-1 Accumulation of plaque leads to gingival inflammation and pocket deepening, which in turn increases the area of plaque accumulation.

Surgical Pocket Therapy

Surgical pocket therapy can be directed toward (1) access surgery to ensure the removal of irritants from the tooth surface or (2) elimination or reduction of the depth of the periodontal pocket.

The effectiveness of periodontal therapy is predicated on success in completely eliminating calculus, plaque, and diseased cementum from the tooth surface. Numerous investigations have shown that the difficulty of this task increases as the pocket becomes deeper.[2,5] The presence of irregularities on the root surface also increase the difficulty of the procedure. As the pocket becomes deeper, the surface to be scaled increases, more irregularities appear on the root surface, and accessibility is impaired.[11,15] The presence of furcations will also create insurmountable problems for scaling the root surface[4] (see Chapter 62).

These problems can be reduced by resecting or displacing the soft tissue wall of the pocket, thereby increasing the visibility and accessibility of the root surface.[3] The flap approach and the gingivectomy technique attain this result.

The need to eliminate or reduce the depth of the pocket is another important consideration. Pocket elimination consists of reducing the depth of periodontal pockets to that of a physiologic sulcus to enable cleansing by the patient. By proper case selection, both resective techniques and regenerative techniques can be used to accomplish this goal. The presence of a pocket produces areas that are impossible for the patient to keep clean, which establishes the vicious cycle depicted in Figure 52-1.

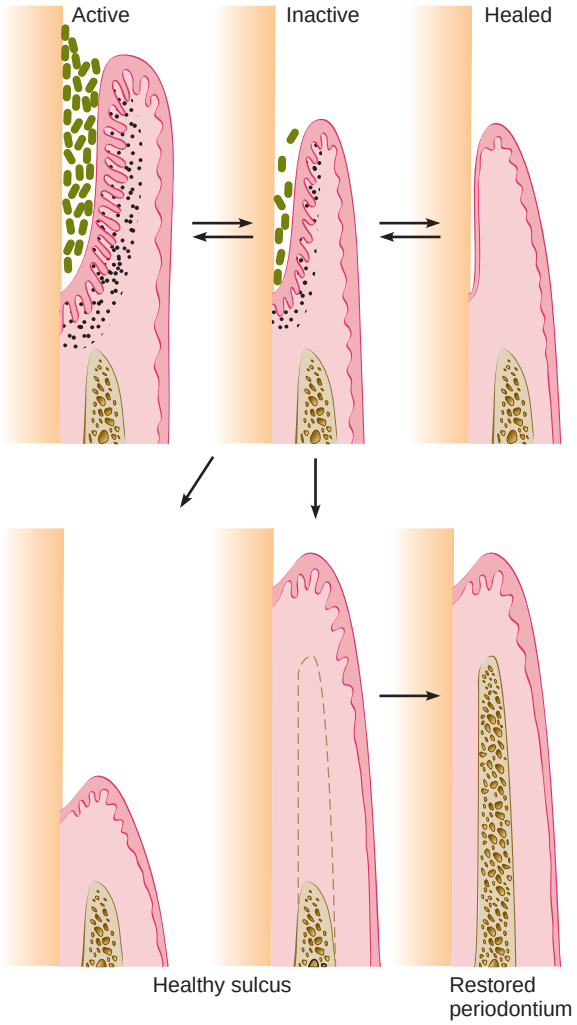

Figure 52-2 Possible results of pocket therapy. An active pocket can become inactive and heal by means of a long junctional epithelium. Surgical pocket therapy can result in a healthy sulcus, with or without gain of attachment. Improved gingival attachment promotes restoration of bone height, with re-formation of periodontal ligament fibers and layers of cementum.

Results of Pocket Therapy

A periodontal pocket can be in an active state or a period of inactivity or quiescence. In an active pocket, underlying bone is being lost (Figure 52-2, *top left*). It often can be diagnosed clinically by bleeding, either spontaneously or on probing. After Phase I therapy the inflammatory changes in the pocket wall subside, rendering the pocket inactive and reducing its depth (Figure 52-2, *top center*). The extent of this reduction depends on the depth before treatment and the degree to which the depth is the result of the edematous and inflammatory component of the pocket wall.

Whether the pocket remains inactive depends on the depth, the individual characteristics of the plaque components and the host response. Recurrence of the initial activity is likely.

Inactive pockets can sometimes heal with a long junctional epithelium (Figure 52-2, *top right*). However, this condition also may be unstable, and the chance of recurrence and re-formation of the original pocket is always present because the epithelial union to the tooth is weak. However, one study in monkeys has shown that the long junctional epithelial union may be as resistant to plaque infection as a normal connective tissue attachment.[9]

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Studies have shown that inactive pockets can be maintained for long periods with little loss of attachment by means of frequent scaling and root-planing procedures.[6,10,12] A more reliable and stable result is obtained, however, by transforming the pocket into a healthy sulcus. The bottom of the healthy sulcus can be located either where the bottom of the pocket was localized or coronal to it. In the first case, there is no gain of attachment (Figure 52-2, *bottom left*), and the area of the root that was previously the tooth wall of the pocket becomes exposed. This does not mean that the periodontal treatment has caused recession but rather that it has uncovered the recession previously induced by the disease.

The healthy sulcus can also be located coronal to the bottom of the preexisting pocket (Figure 52-2, *bottom center* and *right*). This is conducive to a restored marginal periodontium; the result is a sulcus of normal depth with gain of attachment. The creation of a healthy sulcus and a restored periodontium entails a total restoration of the status that existed before periodontal disease began, which is the ideal result of treatment.

POCKET ELIMINATION VERSUS POCKET MAINTENANCE

Pocket elimination (depth reduction to gingival sulcus levels) has traditionally been considered one of the main goals of periodontal therapy. It was considered vital because of the need to improve accessibility to root surfaces for the therapist during treatment and for the patient after healing. The prevalent opinion is the presence of deep pockets after therapy represents a greater risk of disease progression than shallow sites. Individual probing depths are not good predictors of future clinical attachment loss. The absence of deep pockets in treated patients, on the other hand, is an excellent predictor of a stable periodontium.[5]

Longitudinal studies of different therapeutic modalities over the last 30 years have given conflicting results,[7,16] probably because of inherent problems created by the "split-mouth" design. In general, after surgical therapy, pockets that rebound to a shallow or moderate depth can be maintained in a healthy state and without radiographic evidence of advancing bone loss by maintenance visits consisting of scaling and root planing, with oral hygiene reinforcement performed at regular intervals of 3 months or less. In these patients, the residual pocket can be examined with a thin periodontal probe, but no pain, exudate, or bleeding results. This appears to indicate that no plaque has formed on the subgingival root surfaces.

These findings do not alter the indications for periodontal surgery because the results are based on surgical exposure of the root surfaces for thorough elimination of irritants. However, these findings emphasize the importance of the maintenance phase and the close monitoring of both level of attachment and pocket depth, together with the other clinical variables (bleeding, exudation, or tooth mobility). *The transformation of the initial deep, active pocket into a shallower, inactive, maintainable pocket requires some form of definitive pocket therapy and constant supervision thereafter.*

Pocket depth is an extremely useful and widely employed clinical determination, but it must be evaluated together with level of attachment and the presence of bleeding, exudation, and pain. The most important variable for evaluating whether a pocket (or deep sulcus) is progressive is the *level of attachment,* which is measured in millimeters from the cementoenamel junction. The apical displacement of the level of attachment places the tooth in jeopardy, not the increase in pocket depth, which may be caused by coronal displacement of the gingival margin.

Pocket depth remains an important clinical variable that contributes to decisions about treatment selection. Lindhe et al[8] compared the effect of root planing alone and with a modified Widman flap on the resultant level of attachment and in relation to initial pocket depth. They reported that scaling and root-planing procedures induce loss of attachment if performed in pockets shallower than 2.9 mm, whereas gain of attachment occurs in deeper pockets. The modified Widman flap induces loss of attachment if done in pockets shallower than 4.2 mm but results in a greater gain of attachment than root planing in pockets deeper than 4.2 mm. The loss is a true loss of connective tissue attachment, whereas the gain can be considered a false gain because of reduced penetrability of connective tissues apical to the bottom of the pocket after treatment.[9,17]

Furthermore, probing depths established after active therapy and healing (approximately 6 months after treatment) can be maintained unchanged or reduced even further during a maintenance period involving careful prophylaxis once every 3 months.[8]

Ramfjord[12] and Rosling[13] and their colleagues showed that, regardless of the surgical technique used for pocket therapy, a certain pocket depth recurs. *Therefore maintenance of this depth without any further loss of attachment becomes the goal.*

REEVALUATION AFTER PHASE I THERAPY

Longitudinal studies have noted that all patients should be treated initially with scaling and root planing and that a final decision on the need for periodontal surgery should be made only after a thorough evaluation of the effects of Phase I therapy.[5] The assessment is generally made no less than 1 to 3 months and sometimes as much as 9 months after the completion of Phase I therapy.[1] This reevaluation of the periodontal condition should include reprobing the entire mouth. The presence of calculus, root caries, defective restorations, and signs of persistent inflammation should also be evaluated.

CRITICAL ZONES IN POCKET SURGERY

Criteria for the selection of one of the different surgical techniques for pocket therapy are based on clinical findings in the soft tissue pocket wall, tooth surface, underlying bone, and attached gingiva.

Zone 1: Soft Tissue Pocket Wall

The clinician should determine the morphologic features, thickness, and topography of the soft tissue pocket wall and persistence of inflammatory changes in the wall.

Zone 2: Tooth Surface

The clinician should identify the presence of deposits and alterations on the cementum surface and determine the accessibility of the root surface to instrumentation. Phase I therapy should have solved many, if not all, of the problems on the tooth surface. Evaluation of the results of Phase I therapy should determine the need for further therapy and the method to be used.

Zone 3: Underlying Bone

The clinician should establish the shape and height of the alveolar bone next to the pocket wall through careful probing and clinical and radiographic examinations. Bony craters, horizontal or angular bone losses, and other bone deformities are important criteria in selection of the treatment technique.

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Zone 4: Attached Gingiva

The clinician should consider the presence or absence of an adequate band of attached gingiva when selecting the pocket treatment method. Diagnostic techniques for mucogingival problems are described in Chapter 63. An inadequate attached gingiva may be caused by a high frenum attachment, marked gingival recession, or a deep pocket that reaches the level of the mucogingival junction. All these possible conditions should be explored and their influence on pocket therapy determined.

INDICATIONS FOR PERIODONTAL SURGERY

The following findings may indicate the need for a surgical phase of therapy:

1. Areas with irregular bony contours, deep craters, and other defects usually require surgical approach.
2. Pockets on teeth in which a complete removal of root irritants is not considered clinically possible may call for surgery. This occurs frequently in molar and premolar areas.
3. In cases of furcation involvement of grade II or III, a surgical approach ensures the removal of irritants; any necessary root resection or hemisection also requires surgical intervention.
4. Intrabony pockets on distal areas of last molars, frequently complicated by mucogingival problems, are usually unresponsive to nonsurgical methods.
5. Persistent inflammation in areas with moderate to deep pockets may require a surgical approach. In areas with shallow pockets or normal sulci, persistent inflammation may point to the presence of a mucogingival problem that needs a surgical solution.

METHODS OF POCKET THERAPY

The methods for pocket therapy can be classified under the following three main headings:

1. **New attachment techniques** offer the ideal result because they eliminate pocket depth by reuniting the gingiva to the tooth at a position coronal to the bottom of the preexisting pocket. New attachment is usually associated with filling in of bone and regeneration of periodontal ligament and cementum.
2. **Removal of the pocket wall** is the most common method. The wall of the pocket consists of soft tissue and may also include bone in the case of intrabony pockets. It can be removed by the following:
   - *Retraction or shrinkage,* in which scaling and root-planing procedures resolve the inflammatory process, and the gingiva therefore shrinks, reducing the pocket depth.
   - *Surgical removal* performed by the gingivectomy technique or by means of an undisplaced flap.
   - *Apical displacement* with an apically displaced flap.
3. **Removal of the tooth side of the pocket,** which is accomplished by tooth extraction or by partial tooth extraction (hemisection or root resection).

The techniques, what they accomplish, and the factors governing their selection are presented in Chapters 59 to 62.

Criteria for Method Selection

Scientific criteria to establish the indications for each technique are difficult to determine. Longitudinal studies following a significant number of cases over a number of years, standardizing multiple factors and many variables, would be needed. Clinical experience, however, has suggested the criteria for selecting the method to treat the pocket. The selection of a technique for treatment of a particular periodontal lesion is based on the following considerations:

1. Characteristics of the pocket: depth, relation to bone, and configuration.
2. Accessibility to instrumentation, including presence of furcation involvements.
3. Existence of mucogingival problems.
4. Response to Phase I therapy.
5. Patient cooperation, including ability to perform effective oral hygiene. Smokers must be willing to stop their habit.
6. Age and general health of the patient.
7. Overall diagnosis of the case: various types of gingival enlargement and types of periodontitis (e.g., chronic marginal periodontitis, localized aggressive periodontitis, generalized aggressive periodontitis).
8. Esthetic considerations.
9. Previous periodontal treatments.

Each of these variables are analyzed in relation to the pocket therapy techniques available, and a specific technique is selected. Of the many techniques, the one that would most successfully solve the problems with the fewest undesirable effects should be chosen. *Clinicians who adhere to one technique to solve all problems do not use to the advantage of the patient the wide repertoire of techniques at their disposal.*

Approaches to Specific Pocket Problems

Therapy for Gingival Pockets. Two factors are taken into consideration: (1) the character of the pocket wall and (2) the accessibility of the pocket. The pocket wall can be either edematous or fibrotic. *Edematous* tissue shrinks after the elimination of local factors, thereby reducing or totally eliminating pocket depth. Therefore scaling and root planing are the technique of choice in these cases.

Pockets with a *fibrotic* wall are not appreciably reduced in depth after scaling and root planing; therefore they are eliminated surgically. Until recently, gingivectomy was the only technique available; it solves the problem successfully, but in cases of marked gingival enlargement (e.g., severe phenytoin enlargement), it may leave a large wound that goes through a painful and prolonged healing process. In these patients, a modified flap technique can adequately solve the problem with fewer postoperative problems (see Chapter 58).

Therapy for Slight Periodontitis. In slight or incipient periodontitis, a small amount of bone loss has occurred, and pockets are shallow to moderate. In these patients, the conservative approach with good oral hygiene will generally suffice to control the disease. Incipient periodontitis that recurs in previously treated sites may require a thorough analysis of the causes for the recurrence. Occasionally, a surgical approach may be required to correct the problem.

Therapy for Moderate-to-Severe Periodontitis in Anterior Sector. The anterior teeth are important esthetically; therefore the technique that causes the least amount of visual root exposure should be considered. However, the importance of esthetics may be different for different patients, and nonelimination of the pocket may place the tooth in jeopardy. The final decision may have to be a compromise between health and esthetics, not attaining ideal results in either respect.

Anterior teeth offer two main advantages to a conservative approach: (1) they are all single rooted and easily accessible and (2)

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Phase II therapy is used to surgically treat residual periodontal pockets and bone defects remaining after Phase I therapy. All patients treated surgically need to have a preoperative history of adequate plaque control with 20% or more of the tooth surfaces free of plaque after oral hygiene procedures.

Most types of surgical therapy will result in some postsurgical gingival recession and so they are not useful in the anterior segments of the mouth in those patients in whom the esthetic shape of the gingiva is visible. These patients may be best treated with nonsurgical procedures, but new, innovative regeneration surgical techniques using modified papilla preservation approaches, together with minimally invasive procedures, often coupled with a basis of microsurgery, are showing promising results in maintaining and even improving papillary and labial tissue levels.

patient compliance and thoroughness in plaque control are easier to attain. *Therefore scaling and root planing are the technique of choice for the anterior teeth.*

Sometimes, however, a surgical technique may be necessary because of the need for improved accessibility for root planing or regenerative surgery of osseous defects. The papilla preservation flap can be used for both purposes and also offers a better postoperative result, with less recession and reduced soft tissue crater formation interproximally.[14] *The papilla preservation flap is the first choice when a surgical approach is needed.*

When the teeth are too close interproximally, the papilla preservation technique may not be feasible, and a technique that splits the papilla must be used. The *sulcular incision flap* offers good esthetic results and is the next choice.

When esthetics are not the primary consideration, the *modified Widman flap* can be chosen. This technique uses an internal bevel incision about 1 to 2 mm from the gingival margin without thinning the flap and may result in some minor recession.

Infrequently, bone contouring may be needed despite the resultant root exposure. The technique of choice is the *apically displaced flap with bone contouring*.

Therapy for Moderate-to-Severe Periodontitis in Posterior Area. Treatment for premolars and molars usually poses no esthetic problem but frequently involves difficult accessibility. Bone defects occur more often in the posterior than the anterior sector, and root morphologic features, particularly in relation to furcations, may offer insurmountable problems for instrumentation in a close field. Therefore surgery is frequently indicated in the posterior region.

The purpose of surgery in the posterior area is either enhanced accessibility or the need for definitive pocket reduction requiring osseous surgery. Accessibility can be obtained by either the undisplaced or the apically displaced flap.

Most patients with moderate to severe periodontitis have developed osseous defects that require some degree of osseous remodeling or reconstructive procedures. When osseous defects amenable to reconstruction are present, the *papilla preservation flap is the technique of choice* because it better protects the interproximal areas where defects are frequently present. *Second and third choices are the sulcular flap and the modified Widman flap,* maintaining as much of the papilla as possible.

When osseous defects with no possibility of reconstruction are present, such as interdental craters, the technique of choice is the flap with osseous contouring.

Surgical Techniques for Correction of Morphologic Defects. The objectives and rationale for the techniques performed to correct morphologic defects (mucogingival, esthetic, and preprosthetic) are given in Chapter 63.

Surgical Techniques for Implant Placement and Related Problems. The objectives and rationale for these techniques are described in Chapter 71.

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CHAPTER 53

Surgical Anatomy of Periodontal and Implant Areas

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CHAPTER OUTLINE

MANDIBLE
MAXILLA
EXOSTOSES
MUSCLES
ANATOMIC SPACES

A sound knowledge of the anatomy of the periodontium and the surrounding hard and soft structures is essential to determine the scope and possibilities of periodontal and implant surgical procedures and to minimize their risks. The spacial relationship of bones, muscles, blood vessels, and nerves, as well as the anatomic spaces located in the vicinity of the periodontal/implant surgical field, are particularly important. Only those features of periodontal and implant surgery relevance are mentioned in this chapter; the reader is referred to books on oral anatomy for a more comprehensive description of these structures.[4,6]

MANDIBLE

The mandible is a horseshoe-shaped bone connected to the skull by the temporomandibular joints. It presents several landmarks of great surgical importance for both periodontal and implant surgical procedures.

The *mandibular canal,* occupied by the inferior alveolar nerve and vessels, begins at the mandibular foramen on the medial surface of the mandibular ramus and curves downward and forward, becoming horizontal below the apices of the molars (Figure 53-1). The distance from the canal to the apices of the molars is shorter in the third molar area and increases as it goes forward. A small percentage (1%) of mandibular canals bifurcate in the body of the mandible, resulting in two canals and two mental foramen.[5,10] In the premolar area, the mandibular canal divides in two branches, with one exiting the mandible and the other continuing anteriorly: the *incisive canal,* which continues horizontally to the midline, and the *mental canal,* which turns upward and opens in the mental foramen.

The *mental foramen,* from which the mental nerve and vessels emerge, is located on the buccal surface of the mandible below the apices of the premolars, sometimes closer to the second premolar and usually halfway between the lower border of the mandible and the alveolar margin (Figure 53-2). It is often but not always visible on conventional radiographs. The opening of the mental foramen, which may be oval or round in shape, typically faces upward and distally, with its posterosuperior border slanting gradually to the bone surface. An "anterior loop" of the mental foramen has been described, using cadaver dissection, as a reverse turn and looping back of the mental nerve before its exit out of the mental foramen with lengths ranging from 0.5 to 5.0 mm.[10] More recent evaluation of the anterior loop of the mental nerve using cone beam scans and cadaveric dissection reported this loop extension to range from 0.0 to 9.0 mm.[22,23] The anterior loop of the mental nerve has a high prevalence (88%), symmetric occurrence, and a mean length of 4.13 ± 1.08 mm.[18] As it emerges, the *mental nerve* divides into three branches. One branch of the nerve turns forward and downward to supply the skin of the chin. The other two branches course anteriorly and upward to supply the skin and mucous membrane of the lower lip and the mucosa of the labial alveolar surface.

Surgical trauma (e.g., pressure, manipulation, and postsurgical swelling) to the mental nerve can produce paresthesia of the lip, which recovers slowly. Partial or complete cutting of the nerve can result in permanent paresthesia/dysesthesia. Familiarity with the location and appearance of the mental nerve reduces the likelihood of injury (Figure 53-3).

In partially or totally edentulous jaws, the disappearance of the alveolar portion of the mandible brings the mandibular canal and mental foramen closer to the superior border (Figures 53-4 and 53-5). When these patients are evaluated for placement of implants, the distance between the canal and the superior surface of the bone, as well as the location of the mental foramen, must be carefully determined to avoid surgical injury to the nerve (see Chapter 70).

The anterior extension of the inferior alveolar nerve or incisive nerve has been measured using conventional radiographs, computed tomography (CT) scans, cadaver dissections and cone beam scans.[3,10,22,23] This nerve, which is less evident on conventional

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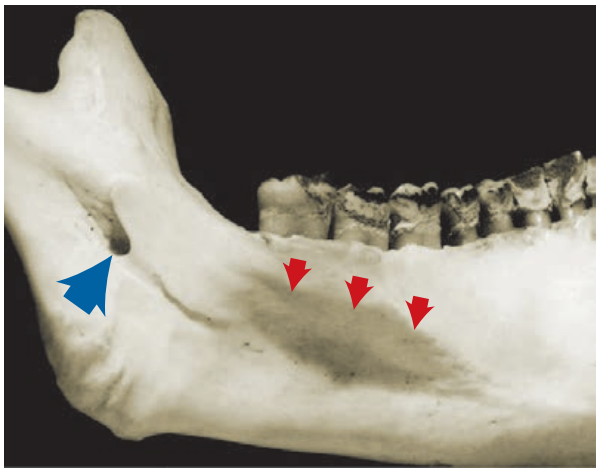

Figure 53-1 Mandible, lingual surface view. Note the lingual, or mandibular, foramen *(blue arrow),* where the inferior alveolar nerve enters the mandibular canal, and the mylohyoid ridge *(red arrows).*

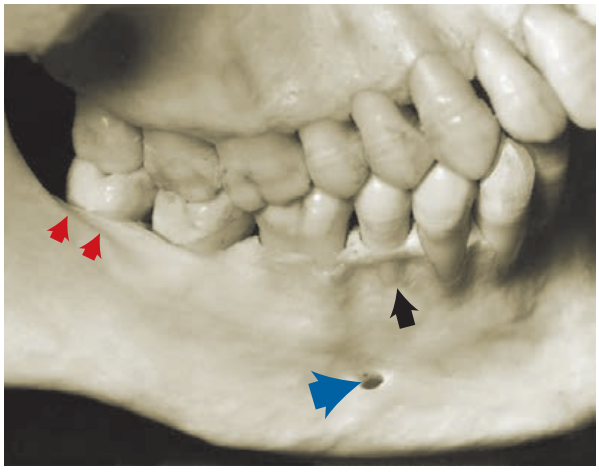

Figure 53-2 Mandible, facial surface view. Note the location of the mental foramen *(blue arrow),* slightly distal and apical to the apex of the second premolar, and the shelflike area in the region of the molars *(red arrows),* created by the external oblique ridge. Note also the fenestration present in the second premolar *(black arrow).*

radiographs and often unnoticed, extends beyond the anterior loop of the mental foramen in a horizontal direction toward the midline. The length of the incisive canal has been reported up to 21.45 mm from the mesial aspect of the mental foramen terminating just 4 mm from the midline.[3]

The *lingual nerve,* along with the inferior alveolar nerve, is a branch of the posterior division of the mandibular nerve and descends along the mandibular ramus medial to and in front of the inferior alveolar nerve. The lingual nerve lies close to the surface of the oral mucosa in the third molar area and goes deeper as it travels forward (Figure 53-6). It can be damaged during anesthetic injections and during oral surgery procedures such as third molar extractions.[14] Less often, the lingual nerve may be injured when a periodontal partial-thickness flap is raised in the third molar region or when releasing incisions are made in the area.

The *alveolar process,* which provides the supporting bone to the teeth, has a narrower distal curvature than the body of the mandible (Figure 53-7), creating a flat surface in the posterior area between the teeth and the anterior border of the ramus. This results in the formation of the *external oblique ridge,* which runs downward and forward to the region of the second or first molar (Figure 53-8), creating a shelflike bony area. Resective osseous therapy may be difficult or impossible in this area because of the amount of bone that must be removed distally toward the ramus to achieve resection of a periodontal osseous defect on the distal aspect of the mandibular second/third molar.

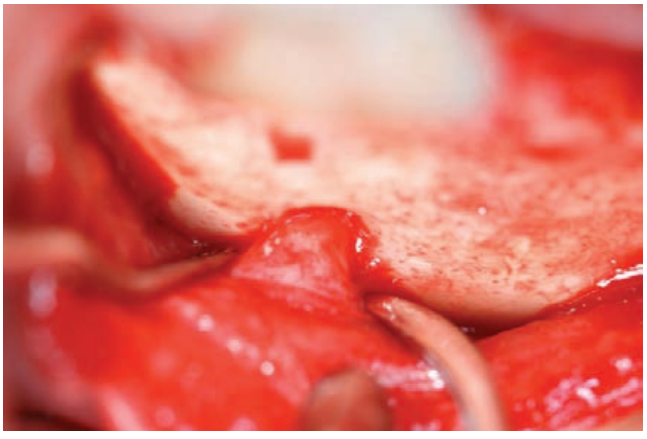

Figure 53-3 Mental nerve emerging from the foramen in the premolar area.

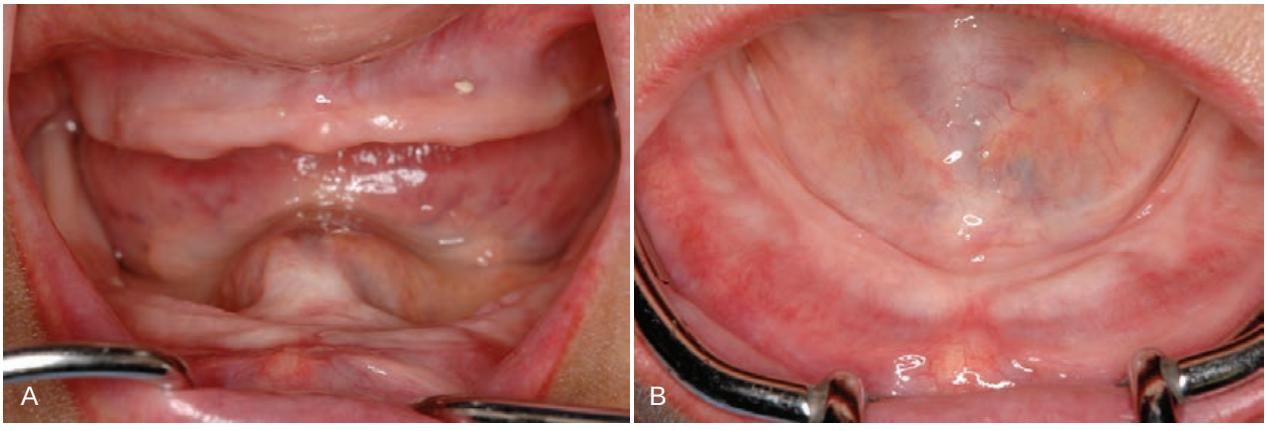

Figure 53-4 Loss of alveolar ridge in edentulous patient brings mental foramen and inferior alveolar nerve canal closer to the surface, which may lead to discomfort for the patient. **A,** Anterior view demonstrating severe loss of vertical alveolar ridge height. **B,** Occlusal view of same patient demonstrating loss of vestibular depth with alveolar bone loss.

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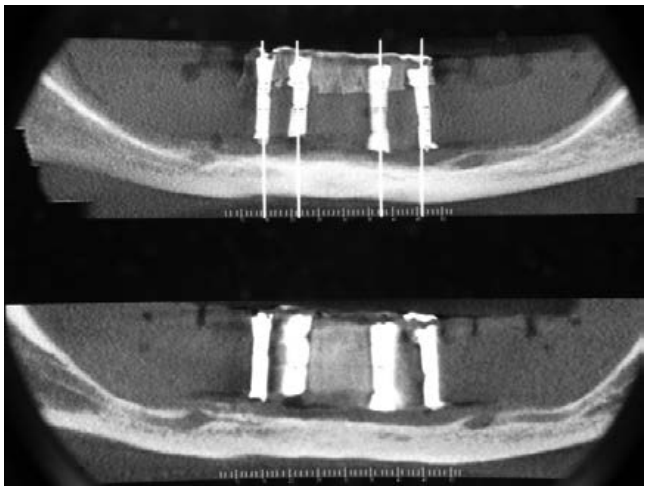

Figure 53-5 Panoramic radiograph of edentulous patient with loss of alveolar bone height resulting in the mental foramen exiting the jaw at the superior aspect of the remaining bone (ridge crest), which is the denture-bearing surface. Pressure from the complete removable denture over this area causes pain.

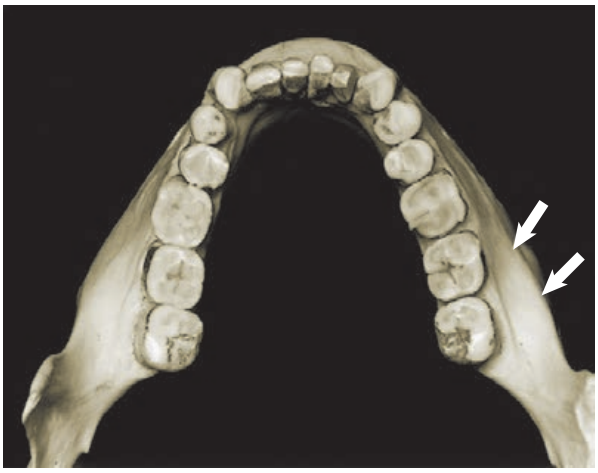

Figure 53-7 Occlusal view of mandible. Note the shelf created in the facial molar areas by the external oblique ridge. *Arrows* show the attachment of the buccinator muscle.

Figure 53-6 Lingual view of mandible showing the pathway of the lingual nerve *(red),* which goes near the gingiva in the third molar area and then continues forward, going deeper and medially.

Figure 53-8 Mandible, occlusal view of ramus and molars. Note the retromolar triangle area distal to the third molar *(arrows).*

Distal to the third molar, the external oblique ridge circumscribes the *retromolar triangle* (see Figure 53-8). This region is occupied by glandular and adipose tissue and covered by unattached, nonkeratinized mucosa. If sufficient space exists distal to the last molar, a band of attached gingiva may be present; only in such a case can a distal flap procedure be performed effectively.

The medial side of the body of the mandible is traversed obliquely by the *mylohyoid ridge,* which starts close to the alveolar margin in the third molar area and continues anteriorly in an apical direction, increasing its distance from the osseous margin as it travels forward (Figure 53-9). The *mylohyoid muscle* inserts along this ridge and separates the *sublingual space,* located above or more anteriorly and superiorly, from the submandibular space, located below or more posteriorly and inferiorly.

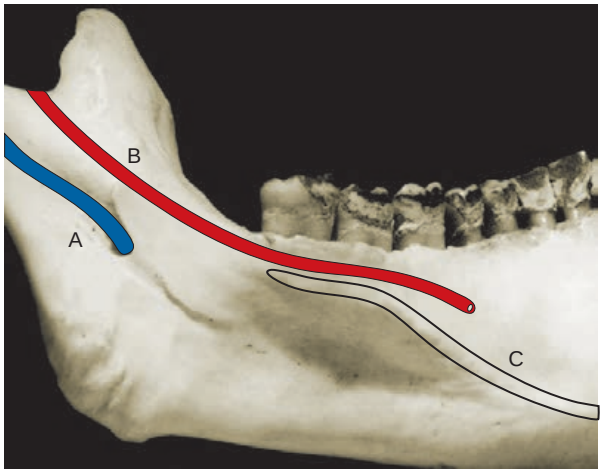

Figure 53-9 Lingual view of mandible showing the inferior alveolar nerve entering the mandibular canal **(A),** the lingual nerve traversing near the lingual surface of the third molar **(B),** and inferiorly, the attachment of the mylohyoid muscle **(C).**

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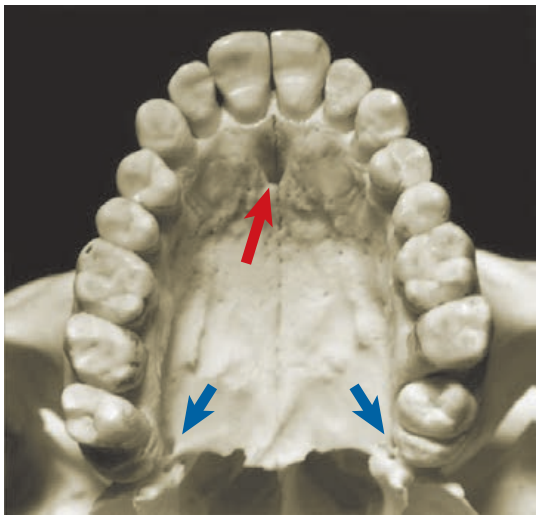

Figure 53-10 Occlusal view of maxilla and palatine bone. Note the opening of the incisive canal or anterior palatine foramen *(red arrow)* and the greater palatine foramen *(blue arrows).*

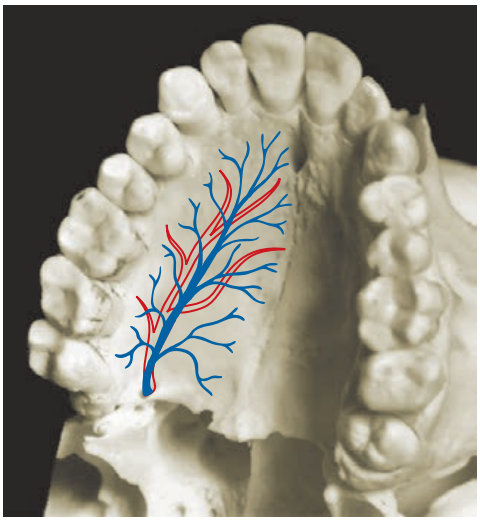

Figure 53-11 Occlusolateral view of palate showing nerves *(red)* and vessels *(blue)* emerging from the greater palatine foramen and continuing anteriorly on the palate.

MAXILLA

The maxilla is a paired bone that is hollowed out by the maxillary sinus and nasal cavity. The maxilla has the following four processes:

- The *alveolar process* contains the sockets for and supports the maxillary teeth.
- The *palatine process* extends horizontally from the alveolar process to meet its counterpart from the opposite maxilla at the midline intermaxillary suture, and it extends posteriorly with the horizontal plate of the palatine bone to form the hard palate.
- The *zygomatic process* extends laterally from the area above the first molar and determines the depth of the vestibular fornix on the lateral aspect of the maxilla.
- The *frontal process* extends in an ascending direction and articulates with the frontal bone at the frontomaxillary suture.

The terminal branches of the nasopalatine nerve and vessels pass through the incisive canal, which opens in the midline anterior area of the palate (Figure 53-10). The mucosa overlying the incisive canal presents a slight protuberance called the *incisive papilla.* Vessels emerging through the incisive canal are of small caliber, and their surgical interference is of little consequence.

The *greater palatine foramen* opens 3 to 4 mm anterior to the posterior border of the hard palate (Figure 53-11). The greater palatine nerve and vessels emerge through this foramen and run anteriorly in the submucosa of the palate, between the palatal and alveolar processes (Figure 53-12). Palatal flaps and donor sites for gingival grafts should be carefully performed and selected to avoid invading these areas because profuse hemorrhage may ensue, particularly if vessels are damaged at the palatine foramen. Vertical incisions in the molar region should be avoided.

The mucous membrane covering the hard palate is firmly attached to the underlying bone. The submucous layer of the palate posterior to the first molars contains the *palatal glands,* which are more compact in the soft palate and extend anteriorly, filling the gap between the mucosal connective tissue and the periosteum and protecting the underlying vessels and nerve (see Figure 53-22).

Figure 53-12 Histologic frontal section of human palate at the level of the first molar, showing the location of vessels and nerve, surrounded by adipose and glandular tissue.

The area distal to the last molar, called the *maxillary tuberosity,* consists of the posterior-inferior angle of the infratemporal surface of the maxilla. Medially it articulates with the pyramidal process of the palatine bone. It is covered by dense, fibrous connective tissue and contains the terminal branches of the middle and posterior palatine nerves. Excision of the area for distal flap surgery may reach medially to the tensor palati muscle. The tensor palati muscle comes from the greater wing of the sphenoid bone and ends in a tendon that forms the palatine aponeurosis, which expands, fanlike, to attach to the posterior border of the hard palate.

The body of the maxilla is occupied by the *maxillary sinus,* which is the largest of the paranasal sinuses. It is an air-filled cavity located

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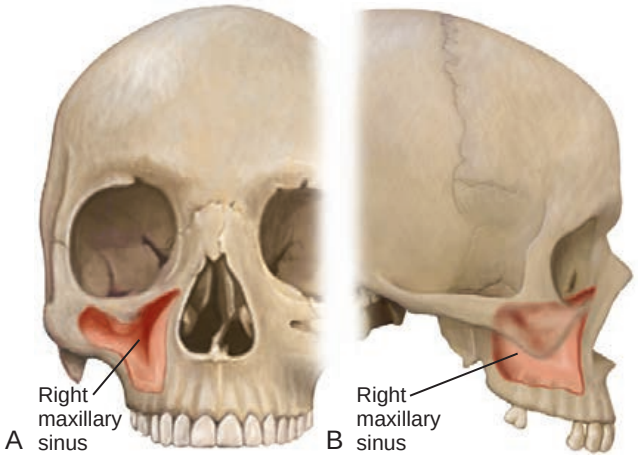

Figure 53-13 Location and anatomy of the maxillary sinus. **A,** Frontal view. **B,** Lateral view.

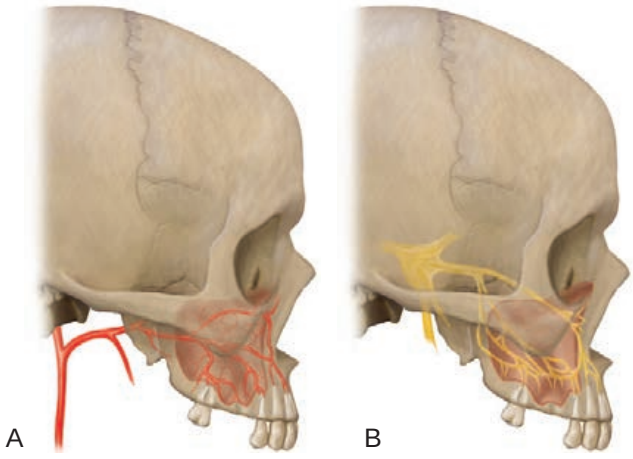

Figure 53-14 Blood supply and innervation of the maxillary sinus. **A,** Arterial blood supply. **B,** Innervation of the maxillary sinus.

in the posterior maxilla superior to the teeth. The lateral wall of the nasal cavity borders the sinus medially; it is bordered superiorly by the floor of the orbit and laterally by the lateral wall of the maxilla, the alveolar process, and the zygomatic arch (Figure 53-13). It is pyramidal in shape, with its apex in the zygomatic arch and its base at the lateral wall of the nasal cavity. The size of the maxillary sinus varies from one individual to another (depending on the individual and age), from very small and narrow to quite large and expansive.

The maxillary sinus is frequently subdivided (incompletely) into recesses by one or more septa. Maxillary sinus septa vary in size and location. Clinical and radiographic examinations suggest that septa are frequently present (up to 39% of sinuses).[8,13,25] CT scans are the preferred method for detecting septa because panoramic radiographs are not reliable (26.5% false diagnosis of the presence or absence of septa).[13,15] Septa are found in the anterior (24%), middle (41%), and posterior (35%) aspects of the maxillary sinus, with the most common location between the second premolar and the first molar.[12,25] The height of septa vary as well, ranging from 0 to 20.6 mm.[25] Only 0.5% of septa form complete separation of sinus spaces into separate chambers.[16]

The entire maxillary sinus is lined with a thin mucosal membrane called the *schneiderian membrane*. The specialized structure of the respiratory mucous membrane, with its motile cilia and rich blood supply, is well adapted to purifying, moistening, and warming air to protect the lungs. The entrance to the maxillary sinus, through the orifice or maxillary duct, is located at the superior medial aspect of the cavity. The orifice is relatively small, measuring only 3 to 6 mm in length and diameter. An accessory opening is occasionally found inferior and posterior to the main opening. The maxillary sinus drains into the middle meatus of the nasal cavity through the maxillary duct, which passes secretions medially to the semilunar hiatus. Normal amounts of secretion are moved from the sinus by the spiral pattern of beating cilia surrounding the orifice. If the maxillary sinus becomes infected or chronically inflamed, swelling of the mucosa around the orifice(s) impairs drainage. The floor of the maxillary sinus extends down below the level of the nasal cavity into the alveolar process.

The roots of the maxillary first and second molars are often close to the floor of the sinus. Less frequently, the roots of the premolars and third molars may protrude into the floor of the sinus. With increasing age the maxillary sinus expands, becoming more and more pneumatized down around the roots of the maxillary teeth, sometimes resulting in exposure of the roots through the bony floor into the sinus, with only the thin mucosal membrane covering the root surface. The ability to perform periodontal osseous surgery in the posterior maxilla may be limited when sinuses are severely pneumatized.

Blood supply to the maxillary sinus arises from the superior alveolar (anterior, middle, and posterior) branches of the maxillary artery (Figure 53-14, *A*).[24] The maxillary artery, a large terminal branch of the external carotid artery, gives off many branches to supply the maxillary sinus, including the infraorbital artery, which travels superiorly and anteriorly and gives off the anterior superior alveolar artery.[9] Branches of the greater palatine artery contribute to a lesser extent. Venous blood drains via the pterygoid plexus. Much of the vasculature travels through channels in the bony walls of the maxillary sinus, with many branches anastomosing with the highly vascularized schneiderian membrane. Innervation of the maxillary sinus is supplied by the superior alveolar (anterior, middle, and posterior) nerves, branches of the maxillary nerve (Figure 53-14, *B*).

Knowledge of the arterial blood supply is particularly important when considering a lateral window approach to sinus floor elevation and bone augmentation. Solar et al found an intraosseous branch of the posterior superior alveolar artery anastomosing with the infraorbital artery in 100% of their human cadaver specimens (134 sinuses, all males).[19] On average, the vessel was located 18.9 mm from the alveolar crest. Using 50 CT scans from 625 patients (both males and females) undergoing sinus bone augmentation, Elian et al, found that the vessel was radiographically evident in 52.9% of sinuses.[7] The vessel was located an average of 16.4 mm from the alveolar crest, which is consistent with the previous study. Human cadaver dissection and CT scan evaluations of the vessels running through the lateral wall of the maxillary sinus reveal that intraosseous vessels are present in the lower two thirds of the anterolateral wall in approximately 10.5% of cases (Figure 53-15).[9] In 57.1% of cases (about 6% of all sinuses), the vessel diameter ranged in size from 1 to 2.5 mm. The location of the artery in relation to the position of the lateral window for sinus augmentation presents a risk for bleeding complications in 10-20% of cases.[7,9]

The inferior wall of the maxillary sinus is frequently separated from the apices and roots of the maxillary posterior teeth by a thin, bony plate (Figure 53-16). In edentulous posterior areas the maxillary sinus bony wall may be only a thin plate in intimate contact

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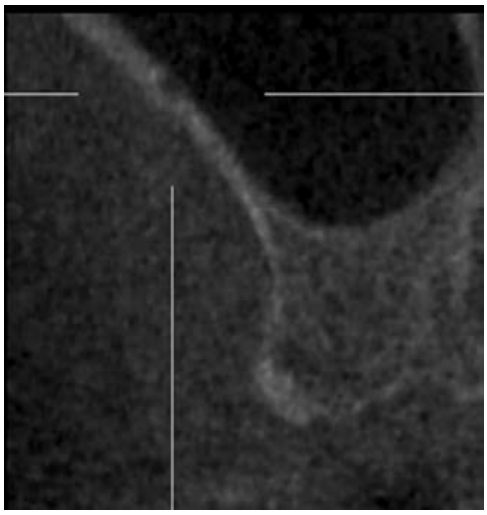

Figure 53-15 Cross-sectional image from a cone-beam computed tomography (CBCT) scan of the maxillary sinus demonstrating the presence of an intraosseous vessel in the lateral wall within the dimensions that a lateral window osteotomy would be created.

with the alveolar mucosa (Figure 53-17). Adequate determination of the extension of the maxillary sinus into the surgical site is important to avoid creating an oroantral communication, particularly in relation to osseous reduction in periodontal surgery or the placement of implants in edentulous areas. Determining the amount of available bone in the anterior area, below the floor of the nasal cavity, is also critical for the placement of implants (see Chapter 70).

EXOSTOSES

Both the maxilla and the mandible may have exostoses or tori, which are considered to be within the normal range of anatomic variation. Sometimes these structures may hinder the removal of plaque by the patient and may have to be removed to improve the prognosis of neighboring teeth. Additional indications for the removal of exostoses include the inability to comfortably wear removable prostheses over these areas. The most common location of a mandibular torus is in the lingual area of canine and premolars, above the mylohyoid muscle (Figure 53-18). Mandibular tori can also be found on the buccal/labial surfaces of the mandibular teeth.

Figure 53-16 Radiograph of maxillary molars and premolars, with the maxillary sinus apparently near the apices.

Figure 53-17 Radiograph of edentulous molar maxillary area demonstrating severe pneumatization of the maxillary sinus. There is only a thin layer of cortical bone separating the sinus from the oral cavity.

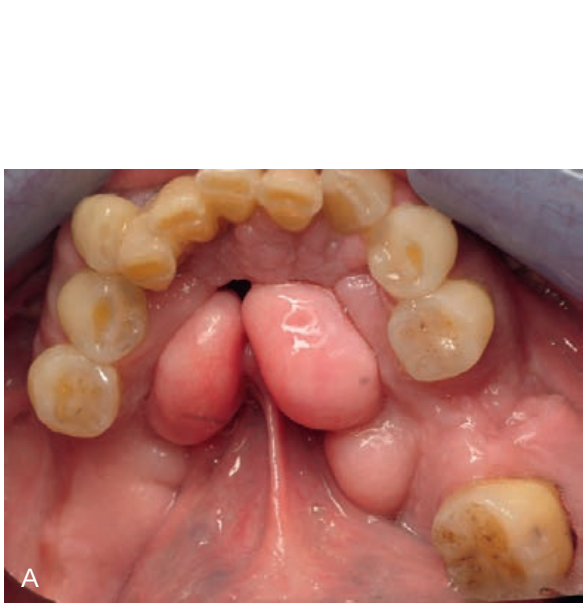

Figure 53-18 **A,** Clinical photograph of large mandibular tori on the lingual aspect of both right and left mandible. **B,** Same patient. Cross-sectional image of mandibular torus in the premolar area.

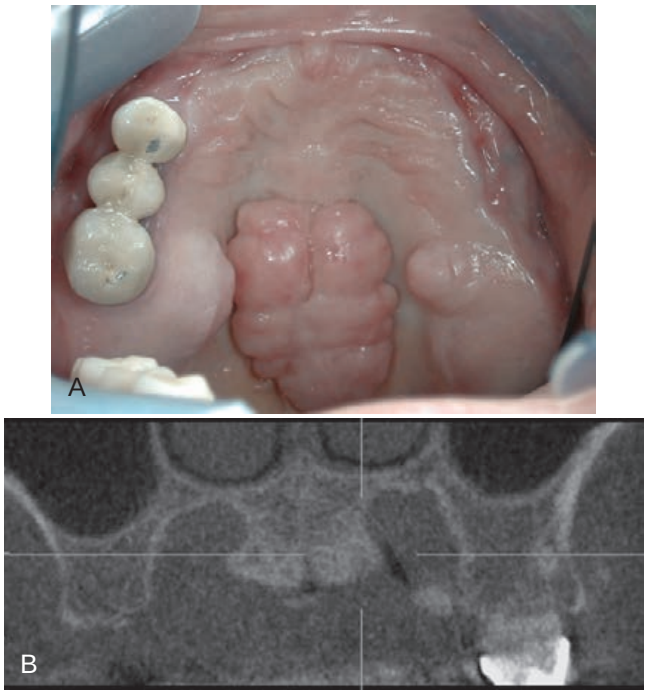

Figure 53-19 **A,** Clinical photograph of large palatal torus, located in the midline of the palate. Notice the large tori on the palatal aspects of the maxillary alveolar ridge as well. **B,** Same patient. Cross-sectional image of maxillary midline torus. Notice torus located on the palatal aspect of the alveolar ridge as well.

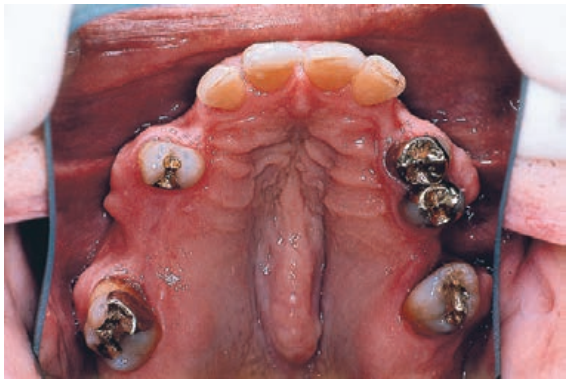

Figure 53-20 Clinical photograph of large buccal exostosis in the maxillary arch. There is also a large midline palatal torus.

Maxillary tori are usually located in the midline of the hard palate (Figure 53-19). Smaller tori may be seen over the palatal roots of the maxillary molars and in the area above the greater palatine foramen (see Figure 53-19) or on the buccal/labial surfaces of the maxillary teeth (Figure 53-20).

MUSCLES

Several muscles may be encountered when performing periodontal and implant flap surgery, particularly in mucogingival surgery and bone augmentation procedures. These are the mentalis, incisivus labii inferioris, depressor labii inferioris, depressor anguli oris (triangularis), incisivus labii superioris, and buccinator muscles. Their bony attachments are shown in Figure 53-21. These muscles provide mobility to the lips and cheeks.

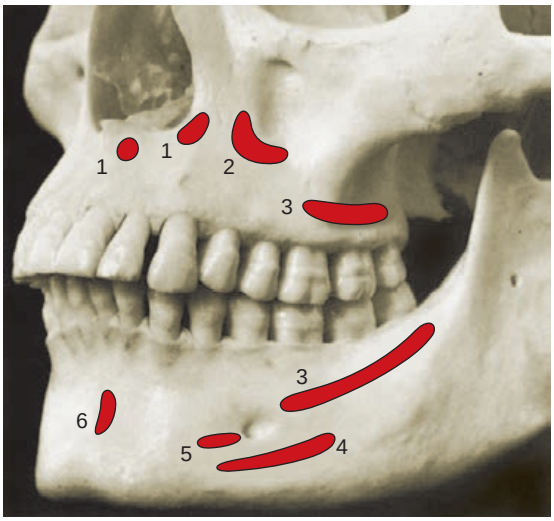

Figure 53-21 Muscle attachments that may be encountered in mucogingival surgery. *1,* Nasalis; *2,* levator anguli oris; *3,* buccinator; *4,* depressor anguli oris; *5,* depressor labii inferioris; *6,* mentalis.

ANATOMIC SPACES

Several anatomic spaces or *compartments* are found close to the operative field of periodontal and implant surgery sites. These spaces contain loose connective tissue but can be easily distended by hemorrhage, inflammatory fluid, and infection.

Surgical invasion of these areas may result in dangerous hemorrhage (intraoperative) or infections (postoperative) and should be carefully avoided. Some of these spaces are briefly described here. For further information, the reader is referred to other sources.[2,11,20,21]

The *canine fossa* contains varying amounts of connective tissue and fat and is bounded superiorly by the quadratus labii superioris muscle, anteriorly by the orbicularis oris, and posteriorly by the buccinator. Infection of this area results in swelling of the upper lip, obliterating the nasolabial fold, and of the upper and lower eyelids, closing the eye.

The *buccal space* is located between the buccinator and the masseter muscles. Infection of this area results in swelling of the cheek but may extend to the temporal space or the submandibular space, with which the buccal space communicates.

The *mental,* or *mentalis, space* is located in the region of the mental symphysis, where the mental muscle, depressor muscle of the lower lip, and depressor muscle of the corner of the mouth are attached. Infection of this area results in large swelling of the chin, extending downward.

The *masticator space* contains the masseter muscle, pterygoid muscles, tendon of insertion of the temporalis muscle, and mandibular ramus and posterior part of the body of the mandible. Infection of this area results in swelling of the face and severe trismus and pain. If the abscess occupies the deepest part of this compartment, facial swelling may not be obvious, but the patient may complain of pain and trismus. Patients may also have difficulty and discomfort when moving the tongue and swallowing.

The *sublingual space* is located below the oral mucosa in the anterior part of the floor of the mouth. It contains the sublingual gland and its excretory duct, the submandibular or Wharton's duct, and is traversed by the lingual nerve and vessels and hypoglossal nerve (Figure 53-22). Its boundaries are the geniohyoid and genioglossus muscles medially and the lingual surface of the mandible and below by the mylohyoid muscle laterally and anteriorly (Figure

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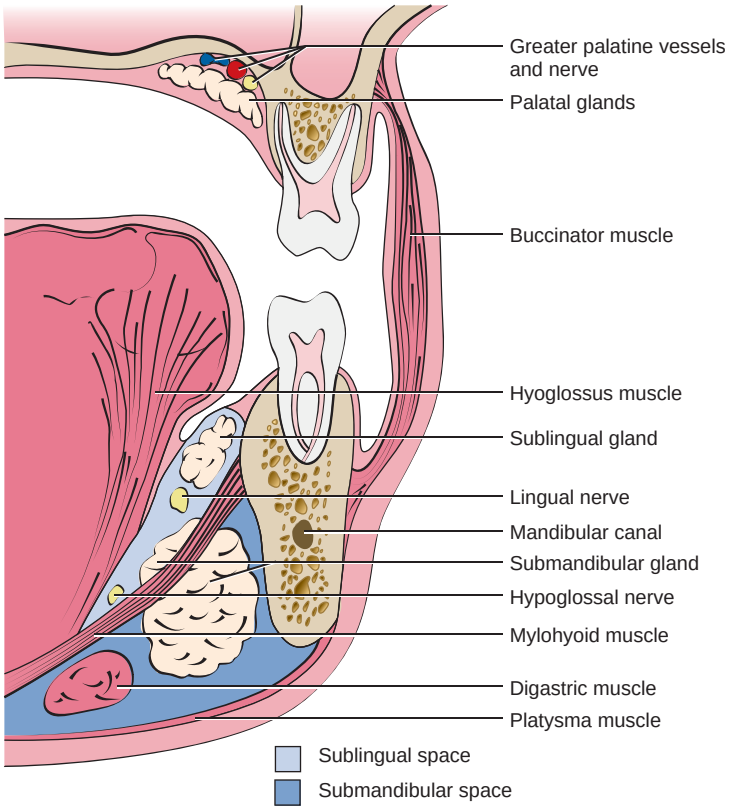

Figure 53-22 Diagram of a frontal section of the human head at the level of the first molars, depicting the most important structures in relation to periodontal surgery. Note the location of the sublingual space, submandibular space, and greater palatine nerve and vessels.

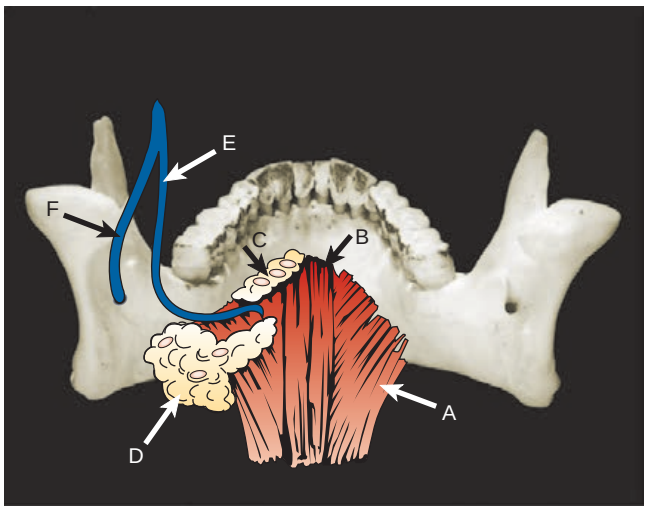

Figure 53-23 Posterior view of mandible, showing the attachment of the mylohyoid muscles **(A);** geniohyoid muscles **(B);** sublingual gland **(C);** submandibular gland **(D),** which extends below and also to some extent above the mylohyoid muscle; and sublingual **(E)** and inferior alveolar **(F)** nerves.

53-23). Infection of this area raises the floor of the mouth and displaces the tongue, resulting in pain and difficulty in swallowing but little facial swelling.

The *submental space* is found between the mylohyoid muscle superiorly and the platysma inferiorly. It is bounded laterally by the mandible and posteriorly by the hyoid bone. It is traversed by the anterior belly of the digastric muscle. Infections of this area arise from the region of the mandibular anterior teeth and result in swelling of the submental region; infections become more dangerous as they proceed posteriorly.

The *submandibular space* is found external to the sublingual space, below the mylohyoid and hyoglossus muscles (see Figures 53-22 and 53-23). This space contains the submandibular gland, which extends partially above the mylohyoid muscle, thus communicating with the sublingual space, and numerous lymph nodes. Infections of this area originate in the molar or premolar area and result in swelling that obliterates the submandibular line and in pain when swallowing. Ludwig's angina is a severe form of infection of the submandibular space that may extend to the sublingual and submental spaces. It results in hardening of the floor of the mouth and may lead to asphyxiation from edema of the neck and glottis. Although the bacteriology of these infections has not been completely determined, they are presumed to be mixed infections with an important anaerobic component.[1,17]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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SCIENCE TRANSFER

Consideration of the surgical anatomy serves as a basis for surgical procedures involving periodontal tissues and implants. Damage to nerves, such as the mental nerve, the mandibular nerve, and the lingual nerve, can be avoided with proper technique and treatment planning that should include three-dimensional radiographs when these structures are likely to be within the vicinity of surgical approaches. Penetration of the mandibular lingual bony plate during implant placement can cause hemorrhage into the sublingual and submandibular spaces, with possible life-threatening elevation of the tissues and respiratory obstruction. Infection spreading into these regions is also dangerous, and clinicians must be aware of these anatomic implications. Inadvertent surgical incision of major blood vessels, such as the greater palatine artery and the middle and posterior superior alveolar artery, can be avoided by knowledge of their anatomic positioning.

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CHAPTER 54

General Principles of Periodontal Surgery

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CHAPTER OUTLINE

OUTPATIENT SURGERY
- Patient Preparation
- Emergency Equipment
- Measures to Prevent Transmission of Infection
- Sedation and Anesthesia
- Tissue Management
- Scaling and Root Planing
- Hemostasis
- Periodontal Dressings (Periodontal Packs)
- Postoperative Instructions
- First Postoperative Week
- Removal of Pack and Return Visit
- Mouth Care between Procedures
- Management of Postoperative Pain

HOSPITAL PERIODONTAL SURGERY (online only)
- Indications
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SURGICAL INSTRUMENTS
- Excisional and Incisional Instruments
- Surgical Curettes and Sickles
- Periosteal Elevators
- Surgical Chisels
- Tissue Forceps
- Scissors and Nippers
- Needleholders

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

All surgical procedures should be carefully planned. The patient should be adequately prepared medically, psychologically, and practically for all aspects of the intervention. This chapter covers the preparation of the patient and the general considerations common to all periodontal surgical techniques. Complications that may occur during or after surgery are also discussed.

Surgical periodontal procedures are usually performed in the dental office. Hospital periodontal surgery is discussed later in this chapter, followed by a review of common surgical instruments.

OUTPATIENT SURGERY

Patient Preparation

Reevaluation after Phase I Therapy. Almost every patient undergoes the so-called initial or preparatory phase of therapy, which basically consists of thorough scaling and root planing and removing all irritants responsible for the periodontal inflammation. These procedures (1) eliminate some lesions entirely; (2) render the tissues more firm and consistent, thus permitting a more accurate and delicate surgery; and (3) acquaint the patient with the office and the operator and assistants, thereby reducing the patient's apprehension and fear.

The reevaluation phase consists of reprobing and reexamining all the pertinent findings that previously indicated the need for the surgical procedure. Persistence of these findings confirms the indication for surgery. The number of surgical procedures, expected outcome, and postoperative care necessary are all decided before therapy. These are discussed with the patient, and a final decision is made, incorporating any necessary adjustments to the original plan.

Premedication.* For patients who are not medically compromised, the value of administering antibiotics routinely for periodontal surgery has not been clearly demonstrated.[29] However, some studies have reported reduced postoperative complications, including reduced pain and swelling, when antibiotics are given before periodontal surgery and continued for 4 to 7 days after surgery.[4,12,21,32]

The prophylactic use of antibiotics in patients who are otherwise healthy has been advocated for bone-grafting procedures and

*Precautions to be taken with medically compromised patients are discussed in Chapter 37.

purported to enhance the chances of new attachment. Although the rationale for such use appears logical, no research evidence is available to support it. In any case, the risks inherent in the administration of antibiotics should be evaluated together with the potential benefits. Other presurgical medications include administration of a nonsteroidal antiinflammatory drug (NSAID) such as ibuprofen (Motrin) 1 hour before the procedure and an oral rinse with 0.12% chlorhexidine gluconate (Peridex, PerioGard).[38]

Smoking. The deleterious effect of smoking on healing of periodontal wounds has been amply documented[20,33,43] (see Chapter26). Patients should be clearly informed of this fact and asked to quit or stop smoking for a minimum of 3 to 4 weeks after the procedure. For patients who are unwilling to follow this advice, an alternate treatment plan that does not include more sophisticated techniques (e.g., regenerative, mucogingival, esthetic) should be considered.

Informed Consent. The patient should be informed at the initial visit about the diagnosis, prognosis, and different possible treatments, with their expected results and all pros and cons of each approach. At surgery the patient should again be informed, verbally and in writing, of the procedure to be performed, and he or she should indicate agreement by signing the consent form.

Emergency Equipment

The operator, all assistants, and office personnel should be trained to handle all the possible emergencies that may arise. Drugs and equipment for emergency use should be readily available at all times.

The most common emergency is *syncope,* or a transient loss of consciousness caused by a reduction in cerebral blood flow. The most common cause is fear and anxiety. Syncope is usually preceded by a feeling of weakness, and then the patient develops pallor, sweating, coldness of the extremities, dizziness, and slowing of the pulse. The patient should be placed in a supine position with the legs elevated; tight clothes should be loosened and a wide-open airway ensured. Administration of oxygen is also useful. Unconsciousness persists for a few minutes. A history of previous syncopal attacks during dental appointments should be explored before treatment is begun, and if these are reported, extra efforts to relieve the patient's fear and anxiety should be made. The reader is referred to other texts for a complete analysis of this important topic.[3]

Measures to Prevent Transmission of Infection

The danger of transmitting infections to the dental team or other patients has become apparent in recent years, particularly with the threat of acquired immunodeficiency syndrome (AIDS) and hepatitis B virus (HBV) infection. Universal precautions, including protective attire, and barrier techniques are strongly recommended and often required by law. These include the use of disposable sterile gloves, surgical masks, and protective eyewear. All surfaces possibly contaminated with blood or saliva that cannot be sterilized (e.g., light handles, unit syringes) must be covered with aluminum foil or plastic wrap. Aerosol-producing devices (e.g., Cavitron) should not be used on patients with suspected infections, and their use should be kept to a minimum in all other patients. Special care should be taken when using and disposing of sharp items such as needles and scalpel blades.

Sedation and Anesthesia

Periodontal surgery should be performed painlessly. The patient should be assured of this at the outset and throughout the procedure. The most reliable means of providing painless surgery is the effective administration of local anesthesia. The area to be treated should be thoroughly anesthetized by means of regional block and local infiltration. Injections directly into the interdental papillae may also be helpful.

Apprehensive and neurotic patients require special management with antianxiety or sedative-hypnotic agents. Modalities for the administration of these agents include inhalation, oral, intramuscular, and intravenous routes. The specific agents and modality of administration are based on the desired level of sedation, anticipated length of the procedure, and overall condition of the patient. Specifically, the patient's medical history and physical and emotional status should be considered when selecting agents and techniques.

Perhaps the simplest, least invasive method to alleviate anxiety in the dental office is *nitrous oxide and oxygen inhalation sedation.* For many individuals, this is quite effective. Advantages include a quick onset of action, the ability to adjust the level of sedation throughout the procedure, a rapid recovery, and little or no concern for postoperative impairment of sensory or motor function. One disadvantage is that a small percentage of patients will not achieve the desired effect. This is especially true for the mentally impaired individual because nitrous oxide and oxygen sedation requires some level of patient cooperation. Overall, inhalation sedation with nitrous oxide and oxygen is a safe, effective, and reliable means of reducing mild anxiety.

For individuals with mild-to-moderate anxiety, oral administration of a *benzodiazepine* can be effective in decreasing anxiety and producing a level of relaxation. Oral administration of a sedative agent can be more effective than inhalation anesthesia because the level of sedation achieved may be more profound. Disadvantages of oral sedative administration include incomplete recovery, an inability to control the level of sedation, and a prolonged period of impaired sensory and motor skills. A variety of benzodiazepine agents are available for oral administration (see Chapter 55).

Intravenous (IV) administration of a benzodiazepine, alone or in combination with other agents, can be used to achieve a greater level of sedation in individuals with moderate to severe levels of anxiety. Furthermore, the onset of action of IV sedation is almost immediate, and the level of sedation can be titrated on an individual basis to the desired effect. The recovery period depends on the half-life of the agent used and the amount given. The operator should receive formal training in the techniques of sedation; this often is required by law. A thorough understanding of the indications, contraindications, and risks of these agents is required.[3] The reader is referred to Chapter 55 and other texts for a more detailed discussion of conscious sedation modalities, agents, and techniques.[26]

Tissue Management

1. *Operate gently and carefully.* In addition to being most considerate to the patient, this is also the most effective way to operate. Tissue manipulation should be precise, deliberate, and gentle. Thoroughness is essential, but roughness must be avoided because it produces excessive tissue injury, causes postoperative discomfort, and delays healing.
2. *Observe the patient at all times.* It is essential to pay careful attention to the patient's reactions. Facial expressions, pallor, and perspiration are distinct signs that may indicate the patient is experiencing pain, anxiety, or fear. The physician's responsiveness to these signs can be the difference between success and failure.
3. *Be certain the instruments are sharp.* Instruments must be sharp to be effective; successful treatment is not possible without sharp instruments. Dull instruments inflict unnecessary trauma because of poor cutting and excessive force applied to

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compensate for their ineffectiveness. A sterile sharpening stone should be available on the operating table at all times.

Scaling and Root Planing

Although scaling and root planing have been performed previously as part of Phase I therapy, all exposed root surfaces should be carefully explored and planed as needed as part of the surgical procedure. In particular, areas of difficult access, such as furcations or deep pockets, often have rough areas or even calculus that was undetected during the preparatory sessions. The assistant who is retracting the tissues and using the aspirator should also check for the presence of calculus and the smoothness of each surface from a different angle.

Hemostasis

Hemostasis is an important aspect of periodontal surgery because good intraoperative control of bleeding permits an accurate visualization of the extent of disease, pattern of bone destruction, and anatomy and condition of the root surfaces. It provides the operator with a clear view of the surgical site, which is essential for wound debridement and scaling and root planing. In addition, good hemostasis also prevents excessive loss of blood into the mouth, oropharynx, and stomach.

Periodontal surgery can produce profuse bleeding, especially during the initial incisions and flap reflection. After flap reflection and removal of granulation tissue, bleeding disappears or is considerably reduced. Typically, control of intraoperative bleeding can be managed with aspiration. Continuous suctioning of the surgical site with an aspirator is indispensable for performing periodontal surgery. Application of pressure to the surgical wound with moist gauze can be a helpful adjunct to control site-specific bleeding. Intraoperative bleeding that is not controlled with these simple methods may indicate a more serious problem and require additional control measures.

Excessive hemorrhaging after initial incisions and flap reflection may be caused by laceration of venules, arterioles, or larger vessels. Fortunately, the laceration of medium or large vessels is rare because incisions near highly vascular anatomic areas, such as the posterior mandible (lingual and inferior alveolar arteries) and the posterior, midpalatal regions (greater palatine arteries), are avoided in incision and flap design. Proper design of the flaps, taking into consideration these areas, avoids accidents (see Chapter 53). However, even when all anatomic precautions are taken, it is possible to cause bleeding from medium or large vessels because anatomic variations do occur and may result in inadvertent laceration. If a medium or large vessel is lacerated, a suture around the bleeding end may be necessary to control hemorrhage. Pressure should be applied through the tissue to determine the location that will stop blood flow in the severed vessel. Then a suture can be passed through the tissue and tied to restrict blood flow. Excessive bleeding from a surgical wound also may result from incisions across a capillary plexus. Minor areas of persistent bleeding from capillaries can be stopped by applying cold pressure to the site with moist gauze (soaked in sterile ice water) for several minutes.

The use of a local anesthetic with a vasoconstrictor may also be useful in controlling minor bleeding from the periodontal flap. Both these methods act through vasoconstriction, thus reducing the flow of blood through incised small vessels and capillaries. This action is relatively short-lived and should not be relied on for long-term hemostasis. It is important to avoid the use of vasoconstrictors to control bleeding before sending a patient home. If a more serious bleeding problem exists or a firm blood clot is not established, bleeding is likely to recur when the vasoconstrictor has metabolized and the patient is no longer in the office.

For slow, constant blood flow and oozing, hemostasis may be achieved with hemostatic agents. Absorbable gelatin sponge (Gelfoam), oxidized cellulose (Oxycel), oxidized regenerated cellulose (Surgicel Absorbable Hemostat), and microfibrillar collagen hemostat (CollaCote, CollaTape, CollaPlug) are useful hemostatic agents for the control of bleeding in capillaries, small blood vessels, and deep wounds (Table 54-1).

Absorbable gelatin sponge is a porous matrix prepared from pork skin that helps stabilize a normal blood clot. The sponge can be cut to the desired dimensions and either sutured in place or positioned

TABLE 54-1 Absorbable Hemostatic Agents

Generic (Brand)	Directions	Adverse Effects	Precautions
Absorbable gelatin sponge (Gelfoam)	May be cut into various sizes and applied to bleeding surfaces.	May form nidus for infection or abscess.	Should not be overpacked into extraction site or wound—may interfere with healing.
Oxidized cellulose (Oxycel)	Most effective when applied to wound dry as opposed to moistened.	May cause foreign body reaction.	Extremely friable and difficult to place; should not be used adjacent to bone—impairs bone regeneration; should not be used as a surface dressing—inhibits epithelialization.
Oxidized regenerated cellulose (Surgicel Absorbable Hemostat)	May be cut to various shapes and positioned over bleeding sites; thick or excessive amounts should not be used.	Encapsulation, cyst formation, and foreign body reaction possible.	Should not be placed in deep wounds—may physically interfere with wound healing and bone formation.
Microfibrillar collagen hemostat (CollaCote, CollaTape, CollaPlug)	May be cut to shape and applied to bleeding surface.	May potentiate abscess formation, hematoma, and wound dehiscence; possible allergic reaction or foreign body reaction.	May interfere with wound healing; placement in extraction sockets has been associated with increased pain.
Thrombin (Thrombostat)	May be applied topically to bleeding surface.	Allergic reaction can occur in patients with known sensitivity to bovine materials.	Must not be injected into tissues or vasculature—can cause severe (possibly fatal) clotting.

within the wound (e.g., extraction socket). It is absorbed in 4 to 6 weeks.

Oxidized cellulose is a chemically modified form of surgical gauze that forms an artificial clot. The material is friable and can be difficult to keep in place. It absorbs in 1 to 6 weeks.

Oxidized regenerated cellulose is prepared from cellulose by reaction with alkali to form a chemically pure, more uniform structure than oxidized cellulose. The material is prepared in a cloth or thin gauze form that can be cut to the desired size and sutured or layered on the bleeding surface. It can be used as a surface dressing because it does not impair epithelialization, and it is bactericidal against many gram-negative and -positive microorganisms, both aerobic and anaerobic. Caution should be used when wounds are infected or have an increased potential to becoming infected (e.g., immunocompromised patients) because the absorbable hemostatic agents can serve as a nidus for infection.

Thrombin is a drug capable of hastening the process of blood clotting. It is intended for topical use only because it is applied as a liquid or powder. Thrombin should never be injected into tissues because it can cause serious, even fatal intravascular coagulation. Also, because thrombin is a bovine-derived material, caution should be used for any patient with known allergic reaction to bovine products.

Finally, it is imperative to recognize that excessive bleeding may be caused by systemic disorders, including (but not limited to) platelet deficiencies, coagulation defects, medications, and hypertension. As a precaution, all surgical patients should be asked about current medications that may contribute to bleeding, any family history of bleeding disorders, and hypertension. All patients, regardless of health history, should have their blood pressure evaluated before surgery, and anyone diagnosed with hypertension must be advised to see a physician before surgery. Patients with known or suspected bleeding deficiencies or disorders must be carefully evaluated before any surgical procedure. A consultation with the patient's physician is recommended, and laboratory tests should be done to assess the risk of bleeding. It may be necessary to refer the patient to a hematologist for a comprehensive workup.

Periodontal Dressings (Periodontal Packs)

In most cases, after the surgical periodontal procedures are completed, the area is covered with a surgical pack. In general, dressings have no curative properties; they assist healing by protecting the tissue rather than providing "healing factors." The pack minimizes the likelihood of postoperative infection and hemorrhage, facilitates healing by preventing surface trauma during mastication, and protects against pain induced by contact of the wound with food or the tongue during mastication. (For a complete literature review on this subject, see Sachs et al.[37])

Zinc Oxide–Eugenol Packs. Packs based on the reaction of zinc oxide and eugenol include the Wondr-Pak developed by Ward[46] in 1923 and several other packs that use modified forms of Ward's original formula. The addition of accelerators, such as zinc acetate, gives the dressing a better working time.

Zinc oxide–eugenol dressings are supplied as a liquid and powder that are mixed before use. Eugenol in this type of pack may induce an allergic reaction that produces reddening of the area and burning pain in some patients.

Noneugenol Packs. The reaction between a metallic oxide and fatty acids is the basis for Coe-Pak, which is the most widely used dressing in the United States. This is supplied in two tubes, the contents of which are mixed immediately before use until a uniform color is obtained. One tube contains zinc oxide, an oil (for plasticity), a gum (for cohesiveness), and Lorothidol (a fungicide); the other tube contains liquid coconut fatty acids thickened with colophony resin (or rosin) and chlorothymol (a bacteriostatic agent).[37,40] This dressing does not contain asbestos or eugenol, thereby avoiding the problems associated with these substances.

Other noneugenol packs include cyanoacrylates[6,19,24] and tissue conditioners (methacrylate gels).[2] However, these are not in common use.

Retention of Packs. Periodontal dressings are usually kept in place mechanically by interlocking in interdental spaces and joining the lingual and facial portions of the pack. In isolated teeth or when several teeth in an arch are missing, retention of the pack may be difficult. Numerous reinforcements and splints and stents for this purpose have been described.[17,18,47] Placement of dental floss tied loosely around the teeth enhances retention of the pack.

Antibacterial Properties of Packs. Improved healing and patient comfort with less odor and taste[6] have been obtained by incorporating antibiotics in the pack. Bacitracin,[5] oxytetracycline (Terramycin),[13] neomycin, and nitrofurazone have been tried, but all may produce hypersensitivity reactions. The emergence of resistant organisms and opportunistic infection has been reported.[35] Incorporation of tetracycline powder in Coe-Pak is generally recommended, particularly when long and traumatic surgeries are performed.

Allergy. Contact allergy to eugenol and rosin has been reported.[34]

Preparation and Application of Dressing. Zinc oxide packs are mixed with eugenol or noneugenol liquids on a wax paper pad with a wooden tongue depressor. The powder is gradually incorporated with the liquid until a thick paste is formed.

Coe-Pak is prepared by mixing equal lengths of paste from tubes containing the accelerator and the base until the resulting paste is a uniform color (Figure 54-1, *A* to *C*). A capsule of tetracycline powder can be added at this time. The pack is then placed in a cup of water at room temperature (Figure 54-1, *D*). In 2 to 3 minutes the paste loses its tackiness and can be handled and molded; it remains workable for 15 to 20 minutes. Working time can be shortened by adding a small amount of zinc oxide to the accelerator (pink paste) before spatulating.

The pack is then rolled into two strips approximately the length of the treated area. The end of one strip is bent into a hook shape and fitted around the distal surface of the last tooth, approaching it from the distal surface (Figure 54-2, *A*). The remainder of the strip is brought forward along the facial surface to the midline and gently pressed into place along the gingival margin and interproximally. The second strip is applied from the lingual surface. It is joined to the pack at the distal surface of the last tooth, then brought forward along the gingival margin to the midline (Figure 54-2, *B*). The strips are joined interproximally by applying gentle pressure on the facial and lingual surfaces of the pack (Figure 54-2, *C*). For isolated teeth separated by edentulous spaces, the pack should be made continuous from tooth to tooth, covering the edentulous areas (Figure 54-3).

When split flaps have been performed, the area should be covered with tin foil to protect the sutures before placing the pack (see Chapter 54).

The pack should cover the gingiva, but overextension onto uninvolved mucosa should be avoided. *Excess pack irritates the mucobuccal*

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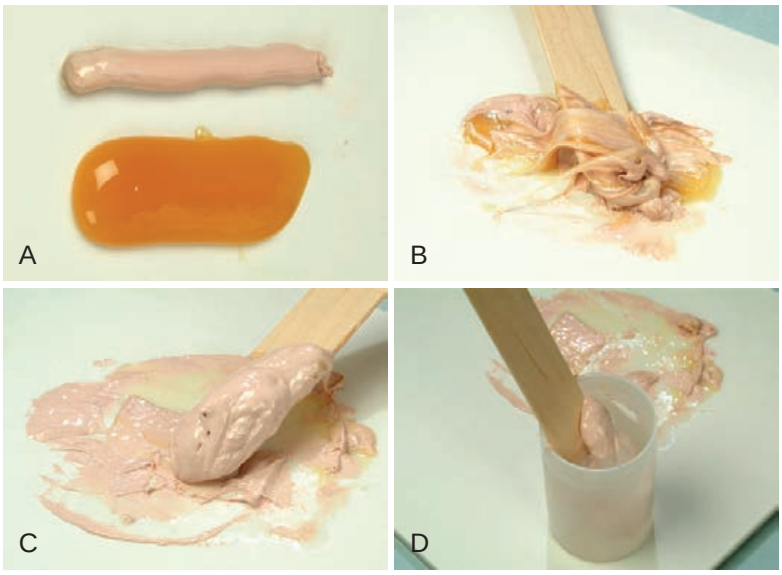

Figure 54-1 Preparing the surgical pack (Coe-Pak). **A,** Equal lengths of the two pastes are placed on a paper pad. **B,** Pastes are mixed with a wooden tongue depressor for 2 or 3 minutes until the paste loses its tackiness **(C). D,** Paste is placed in a paper cup of water at room temperature. With lubricated fingers, it is then rolled into cylinders and placed on the surgical wound.

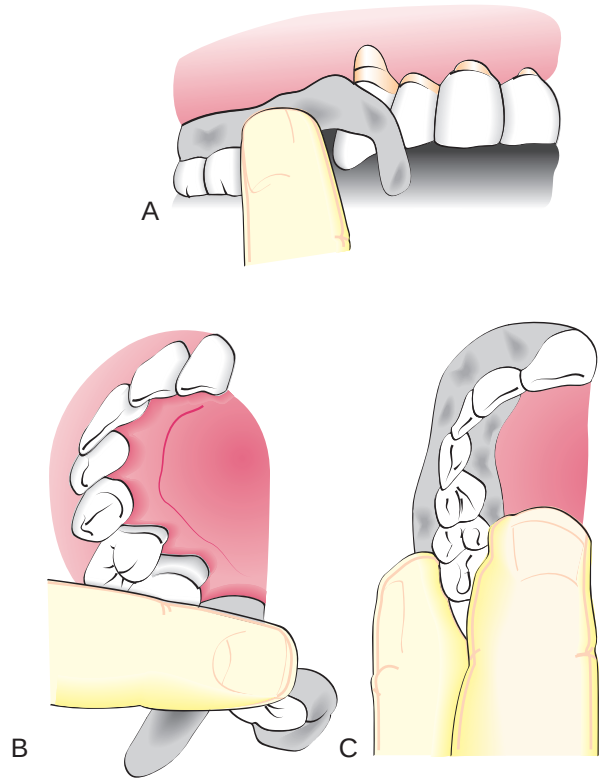

Figure 54-2 Inserting the periodontal pack. **A,** Strip of pack is hooked around the last molar and pressed into place anteriorly. **B,** Lingual pack is joined to the facial strip at the distal surface of the last molar and fitted into place anteriorly. **C,** Gentle pressure on the facial and lingual surfaces joins the pack interproximally.

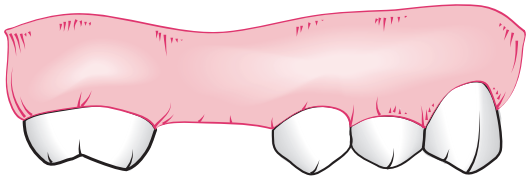

Figure 54-3 Continuous pack covers the edentulous space.

Figure 54-4 Periodontal pack should not interfere with the occlusion.

fold and floor of the mouth and interferes with the tongue. Overextension also jeopardizes the remainder of the pack because the excess tends to break off, taking pack from the operated area with it. *Pack that interferes with the occlusion should be trimmed away before the patient is dismissed* (Figure 54-4). Failure to do this causes discomfort and jeopardizes retention of the pack.

The operator should ask the patient to move the tongue forcibly out and to each side, and the cheek and lips should be displaced in all directions to mold the pack while it is still soft. After the pack has set, it should be trimmed to eliminate all excess.

As a general rule, the pack is kept on for 1 week after surgery. This guideline is based on the usual timetable of healing and clinical experience. It is not a rigid requirement; the period may be extended, or the area may be repacked for an additional week.

Fragments of the surface of the pack may come off during the week, but this presents no problem. If a portion of the pack is lost from the operated area and the patient is uncomfortable, it is usually best to repack the area. The clinician should remove the remaining pack, wash the area with warm water, and apply a topical anesthetic before replacing the pack, which is then retained for 1 week. Again, the patient may develop pain from an overextended margin that irritates the vestibule, floor of the mouth, or tongue. The excess pack should be trimmed away, making sure that the new margin is not rough, before the patient is dismissed.

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BOX 54-1 Patient Instructions after Periodontal Surgery

Instructions for _____ (Patient's Name)

The following information on your gum operation has been prepared to answer questions you may have about how to take care of your mouth. Please read the instructions carefully; our patients have found them very helpful.

Although there will be little or no discomfort when the anesthesia wears off, you should take two acetaminophen (Tylenol) tablets every 6 hours for the first 24 hours. After that, take the same medication if you have some discomfort. Do not take aspirin because this may increase bleeding.

We have placed a periodontal pack over your gums to protect them from irritation. The pack prevents pain, aids healing, and enables you to carry on most of your usual activities in comfort. The pack will harden in a few hours, after which it can withstand most of the forces of chewing without breaking off. It may take a little while to become accustomed to it.

The pack should remain in place until it is removed in the office at the next appointment. If particles of the pack chip off during the week, do not be concerned as long as you do not have pain. If a piece of the pack breaks off and you are in pain, or if a rough edge irritates your tongue or cheek, please call the office. The problem can be easily remedied by replacing the pack.

For the first 3 hours after the operation, avoid hot foods to permit the pack to harden. It is also convenient to avoid hot liquids during the first 24 hours. You can eat anything you can manage, but try to chew on the nonoperated side of your mouth. Semisolid or finely minced foods are suggested. Avoid citrus fruits or fruit juices, highly spiced foods, and alcoholic beverages; these will cause pain. Food supplements or vitamins are generally not necessary.

Do not smoke. The heat and smoke will irritate your gums, and the immunologic effects of nicotine will delay healing and prevent a completely successful outcome of the procedure performed. If possible, use this opportunity to give up smoking. In addition to all other well-known health risks, smokers have more gum disease than nonsmokers.

Do not brush over the pack. Brush and floss the areas of the mouth not covered by the pack as you normally do. Use chlorhexidine (Peridex, PerioGard) oral rinses after brushing (the prescription for this rinse has been given to you).

During the first day, apply ice intermittently on the face over the operated area. It is also beneficial to suck on ice chips intermittently during the first 24 hours. These methods will keep tissues cool and reduce inflammation and swelling.

You may experience a slight feeling of weakness or chills during the first 24 hours. This should not be cause for alarm but should be reported at the next visit. Follow your regular daily activities, but avoid excessive exertion of any type. Golf, tennis, skiing, bowling, swimming, or sunbathing should be postponed for a few days after the operation.

Swelling is not unusual, particularly in areas that required extensive surgical procedures. The swelling generally begins 1 to 2 days after the operation and subsides gradually in 3 or 4 days. If this occurs, apply moist heat over the operated area. If the swelling is painful or appears to become worse, please call the office.

Occasionally, blood may be seen in the saliva for the first 4 or 5 hours after the operation. This is not unusual and will correct itself. If there is considerable bleeding beyond this, take a piece of gauze, form it into the shape of a U, hold it in the thumb and index finger, apply it to both sides of the pack, and hold it there under pressure for 20 minutes. Do not remove it during this period to examine it. If the bleeding does not stop at the end of 20 minutes, please contact the office. Do not try to stop bleeding by rinsing.

After the pack is removed, the gums most likely will bleed more than they did before the operation. This is perfectly normal in the early stage of healing and will gradually subside. Do not stop cleaning because of it. If any other problems arise, please call the office.

Postoperative Instructions

After the pack is placed, printed instructions are given to the patient to be read before he or she leaves the chair (Box 54-1).

First Postoperative Week

Properly performed, periodontal surgery presents no serious postoperative problems. Patients should be told to rinse with 0.12% chlorhexidine gluconate (Peridex, PerioGard) immediately after the surgical procedure and twice daily thereafter until normal plaque control technique can be resumed.[30,38,45] The following complications may arise in the first postoperative week, although they are the exception rather than the rule:

1. *Persistent bleeding after surgery.* The pack is removed, the bleeding points are located, and the bleeding is stopped with pressure, electrosurgery, or electrocautery. After the bleeding is stopped, the area is repacked.
2. *Sensitivity to percussion.* Extension of inflammation into the periodontal ligament may cause sensitivity to percussion. The patient should be questioned regarding the progress of the symptoms. Gradually diminishing severity is a favorable sign. The pack should be removed and the gingiva checked for localized areas of infection or irritation, which should be cleaned or incised to provide drainage. Particles of calculus that may have been overlooked should be removed. Relieving the occlusion is usually helpful. Sensitivity to percussion may also be caused by excess pack, which interferes with the occlusion. Removal of the excess usually corrects the condition.
3. *Swelling.* In the first 2 postoperative days, some patients may report a soft, painless swelling of the cheek in the surgical area. Lymph node enlargement may occur, and the temperature may be slightly elevated. The area of operation itself is usually symptom free. This type of involvement results from a localized inflammatory reaction to the procedure. It generally subsides by the fourth postoperative day, without necessitating removal of the pack. If swelling persists, becomes worse, or is associated with increased pain, amoxicillin (500 mg) should be taken every 8 hours for 1 week, and the patient should also be instructed to apply moist heat intermittently over the area. The antibiotic should also be used as a prophylactic measure after the next procedure, starting before the surgical appointment.
4. *Feeling of weakness.* Occasionally, patients report having experienced a "washed-out," weakened feeling for about 24 hours after surgery. This represents a systemic reaction to a transient bacteremia induced by the procedure. This reaction is prevented by premedication with amoxicillin (500 mg) every 8 hours, beginning 24 hours before the next procedure and continuing for 5 days postoperatively.

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Removal of Pack and Return Visit

When the patient returns after 1 week, the periodontal pack is taken off by inserting a surgical hoe along the margin and exerting gentle lateral pressure. Pieces of pack retained interproximally and particles adhering to the tooth surfaces are removed with scalers. Particles may be enmeshed in the cut surface and should be carefully picked off with fine cotton pliers. The entire area is rinsed with peroxide to remove superficial debris.

Findings at Pack Removal. The following are usual findings when the pack is removed:

- If a *gingivectomy* has been performed, the cut surface is covered with a friable meshwork of new epithelium, which should not be disturbed. If calculus has not been completely removed, red, beadlike protuberances of granulation tissue will persist. The granulation tissue must be removed with a curette, exposing the calculus so that it can be removed and the root can be planed. Removal of the granulation tissue without removal of calculus is followed by recurrence.
  - After a *flap operation*, the areas corresponding to the incisions are epithelialized but may bleed readily when touched; they should not be disturbed. Pockets should not be probed.
  - The facial and lingual mucosa may be covered with a grayish yellow or white granular layer of food debris that has seeped under the pack. This is easily removed with a moist cotton pellet. The root surfaces may be sensitive to a probe or to thermal changes, and the teeth may be stained.
  - *Fragments of calculus delay healing.* Each root surface should be rechecked visually to be certain that no calculus is present. Sometimes the color of the calculus is similar to that of the root. The grooves on proximal root surfaces and the furcations are areas where calculus is likely to be overlooked.

Repacking. After the pack is removed, it is usually not necessary to replace it. However, repacking for an additional week is advised for patients with (1) a low pain threshold who are particularly uncomfortable when the pack is removed, (2) unusually extensive periodontal involvement, or (3) slow healing. Clinical judgment helps in deciding whether to repack the area or leave the initial pack on longer than 1 week.

Tooth Mobility. Tooth mobility is increased immediately after surgery,[8] but it diminishes below the pretreatment level by the fourth week.[25]

Mouth Care between Procedures

Care of the mouth by the patient between the treatment of the first and the final areas, as well as after surgery is completed, is extremely important.[48] These measures should begin after the pack is removed from the first surgery. The patient has been through a presurgical period of instructed plaque control and should be reinstructed at this time.

Vigorous brushing is not feasible during the first week after the pack is removed. However, the patient is informed that plaque and food accumulation impair healing and is advised to try to keep the area as clean as possible by the gentle use of soft toothbrushes and light water irrigation. Rinsing with a chlorhexidine mouthwash or its topical application with cotton-tipped applicators is indicated for the first few postoperative weeks, particularly in advanced cases. Brushing is introduced when healing of the tissues permits it; the vigor of the overall hygiene regimen is increased as healing progresses. Patients should be told that (1) more gingival bleeding will most likely occur than was present before the procedure, (2) this bleeding is perfectly normal and will subside as healing progresses, and (3) it should not deter them from following their oral hygiene regimen.

Management of Postoperative Pain

Periodontal surgery that follows the basic principles outlined here should produce only minor pain and discomfort.[41] One study of 304 consecutive periodontal surgical interventions revealed that 51.3% of the patients reported minimal or no postoperative pain, and only 4.6% reported severe pain. Of these, only 20.1% took 5 or more doses of analgesic.[11] The same study showed that mucogingival procedures result in 6 times more discomfort and osseous surgery in 3.5 times more discomfort than plastic gingival surgery. In the few patients who may have severe pain, its control then becomes an important part of patient management.[29]

A common source of postoperative pain is overextension of the periodontal pack onto the soft tissue beyond the mucogingival junction or onto the frena. Overextended packs cause localized areas of edema, usually noticed 1 to 2 days after surgery. Removal of excess pack is followed by resolution in about 24 hours. Extensive and excessively prolonged exposure and dryness of bone also induce severe pain. For most healthy patients, a preoperative dose of ibuprofen (600 to 800 mg) followed by 1 tablet every 8 hours for 24 to 48 hours is very effective in reducing discomfort after periodontal therapy. Patients are advised to continue taking ibuprofen or change to acetaminophen if needed thereafter. If pain persists, acetaminophen plus codeine (Tylenol #3) can be prescribed. Caution should be used in prescribing or dispensing ibuprofen to patients with hypertension controlled by medications because it can interfere with the effectiveness of the medication. When severe postoperative pain is present, the patient should be seen at the office on an emergency basis. The area is anesthetized by infiltration or topically, the pack is removed, and the wound is examined. Postoperative pain related to infection is accompanied by localized lymphadenopathy and a slight elevation in temperatures.[31] It should be treated with systemic antibiotics and analgesics.

For more information on postoperative root sensitivity and desensitizing agents, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on hospital periodontal surgery, please visit the companion Expert CONSULT website at www.expertconsult.com.

SURGICAL INSTRUMENTS

Periodontal surgery is accomplished with numerous instruments; Figure 54-5 shows a typical surgical cassette. Periodontal surgical instruments are classified as follows:

1. Excisional and incisional instruments
2. Surgical curettes and sickles
3. Periosteal elevators
4. Surgical chisels
5. Surgical files
6. Scissors
7. Hemostats and tissue forceps

Excisional and Incisional Instruments

Periodontal Knives (Gingivectomy Knives). The Kirkland knife is representative of knives typically used for

SCIENCE TRANSFER

The majority of periodontal surgeries can be carried out solely with thorough application of local anesthesia, but clinicians have the obligation to ensure a patient-centered approach that should include oral, intravenous, and inhalational sedation in their spectrum of available services to be used as needed.

An efficient, precise, and minimally traumatic management of tissues is the way to obtain the best clinical outcomes. All patients need oral analgesic support and should be given the necessary pain-relieving medication so that an effective level of analgesic is present in the immediate postsurgical period and thereafter as needed. The use of longer acting local anesthesia agents, such as bupivacaine, and protective periodontal dressings also helps reduce postsurgical pain.

In the immediate postsurgical weeks, plaque control and healing are enhanced by the use of antimicrobial mouthrinses such as chlorhexidine. Postsurgical root sensitivity is well controlled by ensuring plaque control is optimal, and desensitizing agents will be needed only occasionally.

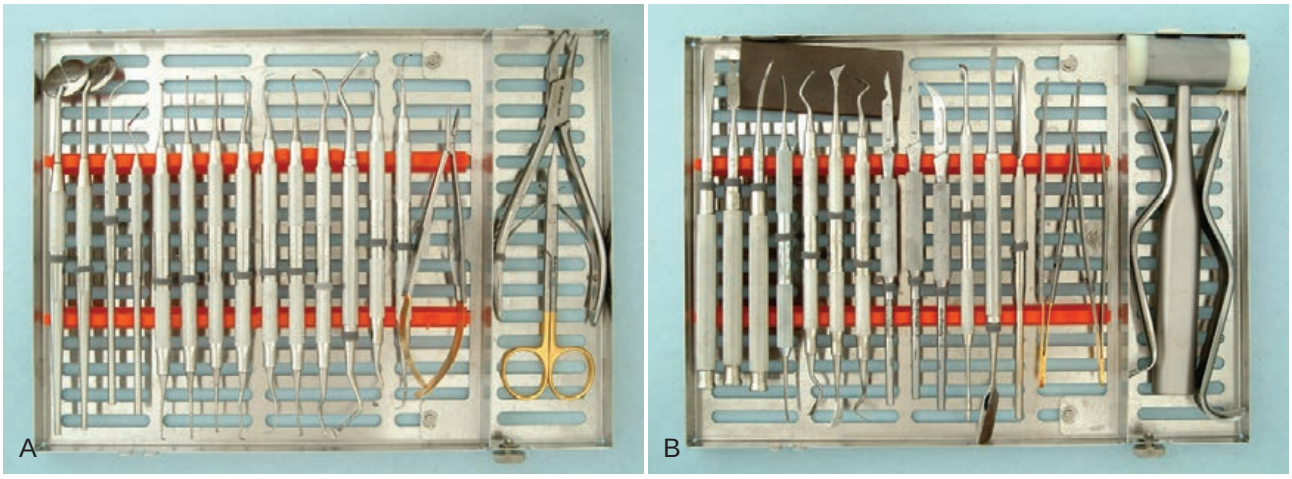

Figure 54-5 Typical series of periodontal surgical instruments, divided into two cassettes. **A,** *From left,* Mirrors, explorer, probe, series of curettes, needleholder, rongeurs, and scissors. **B,** *From left,* Series of chisels, Kirkland knife, Orban knife, scalpel handles with surgical blades (#15C, 15, 12D), periosteal elevators, spatula, tissue forceps, cheek retractors, mallet, and sharpening stone. *(***A** *Courtesy Hu-Friedy, Chicago;* **B** *Courtesy G. Hartzell & Son, Concord, CA.)*

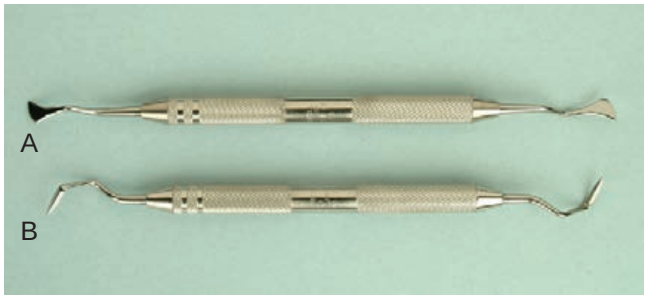

Figure 54-6 Gingivectomy knives. **A,** Kirkland knife. **B,** Orban interdental knife.

Figure 54-7 Surgical blades. *Top to bottom,* #15, #12D, and #15C. These blades are disposable.

gingivectomy. These knives can be obtained as either double-ended or single-ended instruments. The entire periphery of these kidney-shaped knives is the cutting edge (Figure 54-6, *A*).

Interdental Knives. The Orban knife #1-2 (Figure 54-6, *B*) and the Merrifield knife #1, 2, 3, and 4 are examples of knives used for interdental areas. These spear-shaped knives have cutting edges on both sides of the blade and are designed with either double-ended or single-ended blades.

Surgical Blades. Scalpel blades of different shapes and sizes are used in periodontal surgery. The most common blades are #12D, 15, and 15C (Figure 54-7). The #12D blade is a beak-shaped blade with cutting edges on both sides, allowing the operator to engage narrow, restricted areas with both pushing and pulling cutting motions. The #15 blade is used for thinning flaps and general purposes. The #15C blade, a narrower version of the #15 blade, is useful for making the initial, scalloping-type incision. The slim design of this blade allows for incising into the narrow

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Figure 54-8 Prichard surgical curette. Curettes used in surgery have wider blades than those used for conventional scaling and root planing.

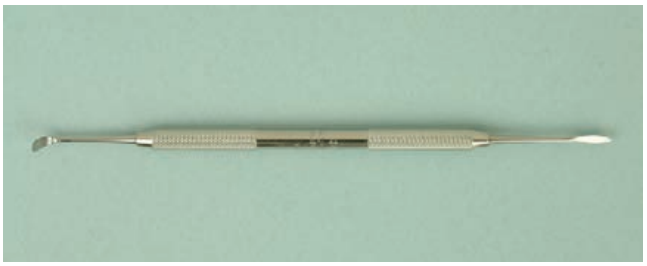

Figure 54-9 Woodson periosteal elevator.

Figure 54-10 Back-action chisel.

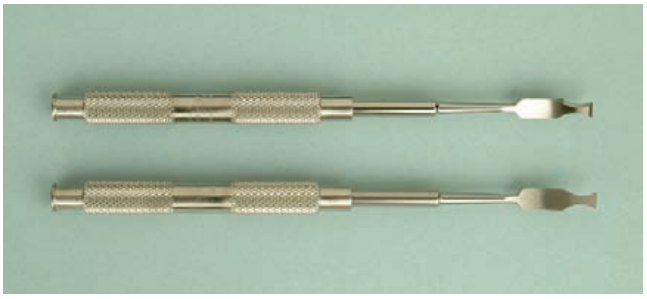

Figure 54-11 Ochsenbein chisels are paired, with the cutting edges in opposite directions.

interdental portion of the flap. All these blades are discarded after one use.

Electrosurgery (Radiosurgery) Techniques and Instrumentation. The term *electrosurgery* or *radiosurgery*[39] is currently used to identify surgical techniques performed on soft tissue using controlled, high-frequency electrical (radio) currents in the range of 1.5 to 7.5 million cycles per second, or megahertz. There are three classes of active electrodes: single-wire electrodes for incising or excising; loop electrodes for planing tissue; and heavy, bulkier electrodes for coagulation procedures.[16,28]

The four basic types of electrosurgical techniques are electrosection, electrocoagulation, electrofulguration, and electrodesiccation.

Electrosection, also referred to as electrotomy or acusection, is used for incisions, excisions, and tissue planing. Incisions and excisions are performed with single-wire active electrodes that can be bent or adapted to accomplish any type of cutting procedure.

Electrocoagulation provides a wide range of coagulation or hemorrhage control by using the electrocoagulation current. Electrocoagulation can prevent bleeding or hemorrhage at the initial entry into soft tissue, but it cannot stop bleeding after blood is present. All forms of hemorrhage must be stopped first by some form of direct pressure (e.g., air, compress, or hemostat). After bleeding has momentarily stopped, final sealing of the capillaries or large vessels can be accomplished by a short application of the electrocoagulation current. The active electrodes used for coagulation are much bulkier than the fine tungsten wire used for electrosection. Electrosection and electrocoagulation are the procedures most often used in all areas of dentistry. The two monoterminal techniques, electrofulguration and electrodesiccation, are not in general use in dentistry.

The most important basic rule of electrosurgery is: *always keep the tip moving*. Prolonged or repeated application of current to tissue induces heat accumulation and undesired tissue destruction, whereas interrupted application at intervals adequate for tissue cooling (5 to 10 seconds) reduces or eliminates heat buildup. Electrosurgery is not intended to destroy tissue; it is a controllable means of sculpturing or modifying oral soft tissue with little discomfort and hemorrhage for the patient.

The indications for electrosurgery in periodontal therapy and a description of wound healing after electrosurgery are presented in Chapter 56. Electrosurgery is contraindicated for patients who have incompatible or poorly shielded cardiac pacemakers.

Surgical Curettes and Sickles

Larger and heavier curettes and sickles are often needed during surgery for the removal of granulation tissue, fibrous interdental tissues, and tenacious subgingival deposits. The Prichard curette (Figure 54-8) and the Kirkland surgical instruments are heavy curettes, whereas the Ball scaler #B2-B3 is a popular heavy sickle. The wider, heavier blades of these instruments make them suitable for surgical procedures.

Periosteal Elevators

The periosteal elevators are needed to reflect and move the flap after the incision has been made for flap surgery. The Woodson and Prichard elevators are well-designed periosteal instruments (Figure 54-9).

Surgical Chisels

The back-action chisel is used with a pull motion Figure 54-10, whereas the straight chisel (e.g., Wiedelstadt, Ochsenbein #1-2) is used with a push motion (Figure 54-11). The Ochsenbein chisel is a useful chisel with a semicircular indentation on both sides of the shank that allows the instrument to engage around the tooth and into the interdental area. The Rhodes chisel is another popular back-action chisel.

Tissue Forceps

The tissue forceps is used to hold the flap during suturing. It is also used to position and displace the flap after the flap has been reflected. The DeBakey forceps is an extremely efficient instrument (Figure 54-12).

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Figure 54-12 DeBakey tissue forceps.

Figure 54-13 Goldman-Fox scissors.

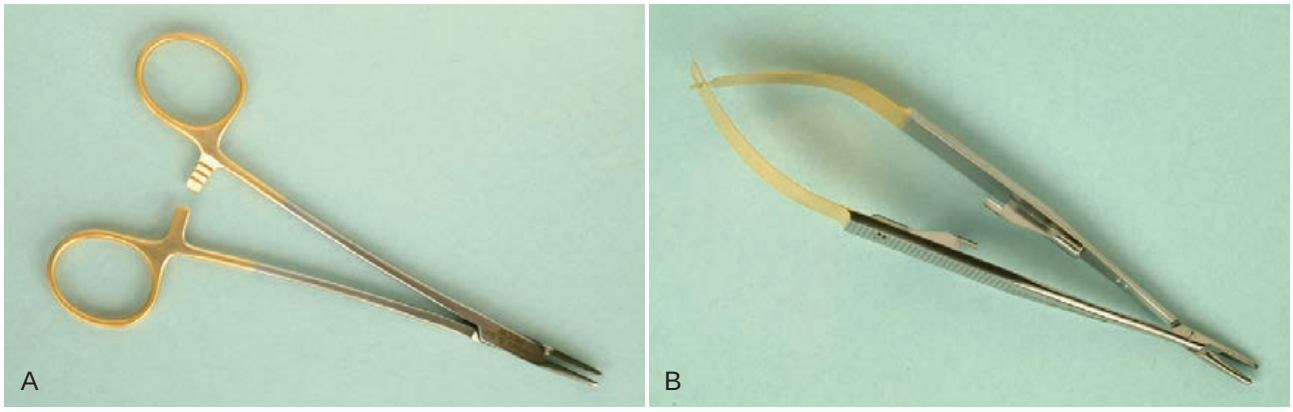

Figure 54-14 **A,** Conventional needleholder. **B,** Castroviejo needleholder.

Scissors and Nippers

Scissors and nippers are used in periodontal surgery to remove tabs of tissue during gingivectomy, trim the margins of flaps, enlarge incisions in periodontal abscesses, and remove muscle attachments in mucogingival surgery. Many types are available, and individual preference determines the choice. The Goldman-Fox #16 scissors has a curved, beveled blade with serrations (Figure 54-13).

Needleholders

Needleholders are used to suture the flap at the desired position after the surgical procedure has been completed. In addition to the regular types of needleholder (Figure 54-14, *A*), the Castroviejo needleholder is used for delicate, precise techniques that require quick and easy release and grasp of the suture (Figure 54-14, *B*).

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 55

Conscious Sedation

Robert L. Merin and Perry R. Klokkevold

CHAPTER OUTLINE

RATIONALE FOR SEDATION DURING PERIODONTAL AND IMPLANT SURGICAL PROCEDURES
ADA POLICY STATEMENT AND GUIDELINES FOR CONSCIOUS SEDATION (online only)
ADA Policy Statement: The Use of Sedation and General Anesthesia by Dentists (online only)
ADA Guidelines for the Use of Sedation and General Anesthesia by Dentists (online only)
DEFINITIONS AND LEVELS OF SEDATION
Pediatric Sedation
Adult Sedation
CLINICAL GUIDELINES FOR MINIMAL AND MODERATE SEDATION[4]
MILD SEDATION (ANXIOLYSIS) (online only)
Oral Sedation (online only)
Inhalation Sedation (online only)
MODERATE (CONSCIOUS) SEDATION (online only)
Oral Sedation (online only)
Combined Oral and Inhalation Sedation (online only)
Intravenous Sedation (online only)
Combined Oral and Intravenous Sedation (online only)
SEDATION FAILURES
EMERGENCY PREPAREDNESS

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Some figures, boxes, and tables may be out of numeric order in this printed chapter.

Periodontal and implant surgery should be performed painlessly and with minimal or no apprehension. The patient should be assured of this at the outset and throughout the procedure. The most reliable means of providing painless surgery is with effective administration of local anesthesia. However, patients who are apprehensive may require treatment under mild or moderate sedation. The use of sedation can help make patients more comfortable during periodontal and implant surgery, especially when the procedure is expected to continue for 2 hours or more. Routes of administration for sedation agents include inhalation, oral, intramuscular, and intravenous (IV). The specific agent(s) and modality of administration are based on the desired level of sedation, anticipated length of the procedure, overall condition of the patient, and training of the clinician and staff. This chapter reviews the rationale, definitions, techniques, and guidelines for the use of mild-to-moderate conscious sedation in the dental office for periodontal and implant surgical procedures.

RATIONALE FOR SEDATION DURING PERIODONTAL AND IMPLANT SURGICAL PROCEDURES

Many patients delay or avoid having needed dental treatment because of fear and anxiety. This avoidance behavior often results in compromised health and quality of life. Anxiety toward dental therapy has not changed significantly over the past 50 years; publications report that about 30% to 50% of patients are at least somewhat fearful of dental procedures.[7,13,14,53] New evidence suggests that genetic variations are associated with anxiety related to dental care, which could help to explain the consistent avoidance patterns despite improved treatment methods.[8] According to a national survey of the Canadian population, more than 68% of patients would prefer to have sedation or general anesthesia for periodontal surgery[9] (Figure 55-1). Anxiety reduction is an important part of delivering advanced periodontal services.[49]

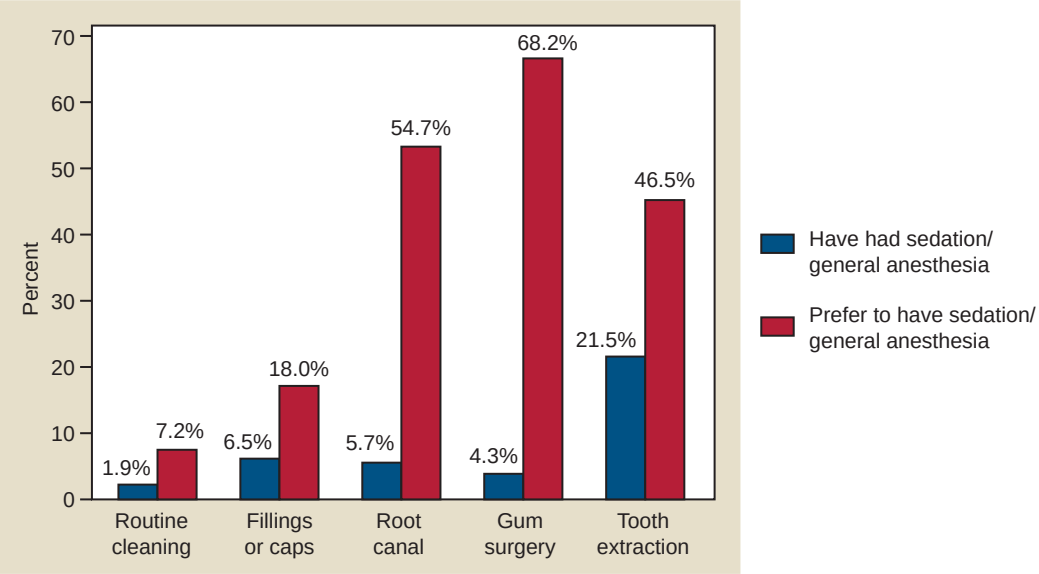

Figure 55-1 Patient preference for sedation or general anesthesia by dental procedure. *(From Chanpong B, Haas DA, Locker D: Need and demand for sedation or general anesthesia in dentistry: a national survey of the Canadian population,* Anesth Prog *52:3-11, 2005.)*

Furthermore, since dental anxiety results in avoidance behavior and is associated with more dental and periodontal problems,[18,38] it is likely that a disproportionate number of patients referred to periodontal specialists will have dental anxiety. Interestingly, there appears to be a close relationship between anxiety and postoperative pain. In fact, preoperative anxiety may be considered a predictor of postoperative pain.[20,23] In addition, high levels of anxiety (stress) can affect wound healing after periodontal treatment[34,37,58] (see Chapter 27). Sedation techniques have been shown to be effective in reducing physiologic markers of stress.[51] For these reasons, it is important for clinicians who provide advanced periodontal and implant therapy to be knowledgeable and skilled in providing sedation in order to reduce anxiety in their patients.

ADA POLICY STATEMENT AND GUIDELINES FOR CONSCIOUS SEDATION

In 2007, the American Dental Association (ADA) released three documents related to the use of sedation and general anesthesia in dentistry. One of these documents, the *ADA Guidelines for the Use of Sedation and General Anesthesia in Dentistry,* adopted the American Society of Anesthesiologists' definitions for levels of sedation (Figure 55-2) and specifically related them to use in dentistry.

For more information on ADA policy statement and guidelines for conscious sedation, please visit the companion Expert CONSULT website at www.expertconsult.com.

DEFINITIONS AND LEVELS OF SEDATION

Pediatric Sedation

Children (aged 12 and under) can become moderately sedated despite an intended level of minimal sedation. The use of preoperative sedatives for children (ages 12 and under) except in extraordinary situations must be avoided because of the risk of unobserved respiratory obstruction during transport by untrained individuals. The management of children with conscious sedation is beyond the scope of this chapter and is not be covered. For children 12 years of age and under, the reader is referred to the *American Academy of Pediatrics/American Academy of Pediatric Dentists Guidelines for Monitoring and Management of Pediatric Patients During and After Sedation for Diagnostic and Therapeutic Procedures.*[2]

Adult Sedation

Minimal Sedation. Minimal sedation is defined as a minimally depressed level of consciousness produced by a pharmacologic method that retains the patients ability to independently and continuously maintain an airway and to respond normally to tactile stimulation and verbal command. Although cognitive function and coordination may be modestly impaired, respiratory and cardiovascular functions are unaffected. When the intent is minimal sedation, the appropriate initial dosing of a single enteral drug is no more than the maximum recommended dose of a drug that can be prescribed for unmonitored home use.

Inhalation sedation with nitrous oxide/oxygen (N_2O/O_2) may be used in combination with a single enteral drug in minimal sedation. It is important to recognize that N_2O/O_2, when used in combination with one or more sedative agent(s), is capable of producing sedation that is minimal, moderate, or deep and in some cases may produce general anesthesia.

Maximum Recommended Dose. The maximum recommended dose (MRD) is the maximum Food and Drug Administration (FDA)-recommended dose of a drug as printed in FDA-approved labeling for unmonitored home use.

Incremental Dosing. Incremental dosing is the administration of multiple doses of a drug until a desired effect is reached.

Supplemental Dosing. During minimal sedation, supplemental dosing is a single additional dose of the sedative drug being used that may be necessary for prolonged procedures. The supplemental dose should not exceed one-half of the initial total dose and should not be administered until the dentist has determined that

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CONTINUUM OF DEPTH OF SEDATION: DEFINITION OF GENERAL ANESTHESIA AND LEVELS OF SEDATION/ANALGESIA*
Committee of Origin: Quality Management and Departmental Administration (Approved by the ASA House of Delegates on October 13, 1999, and amended on October 27, 2004)

	Minimal Sedation/ Analgesia	Moderate Sedation/ "Conscious Sedation"	Deep Sedation/Anxiolysis	General Anesthesia/ Analgesia
Responsiveness	Normal response to verbal stimulation	Purposeful† response to verbal or tactile stimulation	Purposeful† response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

Minimal Sedation (Anxiolysis) is a drug-induced state during which patients respond normally to verbal commands. Although cognitive function and coordination may be impaired, ventilatory and cardiovascular functions are unaffected.

Moderate Sedation/Analgesia ("Conscious Sedation") is a drug-induced depression of consciousness during which patients respond purposefully† to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.

Deep Sedation/Analgesia is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully† following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.

General Anesthesia is a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.

Because sedation is a continuum, it is not always possible to predict how an individual patient will respond. Hence, practitioners intending to produce a given level of sedation should be able to rescue†† patients whose level of sedation becomes deeper than initially intended. Individuals administering Moderate Sedation/Analgesia ("Conscious Sedation") should be able to rescue†† patients who enter a state of Deep Sedation/Analgesia, while those administering Deep Sedation/Analgesia should be able to rescue†† patients who enter a state of General Anesthesia.

*Monitored anesthesia care does not describe the continuum of depth of sedation, rather it describes "a specific anesthesia service in which an anesthesiologist has been requested to participate in the care of a patient undergoing a diagnostic or therapeutic procedure."
†Reflex withdrawal from a painful stimulus is NOT considered a purposeful response.
††Rescue of a patient from a deeper level of sedation than intended is an intervention by a practitioner proficient in airway management and advanced life support. The qualified practitioner corrects adverse physiologic consequences of the deeper-than-intended level of sedation (such as hypoventilation, hypoxia, and hypotension) and returns the patient to the originally intended level of sedation.

Figure 55-2 Continuum of depth of sedation: definition of general anesthesia and levels of sedation/analgesia. (Approved by the ASA House of Delegates on October 27, 2004 and last amended on October 21, 2009. http://www.asahq.org/publicationsAndServices/standards/20.pdf is reprinted with permission of the American Society of Anesthesiologists, 520 N. Northwest Highway, Park Ridge, Illinois 60068-2573.)

the clinical half-life of the initial dose has passed. The total aggregate dose must not exceed 1.5 times the MRD on the day of treatment.

Moderate Sedation. Moderate sedation is a drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilations are adequate. Cardiovascular function is usually maintained. In accord with this particular definition, the drugs and/or techniques used should carry a margin of safety wide enough to render unintended loss of consciousness unlikely. Repeated dosing of an agent before the effects of previous dosing can be fully appreciated may result in greater alteration of the state of consciousness than is the intent of the dentist. Further, a patient whose only response is reflex withdrawal from a painful stimulus is not considered to be in a state of moderate sedation.

Titration. Titration is the administration of incremental doses of a drug until a desired effect is reached. Knowledge of each drug's time of onset, peak response, and duration of action is essential to avoid oversedation. The concept of titration to effect is critical for patient safety. Thus it is important to know whether the previous dose has taken full effect before administering an additional drug increment when the intent is moderate sedation.

Deep Sedation. Deep sedation is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain respiratory function may be impaired. Patients may require assistance in maintaining a patent

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airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.

General Anesthesia. General anesthesia is a drug-induced loss of consciousness during which patients are not aroused, even by painful stimulation. The ability to independently maintain respiratory function is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.

Clinical Guidelines for Minimal and Moderate Sedation

The following clinical guidelines apply to both minimal and moderate sedation.[4] These guidelines include (1) patient evaluation, (2) preoperative preparation, (3) personnel and equipment, (4) monitoring and documentation, and (5) recovery and discharge. Differences between guidelines for minimal and moderate sedation are indicated as appropriate.

Patient Evaluation. Patients must be evaluated to assess their current health status before any sedation procedure. This should include a determination of their American Society of Anesthesiologists (ASA) physical status (Table 55-2). In healthy or medically stable individuals (ASA I or II), a review of their medical history and medication use may be adequate. However, for patients with significant medical considerations (ASA III or IV), a consultation with their primary care physician or consulting medical specialist is indicated. The evaluation should also include baseline vital signs and a focused physical examination of alertness, respiratory function, airway, and appearance, as well as a specific evaluation of any identified medical conditions (Box 55-1 and Figure 55-3).

Preoperative Preparation. The patient (parent, guardian, or caregiver, if patient is a minor) must be informed about the planned procedure under sedation, including benefits, risks, and instructions for sedation (Figure 55-4). An informed consent for the proposed procedure and sedation must be obtained.

Preoperative dietary restrictions must be considered based on the sedative technique prescribed (Boxes 55-2 and 55-3; see also Figure 55-4).

Personnel and Equipment. **Personnel.** At least one additional person trained in Basic Life Support (BSL) for Healthcare Providers must be present in addition to the dentist.

Equipment. Determination of adequate oxygen supply and equipment necessary to deliver oxygen under positive pressure must be completed. A positive-pressure oxygen delivery system suitable for the patient being treated must be immediately available. An

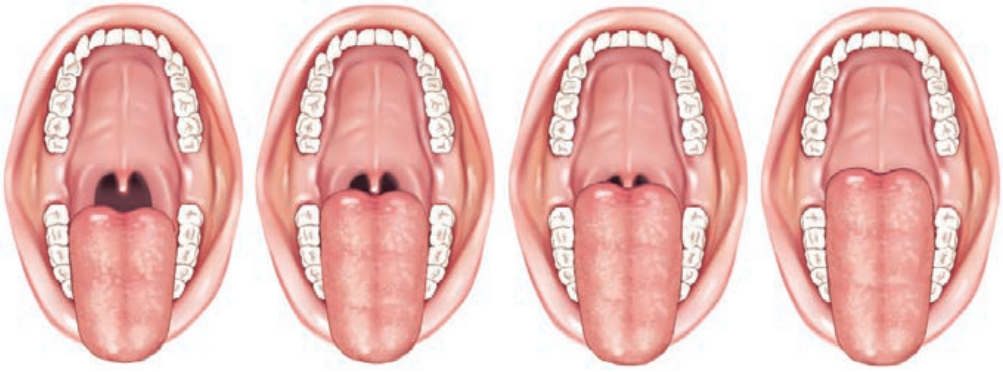
TABLE 55-2 ASA Physical Status Classification System

ASA 1	A normal healthy patient.
ASA 2	A patient with mild systemic disease.
ASA 3	A patient with severe systemic disease.
ASA 4	A patient with severe systemic disease that is a constant threat to life.
ASA 5	A moribund patient who is not expected to survive without the operation.
ASA 6	A declared brain-dead patient whose organs are being removed for donor purposes.

From American Society of Anesthesiologists: ASA Physical Status Classification System, www.asahq.org, 2009.

BOX 55-1 Preoperative Physical Evaluation

1. Blood pressure and pulse
2. Oxygen saturation and respiration. Able to breathe deeply and cough.
3. Mallampati airway classification and neck flexibility
4. Appearance and skin color
5. Alertness
6. Exercise tolerance and ambulation



Class 1; soft palate, fauces, uvula, pillars
Class 2; soft palate, fauces, portion of uvula
Class 3; soft palate, base of uvula
Class 4; hard palate only

Figure 55-3 Mallampati classification. *(Adapted from Mallampati SR, Gatt SP, Gugino LD, et al:* Can Anaesth Soc J *32:429–434, 1985.)*

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An explanation of IV conscious sedation, its purpose, benefits, possible risks, and complications as well as alternative methods of anesthesia has been discussed with you at your consultation, and we obtained your verbal consent to undergo treatment planned for you. Please read this document, which repeats issues we discussed, and provide the appropriate signatures. Please ask us to clarify anything that you do not understand.

PRESEDATION INFORMATION

1. The premedication you will receive by vein is NOT sodium pentothal. Its purpose is not to put you to sleep but to relax and sedate you. In addition, local anesthetics will be administered in your mouth.
2. You must have a responsible adult bring you to the office and drive you home, and you must remain in the company of a responsible adult until you are fully alert. Make arrangements for the person driving you home to come to the waiting room.
3. You should not have any solid food for 6 hours before your appointment. You can have clear liquids up to 2 hours before your appointment. Examples of clear liquids are apple juice, lemonade, Jell-O, or decaffeinated coffee without cream or cream substitutes.
4. Please wear short sleeves or sleeves that may be raised above the elbows.
5. No alcoholic beverages for 12 hours before sedation.
6. Possible risks and side effects: I have been informed and understand that occasionally there are complications associated with IV conscious sedation including, but not limited to, pain, phlebitis (inflammation of the vein), infection, swelling, bleeding, numbness, discoloration, nausea, vomiting, allergic reaction, depressed breathing, and, in extremely rare instances, intraarterial injection with damage to the part of the body supplied by the artery.
7. I have read and understand the presedation instructions and have also read the enclosed postsedation instructions.

Patient's Signature Date

Figure 55-4 Consent form for and explanation of intravenous conscious sedation.

BOX 55-2 Mild Sedation: Suggested Protocol for the Use of Adult Oral Sedative Premedication for Anxious or Fearful Dental Patients

1. The dentist needs to determine the extent of dental treatment, evaluate the patient's medical history, research potential drug interactions, consult with the patient's physician if appropriate, and obtain informed consent.
2. The patient must have a responsible adult companion for travel to and from the dental office. The patient must be escorted by this companion to and from the parking lot to prevent the patient from stumbling.
3. Patients take the prescribed medication according to directions and are instructed to have a light meal such as toast and beverage without caffeine.
4. Patients who have received oral sedatives are monitored visually and never left alone.
5. After the treatment is complete, postoperative directions are given to both the patient and responsible adult, and the patient is released into the care of his or her companion for travel home. The companion is informed that the patient may have psychomotor and cognitive impairment for the rest of the day.

From Merin RL: *J Calif Dent Assoc* 34:959–968, 2006.

BOX 55-3 Mild Sedation: Example of Suggested Patient Pretreatment Instructions

1. The sedative _____(name of medication) is being prescribed to help reduce your anxiety before and during a dental procedure.
2. The medication may make you sleepy and impair your thinking and coordination. You must have a responsible adult companion for travel to and from the dental office.
3. You must be escorted by this companion to and from the parking lot to prevent you from stumbling.
4. You should take the prescribed medication according to directions. You can have a light meal (no fat) such as toast without butter or margarine and beverage without caffeine. No grapefruit juice.
5. After the treatment is complete, a responsible adult must escort you out of the office and take you home. The sedative effects may linger for the rest of the day so you should not plan to do anything and it is advisable to have a responsible adult stay until you are able to take care of yourself.

From Merin RL: *J Calif Dent Assoc* 34:959–968, 2006.

appropriate scavenging system must be available if gases other than oxygen or air are used.

When inhalation equipment is used, it must have a fail-safe system that is appropriately checked and calibrated. The equipment must also have either (1) a functioning device that prohibits the delivery of less than 30% oxygen or (2) an appropriately calibrated and functioning in-line oxygen analyzer with audible alarm.

For moderate sedation, the equipment necessary to establish IV access must be available. This includes a catheter or butterfly needle, an IV drip line, a solution bag (saline or dextrose), tourniquet, and appropriate antiseptic/dermal disinfectant (Figure 55-5).

Monitoring and Documentation

Monitoring. The dentist or an appropriately trained individual, directed by the dentist, must remain in the operatory during active dental treatment with sedation to monitor the patient continuously until he/she meets the criteria for discharge (Box 55-4). The appropriately trained individual must be familiar with monitoring techniques and equipment.

In the case of moderate sedation, a qualified dentist administering moderate sedation must remain in the operatory room to monitor the patient continuously until the patient meets the criteria for recovery. When active treatment concludes and the patient

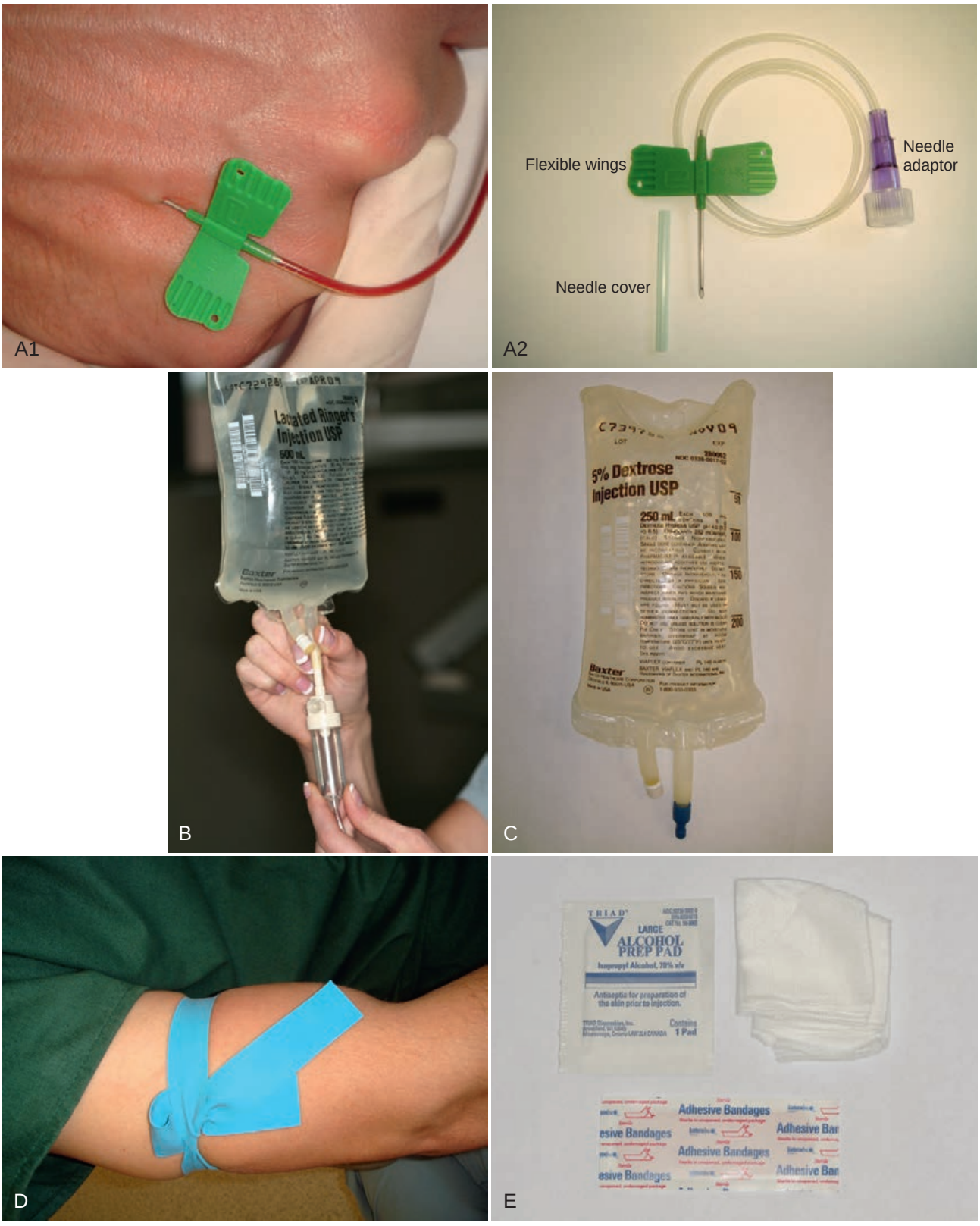

Figure 55-5 Equipment and supplies needed for the administration of moderate intravenous (IV) sedation includes catheter or butterfly needle **(A),** an IV drip line **(B),** a solution bag–saline or dextrose **(C),** tourniquet **(D),** and appropriate antiseptic/dermal disinfectant **(E).** *(From Malamed SF:* Sedation: a guide to patient management, ed 5, *St. Louis, 2010, Elsevier.)*

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BOX 55-4 Discharge Criteria

Alertness: Able to Answer Three Simple Questions Such as the Following:
Who is driving you home?
Where are you?
What day is it?

Breathing
Normal oxygen saturation on room air
Able to breathe deeply and cough

Circulation
Systolic blood pressure within 20% of baseline

Ambulation
Able to walk with minimal assistance

Color
Normal skin color and appearance

TABLE 55-3 Equipment Required For Mild or Moderate Sedation

Mild or Moderate Sedation	Moderate Sedation
• Sphygmomanometer. • Positive pressure oxygen delivery system. • Suction equipment. • When inhalation equipment is used, it must have a fail-safe system and scavenging system.	• Pulse oximeter. • Equipment necessary to establish intravenous access. • Reversal agents for drugs used.

TABLE 55-4 The Observers Assessment of Alertness/Sedation Scale

Category	Observation	Score
Responsiveness	Responds readily to name spoken in normal tone.	5
	Lethargic response to name spoken in normal tone.	4
	Responds only after name is called loudly and/or repeatedly.	3
	Responds only after mild prodding or shaking.	2
	Does not respond to mild prodding or shaking.	1
Speech	Normal.	5
	Mild slowing or thickening.	4
	Slurring or prominent slowing.	3
	Few recognizable words.	2
Facial expression	Normal.	5
	Mild relaxation.	4
	Marked relaxation (slack jaw)	3
Eyes	Clear, no ptosis.	5
	Glazed or mild ptosis (less than half of the eye).	4
	Glazed and marked ptosis (half of the eye or more).	3

Adapted from Chernik DA, Gillings D, Laine H, et al: *J Clin Psychopharmacol* 10:244–251, 1990.

TABLE 55-5 Pulse Oximeter Reading and $Pa0_2$ Tissue Oxygenation

Oximeter $Sp0_2$	Arterial Pressure $Pa0_2$	Interpretation
95%–99%	80–100 mm Hg	Normal oxygenation
90%	60 mm Hg	Alarm goes off; patient hypoxemia
80%	45–50 mm Hg	Severe hypoxemia

recovers to a minimally sedated level, a qualified auxiliary may be directed by the dentist to remain with the patient and continue to monitor him or her as explained in the guidelines until discharged from the facility. The dentist must not leave the facility until the patient meets the criteria for discharge and is discharged to go home with a responsible adult (see Box 55-4).

Monitoring equipment must include a sphygmomanometer, positive pressure oxygen delivery system, suction, and if inhalation sedation is used, a fail-safe and scavenging system. In the case of moderate sedation, a pulse oximeter, equipment for IV access, and reversal agents for drugs used must also be available (Table 55-3).

In the case of moderate sedation, the level of consciousness (e.g., responsiveness to verbal command) must be continually assessed (Table 55-4).

The color of the mucosa, skin, or blood must be evaluated continuously to assess oxygenation. Oxygen saturation by pulse oximetry is clinically useful and should be considered for minimal sedation. For moderate sedation, oxygen saturation must be continuously evaluated by pulse oximetry. Approximately 98% to 99% of the total oxygen content in arterial blood is bound to hemoglobin within red blood cells. The pulse oximeter measures the degree hemoglobin is saturated with oxygen ($Sp0_2$). The remaining 1% to 2% of oxygen is dissolved in plasma and produces a gas pressure referred to as arterial oxygen tension ($Pa0_2$). The $Pa0_2$ level is what determines how much oxygen is entering the body tissues and is referred to as oxygenation. Normally, the $Sp0_2$ level is at 98% to 99% and sustains a $Pa0_2$ level of about 95%. Normal oxygenation is defined as a $Pa0_2$ of 80 to 100 mm Hg. There is a nonlinear relationship between the $Sp0_2$ and the $Pa0_2$. $Sp0_2$ readings of 95% and higher maintain $Pa0_2$ at or above 80 mm Hg and prevent hypoxemia (Table 55-5).

The dentist and/or appropriately trained individual must monitor respirations continually. This can be accomplished by auscultation of breath sounds with a precordial stethoscope, monitoring end-tidal carbon dioxide (CO_2) or by verbal communication with the patient.

Blood pressure and heart rate should be evaluated preoperatively, postoperatively, and intraoperatively as necessary in cases of minimal sedation. For moderate sedation, the dentist must continually evaluate blood pressure and heart rate. Continuous electrocardiograph (ECG) monitoring of patients with significant cardiovascular disease should be considered.

Documentation. An appropriate, time-oriented anesthetic record must be maintained with the names and dosages of all drugs administered (including local anesthetics) and monitored

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Patient's Name: S.S. #: Age: DATE ____/____/____

Medical Hx: CVS
Respiratory System
CNS
Liver
Kidneys
Other

Current Medications:

Allergy:

Base Line Vital Signs:
Date of V.S.:
B.P.: P.R.:
R.: T.:
Ht.: Wt.:
Age:

ASA: I, II, III, IV

Reason for Sedation:
Evaluator:
Name of Driver:

IV started at ____ a.m./p.m.
Venipuncture Site
Type of Needle
IV d/c'd at ____ a.m./p.m.
IV solution & volume ____ ml

DRUGS ADMINISTERED – SUMMARY –

DRUGS DISCARDED – SUMMARY –

DENTISTRY TREATMENT

Start ____ Finish ____
Name of person discharged to:
Post-Op Medications (if any)

COMMENTS

	PREOPERATIVE *time*	INTRAOPERATIVE *time*	*time*	*time*	*time*	*time*	POSTOP *time*	DISCHARGE *time*
Blood Pressure								
Heart Rate								
Respirations								
O_2 LPM								
N_2O LPM								
List all drugs and route of administration (IV, IM) (mg)								
(mg)								
(mg)								
(mg)								
(mg)								
(mg)								
(mg)								

Additional Monitoring: precordial stethoscope ____ pulse oximeter ____
(check as appropriate) ECG ____ automatic blood pressure ____

Student Doctor: ____ AMED Faculty: ____ ☐ Informed Consent
IV Student: ____ Assistants: ____ ☐ Postoperative Instructions

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Figure 55-6 Moderate sedation record sample. *(From Malamed SF:* Sedation: a guide to patient management, ed 5, *St. Louis, 2010, Elsevier.)*

physiologic parameters, including blood pressure, pulse, and respiratory rate (Figure 55-6). In cases of moderate sedation, oxygen saturation must be monitored and recorded continuously (e.g., pulse oximetry).

Recovery and Discharge. Oxygen and suction equipment must be immediately available in the treatment room and the recovery room (if a separate recovery area is utilized). The qualified dentist or appropriately trained clinical staff must continually monitor the patients blood pressure, heart rate, and respirations. In cases of moderate sedation, oxygen saturation and level of consciousness must be evaluated continuously. The qualified dentist must determine and document that the level of consciousness, oxygenation, ventilation, and circulation are satisfactory before discharge (see Box 55-4).

Postoperative verbal and written instruction must be given to the patient and a responsible adult (e.g., parent, escort, guardian, or caregiver).

If a reversal agent is administered before discharge criteria have been met, the patient must be monitored until recovery is assured. A potential problem when using reversal agents is the possibility that the duration of action of the reversal agent may be shorter than the sedative agent used, and the patient may become sedated again. It is critical for the clinician to understand and appreciate the duration of action of all sedative and reversal agents used.

For more information on mild sedation/anxiolysis including guidelines, please visit the companion Expert CONSULT website at www.expertconsult.com.

To view sample prescriptions for zaleplon, triazolam, and lorazepam, please visit the companion Expert CONSULT website at www.expertconsult.com.

When using a very short-acting agent, such as zaleplon, there is a risk that recovery of psychomotor functions may take longer than 1 hour after the onset of action despite the reported duration of action. Also, when using triazolam with anxiolytic doses (generally higher than the hypnotic dose), there is a risk that full recovery to a normal state of consciousness may take longer than 4 hours. Ganzberg found that 28.5% of the zaleplon-sedated patients and 78.5% of the triazolam-sedated patients felt that the sedation effects lingered for the rest of the day.[26] Consequently, zaleplon and triazolam patients must be accompanied by a responsible adult and not resume normal activities for the remainder of the day. The opposite problem can occur when using short-acting sedative agents. Namely, the sedation effects of the short-acting agent may wear off quickly. For this reason, the dentist must prevent delays or interruptions so that treatment can be completed in a timely

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SCIENCE TRANSFER

As periodontal procedures become more complex, there is an increased need for the use of conscious sedation techniques for patients with fear and anxiety. The American Dental Association (ADA) has guidelines and policy outlined in documents that need to be followed. The use of mild sedation with nitrous oxide inhalation techniques must be done with appropriate patient monitoring; the doctor should have a minimum of 14 hours instruction including a clinical component. After appropriate recovery, patients can generally be allowed to function normally and do not need another adult to manage them. Mild sedation using oral agents, such as triazolam in one dose, should not exceed the maximum recommended dose (MRD) for home use, but these patients, when recovered, cannot drive and need another adult to supervise them. Medium-to-deep sedation should only be performed by practitioners with appropriate postdoctoral training in an Accredited Advanced Education Program and carries with it additional responsibilities for patient monitoring, restrictions on food and liquid intake, recovery, and additional trained support staff.

Patients with systemic problems should get preoperative evaluations from a physician, and elderly patients frequently need downward adjustments of normal dosage. Children require special care from trained doctors, and preoperative sedation in children younger than 12 years of age should be carried out by specialists in pediatric anesthesia. Patients with significant systemic problems are best treated in a hospital environment rather than in an outpatient surgical center.

manner. If procedures are expected to be longer than 2 hours, it may be best to use a longer acting agent such as lorazepam.

For more information on inhalation sedation; induction, treatment, and recovery phases; and moderate (conscious) sedation, please visit the companion Expert CONSULT website at www.expertconsult.com.

SEDATION FAILURES

When performing outpatient mild or moderate sedation, the clinician must realize that sedation success will not be 100% effective for all patients. A certain percentage of patients will not respond appropriately to minimal and moderate sedation protocols (Table 55-9). If a patient is not responding to the sedation procedure, it is extremely hazardous to go beyond dose limits or to attempt putting the patient into a deeper level of sedation. It is best to abort the procedure, and reschedule the appointment for another day with a different technique or with a dental anesthesiologist.

TABLE 55-9 Sedation Failures in Dentistry by Route of Administration[50]

Technique	Expected Failure Rate
Oral (child): older	40% to 50%
Oral (child): younger	50% to 65%
Oral (adult)	20% to 50%
Inhalation	15% to 20%
Oral + inhalation	5% to 10%
Intravenous	5%
Oral + intravenous	2% to 3%
Deep sedation/general anesthesia	1.6%[52]

Data from Malamed SF: *Alpha Omegan* 99:70, 2006.

EMERGENCY PREPAREDNESS

Because sedation and general anesthesia are a continuum of drug-induced depression of consciousness to loss of consciousness, it is not always possible to predict how an individual patient will respond. Thus practitioners intending to produce a given level of sedation should be able to diagnose and manage the physiologic consequences (rescue) for patients whose level of sedation becomes deeper than initially intended. For all levels of sedation, the practitioner must have the training, skills, drugs, and equipment to identify and manage such an occurrence until either assistance arrives (emergency medical service) or the patient returns to the intended level of sedation without airway or cardiovascular complications. The qualified dentist is responsible for the sedation management, the adequacy of the facility, the competence of the staff, the diagnosis and treatment of emergencies related to the administration of sedation, and for providing and maintaining the equipment and protocols for patient rescue.

If a patient enters a deeper level of sedation than the dentist is qualified to provide, the dentist must stop the dental procedure and focus his or her attention on managing the patient until the patient's condition returns to the intended level of sedation. This could involve simply monitoring the patient, airway management and support, reversal agents, or activating the emergency medical services.

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 56

Gingival Surgical Techniques

Henry H. Takei, Fermin A. Carranza, and Kitetsu Shin

CHAPTER OUTLINE

GINGIVAL CURETTAGE
- Rationale
- Indications
- Procedure
- Healing after Scaling and Curettage
- Clinical Appearance after Scaling and Curettage

GINGIVECTOMY
- Indications and Contraindications
- Surgical Gingivectomy
- Gingivoplasty
- Healing after Surgical Gingivectomy
- Gingivectomy by Electrosurgery **(online only)**
- Laser Gingivectomy **(online only)**
- Gingivectomy by Chemosurgery **(online only)**

SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Periodontal pocket reduction surgery limited to the gingival tissues only and not involving the underlying osseous structures, without the use of flap surgery, can be classified as **gingival curettage** and **gingivectomy.** Current understanding of disease etiology and therapy limits the use of both techniques, but their place in surgical therapy is essential.

GINGIVAL CURETTAGE

The word *curettage* is used in periodontics to mean the scraping of the gingival wall of a periodontal pocket to remove diseased soft tissue. *Scaling* refers to the removal of deposits from the root surface, whereas *planing* means smoothing the root to remove infected and necrotic tooth substance. Scaling and root planing may inadvertently include various degrees of curettage. However, they are different procedures with different rationales and indications. Both should be considered separate parts of periodontal treatment.

Curettage in periodontics has been defined as gingival and subgingival curettage (Figure 56-1). *Gingival curettage* consists of the removal of the inflamed soft tissue lateral to the pocket wall and the junctional epithelium. *Subgingival curettage* refers to the procedure that is performed apical to the junctional epithelium and severing the connective tissue attachment down to the osseous crest.

It should also be understood that some degree of curettage is accomplished unintentionally during scaling and root planing and is referred to *inadvertent curettage.* This chapter refers to the intentional curettage performed during the same visit as scaling and root planing, or as a separate procedure to reduce pocket depth by enhancing gingival shrinkage, new connective tissue attachment, or both.

Rationale

Curettage accomplishes the removal of the chronically inflamed granulation tissue that forms in the lateral wall of the periodontal pocket. This tissue, in addition to the usual components of granulation tissues (fibroblastic and angioblastic proliferation), contains areas of chronic inflammation and may also have pieces of dislodged calculus and bacterial colonies. The latter may perpetuate the pathologic features of the tissue and hinder healing.

This inflamed granulation tissue is lined by epithelium, and deep strands of epithelium penetrate into the tissue. The presence of this epithelium is construed as a barrier to the attachment of new fibers in the area.

When the root is thoroughly planed, the major source of bacteria disappears, and the pathologic changes in the tissues adjacent to the pocket resolve with no need to eliminate the inflamed granulation tissue by curettage. The existing granulation tissue is slowly resorbed and the bacteria present in the tissue without replenishment of their numbers from the plaque in the pocket are

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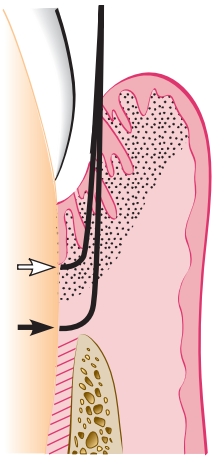

Figure 56-1 Extent of gingival curettage *(white arrow)* and subgingival curettage *(black arrow)*.

destroyed by the defense mechanisms of the host. *Therefore the need for curettage to eliminate the inflamed granulation tissue appears questionable.** It has been shown that scaling and root planing with additional curettage do not improve the condition of the periodontal tissues beyond the improvement resulting from scaling and root planing alone.

Curettage may also eliminate all or most of the epithelium that lines the pocket wall and the underlying junctional epithelium. Curettage for this purpose is still valid, particularly when an attempt is made for new attachment, as occurs in intrabony pockets. However, opinions differ regarding whether scaling and curettage consistently remove the pocket lining and the junctional epithelium. Some investigators report that scaling and root planing tear the epithelial lining of the pocket without removing either it or the junctional epithelium.[33] Others claim that both epithelial structures[6,7,32] and sometimes the underlying inflamed connective tissue[34] are removed by curettage. Some investigators have reported that the removal of the pocket lining and junctional epithelium by curettage is not complete.[51,54,57]

Curettage and Esthetics. The awareness of esthetics in periodontal therapy has become an integral part of care in the modern practice of periodontics. In the past, pocket elimination was the primary goal of therapy and little regard was given to the esthetic result. Rapid shrinkage of the gingival tissue was the goal to eliminate the pocket. Currently, esthetics is a major consideration of therapy, especially in the maxillary anterior area (teeth #6 to 11), and every effort is made to minimize gingival tissue shrinkage and the preservation of the interdental papilla.

A compromise therapy is feasible in the anterior maxilla; this therapy consists of thorough subgingival root planing while attempting to not detach the connective tissue attachment beneath the junctional epithelium. Gingival curettage should be avoided. The granulation tissue in the lateral wall of the pocket, in an environment free of plaque and calculus, becomes connective tissue, thereby minimizing gingival shrinkage. Thus, although complete pocket elimination is not accomplished, the inflammatory changes are reduced or eliminated and the interdental papilla and the esthetic appearance of the area are preserved.

There are many instances in which a surgical flap is necessary for access to the root surface for scaling and root planing. A surgical technique specially designed to minimize gingival recession and preserve the interdental papilla is the *papilla preservation technique* (see Chapter 57).

Another important precaution is to avoid root planing apical to the base of the pocket to the osseous crest The removal of the junctional epithelium and disruption of the connective tissue attachment exposes the nondiseased portion of the cementum. Root planing and the removal of the nondiseased cementum may result in excessive shrinkage of the gingiva, which results in increased gingival recession.

Indications

Indications for curettage are very limited. It can be used after scaling and root planing for the following purposes:

1. Curettage can be performed as part of new attachment attempts in moderately deep intrabony pockets located in accessible areas in which a nonflap type of "closed" surgery is indicated.
2. Curettage can be attempted as a nondefinitive procedure to reduce inflammation when aggressive surgical techniques (e.g., flaps) are contraindicated in patients because of their age, systemic problems, psychologic problems, or other factors. It should be understood that in these patients, the goal of pocket elimination is compromised and prognosis is impaired. The clinician should attempt this approach only when the indicated surgical techniques cannot be performed and both the clinician and the patient have a clear understanding of its limitations.
3. Curettage is also frequently performed on recall visits[45] as a method of maintenance treatment for areas of recurrent inflammation and pocket depth, especially where pocket reduction surgery has previously been performed. Careful probing should establish the extent of the required root planing and curettage.

Procedure

Basic Technique. Curettage does not eliminate the causes of inflammation (i.e., bacterial plaque and deposits). *Therefore curettage should always be preceded by scaling and root planing,* which is the basic periodontal therapy procedure (see Chapter 45). The use of local infiltrative anesthesia for scaling and root planing is optional. However, gingival curettage will always require some type of local anesthesia.

The curette is selected so that the cutting edge is against the tissue (e.g., Gracey #13-14 for mesial surfaces, Gracey #11-12 for distal surfaces). Curettage can also be performed with a 4R-4L Columbia Universal curette. The instrument is inserted to engage the inner lining of the pocket wall and is carried along the soft tissue, usually in a horizontal stroke (Figure 56-2). The pocket wall may be supported by gentle finger pressure on the external surface. The curette is then placed under the cut edge of the junctional epithelium to undermine it.

In subgingival curettage, the tissues attached between the bottom of the pocket and the alveolar crest are removed with a scooping motion of the curette to the tooth surface (Figure 56-3). The area is flushed to remove debris, and the tissue is partly adapted to the tooth by gentle finger pressure. In some cases, suturing of

*This should not be confused with elimination of granulation tissue during flap surgery. The reason for the latter is to remove the bleeding tissue that obstructs the view and prevents the necessary examination of the root surface and the bone morphology. Thus removal of granulation tissue during surgery is accomplished for technical rather than biologic reasons.

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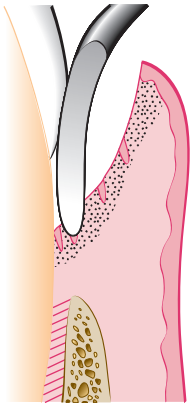

Figure 56-2 Gingival curettage performed with a horizontal stroke of the curette.

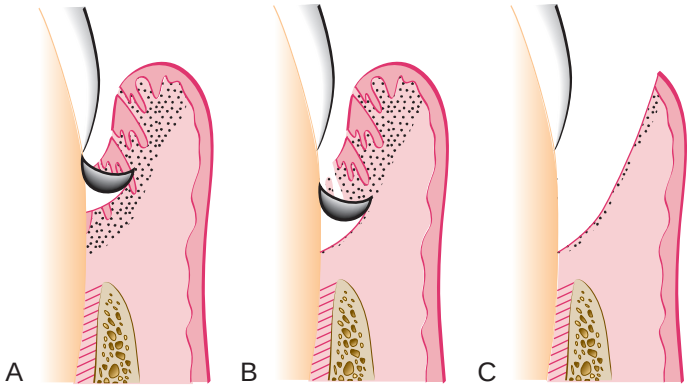

Figure 56-3 Subgingival curettage. **A,** Elimination of pocket lining. **B,** Elimination of junctional epithelium and granulation tissue. **C,** Procedure completed.

separated papillae and application of a periodontal pack may be indicated.

Other Techniques. Other techniques for gingival curettage include the excisional new attachment procedure, ultrasonic curettage, and the use of caustic drugs:

Excisional New Attachment Procedure. The excisional new attachment procedure (ENAP) has been developed and used by the United States (US) Naval Dental Corps.[40,62,63] It is a definitive subgingival curettage procedure performed with a knife. The ENAP technique is as follows:

1. After adequate anesthesia, make an internal bevel incision from the margin of the free gingiva apically to a point below the bottom of the pocket (Figure 56-4). Carry the incision interproximally on both the facial and the lingual side, attempting to retain as much interproximal tissue as possible. The intention is to cut the inner portion of the soft tissue wall of the pocket, all around the tooth.
2. Remove the excised tissue with a curette, and carefully perform root planing on all exposed cementum to achieve a smooth, hard consistency. Preserve all connective tissue fibers that remain attached to the root surface.
3. Approximate the wound edges; if they do not meet passively, recontour the bone until good adaptation of the wound edges is achieved. Place sutures and a periodontal dressing.

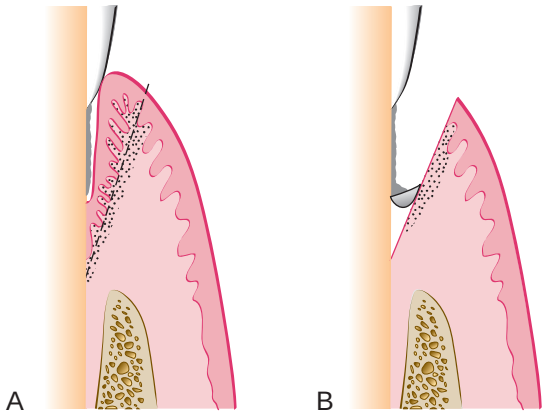

Figure 56-4 Excisional new attachment procedure. **A,** Internal bevel incision to point below bottom of pocket. **B,** After excision of tissue, scaling and root planing are performed.

Ultrasonic Curettage. The use of ultrasonic devices has been recommended for gingival curettage.[35] When applied to the gingiva of experimental animals, ultrasonic vibrations disrupt tissue continuity, lift off the epithelium, dismember collagen bundles, and alter the morphologic features of fibroblast nuclei.[20] Ultrasound is effective for debriding the epithelial lining of periodontal pockets. This results in a narrow band of necrotic tissue (microcauterization), which strips off the inner lining of the pocket.

The Morse scaler-shaped and rod-shaped ultrasonic instruments are used for this purpose. Some investigators found ultrasonic instruments to be as effective as manual instruments for curettage[35,50,64] but resulted in less inflammation and less removal of underlying connective tissue. The gingiva can be made more rigid for ultrasonic curettage by injecting anesthetic solution directly into it.[10]

Caustic Drugs. Since early in the development of periodontal procedures,[53,61] the use of caustic drugs has been recommended to induce a chemical curettage of the lateral wall of the pocket or even the selective elimination of the epithelium. Drugs, such as sodium sulfide, alkaline sodium hypochlorite solution (Antiformin),[8,24,26] and phenol,[4,9] have been proposed and then discarded after studies indicated their ineffectiveness.[5,18,26] The extent of tissue destruction with these drugs cannot be controlled, and they may increase rather than reduce the amount of tissue to be removed by enzymes and phagocytes.

Healing after Scaling and Curettage

Immediately after curettage, a blood clot fills the pocket area, which is totally or partially devoid of epithelial lining. Hemorrhage is also present in the tissues with dilated capillaries and abundant polymorphonuclear leukocytes (PMNs), which appear on the wound surface. This is followed by a rapid proliferation of granulation tissue with a decrease in the number of small blood vessels as the tissue matures.

Restoration and epithelialization of the sulcus generally require 2 to 7 days,[27,34,37,57] and restoration of the junctional epithelium occurs in animals as early as 5 days after treatment. Immature collagen fibers appear within 21 days. Healthy gingival fibers inadvertently severed from the tooth and tears in the epithelium[33,46] are

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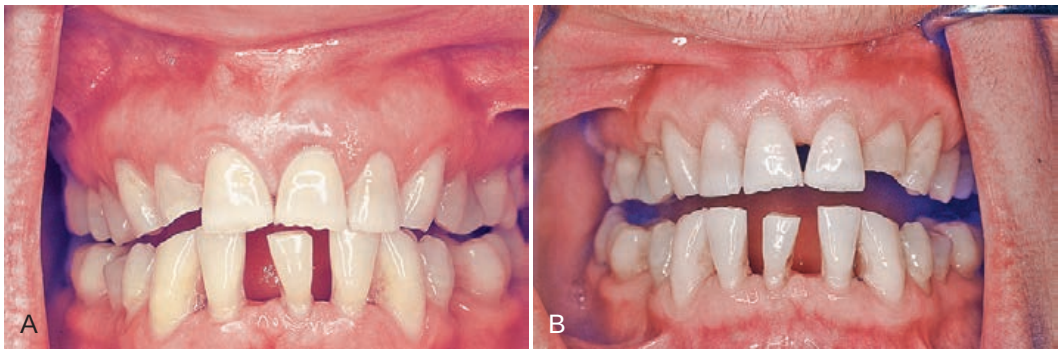

Figure 56-5 Results obtained by treating a suprabony pocket with gingivectomy. **A,** Before treatment. **B,** After treatment.

repaired in the healing process. Several investigators have reported that in monkeys[11,62] and humans[58] treated by scaling and curettage, healing results in the formation of a long, thin junctional epithelium with no new connective tissue attachment. In some cases, this long epithelium is interrupted by "windows" of connective tissue attachment.[11]

Clinical Appearance after Scaling and Curettage

Immediately after scaling and curettage, the gingiva appears hemorrhagic and bright red. *After 1 week*, the gingiva appears reduced in height because of an apical shift in the position of the gingival margin. The gingiva is also darker red than normal but much less so than on previous days. *After 2 weeks* and with proper oral hygiene, the normal color, consistency, surface texture, and contour of the gingiva are attained and the gingival margin is well adapted to the tooth.

GINGIVECTOMY

Gingivectomy means excision of the gingiva. By removing the pocket wall, gingivectomy provides visibility and accessibility for complete calculus removal and thorough smoothing of the roots (Figure 56-5). This creates a favorable environment for gingival healing and restoration of a physiologic gingival contour.

The gingivectomy technique was widely performed in the past. Improved understanding of healing mechanisms and the development of more sophisticated flap methods have relegated the gingivectomy to a lesser role in the current repertoire of available techniques. However, it remains an effective form of treatment when indicated (see Figure 56-5).

Indications and Contraindications

The gingivectomy technique may be performed for the following indications[16]:

1. Elimination of suprabony pockets, regardless of their depth, if the pocket wall is fibrous and firm.
2. Elimination of gingival enlargements.
3. Elimination of suprabony periodontal abscesses.

Contraindications to gingivectomy include the following:

1. The need for bone surgery or examination of the bone shape and morphology.
2. Situations in which the bottom of the pocket is apical to the mucogingival junction.
3. Esthetic considerations, particularly in the anterior maxilla.

The gingivectomy technique may be performed by means of scalpels, electrodes, lasers, or chemicals. All these techniques are reviewed here, although the surgical method is the only technique recommended.

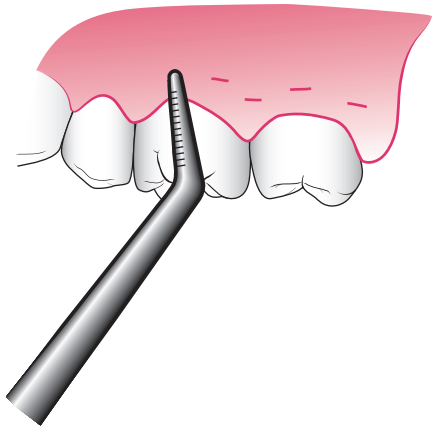

Figure 56-6 Pocket marker makes pinpoint perforations that indicate pocket depth.

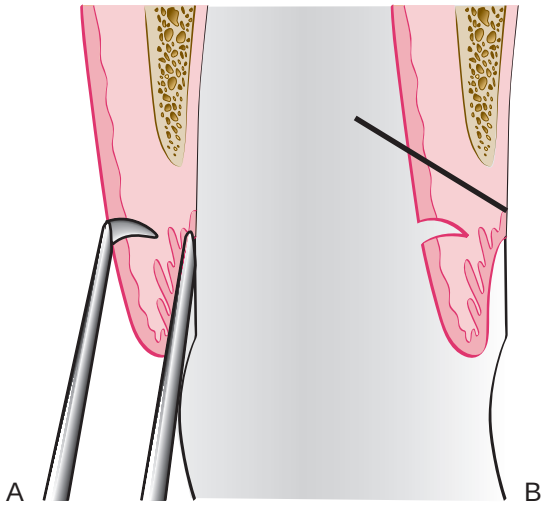

Figure 56-7 Marking the depth of suprabony pocket. **A,** Pocket marker in position. **B,** Beveled incision extends apical to the perforation made by the pocket marker.

Surgical Gingivectomy

Step 1. The pockets on each surface are explored with a periodontal probe and marked with a pocket marker (Figures 56-6 and 56-7). Each pocket is marked in several areas to outline its course on each surface.

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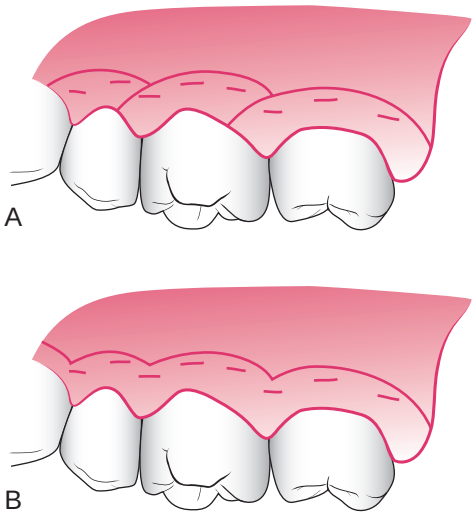

Figure 56-8 **A,** Discontinuous incision apical to bottom of the pocket indicated by pinpoint markings. **B,** Continuous incision begins on the molar and extends anteriorly without interruption.

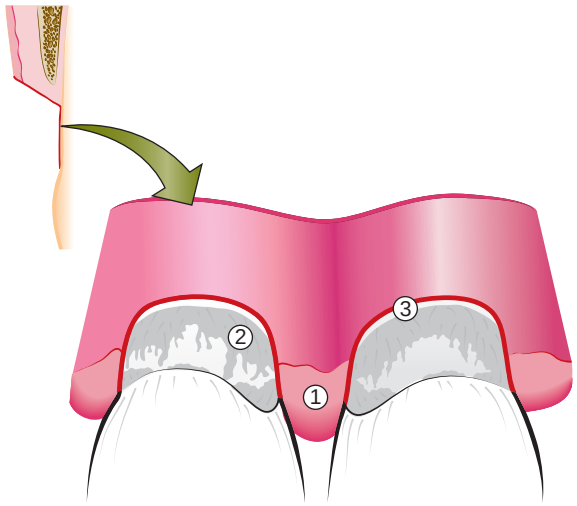

Figure 56-9 Field of operation immediately after removing pocket wall. *1*, Granulation tissue; *2*, calculus and other root deposits; *3*, clear space where junctional epithelium was attached.

Step 2. Periodontal knives (e.g., Kirkland knives) are used for incisions on the facial and lingual surfaces and those distal to the terminal tooth in the arch. Orban periodontal knives are used for interdental incisions. Bard-Parker blades #12 and #15, as well as scissors, are used as auxiliary instruments.

The incision is started apical to the points marking the course of the pockets[48,56] and is directed coronally to a point between the base of the pocket and the crest of the bone. It should be as close as possible to the bone without exposing it, to remove the soft tissue coronal to the bone. Exposure of bone is undesirable. If it occurs, healing usually presents minimal complications if the area is adequately covered by the periodontal pack.

Either interrupted or continuous incisions may be used (Figure 56-8). The incision should be beveled at approximately 45 degrees to the tooth surface and recreate the normal festooned pattern of the gingiva. Failure to bevel the incision will leave a broad, fibrous plateau, which will take a longer time to develop a physiologic contour. In the interim, plaque and calculus may lead to the recurrence of pockets.

Step 3. Remove the excised pocket wall, clean the area, and closely examine the root surface. The most apical zone consists of a light, bandlike zone where the tissues were attached. Coronally, calculus remnants, root caries, or resorption may be found. Granulation tissue may be seen on the excised soft tissue (Figure 56-9).

Step 4. Carefully curette the granulation tissue and remove any remaining calculus and necrotic cementum to leave a smooth and clean surface.

Step 5. Cover the area with a surgical pack (see Chapter 54).

Gingivoplasty

Gingivoplasty is similar to gingivectomy, but its objective is different. Gingivectomy is performed to eliminate periodontal pockets and includes reshaping as part of the technique. Gingivoplasty is a reshaping of the gingiva to create physiologic gingival contours with the sole purpose of *recontouring the gingiva* in the absence of pockets.[19]

Gingival and periodontal disease often produces deformities in the gingiva that is conducive for plaque accumulation and food debris, which prolongs and aggravates the disease process. Such deformities include (1) gingival clefts and craters, (2) craterlike interdental papillae caused by acute necrotizing ulcerative gingivitis, and (3) gingival enlargements.

Gingivoplasty may be accomplished with a periodontal knife, a scalpel, rotary coarse diamond stones,[44] or electrodes.[13] The technique resembles that of festooning of a artificial denture, which consists of tapering the gingival margin, creating a scalloped marginal outline, thinning the attached gingiva , creating vertical interdental grooves, and shaping the interdental papillae.

Healing after Surgical Gingivectomy

The *initial response* after gingivectomy is the formation of a protective surface blood clot. The underlying tissue becomes acutely inflamed with necrosis. The clot is then replaced by granulation tissue. *In 24 hours,* there is an increase in new connective tissue cells, which are mainly angioblasts beneath the surface layer of inflammation and necrotic tissue. *By the third day,* numerous young fibroblasts are located in the area.[47] The highly vascular granulation tissue grows coronally, creating a new free gingival margin and sulcus.[41] Capillaries derived from blood vessels of the periodontal ligament migrate into the granulation tissue and *within 2 weeks,* they connect with the gingival vessels.[59]

After 12 to 24 hours, epithelial cells at the margins of the wound begin to migrate over the granulation tissue, separating it from the contaminated surface layer of the clot. Epithelial activity at the margins reaches a peak in 24 to 36 hours.[13]

The new epithelial cells arise from the basal and deeper spinous layers of the epithelial wound edge and migrate over the wound over a fibrin layer that is later resorbed and replaced by a connective tissue bed.[25] The epithelial cells advance by a tumbling action with the cells becoming fixed to the substrate by hemidesmosomes and a new basement lamina.[23,28]

After 5 to 14 days, surface epithelialization is generally complete. During the first 4 weeks after gingivectomy, keratinization is less than it was before surgery. Complete epithelial repair takes about 1 month.[52] Vasodilation and vascularity begin to decrease after the

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SCIENCE TRANSFER

Gingival curettage procedures involve root planing plus a deliberate curettage of the soft tissues lining the pocket carried out under local anesthesia. These procedures result in improvement of periodontal health but generally are not as effective as periodontal surgical approaches. Therefore these treatments are used in patients in which surgical procedures are contraindicated by the patient's systemic status or patients who will not accept surgical therapy. In the anterior segments, gingival curettage gives a better esthetic outcome than periodontal surgery and may be used even though the pocket depth reduction is compromised. The excisional new attachment procedure (ENAP) removes the soft tissue pocket lining with a scalpel and then sutures the tissue at its original level. Recently, ENAP has been done with a neodymium:yttrium-aluminum-garnet (Nd-YAG) laser, and preliminary reports show evidence of periodontal regeneration.

Gingivectomy is rarely used to treat periodontitis and even patients with extensive gingival hyperplasia are best treated with flap surgery so that the underlying bone defects can be visualized, treated, and then covered with soft tissue. A few patients needing crown lengthening can be treated with gingivectomy when there is no need for osseous surgery to establish an adequate biologic width.

Laser gingivectomy with carbon dioxide (CO_2) laser or Nd-YAG laser gives a more precise tissue contouring than tissue removal with surgical blades and may be another treatment option. Electrosurgical gingivectomies may cause delayed healing, and the surgeon must be careful not to place the electrodes close to the bone margins because sequestration of bone can occur.

fourth day of healing and appear to be almost normal by the sixteenth day.[36] Complete repair of the connective tissue takes about 7 weeks.[52]

The flow of gingival fluid in humans is initially increased after gingivectomy and diminishes as healing progresses.[1,49] Maximal flow is reached after 1 week, coinciding with the time of maximal inflammation.

Although the tissue changes that occur in postgingivectomy healing are the same in all individuals, the time required for complete healing varies considerably, depending on the area of the incised surface and interference from local irritation and infection. In patients with physiologic gingival melanosis, the pigmentation is diminished in the healed gingiva.

For more information on gingivectomy by electrosurgery, laser, and chemosurgery, please visit the companion Expert CONSULT website at www.expertconsult.com.

SUMMARY

The gingivectomy surgical technique has a long history of use in periodontal surgery. This technique has been attempted using scalpels, electrosurgery, laser, and chemical cautery. Even though this technique may have some use for the minimal reduction of redundant gingival tissue, many limiting factors must be considered. Current periodontal surgery must consider the (1) conservation of keratinized gingiva, (2) minimal gingival tissue loss to maintain esthetics, (3) adequate access to the osseous defects for definitive defect correction, and (4) minimal postsurgical discomfort and bleeding by attempting surgical procedures that will allow primary closure. The gingivectomy surgical technique has limited use in current surgical therapy because it does not satisfy these considerations in periodontal therapy. The clinician must carefully evaluate each case as to the proper application of this surgical procedure.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 57

The Periodontal Flap

Henry H. Takei and Fermin A. Carranza

CHAPTER OUTLINE

CLASSIFICATION OF FLAPS
FLAP DESIGN
INCISIONS
Horizontal Incisions
Vertical Incisions
ELEVATION OF THE FLAP
SUTURING TECHNIQUES (online only)
Technique
Ligation
Types of Sutures
HEALING AFTER FLAP SURGERY
SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures and boxes may be out of numeric order in this printed chapter.

A periodontal flap is a section of gingiva and/or mucosa surgically separated from the underlying tissues to provide visibility of and access to the bone and root surface. The flap also allows the gingiva to be displaced to a different location in patients with mucogingival involvement.

CLASSIFICATION OF FLAPS

Periodontal flaps can be classified based on the following:

- Bone exposure after flap reflection
- Placement of the flap after surgery
- Management of the papilla

Based on bone exposure after reflection, the flaps are classified as either full-thickness (mucoperiosteal) or partial-thickness (mucosal) flaps (Figure 57-1).

In *full-thickness flaps,* all the soft tissue, including the periosteum, is reflected to expose the underlying bone. This complete exposure of and access to the underlying bone is indicated when resective osseous surgery is contemplated.

The *partial-thickness flap* includes only the epithelium and a layer of the underlying connective tissue. The bone remains covered by a layer of connective tissue, including the periosteum. This type of flap is also called the *split-thickness flap.* The partial-thickness flap is indicated when the flap is to be positioned apically or when the operator does not want to expose bone.

Conflicting data surround the advisability of uncovering the bone when this is not actually needed. When bone is stripped of its periosteum, a loss of marginal bone occurs, and this loss is prevented when the periosteum is left on the bone.[4] Although usually not clinically significant,[7] the differences may be significant in some cases (Figure 57-2). The partial-thickness flap may be necessary when the crestal bone margin is thin and exposed with an apically placed flap, or when dehiscences or fenestrations are present. The periosteum left on the bone may also be used for suturing the flap when it is displaced apically.

Based on flap placement after surgery, flaps are classified as (1) *nondisplaced flaps,* when the flap is returned and sutured in its original position, or (2) *displaced flaps,* which are placed apically, coronally, or laterally to their original position. Both full-thickness and partial-thickness flaps can be displaced, but to do so, the attached gingiva must be totally separated from the underlying bone, thereby enabling the unattached portion of the gingiva to be movable. However, palatal flaps cannot be displaced because of the absence of unattached gingiva.

Apically displaced flaps have the important advantage of preserving the outer portion of the pocket wall and transforming it into attached gingiva. Therefore these flaps accomplish the double objective of eliminating the pocket and increasing the width of the attached gingiva.

Based on management of the papilla, flaps can be conventional or papilla preservation flaps. In the *conventional flap,* the interdental papilla is split beneath the contact point of the two approximating teeth to allow reflection of the buccal and lingual flaps. The incision is usually scalloped to maintain gingival morphology and retain as much papilla as possible. The conventional flap is used (1) when the interdental spaces are too narrow, thereby precluding the

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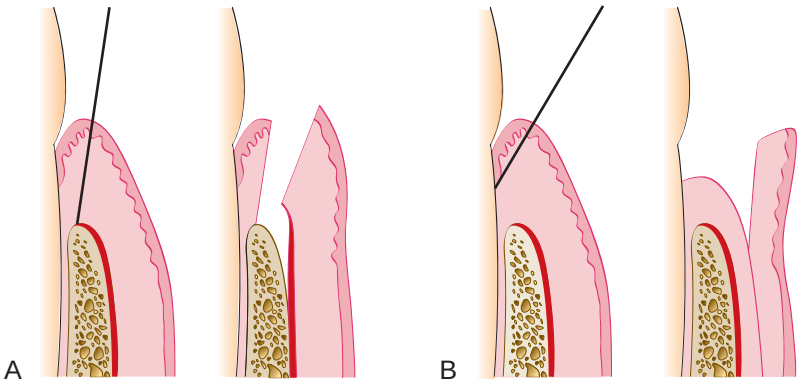

Figure 57-1 **A,** Diagram of the internal bevel incision (first incision) to reflect a full-thickness (mucoperiosteal) flap. Note that the incision ends on the bone to allow for the reflection of the entire flap. **B,** Diagram of the internal bevel incision to reflect a partial-thickness flap. Note that the incision ends on the root surface to preserve the periosteum on the bone.

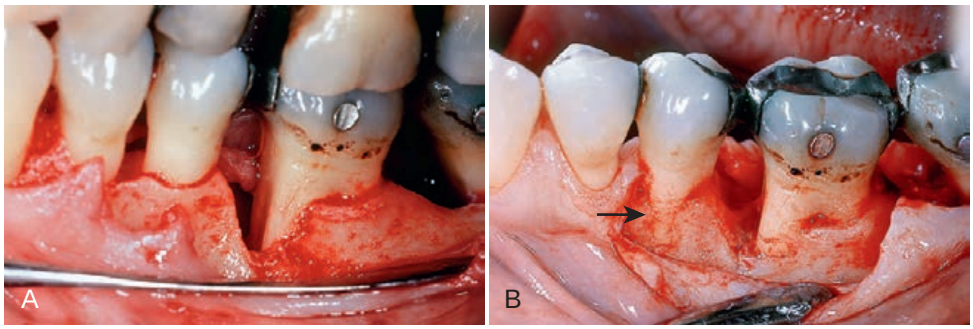

Figure 57-2 Loss of marginal bone as a result of uncovering the osseous crest. **A,** Mucoperiosteal flap elevated as part of a clinical study. **B,** Reentry performed 6 months later reveals loss of marginal bone facial to second premolar *(arrow). (Courtesy Dr. Silvia Oreamuno, San Jose, Costa Rica.)*

possibility of preserving the papilla and (2) when the flap is to be displaced.

Conventional flaps include the modified Widman flap, the undisplaced flap, the apically displaced flap, and the flap for reconstructive procedures. These techniques are described in detail in Chapter 59.

The *papilla preservation flap* incorporates the entire papilla in one of the flaps by means of crevicular interdental incisions to sever the connective tissue attachment and a horizontal incision at the base of the papilla, leaving it connected to one of the flaps.

FLAP DESIGN

The design of the flap is dictated by the surgical judgment of the operator and may depend on the objectives of the procedure. The necessary degree of access to the underlying bone and root surfaces and the final position of the flap must be considered in designing the flap. Preservation of good blood supply to the flap is also an important consideration.

Two basic flap designs are used. Depending on how the interdental papilla is managed, flaps can either split the papilla (conventional flap) or preserve it (papilla preservation flap).

In the conventional flap procedure, the incisions for the facial and the lingual or palatal flap reach the tip of the interdental papilla or its vicinity, thereby splitting the papilla into a facial half and a lingual or palatal half (Figures 57-3 and 57-4).

The entire surgical procedure should be planned in every detail before the procedure is initiated.. This should include the type of flap, exact location and type of incisions, management of the underlying bone, and final closure of the flap and sutures. Although some details may be modified during the actual performance of the procedure, detailed planning allows for a better clinical result.

INCISIONS

Periodontal flaps use horizontal and vertical incisions.

Horizontal Incisions

Horizontal incisions are directed along the margin of the gingiva in a mesial or a distal direction. Two types of horizontal incisions have been recommended: the internal bevel incision,[6] which starts at a distance from the gingival margin and is aimed at the bone crest, and the crevicular incision, which starts at the bottom of the pocket and is directed to the bone margin. In addition, the interdental incision is performed after the flap is elevated.

The *internal bevel incision* is basic to most periodontal flap procedures. It is the incision from which the flap is reflected to expose the underlying bone and root. The internal bevel incision accomplishes three important objectives: (1) it removes the pocket lining; (2) it conserves the relatively uninvolved outer surface of the gingiva, which, if apically positioned, becomes attached gingiva; and (3) it produces a sharp, thin flap margin for adaptation to the bone-tooth junction. This incision has also been termed the *first incision* because it is the initial incision in the reflection of a periodontal flap, and the *reverse bevel incision* because its bevel is in reverse direction from that of the gingivectomy incision. The #15C or the #15 surgical blade is used most often to make this incision. That portion of the gingiva left around the tooth contains the epithelium of the pocket lining and the adjacent granulomatous tissue. It is discarded after the crevicular (second) and interdental (third) incisions are performed (Figure 57-5).

The internal bevel incision starts from a designated area on the gingiva and is directed to an area at or near the crest of the bone (Figure 57-6). The starting point on the gingiva is determined by

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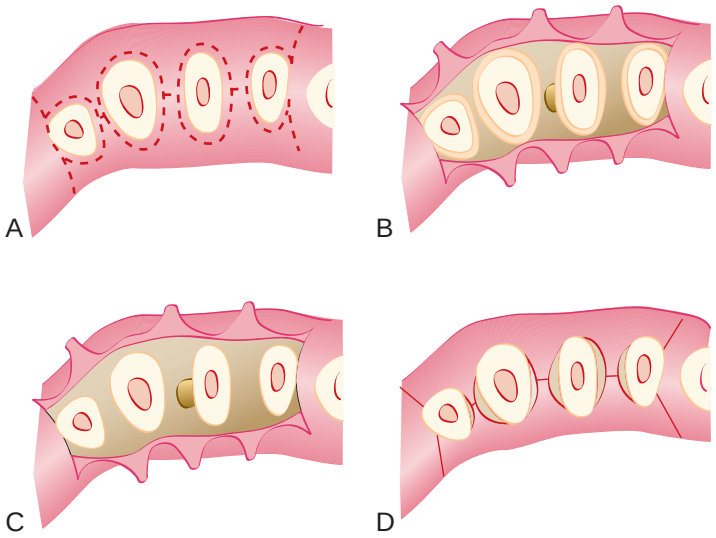

Figure 57-3 Flap design for conventional or traditional flap technique. **A,** Design of incisions: internal bevel incision, splitting the papilla, and vertical incisions are drawn in interrupted lines. **B,** The flap has been elevated, and the wedge of tissue next to the tooth is still in place. **C,** All marginal tissue has been removed, exposing the underlying bone (see defect in one space). **D,** Tissue returned to its original position. Proximal areas are not totally covered.

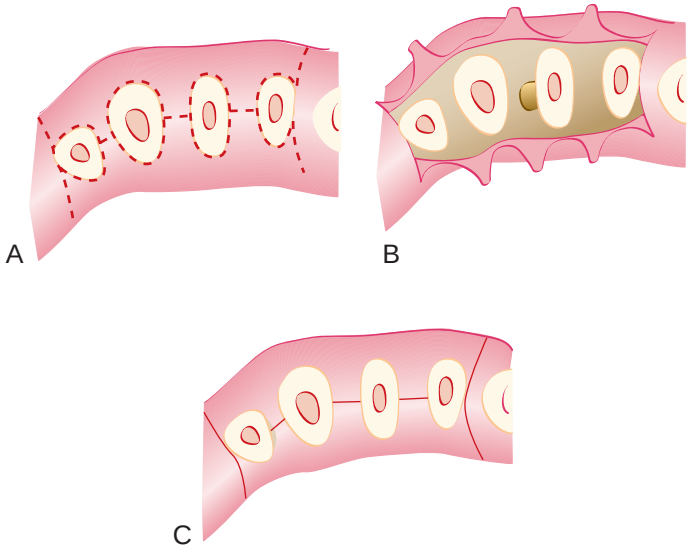

Figure 57-4 Flap design for sulcular incision flap. **A,** Design of incisions: sulcular incisions and vertical incisions are depicted by interrupted lines. **B,** The flap has been elevated, exposing the underlying bone (see defect in one space). **C,** Tissue returned to its original position covers the entire interdental spaces.

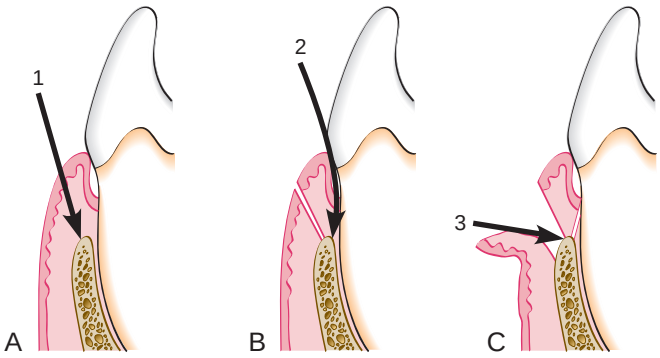

Figure 57-5 Three incisions necessary for flap surgery. **A,** First (internal bevel) incision; **B,** second (crevicular) incision; **C,** third (interdental) incision.

Figure 57-6 Position of knife in performing internal bevel incision.

whether the flap is apically displaced or not displaced (Figure 57-7).

The *crevicular incision*, also termed the *second incision*, is made from the base of the pocket to the crest of the bone (Figure 57-8). This incision, together with the initial reverse bevel incision, forms a V-shaped wedge ending at or near the crest of bone. This wedge of tissue contains most of the inflamed and granulomatous areas that constitute the lateral wall of the pocket, as well as the junctional epithelium and the connective tissue fibers that still persist between the bottom of the pocket and the crest of the bone. The incision is carried around the entire tooth. The beak-shaped #12D blade is usually used for this incision.

A periosteal elevator is inserted into the initial internal bevel incision, and the flap is separated from the bone. The most apical end of the internal bevel incision is exposed and visible. With this access, the surgeon is able to make the *third incision,* or *interdental incision,* to separate the collar of gingiva that is left around the tooth. The Orban knife is usually used for this incision. The incision is made not only around the facial and lingual radicular area but also interdentally, connecting the facial and lingual segments to free the gingiva completely around the tooth (Figure 57-9; see also Figure 57-5).

These three incisions allow the removal of the gingiva around the tooth (i.e., the pocket epithelium and the adjacent granulomatous tissue). A curette or a large scaler (U15/30) can be used for this purpose. After removal of the large pieces of tissue, the remaining connective tissue in the osseous lesion should be carefully

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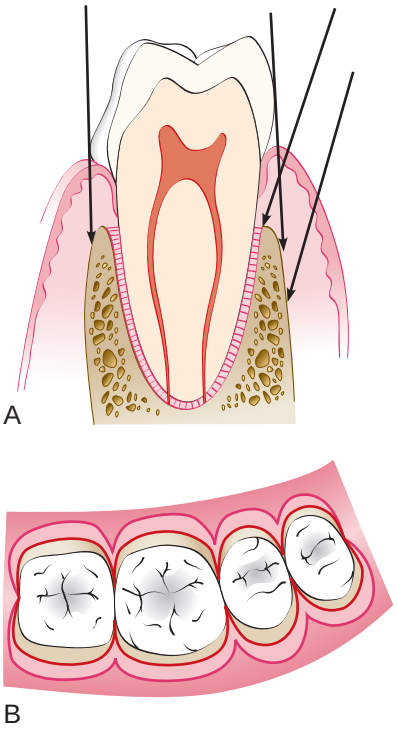

Figure 57-7 **A,** Internal bevel (first) incision can be made at varying locations and angles according to the different anatomic and pocket situations. **B,** Occlusal view of the different locations where the internal bevel incision can be made. Note the scalloped shape of the incisions.

Figure 57-8 Position of knife in performing crevicular (second) incision.

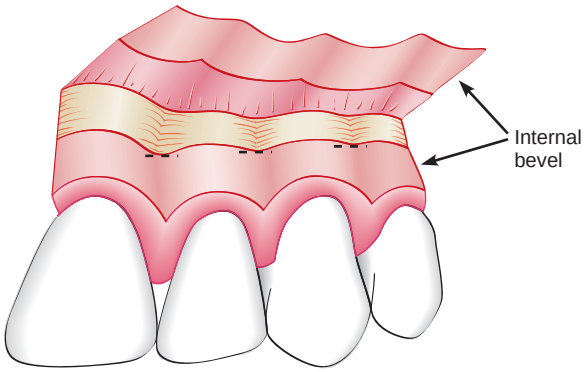

Figure 57-9 After the flap has been elevated, a wedge of tissue remains on the teeth, attached by the base of the papillae. An interdental (third) incision along the horizontal lines seen in the interdental spaces will sever these connections.

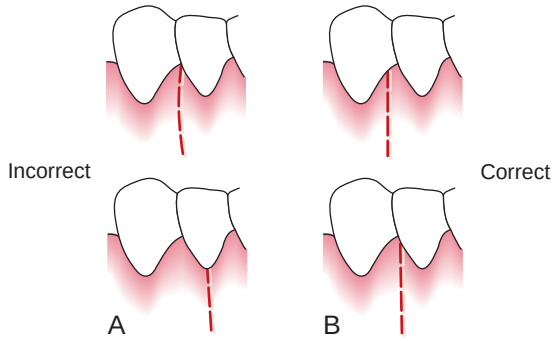

Figure 57-10 Incorrect **(A)** and correct **(B)** locations of a vertical incision. This incision should be made at the line angles to prevent splitting of a papilla or incising directly over a radicular surface.

curetted so that the entire root and the bone surface adjacent to the teeth can be observed.

Flaps can be reflected using only the horizontal incision if sufficient access can be obtained in this way and if apical, lateral, or coronal displacement of the flap is not anticipated. If vertical incisions are not made, the flap is called an *envelope flap.*

Vertical Incisions

Vertical or oblique releasing incisions can be used on one or both ends of the horizontal incision, depending on the design and purpose of the flap. Vertical incisions at both ends are necessary if the flap is to be apically displaced. Vertical incisions must extend beyond the mucogingival line, reaching the alveolar mucosa, to allow for the release of the flap to be displaced (see Chapter 59).

In general, vertical incisions in the lingual and palatal areas are avoided. Facial vertical incisions should not be made in the center of an interdental papilla or over the radicular surface of a tooth. Incisions should be made at the line angles of a tooth either to include the papilla in the flap or to avoid it completely (Figure 57-10). The vertical incision should also be designed to avoid short flaps (mesiodistal) with long, apically directed incisions because this could jeopardize the blood supply to the flap.

Several investigators proposed the *interdental denudation procedure,* which consists of horizontal, internal bevel, nonscalloped incisions to remove the gingival papillae and denude the interdental space.[1,2,11,12] This technique completely eliminates the inflamed interdental tissue. Healing is by secondary intention and results in excellent gingival contour. It is contraindicated when bone grafts are used for the graft material placed interdentally will not be covered.

ELEVATION OF THE FLAP

When a full-thickness flap is desired, reflection of the flap is accomplished by blunt dissection. A periosteal elevator is used to separate the mucoperiosteum from the bone by moving it mesially, distally, and apically until the desired reflection is accomplished (Figure 57-11).

Sharp dissection is necessary to reflect a partial-thickness flap. A surgical scalpel (#15) is used (Figure 57-12).

A combination of full-thickness and partial-thickness flaps may be indicated to obtain the advantages of both. The flap is started as a full-thickness procedure, then a partial-thickness flap is made at the apical portion. In this way the coronal portion of the bone, which may be subject to osseous remodeling, is exposed while the remaining bone is protected by the periosteum.

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Figure 57-11 Elevation of the flap with a periosteal elevator to obtain a full-thickness flap.

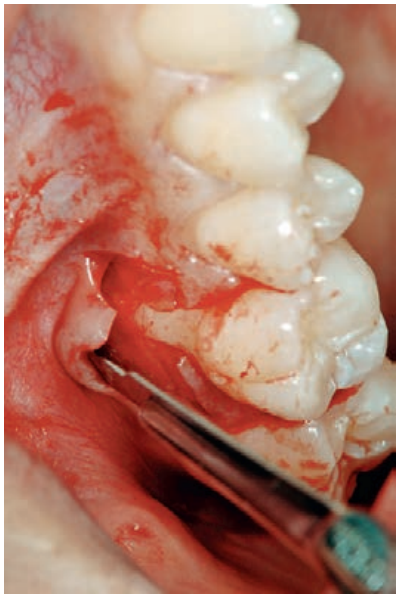

Figure 57-12 Elevation of the flap with a Bard-Parker knife to obtain a split-thickness flap.

For more information on suturing techniques, please visit the companion Expert CONSULT website at www.expertconsult.com.

HEALING AFTER FLAP SURGERY

Immediately after suturing (up to 24 hours), a connection between the flap and the tooth or bone surface is established by a blood clot, which consists of a fibrin reticulum with many polymorphonuclear leukocytes, erythrocytes, debris of injured cells, and capillaries at the edge of the wound.[3] Bacteria and an exudate or transudate also result from tissue injury.

One to 3 days after flap surgery, the space between the flap and the tooth or bone is thinner and epithelial cells migrate over the border of the flap, usually contacting the tooth at this time. When the flap is closely adapted to the alveolar process, there is minimal inflammatory response.[3]

One week after surgery, an epithelial attachment to the root has been established by means of hemidesmosomes and a basal lamina. The blood clot is replaced by granulation tissue derived from the gingival connective tissue, the bone marrow, and the periodontal ligament.

Two weeks after surgery, collagen fibers begin to appear parallel to the tooth surface.[3] Union of the flap to the tooth is still weak because of the presence of immature collagen fibers, although the clinical aspect may be almost normal.

One month after surgery, a fully epithelialized gingival crevice with a well-defined epithelial attachment is present. There is a beginning functional arrangement of the supracrestal fibers.

Full-thickness flaps, which denude the bone, result in a superficial bone necrosis at 1 to 3 days. Osteoclastic resorption follows and reaches a peak at 4 to 6 days, declining thereafter.[13] This results in a loss of bone of about 1 mm^3 and the bone loss is greater if the bone is thin.[14,15]

Osteoplasty (thinning of the buccal bone) using diamond burs, included as part of the surgical technique, results in areas of bone necrosis with reduction in bone height, which is later remodeled by new bone formation. Therefore, the final shape of the crest is determined more by osseous remodeling than by surgical reshaping.[8]

SCIENCE TRANSFER

Periodontal flaps are designed to preserve gingival integrity and to gain access to root surfaces for residual calculus removal and to thoroughly remove granulation tissue so bone defects can be visualized and treated. Gentle and efficient procedures result in optimum healing and minimal postoperative pain. When flaps need to be repositioned apically or less often, coronally, then the flaps must sit passively at the appropriate level before suturing. To ensure this, buccal and lingual flaps need to be elevated beyond the mucogingival junction so the elasticity of the mucosa allows for flap mobility. Sometimes it may be necessary to extend the flap elevation apically with a split incision approach to minimize the effect of the less elastic periosteum. Vertical incisions can aid in flap positioning by allowing the clinician to suture the flap at a different level to the adjacent untreated gingiva.

Palatal flaps are less mobile because of the absence of oral mucosa so that the apical position of the flap depends on how much marginal gingival tissue is discarded using a reverse bevel incision. The more apical positioning desired, the more extensive is the reverse bevel cut. Palatal flaps are more difficult to position coronally than buccal or lingual flaps and thus if it is required to position flaps coronally or even at their original levels, then a sulcular incision is used. In osseous periodontal surgery, flaps are apically positioned to minimize postoperative pocket depth. In regenerative periodontal surgery, soft tissue coverage of bony defects, graft materials, membranes, and biologic agents is important so sulcular incisions and tight suturing techniques are crucial.

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This may not be the case when osseous remodeling does not include excessive thinning of the radicular bone.[9] Bone repair reaches its peak at 3 to 4 weeks.[15]

Loss of bone occurs in the initial healing stages both in radicular bone and in interdental bone areas. However, in interdental areas, which have cancellous bone, the subsequent repair stage results in total restitution without any loss of bone, whereas in radicular bone, particularly if thin and unsupported by cancellous bone, bone repair results in loss of marginal bone.[15]

SUMMARY

The periodontal flap surgery is the most widely utilized surgical procedure to reduce the pocket depth and to access the subgingival root surfaces for scaling and root planing. The diagnosis of the periodontal lesion and the objective of the surgery will dictate the type of flap procedure which will be utilized to obtain the best result. The incisions, type of flap and the selection of suturing design must be planned and executed to fit the problem.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 58

Treatment of Gingival Enlargement

Paulo M. Camargo, Fermin A. Carranza, and Henry H. Takei

CHAPTER OUTLINE

CHRONIC INFLAMMATORY ENLARGEMENT
PERIODONTAL AND GINGIVAL ABSCESSES
DRUG-ASSOCIATED GINGIVAL ENLARGEMENT
Treatment Options
LEUKEMIC GINGIVAL ENLARGEMENT
GINGIVAL ENLARGEMENT IN PREGNANCY
Timing of Treatment and Indications
GINGIVAL ENLARGEMENT IN PUBERTY
RECURRENCE OF GINGIVAL ENLARGEMENT

Treatment of gingival enlargement is based on an understanding of the cause and underlying pathologic changes (see Chapter 9). Gingival enlargements are of special concern to the patient and dentist because they pose problems in plaque control, function (including mastication, tooth eruption, and speech), and esthetics. Because gingival enlargements differ in cause, treatment of each type is best considered individually.

CHRONIC INFLAMMATORY ENLARGEMENT

Chronic inflammatory enlargements are soft and discolored and are caused principally by edema and cellular infiltration. These gingival enlargements are treated by scaling and root planing, provided the size of the enlargement does not interfere with complete removal of deposits from the involved tooth surfaces.

When chronic inflammatory gingival enlargements include a significant fibrotic component that does not undergo shrinkage after scaling and root planing or are of such size that they obscure deposits on the tooth surfaces and interfere with access to them, *surgical removal* is the treatment of choice. Two techniques are available for this purpose: gingivectomy and flap operation.

Selection of the appropriate technique depends on the size of the enlargement and character of the tissue. When the enlarged gingiva remains soft and friable even after scaling and root planing, a *gingivectomy* is used to remove it because a flap requires a firmer tissue to perform the incisions and other steps in the technique. However, if the gingivectomy incision removes all of the attached, keratinized gingiva, which will create a mucogingival problem, then the *flap technique* is indicated.

Tumorlike inflammatory enlargements are treated by gingivectomy as follows:

1. With the patient under local anesthesia, the tooth surfaces beneath the mass are scaled to remove calculus and other debris.
2. The lesion is separated from the mucosa at its base with a #12 Bard-Parker blade. If the lesion extends interproximally, the interdental gingiva is included in the incision to ensure exposure of the root deposits.
3. After the lesion is removed, the involved root surfaces are scaled and planed and the area is cleansed and irrigated with warm water.
4. A periodontal dressing is applied. It is removed after 1 week, at which time the patient is instructed in plaque control techniques.

For the flap operation, see Chapters 57 and 59 and the following discussion of the flap technique for drug-induced enlargements.

PERIODONTAL AND GINGIVAL ABSCESSES

The reader is referred to Chapter 42 for a complete discussion of abscess treatment.

DRUG-ASSOCIATED GINGIVAL ENLARGEMENT

Gingival enlargement has been associated with the administration of three different types of drugs: anticonvulsants, calcium channel blockers, and the immunosuppressant, cyclosporine. Chapter 9 provides a comprehensive review of the clinical and microscopic features and pathogenesis of gingival enlargement induced by these drugs.

Examination of cases of drug-induced gingival enlargement reveals the overgrown tissues to have two components: a *fibrotic* type caused by the drug and an *inflammatory* type induced by bacterial plaque. Although the fibrotic and inflammatory components present in the enlarged gingiva are the result of distinct pathologic processes, they almost always are observed in combination. The role of bacterial plaque in the overall pathogenesis of drug-induced

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gingival enlargement is not clear. Some studies indicate that plaque is a prerequisite for gingival enlargement,[10] whereas others suggest that the presence of plaque is a consequence of its accumulation caused by the enlarged gingiva.

Treatment Options

Treatment of drug-induced gingival enlargement should be based on the medication being used and the clinical features of the case.

First, consideration should be given to the possibility of discontinuing the drug[8,11] or changing the medication. These possibilities should be examined with the patient's physician. Simple discontinuation of the offending drug is usually not practical, but its substitution with another medication might be an option. If any drug substitution is attempted, it is important to allow a 6- to 12-month period to elapse between discontinuation of the offending drug and the possible resolution of gingival enlargement. The decision to implement surgical treatment is made after this time period has elapsed.

Alternative medications to the anticonvulsant phenytoin include carbamazepine[7] and valproic acid, both of which have been reported to have a lesser effect in inducing gingival enlargement.

For patients taking *nifedipine,* which has a reported prevalence of gingival enlargement of up to 44%, other calcium channel blockers, such as diltiazem or verapamil, may be viable alternatives.[22] Their reported prevalence of inducing gingival enlargement is 20% and 4%, respectively.[4,9,15] Also, consideration may be given to the use of another class of antihypertensive medications rather than calcium channel blockers, none of which is known to induce gingival enlargement.

Drug substitutions for *cyclosporine* are more limited. Tacrolimus is another immunosuppressant that has been used on organ transplant recipients. The incidence of gingival enlargement in patients under tacrolimus therapy is approximately 65% lower than in those taking cyclosporine.[2] Clinical trials have also shown that the substitution of cyclosporine by tacrolimus results in a significant decrease in the severity of gingival enlargement when compared to patients who are kept on cyclosporine therapy[24]; in another study,[13] the same drug substitution resulted in a strong decrease or complete resolution of gingival enlargement in over 70% of the patients initially presenting with cyclosporine-induced gingival enlargement.[13] Therefore the dental practitioner should consult with the treating transplantation physician to investigate the possibility of a change in immunosuppressant therapy as one of the steps in the treatment of cyclosporine-induced gingival enlargement.

Administration of the antibiotic azithromycin has been shown to decrease the severity of gingival enlargement induced by administration of cyclosporine. A 3-day course of systemic azithromycin significantly decreased gingival enlargement, and the effect was observed as early as 7 to 30 days after initiation of antibiotic therapy.[23] The effect of azithromycin in decreasing cyclosporine-induced gingival enlargement is significantly greater than that observed with an improvement in oral hygiene.[18] Topical administration of azithromycin in the form of a toothpaste also decreased the severity of cyclosporine-induced gingival enlargement.[2]

Second, the clinician should emphasize plaque control as the first step in the treatment of drug-induced gingival enlargement. Although the exact role played by bacterial plaque is not well understood, evidence suggests that good oral hygiene and frequent professional removal of plaque decrease the degree of gingival enlargement and improve overall gingival health.[8,10,22] The presence of drug-induced enlargement is associated with pseudopocket formation, frequently with abundant plaque accumulation. This may lead to the development of periodontitis. Therefore meticulous plaque control helps maintain attachment levels. Also, adequate plaque control may aid in preventing the recurrence of gingival enlargement in surgically treated cases.

Third, in some patients, gingival enlargement persists even after careful consideration of the previous approaches. These patients may require surgery, either gingivectomy or the periodontal flap.

Figure 58-1 presents a decision tree outlining the sequence of events and options in the treatment of drug-induced gingival enlargement.

Gingivectomy. Gingivectomy has the advantage of simplicity and quickness but presents the disadvantages of postoperative discomfort and increased chance of postoperative bleeding. It also sacrifices keratinized tissue and does not allow for osseous recontouring if it is necessary. The clinician's decision between the two surgical techniques available must consider the extent of the area to be operated, the presence of periodontitis and osseous defects, and the location of the base of the pockets in relation to the mucogingival junction.

In general, small areas (up to six teeth) of drug-induced gingival enlargement with no evidence of attachment loss (and therefore no anticipated need for osseous surgery) can effectively be treated with the gingivectomy technique. An important consideration is the amount of keratinized tissue that is present. At least 3 mm of keratinized gingiva in the apicocoronal direction should remain after the surgery is completed.

Chapter 56 describes the gingivectomy technique in detail. Figures 56-6 to 56-8 and Figure 58-2 depict the procedure diagrammatically, and Figure 58-3 illustrates a case of cyclosporine-induced gingival enlargement treated with the gingivectomy technique.

Gingivectomy or gingivoplasty can also be performed with electrosurgery, using a laser device (see Chapter 56). There is some preliminary evidence that the recurrence of drug-induced gingival enlargement is slower on patients treated via laser when compared to conventional gingivectomy or flap surgery.[14]

Flap Technique. Larger areas of gingival enlargement (more than six teeth) or areas where attachment loss and osseous defects are present should be treated by the flap technique, as should any situation in which the gingivectomy technique may create a mucogingival problem.

The periodontal flap technique used for the treatment of gingival enlargements is a simple variation of the one used to treat periodontitis (see Chapters 57 and 59). Figure 58-4 describes the basic steps in the technique, described as follows:

1. After anesthetizing the area, sounding of the underlying alveolar bone is performed with a periodontal probe to determine the presence and extent of osseous defects.
2. With a #15 Bard-Parker blade, the initial scalloped internal bevel incision is made at least 3 mm coronal to the mucogingival junction, including the creation of new interdental papillae.
3. The same blade is used to thin the gingival tissues in a buccolingual direction to the mucogingival junction. At this point the blade establishes contact with the alveolar bone, and a full-thickness or a split-thickness flap is elevated.
4. Using an Orban knife, the base of each papilla connecting the facial and the lingual incisions is incised.
5. The excised marginal and interdental tissues are removed with curettes.
6. Tissue tabs are removed, the roots are thoroughly scaled and planed, and the bone is recontoured as needed.

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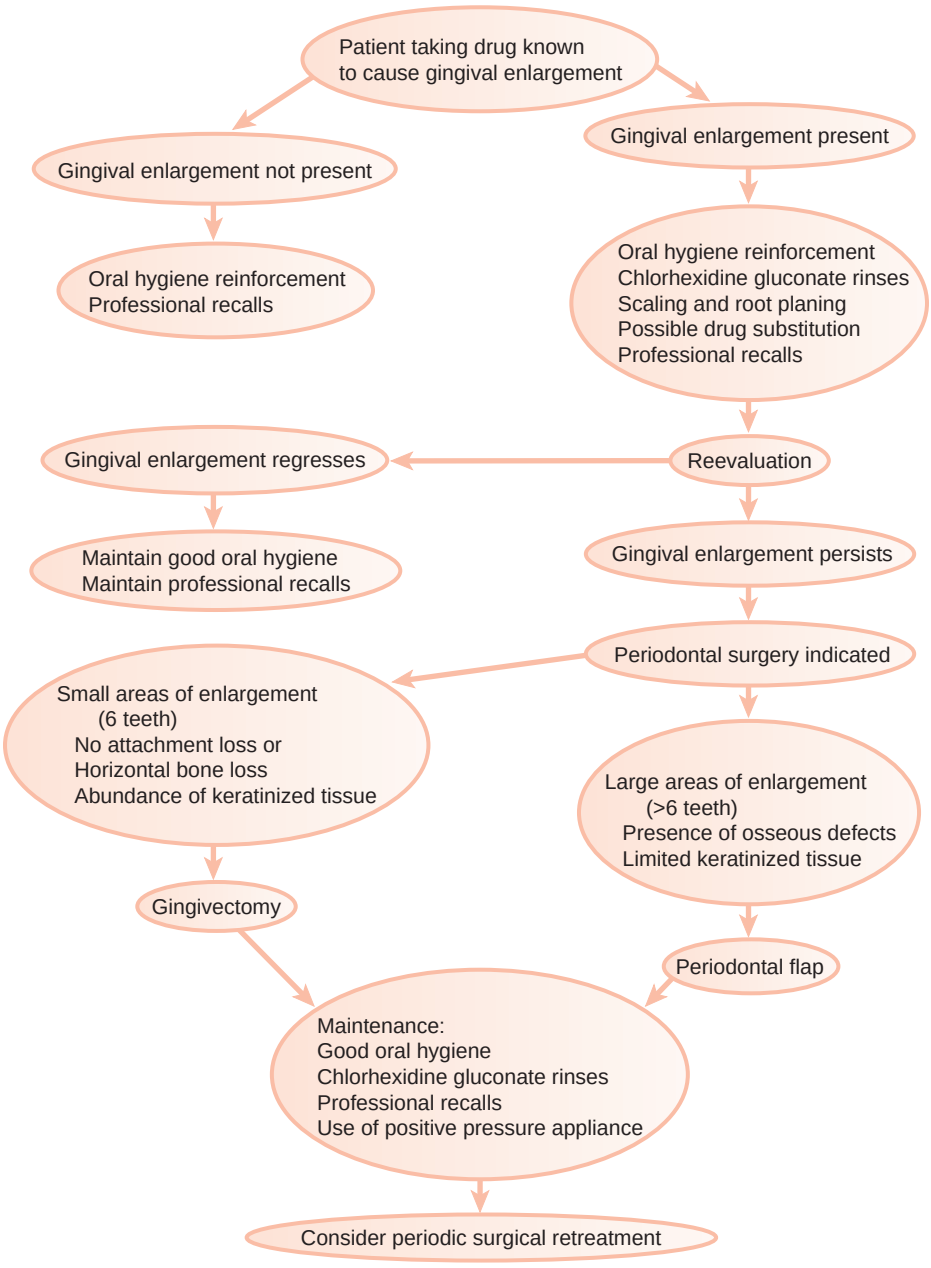

Figure 58-1 Decision tree for treatment of drug-induced gingival enlargements.

7. The flap is replaced and if necessary, trimmed to reach the bone-tooth junction. The flap is then sutured with an interrupted or a continuous mattress technique. The surgical area is covered with a periodontal dressing.

Sutures and dressing are removed after 1 week. The patient is then instructed to initiate plaque control methods. Usually it is convenient for the patient to use chlorhexidine oral rinses once or twice daily for several weeks. Figure 58-5 illustrates a patient treated with the flap technique.

Recurrence of drug-induced gingival enlargement is a reality in surgically treated cases.[19] As stated previously, meticulous home care,[6,16] chlorhexidine gluconate rinses,[16,21] and professional recall therapy can decrease the rate and the degree to which recurrence occurs. A hard, natural rubber fitted bite guard worn at night may help to control the recurrence.[1,3]

Even though the periodontal flap approach may be technically more difficult than the gingivectomy procedure, the postsurgical healing of the flap technique presents less discomfort and alleviates hemorrhagic problems. The primary closure of the surgical site with the flap procedure is a great advantage over the secondary open wound resulting from the gingivectomy technique. Also, postsurgical home care can be instituted earlier with the periodontal flap.[5]

Recurrence may occur as early as 3 to 6 months after the surgical treatment. In general, surgical results are maintained for at least 12 months. In one study, 6-month postsurgical examination of the recurrence of cyclosporine-induced gingival enlargement after periodontal flap surgery or gingivectomy determined the return of increased pocket depth was slower with the flap technique.[17] However, the recurrence of increased thickness of the periodontal tissue has not been objectively evaluated.

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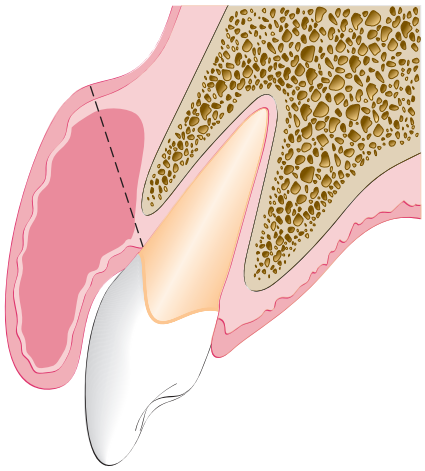

Figure 58-2 Gingivectomy technique as used in treating patients with drug-induced gingival enlargement. The dotted line represents the external bevel incision, and the shaded area corresponds to the tissue to be excised. Gingivectomy incision may not remove the entire hyperplastic tissue *(shaded area)* and may leave a wide wound of exposed connective tissue.

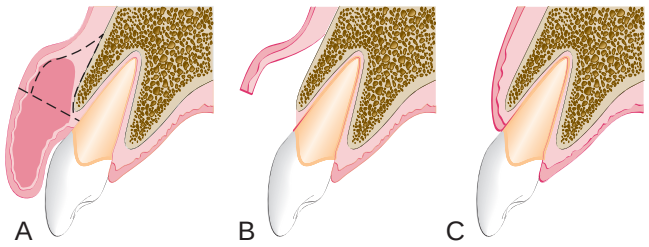

Figure 58-4 Diagram of periodontal flap treatment for drug-induced gingival enlargement. **A,** Initial reverse bevel incision followed by thinning of the enlarged gingival tissue; dotted lines represent incisions, and the shaded area represents the tissue portion to be excised. **B,** After flap elevation, enlarged portion of the gingival tissue is removed. **C,** The flap is placed on top of the alveolar bone and sutured.

Figure 58-3 Surgical treatment of cyclosporine-induced gingival enlargement using the gingivectomy technique on a 16-year-old girl who had received a kidney allograft 2 years earlier. **A,** Presence of enlarged gingival tissues and pseudopocket formation; no attachment loss or evidence of vertical bone loss existed. **B,** Initial external bevel incision performed with a Kirkland knife. **C,** Interproximal tissue release achieved with an Orban knife. **D** and **E,** Gingivoplasty performed with tissue nippers and a round diamond at high speed with abundant refrigeration. **F,** Aspect of the surgical wound at conclusion of the surgical procedure. **G,** Placement of noneugenol periodontal dressing. **H,** Surgical area 3 months postoperatively. Note the successful elimination of enlarged gingival tissue, restoration of a physiologic gingival contour, and maintenance of an adequate band of keratinized tissue.

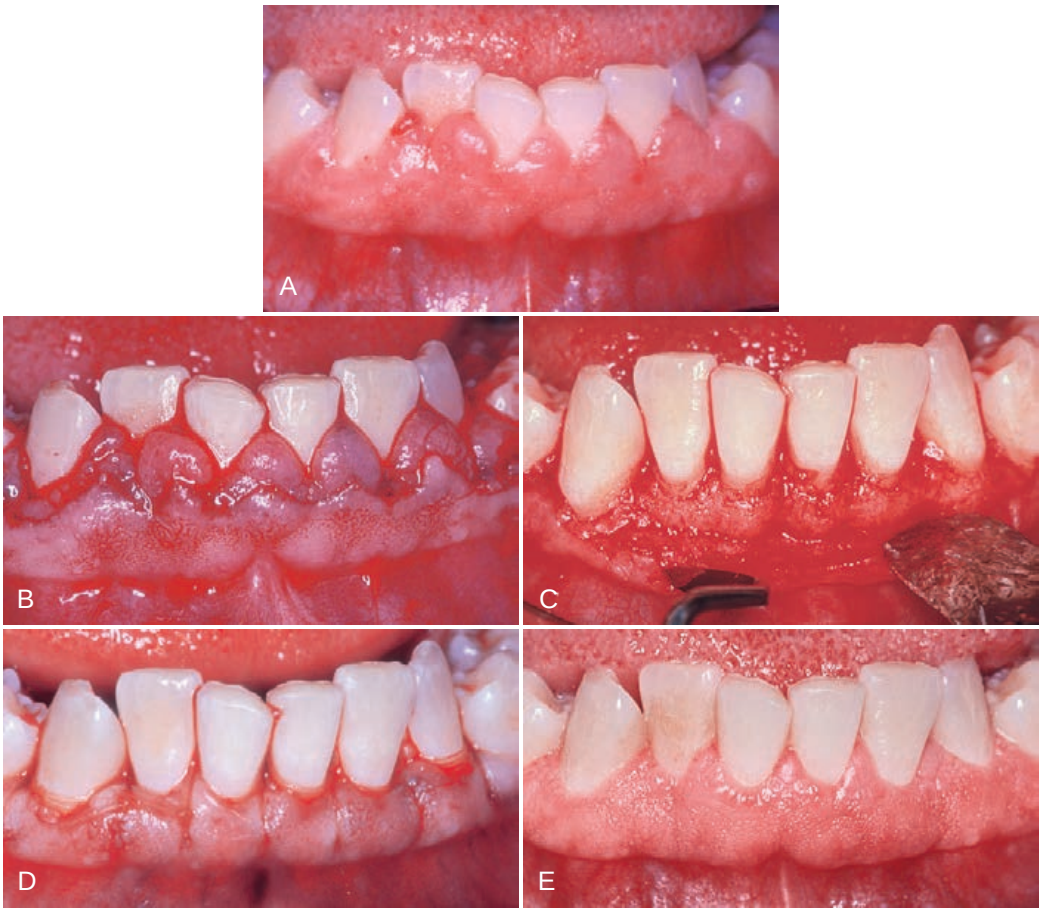

Figure 58-5 Treatment of combined cyclosporine and nifedipine–induced gingival enlargement with a periodontal flap on a 35-year-old female who had received a kidney allograft 3 years earlier. **A,** Presurgical clinical aspect of the lower anterior teeth, showing severe gingival enlargement. **B,** Initial scalloped reverse bevel incision, including maintenance of keratinized tissue and creation of surgical papillae. **C,** Elevation of a full-thickness flap and removal of the inner portion of the previously thinned gingival tissue. After scaling and root planing, osseous recontouring can be performed if necessary. **D,** The flap is positioned on top of the alveolar crest. **E,** Postsurgical aspect of the treated area at 12 months. Note the reduction of enlarged tissue volume and acceptable gingival health.

LEUKEMIC GINGIVAL ENLARGEMENT

Leukemic enlargement occurs in acute or subacute leukemia and is uncommon in the chronic leukemic state. The medical care of leukemic patients is often complicated by gingival enlargement and superimposed painful acute necrotizing ulcerative gingivitis. This interferes with masticating and creates toxic systemic reactions. The patient's bleeding and clotting times and platelet count should be checked, and the hematologist should be consulted before periodontal treatment is instituted (see Chapter 54).

Treatment of acute gingival involvement is described in Chapter 41. After acute symptoms subside, attention is directed to correction of the gingival enlargement. The rationale for therapy is to remove the local irritating factors to control the inflammatory component of the enlargement.

The lesion is treated by scaling and root planing performed in stages with topical and local anesthesia. The initial treatment consists of gently removing the loose accumulations of bacterial plaque, performing superficial scaling, and instructing the patient in oral hygiene for plaque control. This portion of the therapy may include, at least initially, the daily use of chlorhexidine mouthwashes. Oral hygiene procedures are extremely important in these patients and should be performed by the nurse if necessary.

Progressively deeper scaling is carried out at subsequent visits. Treatments are confined to a small area of the mouth to facilitate the control of bleeding. Antibiotics are administered systemically the evening before and for 48 hours after each treatment to reduce the risk of infection.

GINGIVAL ENLARGEMENT IN PREGNANCY

Treatment requires elimination of all local irritants responsible for precipitating the gingival changes in pregnancy. Elimination of local irritants early in pregnancy is a preventive measure against gingival disease. This is preferable to treatment of gingival enlargement after it occurs. Marginal and interdental gingival inflammation and enlargement are treated by scaling and root planing (see Chapters 45 and 46). Treatment of tumorlike gingival enlargements consists of surgical excision and scaling and planing of the tooth surface. The enlargement will recur unless all of the irritants are removed. Food impaction is frequently an inciting factor.

Timing of Treatment and Indications

Gingival lesions in pregnancy should be treated as soon as they are detected, although not necessarily by surgical means. Scaling and root-planing procedures and adequate oral hygiene measures may

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SCIENCE TRANSFER

Although gingivectomy has been appropriate in the past, most patients with gingival enlargement are better treated with a flap approach that includes resection of hyperplastic tissues. This allows access to bone defects for management, ensures an adequate postsurgical band of keratinized gingiva, and minimizes the risk of postsurgical bleeding.

Some disease entities that cause gingival enlargement have a strong tendency to recur after surgical removal. This can be lessened by adequate posttreatment control of plaque and calculus, and repeated surgical interventions should be restricted to those cases in which functional and esthetic problems caused by excessive gingival tissue are manifest or where there is evidence of ongoing loss of attachment. Gingival enlargements seen in pregnancy tend to resolve postpartum when hormonal balance of estrogen and progesterone occurs, thus, in most of these patients, surgical intervention is not needed during pregnancy.

reduce the size of the enlargement. Gingival enlargements will shrink after pregnancy but may not completely disappear. After pregnancy, the entire mouth should be reevaluated, a full set of radiographs taken, and the necessary treatment undertaken.

Lesions should be removed surgically during pregnancy only if they interfere with mastication or produce an esthetic disfigurement that the patient wants removed.

In pregnancy, the emphasis should be on preventing gingival disease before it occurs and treating existing gingival disease before it worsens. All patients should be seen as early as possible in pregnancy. Those without gingival disease should be checked for potential sources of local irritation and should be instructed in plaque control procedures. Those with gingival disease should be treated promptly, before the effects of pregnancy on the gingiva become apparent. Chapter 38 presents the necessary precautions for periodontal treatment of pregnant women.

Every pregnant patient should be scheduled for periodic dental visits. At these appointments, the importance of prevention should be stressed to avoid serious periodontal problems during pregnancy.

GINGIVAL ENLARGEMENT IN PUBERTY

Gingival enlargement in puberty is treated by performing scaling and curettage, removing all sources of irritation, and controlling plaque. Surgical removal may be required in severe cases. The problem in these patients is recurrence that results from poor oral hygiene. Chapter 11 discusses the problems encountered during puberty.

RECURRENCE OF GINGIVAL ENLARGEMENT

Recurrence after treatment is the most common problem in the management of gingival enlargement. Residual local irritation and systemic or hereditary conditions causing noninflammatory gingival hyperplasia are the responsible factors.

Recurrence of chronic inflammatory enlargement immediately after treatment indicates that all irritants have not been removed. Contributory local conditions, such as food impaction and overhanging margins of restorations, are often overlooked. If the enlargement recurs after healing is complete and normal contour is attained, inadequate plaque control by the patient is the most common cause.

Recurrence during the healing period is manifested as red, beadlike, granulomatous masses that bleed on slight provocation. This is a proliferative vascular inflammatory response to local irritation, usually a fragment of calculus on the root. The condition is corrected by removing the granulation tissue and scaling and planing the root surface.

Familial, hereditary, or idiopathic gingival enlargement recurs after surgical removal even after all local irritants have been removed. The enlargement can be maintained at minimal size by preventing secondary inflammatory involvement.

The use of escharotic drugs has been recommended in the past for the removal of gingival enlargements. Their use is currently not recommended because the destructive action of the drugs is difficult to control. Injury to healthy tissue and root surfaces, delayed healing, and excessive postoperative pain are complications that often occur.

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CHAPTER 59

The Flap Technique for Pocket Therapy

Fermin A. Carranza, Henry H. Takei, and Kitetsu Shin

CHAPTER OUTLINE

OVERVIEW
Technique for Access and Pocket Depth Reduction/Elimination
Incisions
Reconstructive Techniques
MODIFIED WIDMAN FLAP
UNDISPLACED FLAP
Palatal Flap
APICALLY DISPLACED FLAP
FLAPS FOR RECONSTRUCTIVE SURGERY
Papilla Preservation Flap
Conventional Flap
DISTAL MOLAR SURGERY
Maxillary Molars
Mandibular Molars

Several techniques can be used for the treatment of periodontal pockets. The periodontal flap is one of the most frequently employed procedures, particularly for moderate and deep pockets in posterior areas (see Chapter 52).

OVERVIEW

Flaps are used for pocket therapy to accomplish the following:

1. Increase accessibility to root deposits for scaling and root planing.
2. Eliminate or reduce pocket depth by resection of the pocket wall.
3. Gain access for osseous resective surgery if it is necessary.
4. Expose the area to perform regenerative methods.

To fulfill these purposes, several flap techniques are available and in current use.

Technique for Access and Pocket Depth Reduction/Elimination

The three different categories of flap techniques used in periodontal flap surgery are (1) the modified Widman flap, (2) the undisplaced flap, and the (3) apically displaced flap.

The modified Widman flap facilitates instrumentation for root therapy. It does not attempt to reduce the pocket depth, but it does eliminate the pocket lining. The original intent of the surgery was to access the root surface for scaling and root planing. The objectives for the other two flap procedures, the nondisplaced flap and the apically displaced flap, include root surface access and the reduction or elimination of the pocket depth. The decision on which procedure to utilize depends on two important anatomic landmarks: the pocket depth and location of the mucogingival junction. These landmarks establish the presence and width of the attached gingiva, which is the basis for the decision.

The *modified Widman flap* has been described for exposing the root surfaces for meticulous instrumentation and for removal of the pocket lining.[6] Again, it is not intended to eliminate or reduce pocket depth, except for the reduction that occurs in healing by tissue shrinkage.

The *undisplaced (unrepositioned) flap* improves accessibility for instrumentation, but it also removes the pocket wall, thereby reducing or eliminating the pocket. This is essentially an excisional procedure of the gingiva.

The *apically displaced flap* provides accessibility and eliminates the pocket, but it does the latter by apically positioning the soft tissue wall of the pocket.[2] Therefore it preserves or increases the width of the attached gingiva by transforming the previously unattached keratinized pocket wall into attached tissue. This increase in the width of the attached gingiva is based on the apical shift of the mucogingival junction, which may include the apical displacement of the muscle attachments. A study made before and 18 years after apically displaced flaps failed to show a permanent relocation of the mucogingival junction.[1]

Incisions

All three flap techniques just discussed use the basic incisions described in Chapter 57: the internal bevel incision, the crevicular incision, and the interdental incision. However, there are important variations in the way these incisions are performed for the different types of flaps (Figures 59-1 and 59-2). The internal beveled incision for the modified Widman flap closely follows the scalloped outline of the dentition to minimize the loss of the attached keratinized gingiva. This incision is made 1 to 2 mm from the teeth. The

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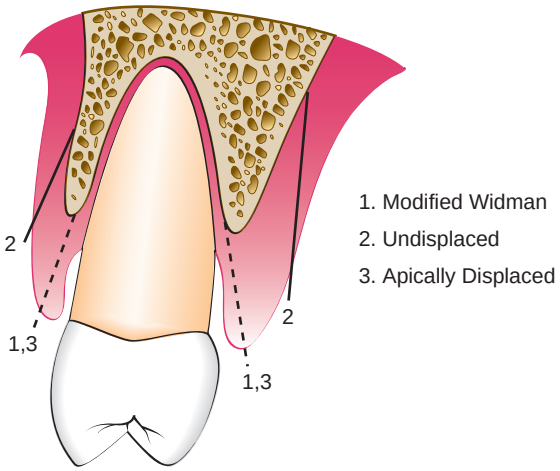

Figure 59-1 Locations of the internal bevel incisions for the different types of flaps.

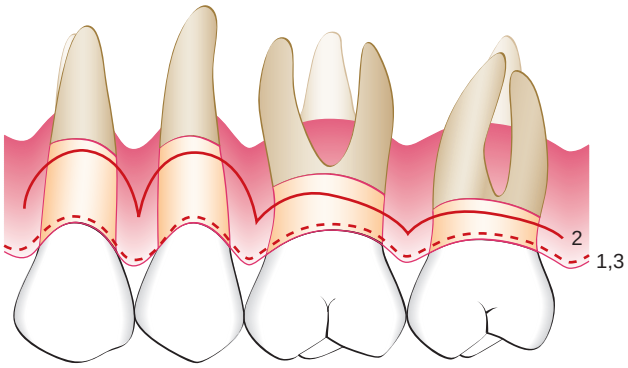

Figure 59-2 Scallopings required for the different types of flaps (see Figure 59-1).

gingival margin is removed and the flap is reflected to gain access for root therapy.

The apically displaced flap technique is selected for cases that present a minimal amount of keratinized, attached gingiva. For this reason, the internal bevel incision should be made as close to the tooth as possible, 0.5 to 1.0 mm (see Figure 59-1). There is no need to determine where the bottom of the pocket is in relation to the incision for the apically displaced flap, as one would for the undisplaced flap. The flap is placed at the tooth-bone junction by apically displacing the flap. Its final position is not determined by the placement of the first incision.

For the undisplaced flap, the internal bevel incision is initiated at or near a point just coronal to where the bottom of the pocket is projected on the outer surface of the gingiva (see Figure 59-1). This incision can be accomplished only if sufficient attached gingiva remains apical to the incision. Therefore the two anatomic landmarks, the pocket depth and the location of the mucogingival junction, must be considered to evaluate the amount of attached gingiva that will remain after the surgery has been completed. Because the pocket wall is not displaced apically, the initial incision should eliminate the pocket wall. Therefore an incision should not be made too close to the tooth because it will not eliminate the pocket wall and may result in the re-creation of the soft tissue pocket. If the tissue is too thick, the flap margin should be thinned with the initial incision. The proper placement of the flap margin at the tooth-bone junction during closure is important to prevent either recurrence of the pocket or result in the exposure of bone.

The internal bevel incision should be scalloped into the interdental area to preserve the interdental papilla (see Figure 59-2). This will allow better coverage of the bone at both the radicular and the interdental areas.

If the surgeon contemplates osseous surgery, the first incision should be placed in such a way to compensate for the removal of the bone tissue so the flap can be placed at the tooth-bone junction.

Reconstructive Techniques

The techniques used to achieve reconstructive and regenerative objectives are the *papilla preservation flap*[8] and the *conventional flap* using only crevicular or pocket incisions. This will allow the clinician to retain the maximum amount of gingival tissue, including the papilla, which is essential for graft or membrane coverage.

MODIFIED WIDMAN FLAP

In 1965, Morris[4] revived a technique described early in the twentieth century in the periodontal literature; he called it the "unrepositioned mucoperiosteal flap." Essentially, the same procedure was presented in 1974 by Ramfjord and Nissle,[6] who called it the "modified Widman flap" (Figure 59-3). This technique offers the possibility of establishing an intimate postoperative adaptation of healthy collagenous connective tissue to tooth surfaces[2,3,5,6] and provides access for adequate instrumentation of the root surfaces and immediate closure of the area. The following steps outline the modified Widman flap technique.

Step 1: The initial incision is an internal bevel incision to the alveolar crest starting 0.5 to 1 mm away from the gingival margin (Figure 59-3, *C*). Scalloping follows the gingival margin. Care should be taken to insert the blade in such a way that the papilla is left with a thickness similar to that of the remaining facial flap. Vertical relaxing incisions are usually not needed.

Step 2: The gingiva is reflected with a periosteal elevator (Figure 59-3, *D*).

Step 3: A crevicular incision is made from the bottom of the pocket to the bone, circumscribing the triangular wedge of tissue containing the pocket lining.

Step 4: After the flap is reflected, a third incision is made in the interdental spaces coronal to the bone with a curette or an interproximal knife and the gingival collar is removed (Figure 59-3, *E* and *F*).

Step 5: Tissue tags and granulation tissue are removed with a curette. The root surfaces are checked, then scaled and planed if needed (Figure 59-3, *G* and *H*). Residual periodontal fibers attached to the tooth surface should not be disturbed.

Step 6: Bone architecture is not corrected, except if it prevents good tissue adaptation to the necks of the teeth. Every effort is made to adapt the facial and lingual interproximal tissue adjacent to each other in such a way that no interproximal bone remains exposed at the time of suturing .The flaps may be thinned to allow for close adaptation of the gingiva around the entire circumference of the tooth and to each other interproximally.

Step 7: Continuous, independent sling sutures are placed in both the facial and palatal (Figure 59-3, *I* and *J*) and covered with a periodontal surgical pack.

Ramfjord and Nissle[6] performed an extensive longitudinal study comparing the Widman procedure, as modified by them, with the curettage technique and the pocket elimination methods that

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Figure 59-3 Modified Widman flap technique. **A,** Facial view before surgery. Probing of pockets revealed interproximal depths ranging from 4 to 8 mm and facial and palatal depths of 2 to 5 mm. **B,** Radiographic survey of area. Note generalized horizontal bone loss. **C,** Facial internal bevel incision. **D,** Palatal incision. **E,** Elevation of the flap, leaving a wedge of tissue still attached to its base. **F,** Removal of tissue. **G,** Tissue removed and ready for scaling and root planing. **H,** Scaling and root planing of exposed root surfaces. **I,** Continuous, independent sling suture of facial portion of surgery. **J,** Continuous, independent sling suture of palatal portion of surgery. K, Post surgical result. *(Courtesy Dr. Kitetsu Shin, Saitama, Japan).*

include bone contouring when needed. The patients were assigned randomly to one of the techniques, and results were analyzed yearly up to 7 years after therapy. They reported approximately similar results with the three methods tested. Pocket depth was initially similar for all methods but was maintained at shallower levels with the Widman flap; the attachment level remained higher with the Widman flap.

UNDISPLACED FLAP

Currently, the undisplaced flap may be the most frequently performed type of periodontal surgery. It differs from the modified Widman flap in that the soft tissue pocket wall is removed with the initial incision; thus it may be considered an "internal bevel gingivectomy." The undisplaced flap and the gingivectomy are the two techniques that surgically remove the pocket wall. To perform this technique without creating a mucogingival problem, the clinician should determine that enough attached gingiva will remain after removal of the pocket wall. The following steps outline the undisplaced flap technique.

Step 1: The pockets are measured with the periodontal probe, and a bleeding point is produced on the outer surface of the gingiva to mark the pocket bottom.

Step 2: The initial, or internal bevel, incision is made (Figure 59-4) after scalloping the bleeding marks on the gingiva (Figure 59-5). The incision is usually carried to a point apical to the alveolar crest, depending on the thickness of the tissue. The thicker the tissue is the more apical the ending point of the incision (see Figure 59-4). In addition, thinning of the flap should be done with the initial incision because it is

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easier to accomplish at this time than later, with a loose, reflected flap that is difficult to manage. (Use of this technique in palatal areas is considered in the following discussion.)

Step 3: The second, or crevicular, incision is made from the bottom of the pocket to the bone to detach the connective tissue from the bone.

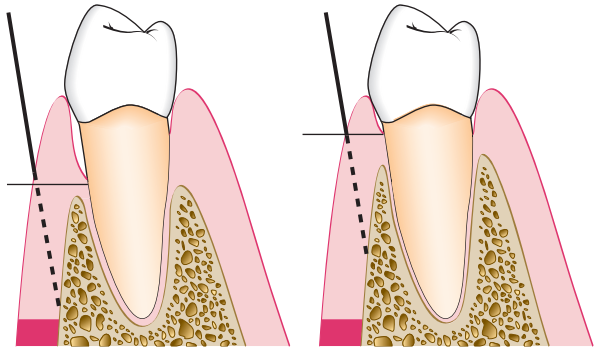

Figure 59-4 Diagram showing the location of two different areas where the internal bevel incision is made in an undisplaced flap. The incision is made at the level of the pocket to discard the tissue coronal to the pocket if there is sufficient remaining attached gingiva.

Step 4: The flap is reflected with a periosteal elevator (blunt dissection) from the internal bevel incision. Usually there is no need for vertical incisions because the flap is not displaced apically.

Step 5: The third, or interdental, incision is made with an interdental knife, separating the connective tissue from the bone.

Step 6: The triangular wedge of tissue created by the three incisions is removed with a curette.

Step 7: The area is debrided, removing all tissue tags and granulation tissue using sharp curettes.

Step 8: After the necessary scaling and root planing, the flap edge should rest on the root-bone junction. If this is not the case, because of improper location of the initial incision or the unexpected need for osseous surgery, the edge of the flap is scalloped again and trimmed to allow the flap edge to end at the root-bone junction.

Step 9: A continuous sling suture is used to secure the facial and the lingual or palatal flaps. This type of suture, using the tooth as an anchor, is advantageous to position and hold the flap edges at the root-bone junction. The area is covered with a periodontal pack.

Palatal Flap

The surgical approach to the palatal area differs from that for other areas because of the character of the palatal tissue and the

Figure 59-5 Undisplaced flap. **A** and **B,** Preoperative facial and palatal views. **C** and **D,** Internal bevel incisions in the facial and palatal aspects. Note the deeper scalloping palatally for the replaced flap. **E** and **F,** Flap elevated showing osseous defects. **G** and **H,** Osseous surgery has been performed. **I** and **J,** Flaps have been placed in their original site and sutured. **K** and **L,** Postoperative results. *(Courtesy Dr. Paulo Camargo, University of California, Los Angeles.)*

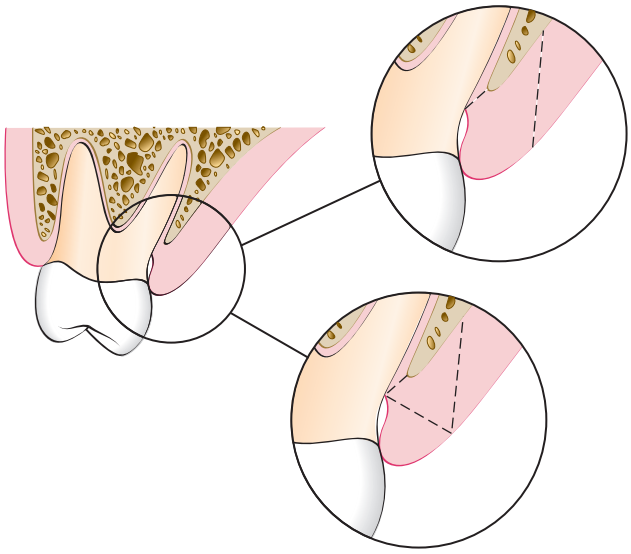

Figure 59-6 Examples of two methods for eliminating a palatal pocket. One incision is an internal bevel incision made at the area of the apical extent of the pocket. The other procedure uses a gingivectomy incision, which is followed by an internal bevel incision.

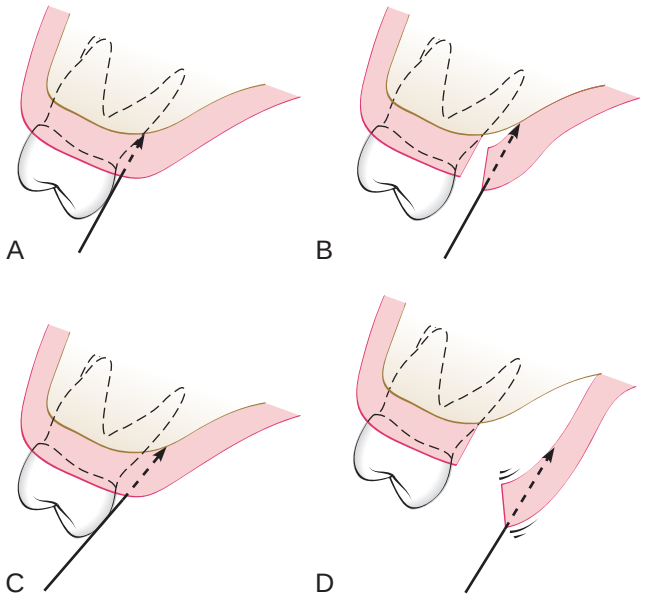

Figure 59-7 Diagrams illustrating the angle of the internal bevel incision in the palate and the different ways to thin the flap. **A,** Usual angle and direction of the incision. **B,** Thinning of the flap after it has been slightly reflected with a second internal incision. **C,** Beveling and thinning of the flap with the initial incision if the position and contour of the tooth allow. **D,** The problem encountered in thinning the flap once it has been reflected. The flap is too loose and free for proper positioning and incision.

anatomy of the area. The palatal tissue is all attached, keratinized tissue and has none of the elastic properties associated with other gingival tissues. Therefore the palatal tissue cannot be apically displaced, and a partial-thickness (split-thickness) flap cannot be accomplished.

The initial incision for the palatal flap should allow the flap, when sutured, to be precisely adapted at the root-bone junction. The flap cannot be moved apically or coronally to adapt to the root-bone junction, as can be done with the flaps in other areas. Therefore the location of the initial incision is important for the final placement of the flap.

The palatal tissue may be thin or thick, it may or may not have osseous defects, and the palatal vault may be high or low. These anatomic variations may require changes in the location, angle, and design of the incision.

The initial incision for a flap varies with the anatomic situation. As shown in Figure 59-6, the initial incision may be the usual internal bevel incision, followed by crevicular and interdental incisions. If the tissue is thick, a horizontal gingivectomy incision may be made, followed by an internal bevel incision that starts at the edge of this incision and ends on the lateral surface of the underlying bone. The placement of the internal bevel incision must be done in such a way that the flap fits around the tooth without exposing the bone.

Before the flap is reflected to the final position for scaling and management of the osseous lesions, its thickness must be checked. Flaps should be thin to adapt to the underlying osseous tissue and provide a thin, knifelike gingival margin. Flaps, particularly palatal flaps, often are too thick; they may have a propensity to separate from the tooth and may delay and complicate healing. It is best to thin the flaps before their complete reflection because a free, mobile flap is difficult to hold for thinning (Figure 59-7). A sharp, thin papilla positioned properly around the interdental areas at the tooth-bone junction is essential to prevent recurrence of soft tissue pockets.

The purpose of the palatal flap should be considered before the incision is made. If the intent of the surgery is debridement, the internal bevel incision is planned so that the flap adapts at the root-bone junction when sutured. If osseous resection is necessary, the incision should be planned to compensate for the lowered level of the bone when the flap is closed. Probing and sounding of the osseous level and the depth of the intrabony pocket should be used to determine the position of the incision.

The apical portion of the scalloping should be narrower than the line-angle area because the palatal root tapers apically. A rounded scallop results in a palatal flap that does not fit snugly around the root. This procedure should be done before the complete reflection of the palatal flap because a loose flap is difficult to grasp and stabilize for dissection.

It is sometimes necessary to thin the palatal flap after it has been reflected. This can be accomplished by holding the inner portion of the flap with a mosquito hemostat or Adson forceps as the inner connective tissue is carefully dissected away with a sharp #15 scalpel blade. Care must be taken not to perforate or overthin the flap. The edge of the flap should be thinner than the base; therefore the blade should be angled toward the lateral surface of the palatal bone. The dissected inner connective tissue is removed with a hemostat. As with any flap, the triangular papilla portion should be thin enough to fit snugly against the bone and into the interdental area (Figure 59-8).

The principles for the use of vertical releasing incisions are similar to those for using other incisions. Care must be exercised so that the length of the incision is minimal to avoid the numerous vessels located in the palate.

APICALLY DISPLACED FLAP

With some variants, the apically displaced flap technique can be used for (1) pocket eradication and/or (2) widening the zone of attached gingiva. Depending on the purpose, it can be a full-thickness (mucoperiosteal) or a split-thickness (mucosal) flap. The

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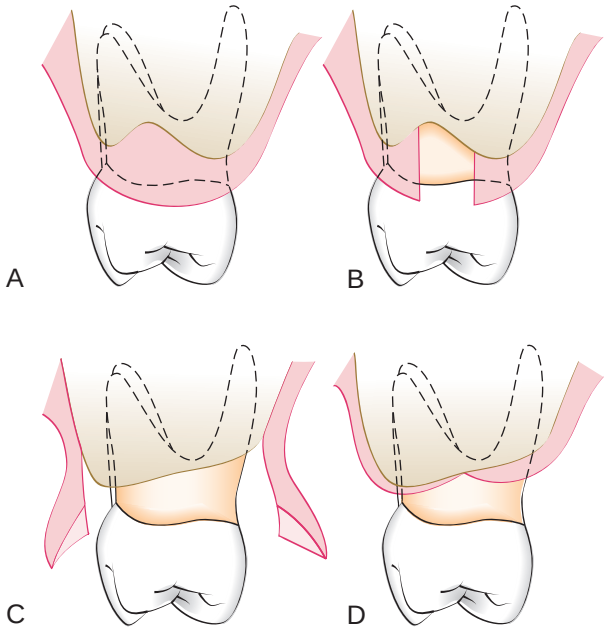

Figure 59-8 **A,** Distal view of incisions made to eliminate a pocket distal to the maxillary second molar. **B,** Two parallel incisions and the removal of the intervening tissue. **C,** Thinning of the flap and contouring of the bone. **D,** Approximation of the buccal and palatal flaps.

split-thickness flap requires more precision and time, as well as a gingival tissue thick enough to split, but it can be more accurately positioned and sutured in an apical position using a periosteal suturing technique, as follows:

Step 1: An internal bevel incision is made (Figure 59-9). To preserve as much of the keratinized and attached gingiva as possible, it should be no more than about 1 mm from the crest of the gingiva and directed to the crest of the bone (see Figure 59-1). The incision is made after the existing scalloping, and there is no need to mark the bottom of the pocket in the external gingival surface because the incision is unrelated to pocket depth. It is also not necessary to accentuate the scallop interdentally because the flap is displaced apically and not placed interdentally.

Step 2: Crevicular incisions are made, followed by initial elevation of the flap; then interdental incisions are performed, and the wedge of tissue that contains the pocket wall is removed.

Step 3: Vertical incisions are made extending beyond the mucogingival junction. If the objective is a full-thickness flap, it is elevated by blunt dissection with a periosteal elevator. If a split-thickness flap is required, it is elevated using sharp dissection with a Bard-Parker knife to split it, leaving a layer of connective tissue, including the periosteum, on the bone.

Step 4: After removal of all granulation tissue, scaling and root planing, and osseous surgery if needed, the flap is displaced apically. It is important that the vertical incisions and

Figure 59-9 Apically displaced flap. **A** and **B,** Facial and lingual preoperative views. **C** and **D,** Facial and lingual flaps elevated. **E** and **F,** After debridement of the areas. **G** and **H,** Sutures in place. **I** and **J,** Healing after 1 week. **K,** Healing after 2 months. Note the preservation of attached gingiva displaced to a more apical position. *(Courtesy of Dr. Thomas Han, Los Angeles.)*

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therefore the flap elevation, reach past the mucogingival junction to provide adequate mobility to the flap for its apical displacement.

Step 5: If a full-thickness flap was performed, a sling suture around the tooth prevents the flap from sliding to a position more apical than that desired, and the periodontal dressing can avoid its movement in a coronal direction. A partial-thickness flap is sutured to the periosteum using a direct loop suture or a combination of loop and anchor suture. A dry foil is placed over the flap before covering it with the dressing to prevent the introduction of pack under the flap.

After 1 week, dressings and sutures are removed. The area is usually repacked for another week, after which the patient is instructed to use chlorhexidine mouth rinse or to apply chlorhexidine topically with cotton-tipped applicators for another 2 or 3 weeks.

FLAPS FOR RECONSTRUCTIVE SURGERY

In current reconstructive therapy, bone grafts, membranes, or a combination of these, with or without other agents, are used for a successful outcome (see Chapter 61). The flap design should therefore be set up so that the maximum amount of gingival tissue and papilla are retained to cover the material(s) placed in the pocket.

Two flap designs are available for reconstructive surgery: the papilla preservation flap and the conventional flap with only crevicular incisions. *The flap design of choice is the papilla preservation flap,* which retains the entire papilla covering the lesion. However, to use this flap, there must be adequate interdental space to allow the intact papilla to be reflected with the facial or lingual/palatal flap. When the interdental space is very narrow, making it impossible to perform a papilla preservation flap, a conventional flap with only crevicular incisions is made.

Papilla Preservation Flap

The technique for employing a papilla preservation flap (Figures 59-10 and 59-11) is as follows:

Step 1: A crevicular incision is made around each tooth with no incisions across the interdental papilla.

Step 2: The preserved papilla can be incorporated into the facial or lingual/palatal flap, although it is most often integrated into the facial flap. In these cases, the lingual or palatal incision consists of a semilunar incision across the interdental papilla in its palatal or lingual aspect; this incision dips apically from the line angles of the tooth so that the papillary incision is at least 5 mm from the crest of the papilla.

Step 3: An Orban knife is then introduced into this incision to sever half to two-thirds the base of the interdental papilla. The papilla is then dissected from the lingual or palatal aspect and elevated intact with the facial flap.

Step 4: The flap is reflected without thinning the tissue.

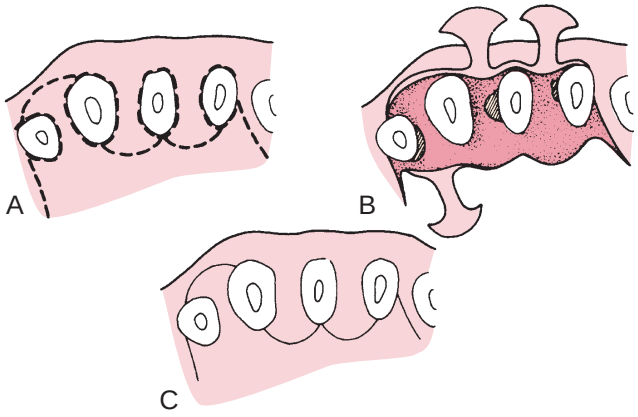

Figure 59-10 Flap design for a papilla preservation flap. **A,** Incisions for this type of flap are depicted by interrupted lines. The preserved papilla can be incorporated into the facial or the lingual-palatal flap. **B,** Reflected flap exposes the underlying bone. Several osseous defects are seen. **C,** Flap returned to its original position, covering the entire interdental spaces.

Conventional Flap

The technique for employing a conventional flap for reconstructive surgery is as follows:

Step 1: Using a #12 blade, incise the tissue at the bottom of the pocket and to the crest of the bone, splitting the papilla below the contact point. Every effort should be made to retain as much tissue as possible to protect the area subsequently.

Step 2: Reflect the flap, maintaining it as thick as possible, not attempting to thin it as is done for resective surgery. The maintenance of a thick flap is necessary to prevent exposure of the graft or the membrane resulting from necrosis of the flap margins.

DISTAL MOLAR SURGERY

Treatment of periodontal pockets on the distal surface of terminal molars is often complicated by the presence of bulbous fibrous tissue over the maxillary tuberosity or prominent retromolar pads in the mandible. Deep vertical defects are also often present in conjunction with the redundant fibrous tissue. Some of these osseous lesions may result from incomplete repair after the extraction of impacted third molars (Figure 59-12).

The gingivectomy incision is the most direct approach in treating distal pockets that have adequate attached gingiva and no osseous lesions. However, the flap approach is less traumatic postsurgically because it produces a primary closure wound rather than the open secondary wound left by a gingivectomy incision. In addition, it results in attached gingiva and provides access for examination and if needed, correction of the osseous defect. Procedures for this purpose were described by Robinson[7] and Braden[2] and modified by several other investigators. Some representative procedures are discussed here.

Maxillary Molars

The treatment of distal pockets on the maxillary arch is usually simpler than the treatment of a similar lesion on the mandibular arch because the tuberosity presents a greater amount of fibrous attached gingiva than does the area of the retromolar pad. In addition, the anatomy of the tuberosity extending distally is more adaptable to pocket elimination than is that of the mandibular molar arch, where the tissue extends coronally. However, the lack of a broad area of attached gingiva and the abruptly ascending tuberosity sometimes complicate therapy (Figure 59-13).

The following considerations determine the location of the incision for distal molar surgery: accessibility, amount of attached gingiva, pocket depth, and available distance from the distal aspect of the tooth to the end of the tuberosity or retromolar pad.

Technique. Two parallel incisions, beginning at the distal portion of the tooth and extending to the mucogingival junction distal to the tuberosity or retromolar pad, are made (Figure 59-14).

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Figure 59-11 Papilla preservation flap. **A,** Facial view after sulcular incisions have been made. **B,** Straight-line incision in the palatal area about 3 mm from gingival margins. This incision is then connected to the margins with vertical incisions in the midpart of each tooth. **C,** Papillae are reflected with the facial flap. **D,** Lingual view after reflection of the flap. **E,** Lingual view after the flap is brought back to its original position. It is then sutured with independent sutures. **F,** Facial view after healing. **G,** Palatal view after healing. *(Courtesy of Dr. Thomas Han, Los Angeles.)*

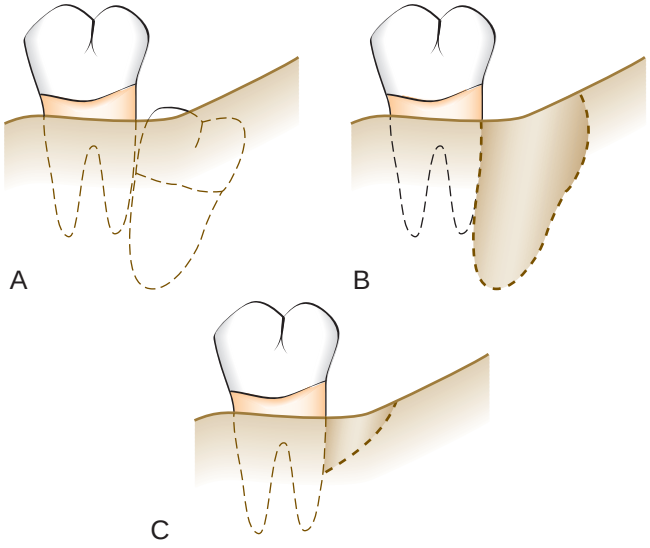

Figure 59-12 **A,** Impaction of a third molar distal to a second molar with little or no interdental bone between the two teeth. **B,** Removal of the third molar creates a pocket with little or no bone distal to the second molar. This often leads to a vertical osseous defect distal to the second molar **(C).**

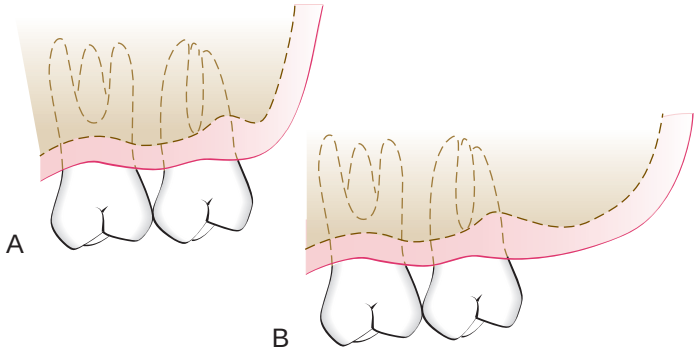

Figure 59-13 **A,** Removal of a pocket distal to the maxillary second molar may be difficult if there is minimal attached gingiva. If the bone ascends acutely apically, the removal of this bone may make the procedure easier. **B,** Long distal tuberosity with abundant attached gingiva is an ideal anatomic situation for distal pocket eradication.

Figure 59-14 **A,** Distal pocket eradication procedure with the incision distal to the molar. **B,** Scalloped incision around the remaining teeth. **C,** Flap reflected and thinned around the distal incision. **D,** Flap in position before suturing. It should be closely approximated. **E,** Flap sutured both distally and over the remaining surgical area.

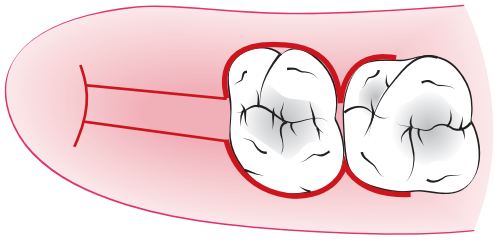

Figure 59-15 Typical incision design for a surgical procedure distal to the maxillary second molar.

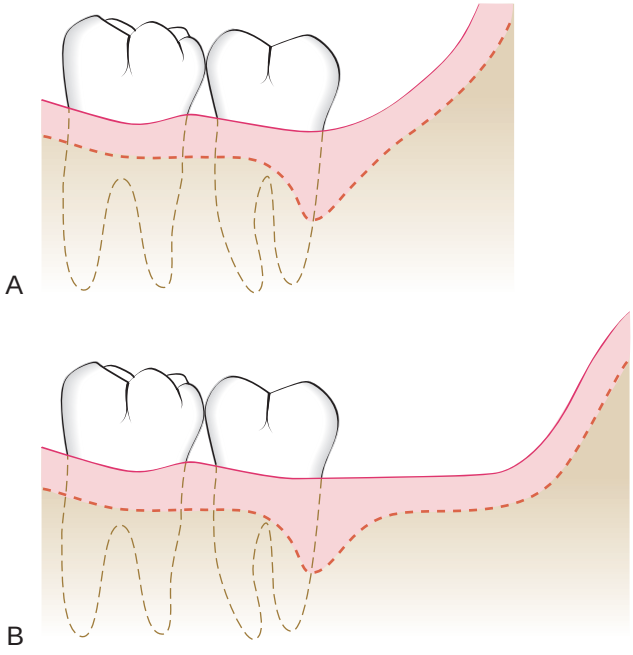

Figure 59-16 **A,** Pocket eradication distal to a mandibular second molar with minimal attached gingiva and a close ascending ramus is anatomically difficult. **B,** For surgical procedures distal to a mandibular second molar, abundant attached gingiva and distal space are ideal.

The faciolingual distance between these two incisions depends on the depth of the pocket and the amount of fibrous tissue involved. The deeper the pocket, the greater is the distance between the two parallel incisions. Importantly, when the tissue between the two incisions is removed and the flaps are thinned, the two flap edges must approximate each other at a new apical position without overlapping.

When the depth of the pocket cannot be easily estimated, it is better to err on the conservative side, leaving overlapping flaps rather than flaps that are too short and result in exposure of bone. When the two flaps overlap after the surgery is completed, they should be placed one over the other, and the overlapping portion of one of them is grabbed with a hemostat. A sharp knife or scissors is then used to cut the excess.

A transversal incision is made at the distal end of the two parallel incisions so that a long, rectangular piece of tissue can be removed. These incisions are usually interconnected with the incisions for the remainder of the surgery in the quadrant involved. The parallel distal incisions should be confined to the attached gingiva because bleeding and flap management become problems when the incision is extended into the alveolar mucosa. If access is difficult, especially if the distance from the distal aspect of the tooth to the mucogingival junction is short, a vertical incision can be made at the end of the parallel incisions.

In treating the tuberosity area, the two distal incisions are usually made at the midline of the tuberosity (Figure 59-15). In most cases, no attempt is made to undermine the underlying tissue at this time. These incisions are made straight down into the underlying bone where access is difficult. A #12B blade is generally used. It is easier to dissect out the underlying redundant tissue when the flap is partially reflected. When the distal flaps are placed back on the bone, the two flap margins should closely approximate each other.

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SCIENCE TRANSFER

Periodontal flap procedures for pocket therapy include flaps solely for access to root surfaces and bone margins, flaps for the precise processes of osseous surgery, and flaps for periodontal regeneration. Each of these approaches have specific flap designs, step-by-step elements, and all of them have calculus removal and root planing as an essential treatment protocol. Flaps should allow adequate access and should be reflected so that at least 3 mm of bone crestal is exposed. If flaps are to be positioned apically, flap mobility is obtained by extending facial and lingual flap elevation beyond the mucogingival junction, which enables the elasticity of the mucosa to be applied. Palatal flaps are much less mobile; thus inverse bevel incisions are used to remove marginal gingival tissues so that at the time of suturing, the flap margin is positioned apically, if so indicated.

Papilla preservation techniques with various modifications are applied to those areas in which gingival recession needs to be minimized. In some esthetic locations, nonsurgical therapy may be more appropriate than flap surgery.

Postoperative plaque control is essential for successful outcomes, and clinicians should ensure that patients have demonstrated adequate oral hygiene during the presurgical Phase I events and continue this after surgery. Adjacent edentulous areas are incorporated into the flap design using wedge incisions that enable access to adjacent root surfaces for complete root planing and allow clinicians to visualize and treat the associated bone defects.

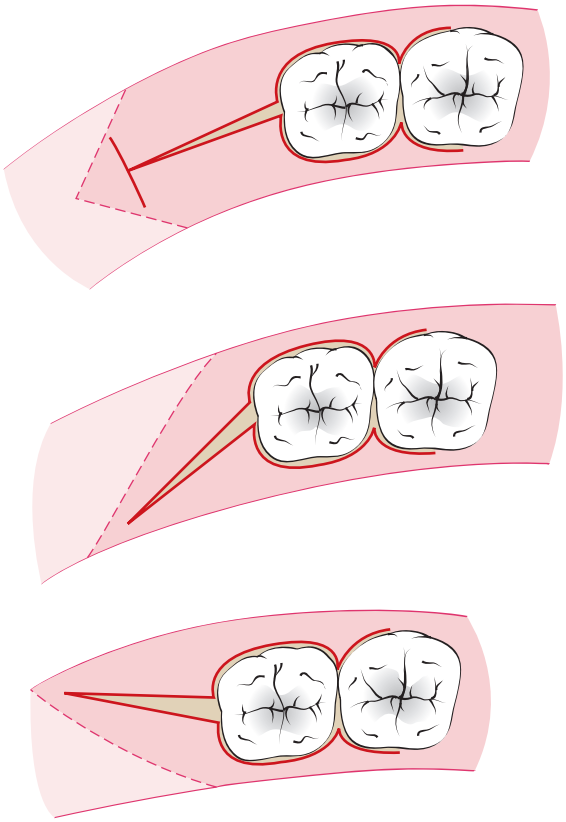

Figure 59-17 Incision designs for surgical procedures distal to the mandibular second molar. The incision should follow the areas of greatest attached gingiva and underlying bone.

Mandibular Molars

Incisions for the mandibular arch differ from those used for the tuberosity because of differences in the anatomy and histologic features of the areas. The retromolar pad area does not usually present as much fibrous attached gingiva. The keratinized gingiva, if present, may not be found directly distal to the molar. The greatest amount may be distolingual or distofacial and may not be over the bony crest. The ascending ramus of the mandible may also create a short, horizontal area distal to the terminal molar (Figure 59-16). The shorter this area, the more difficult it is to treat any deep distal lesion around the terminal molar.

The two incisions distal to the molar should follow the area with the greatest amount of attached gingiva (Figure 59-17). Therefore the incisions could be directed distolingually or distofacially, depending on which area has more attached gingiva. Before the flap is completely reflected, it is thinned with a #15 blade. It is easier to thin the flap before it is completely free and mobile. After the reflection of the flap and the removal of the redundant fibrous tissue, any necessary osseous surgery is performed. The flaps are approximated similarly to those in the maxillary tuberosity area.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 60

Resective Osseous Surgery

Thomas N. Sims

CHAPTER OUTLINE

SELECTION OF TREATMENT TECHNIQUE
RATIONALE
NORMAL ALVEOLAR BONE MORPHOLOGY
TERMINOLOGY
FACTORS IN SELECTION OF RESECTIVE OSSEOUS SURGERY
EXAMINATION AND TREATMENT PLANNING (online only)
METHODS OF RESECTIVE OSSEOUS SURGERY (online only)
OSSEOUS RESECTION TECHNIQUE (online only)
Instrumentation
Vertical Grooving
Radicular Blending
Flattening Interproximal Bone
Gradualizing Marginal Bone
FLAP PLACEMENT AND CLOSURE (online only)
POSTOPERATIVE MAINTENANCE (online only)
SPECIFIC OSSEOUS RESHAPING SITUATIONS (online only)
SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

The damage resulting from periodontal disease manifests in variable destruction of the tooth-supporting bone. Generally, bony deformities are not uniform; they are not indicative of the alveolar housing of the tooth before the disease process and do not reflect the overlying gingival architecture. Bone loss has been classified as either "horizontal" or "vertical," but in fact, bone loss is most often a combination of horizontal and vertical loss. Horizontal bone loss generally results in a relative thickening of the marginal alveolar bone because bone tapers as it approaches its most coronal margin.

The effects of this thickening and the development of vertical defects leave the alveolar bone with countless combinations of bony shapes. If these various topographic changes are to be altered to provide a more physiologic bone pattern, a method for osseous recontouring must be followed.

Osseous surgery may be defined as the procedure by which changes in the alveolar bone can be accomplished to rid it of deformities induced by the periodontal disease process or other related factors such as exostosis and tooth supraeruption.

Osseous surgery can be either additive or subtractive in nature. *Additive osseous surgery* includes procedures directed at restoring the alveolar bone to its original level, whereas *subtractive osseous surgery* is designed to restore the form of preexisting alveolar bone to the level present at the time of surgery or slightly more apical to this level (Figure 60-1).

Additive osseous surgery brings about the ideal result of periodontal therapy; it implies regeneration of lost bone and reestablishment of the periodontal ligament, gingival fibers, and junctional epithelium at a more coronal level. This type of osseous surgery is discussed in Chapter 61.

Subtractive osseous surgery procedures provide an alternative to additive methods and should be used when additive procedures are not feasible. These subtractive procedures are discussed in this chapter.

SELECTION OF TREATMENT TECHNIQUE

The morphology of the osseous defect largely determines the treatment technique to be used. *One-wall angular defects* usually need to be recontoured surgically. *Three-wall defects,* particularly if they are narrow and deep, can be successfully treated with techniques that strive for new attachment and bone reconstruction. *Two-wall angular defects* can be treated with either method, depending on their depth, width, and general configuration. Therefore, except for one-wall defects and wide, shallow two-wall defects, along with

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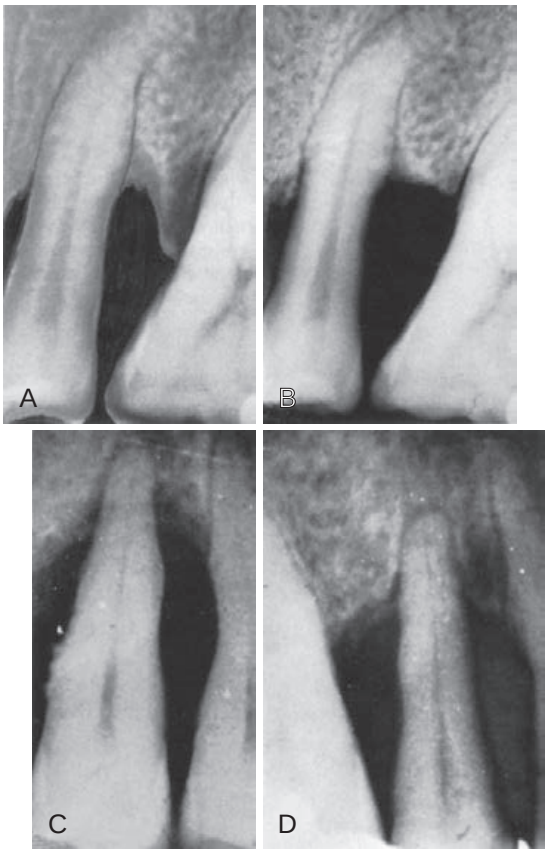

Figure 60-1 Additive and subtractive osseous surgery. **A,** Before, and **B,** immediately after subtractive osseous surgery; the osseous wall of the two adjoining infrabony pockets has been removed. **C,** Before, and **D,** 1 year after additive osseous surgery; the area has been flapped and thoroughly instrumented, resulting in reconstruction of the interdental and periapical bone. *(Courtesy Drs. E.A. Albano and B.O. Barletta, Buenos Aires, Argentina.)*

interdental craters, osseous defects are treated with the objective of obtaining optimal repair by natural healing processes.

RATIONALE

Osseous resective surgery necessitates following a series of strict guidelines for proper contouring of alveolar bone and subsequent management of the overlying gingival soft tissues. The specifics of these techniques are discussed later in this chapter. The techniques discussed here for osseous resective surgery have limited applicability in deep intrabony or hemiseptal defects, which could be treated with a different surgical approach (see Chapter 61). Osseous surgery provides the purest and surest method for reducing pockets with bony discrepancies that are not overly vertical and also remains one of the principal periodontal modalities because of its long-term success and predictability.

Osseous resective surgery is the most predictable pocket reduction technique.[10–12] However, more than any other surgical technique, osseous resective surgery is performed at the expense of bony tissue and attachment level.[1,2,8] Thus its value as a surgical approach is limited by the presence, quantity, and shape of the bony tissues and by the amount of attachment loss that is acceptable.

The major rationale for osseous resective surgery is based on the tenet that discrepancies in level and shapes of the bone and gingiva predispose patients to the recurrence of pocket depth postsurgically.[6] Although this concept is not universally accepted[3,5] and the procedure induces loss of radicular bone in the healing phase, recontouring of bone is the only logical treatment choice in some cases. The goal of osseous resective therapy is to reshape the marginal bone to resemble that of the alveolar process undamaged by periodontal disease. The technique is performed in combination with apically positioned flaps, and the procedure eliminates periodontal pocket depth and improves tissue contour to provide a more easily maintainable environment. The relative merits of pocket reduction procedures are discussed in Chapters 52 and 59; this chapter discusses the osseous resective technique and how and where it may be accomplished.

Figure 60-2 Photograph of a healthy bony periodontium in a skull. Although a slight amount of attachment may have been lost, this skull demonstrates the characteristics of normal form.

It is proposed that the conversion of the periodontal pocket to a shallow gingival sulcus enhances the patient's ability to remove plaque and oral debris from the dentition. Likewise, the ability of dental professionals to maintain the periodontium in a state free of gingivitis and periodontitis is more predictable in the presence of shallow sulci. The more effective the periodontal maintenance therapy, the greater is the longitudinal stability of the surgical result. The efficacy of osseous surgery therefore depends on its ability to affect pocket depth and to promote periodontal maintenance.[11,22,23] The merits of resection versus other treatment procedures are discussed in Chapter 52.

NORMAL ALVEOLAR BONE MORPHOLOGY

Knowledge of the morphology of the bony periodontium in a state of health is required to perform resective osseous surgery correctly (Figure 60-2). The characteristics of a normal bony form are as follows:

1. The interproximal bone is more coronal in position than the labial or lingual/palatal bone and pyramidal in form.
2. The form of the interdental bone is a function of the tooth form and the embrasure width. The more tapered the tooth, the more pyramidal is the bony form. The wider the embrasure, the more flattened is the interdental bone mesiodistally and buccolingually.
3. The position of the bony margin mimics the contours of the cementoenamel junction. The distance from the facial bony margin of the tooth to the interproximal bony crest is more flat in the posterior than the anterior areas. This "scalloping" of the bone on the facial surfaces and lingual/palatal surfaces is related to tooth and root form, as well as tooth position

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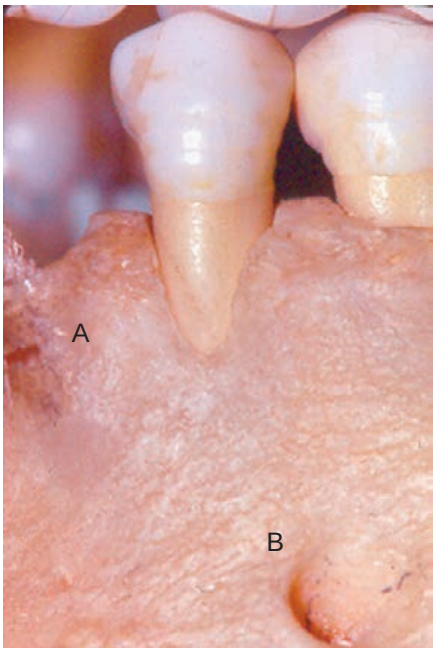

Figure 60-3 Effects of tooth position on facial bony contours. **A,** Bony fenestration. **B,** Bony dehiscence. These deformities can and should be detected by palpation, probing, and sounding before flap surgery.

within the alveolus. Teeth with prominent roots or those displaced to the facial or lingual side may also have fenestrations or dehiscences (Figure 60-3). The molar teeth have less scalloping and a more flat profile than bicuspids and incisors.

Although these general observations apply to all patients, the bony architecture may vary from patient to patient in the extent of contour, configuration, and thickness. These variations may be both normal and healthy.

TERMINOLOGY

Numerous terms have been developed to describe the topography of the alveolar housing, the procedure for its removal, and the resulting correction. These terms should be clearly defined.

Procedures used to correct osseous defects have been classified in two groups: osteoplasty and ostectomy.[7] *Osteoplasty* refers to reshaping the bone without removing tooth-supporting bone. *Ostectomy,* or *osteoectomy,* includes the removal of tooth-supporting bone. One or both of these procedures may be necessary to produce the desired result.

Terms that describe the bone form after reshaping can refer to morphologic features or to the thoroughness of the reshaping performed. Examples of morphologically descriptive terms include *negative, positive, flat,* and *ideal*. These terms all relate to a preconceived standard of ideal osseous form.

Positive architecture and *negative architecture* refer to the relative position of interdental bone to radicular bone (Figure 60-4). The architecture is "positive" if the radicular bone is apical to the interdental bone. The bone has "negative" architecture if the interdental bone is more apical than the radicular bone. *Flat architecture* is the reduction of the interdental bone to the same height as the radicular bone.

Osseous form is considered to be "ideal" when the bone is consistently more coronal on the interproximal surfaces than on the facial and lingual surfaces. The ideal form of the marginal bone has similar interdental height, with gradual, curved slopes between interdental peaks (Figure 60-5).

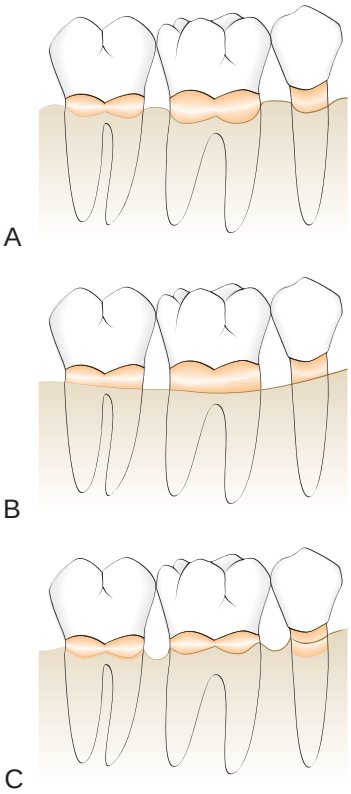

Figure 60-4 Diagram of types of bony architecture. **A,** Positive bony architecture. **B,** Flat bony architecture. **C,** Reversed, or negative, bony form.

Figure 60-5 Skull photograph of healthy periodontium. Note the shape of the alveolar bone housing. This bone is considered to have ideal form. It is more coronal in the interproximal areas, with a gradual slope around and away from the tooth.

Terms that relate to the thoroughness of the osseous reshaping techniques include "definitive" and "compromise." *Definitive osseous reshaping* implies that further osseous reshaping would not improve the overall result. *Compromise osseous reshaping* indicates a bone pattern that cannot be improved without significant osseous removal that would be detrimental to the overall result. References to compromise and definitive osseous architecture can be useful to

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the clinician, not as description of a morphologic feature, but as terms that express the expected therapeutic result.

FACTORS IN SELECTION OF RESECTIVE OSSEOUS SURGERY

The relationship between the depth and configuration of the bony lesion(s) to root morphology and the adjacent teeth determines the extent that bone and attachment is removed during resection. Bony lesions have been classified according to their configuration and number of bony walls.[9] The technique of ostectomy is best applied to patients with early to moderate bone loss (2 to 3 mm) with moderate-length root trunks[18] that have bony defects with one or two walls. These shallow-to-moderate bony defects can be effectively managed by osteoplasty and osteoectomy. Patients with advanced attachment loss and deep intrabony defects are not candidates for resection to produce a positive contour. To simulate a normal architectural form, so much bone would have to be removed that the survival of the teeth could be compromised.

Two-walled defects, or craters, occur at the expense of the interseptal bone. As a result, they have buccal and lingual/palatal walls that extend from one tooth to the adjacent tooth. The interdental loss of bone exposes the proximal aspects of both adjacent teeth. The buccal-lingual interproximal contour that results is opposite to the contour of the cementoenamel junction of the teeth (Figure 60-6, *A* and *B*). *Two-walled defects (craters) are the most common bony defects found in patients with periodontitis.*[14,20] If the facial and lingual plates of this bone are resected, the resultant interproximal contour would become more flattened or ovate (Figure 60-6, *C* and *D*). However, confining resection only to ledges and the interproximal lesion results in a facial and lingual bone form in which the interproximal bone is located more apically than the bone on the facial or lingual aspects of the tooth. This resulting anatomic form is reversed, or negative, architecture[17,18,22] (Figure 60-6, *C* and *D*).

Although the production of a reversed architecture minimizes the amount of ostectomy that is performed, it is not without consequences.[5] Peaks of bone typically remain at the facial and lingual/palatal line angles of the teeth *(widow's peaks).* During healing, the soft tissue tends to bridge the embrasure from the most coronal height of the bone on one tooth to the most coronal heights on the adjacent teeth. The result is therefore the tendency to replicate the attachment contour on the tooth. The interproximal soft tissues invest these peaks of bone, which may subsequently resorb with a tendency to rebound without gain in attachment over time. Interproximal pocket depth can recur.[22,24]

Ostectomy to a positive architecture requires the removal of the line-angle inconsistencies (widow's peaks), as well as some of the facial, lingual, and palatal and interproximal bone. The result is a loss of some attachment on the facial and lingual root surfaces but a topography that more closely resembles normal bone form before disease (Figure 60-6, *E* and *F*). Proponents of osseous resection to create a positive contour believe that this architecture, devoid of sharp angles and spines, is conducive to the formation of a more uniform and reduced soft tissue dimension postoperatively.[17,21] The therapeutic result is less pocket depth and increased ease of periodontal maintenance by the patient, dental hygienist, or dentist.

The amount of attachment lost from the use of ostectomy varies with the depth and configuration of the treated osseous defects. Osseous resection applied to two-wall intrabony defects (craters), the most common osseous defects, results in attachment loss at the proximal line angles and the facial and lingual aspects of the affected teeth without affecting the base of the pocket. The extent of attachment loss during resection to a positive architecture has been measured. When the technique is properly applied to appropriate patients, the mean reduction in attachment circumferentially around the tooth has been determined to be 0.6 mm at six probing sites.[22] In practical terms, this means that the technique is best applied to interproximal lesions 1 to 3 mm deep in patients with moderate-to-long root trunks.[17] Patients with deep, multiwalled defects are not candidates for resective osseous surgery. They are better treated with regenerative therapies or by combining osteoplasty to reduce bony ledges and to facilitate flap closure with new attachment and regeneration procedures.

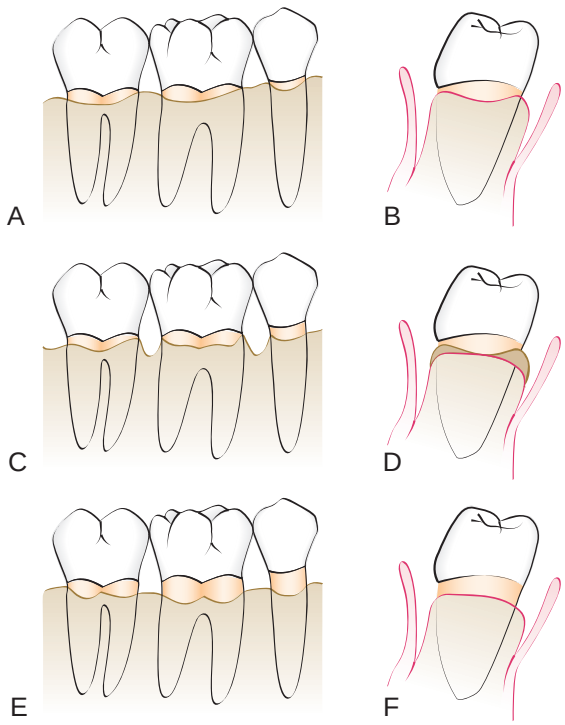

Figure 60-6 Effect of correction of craters. **A** and **B,** Diagram of facial and interproximal bony contours after flap reflection. Note the loss of some interproximal bone and cratering. **C** and **D,** Line angles; this is only osteoplasty and has resulted in a reversed architecture. **E** and **F,** Ostectomy on the facial and lingual bone and the removal of the residual widow's peaks to produce a positive bony architecture.

For more information on examination and treatment planning of osseous surgery, please visit the companion Expert CONSULT website at www.expertconsult.com.

SUMMARY

Although osseous surgical techniques cannot be applied to every bony abnormality or topographic modification, it has been clearly demonstrated that properly used osseous surgery can eliminate and modify defects, as well as gradualize excessive bony ledges, irregular alveolar bone, early furcation involvement, excessive bony exostosis, and circumferential defects. When properly performed, resective osseous surgery achieves a physiologic architecture of marginal alveolar bone conducive to gingival flap adaptation with minimal probing depth. The advantages of this surgical modality include a

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SCIENCE TRANSFER

Osseous surgery is an effective treatment for patients with moderate bone loss and craters up to 3 mm deep and/or early furcation defects, as well as for bone contour irregularities such as exostoses and ledges. It results in some attachment loss, but by developing bone contours that parallel healthy gingival margin shapes, it gives a stable, new periodontium that can be maintained for years.

The most important determinant for successful periodontal surgical therapy is adequate plaque control, thus all patients must demonstrate satisfactory oral hygiene levels before and after resective osseous surgery. If a patient is unable to achieve 80% or more plaque-free surfaces, surgical therapy is contraindicated and nonsurgical approaches and maintenance are the endpoints of treatment until plaque scores are improved. In areas of esthetic concern, resective osseous surgery can result in increased gingival recession and should not be used in these patients. Patients with advanced bone loss are better treated with regenerative surgical techniques, but in many cases, it is possible to combine resective osseous surgery and regenerative techniques for different periodontal defects treated during the same flap surgery.

predictable amount of pocket reduction that can enhance oral hygiene and periodic maintenance. It also preserves the width of the attached tissue while removing granulomatous tissue and providing access for debridement of the radicular surfaces. In addition, the osseous resection technique permits recontouring of bony abnormalities, including hemiseptal defects, tori, and ledges. Its substantial benefits include proper assessment for restorative procedures (e.g., crown lengthening) and assessment of restorative overhangs and tooth abnormalities (e.g., enamel projections, enamel pearls, perforations, fractures). Therefore resective osseous surgery can be an important technique in the armamentarium necessary to provide a maintainable periodontium for periodontal patients.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 61

Reconstructive Periodontal Surgery

Henry H. Takei, Marc L. Nevins, David L. Cochran, Fermin A. Carranza, and Mark A. Reynolds

CHAPTER OUTLINE

EVALUATION OF NEW ATTACHMENT AND PERIODONTAL RECONSTRUCTION (online only)
- Clinical Methods
- Radiographic Methods
- Surgical Reentry
- Histologic Methods

RECONSTRUCTIVE SURGICAL TECHNIQUES
- Non–Bone Graft–Associated Procedures
- Graft Materials and Procedures
- Biologic Mediators
- Enamel Matrix Proteins
- Combined Techniques

SUMMARY

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content and a supplement to this chapter.

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content on the evaluation of new attachment and periodontal reconstruction.

RECONSTRUCTIVE SURGICAL TECHNIQUES

Reconstructive techniques can be subdivided into two major types: non–bone graft–associated new attachment and bone graft–associated new attachment. Many procedures combine both approaches.[71,83]

All recommended techniques include careful case selection and complete removal of all irritants on the root surface. Although this can be done in some cases as a closed procedure, in the great majority of cases it should be done after exposure of the area with a flap.[3,4] Flap design and incisions should follow the description given in Chapter 59 for flaps used in reconstructive surgery. Trauma from occlusion, as well as other factors, may impair posttreatment healing of the supporting periodontal tissues, reducing the likelihood of new attachment. Occlusal adjustment, if needed, is therefore indicated.

Systemic antibiotics are generally used after reconstructive periodontal therapy, although definitive information on the advisability of this measure is still lacking. Case reports have shown extensive reconstruction of periodontal lesions after scaling, root planing, and curettage, with systemic and local treatment using penicillin or tetracycline, in combination with other forms of therapy.[28,132]

Non–Bone Graft–Associated Procedures

Periodontal reconstruction can be attained without the use of bone grafts in meticulously treated three-wall defects (intrabony defects) and in periodontal and endodontic abscesses.[28,89,153] New attachment is more likely to occur when the destructive process has occurred rapidly such as after treatment of pockets complicated by acute periodontal abscesses and after treatment of acute necrotizing ulcerative lesions.[136]

The following sections discuss the rationale and techniques that must be considered for a successful outcome in periodontal bone regeneration; some of which are: (1) the removal of the junctional and pocket epithelium; (2) the prevention of their migration into the healing area after therapy; (3) clot stabilization, wound protection, and space creation; (4) guided tissue regeneration; (5) the biomodification of the root surface; (6) selection of the proper graft materials; (7) biologic mediators (growth factors) and enamel matrix proteins to enhance or direct healing; and finally (8) the combination of graft materials, membranes, and biologic mediators used to enhance new attachment and bone growth.

Removal of Junctional and Pocket Epithelium. Since the earliest attempts at periodontal new attachment, the presence

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of junctional and pocket epithelium has been perceived as a barrier to successful therapy because its presence interferes with the direct apposition of connective tissue and cementum, thus limiting the height to which periodontal fibers can insert to the cementum.[86,131,156,212]

Several methods have been recommended to remove the junctional and pocket epithelium. These include curettage, chemical agents, ultrasonics, lasers, and surgical techniques.

Curettage. Results of removal of the epithelium by means of curettage vary from complete removal to persistence of as much as 50%.[183] Therefore curettage is not a reliable procedure. Ultrasonic methods, lasers, and rotary abrasive stones have also been used, but their effects cannot be controlled because of the clinician's lack of vision and tactile sense when using these methods.

Chemical Agents. Chemical agents have also been used to remove pocket epithelium, usually in conjunction with curettage. The drugs used most often have been sodium sulfide, phenol camphor, Antiformin, and sodium hypochlorite. However, the effect of these agents is not limited to the epithelium, and their depth of penetration cannot be controlled. These drugs are mentioned here for their historic interest.

Surgical Techniques. Surgical techniques have been recommended to eliminate the pocket and junctional epithelium. The excisional new attachment procedure consists of an internal bevel incision performed with a surgical knife, followed by removal of the excised tissue.[213] No attempt is made to elevate a flap. After careful scaling and root planing, interproximal sutures are used to close the wound (see Chapter 57).

Glickman[74] and Prichard[152] have advocated performing a *gingivectomy to the crest of the alveolar bone* and debriding the defect. Excellent results have been obtained with this technique in uncontrolled human studies.[8,153]

The modified Widman flap, as described by Ramfjord and Nissle,[157] is similar to the excisional new attachment procedure but is followed by elevation of a flap for better exposure of the area. The internal bevel incision eliminates the pocket epithelium (see Chapter 59).

Prevention or Impeding the Epithelial Migration. Elimination of the junctional and pocket epithelium may not be sufficient because the epithelium from the excised margin may rapidly proliferate to become interposed between the healing connective tissue and the cementum.

For experimental purposes, several investigators have analyzed in animals and humans the effect of excluding the epithelium by amputating the crown of the tooth and covering the root with the flap ("root submergence").[12,13,17] This experimental technique not only excludes the epithelium but also prevents microbial contamination of the wound during the reparative stages. Successful repair of osseous lesions in the submerged environment was reported, but obviously this method has little or no clinical application.

Two other methods have been proposed to prevent or impede the migration of the epithelium. One consists of the total removal of the interdental papilla covering the defect and its replacement with a free autogenous graft obtained from the palate.[54] During healing, the graft epithelium necroses and is slowly replaced by proliferating epithelium from the gingival surface. The graft simply delays the epithelium from proliferating into the healing area. This method has not been widely used.

The second approach is the use of *coronally displaced flaps,* which increase the distance between the epithelial wound edge and the healing area. This technique is particularly suitable for the treatment of mandibular molar furcations and has been used mostly in conjunction with citric acid treatment of the roots.[69,114] Periodontal regeneration after the use of this technique has been demonstrated histologically in humans.[180]

Clot Stabilization, Wound Protection, and Space Creation. Some investigators have attributed the successful results reported with graft materials, barrier membranes, and coronally displaced flaps to the fact that these techniques protect the wound and create a space for undisturbed and stable maturation of the clot.[70,84,85] This hypothesis suggests that preservation of the root surface fibrin clot interface prevents apical migration of the gingival epithelium and allows for connective tissue attachment during the early wound-healing period.[70,208]

The importance of space creation for bone repair has long been recognized in orthopedic and maxillofacial surgery. Transference of this concept to periodontal therapy has been explored for treatment of periodontal and periimplant osseous defects and for root coverage. The space can be created by using a titanium-reinforced expanded polytetrafluoroethylene (ePTFE) membrane to prevent its collapse. For the study of reconstructive techniques, these membranes were placed over experimentally created supraalveolar bone defects in dogs, and considerable bone reconstruction was reported.[182] The following is a discussion of the guided tissue regeneration technique.

Guided Tissue Regeneration. The method for the prevention of epithelial migration along the cemental wall of the pocket and maintaining space for clot stabilization is a technique called *guided tissue regeneration* (GTR). This method is derived from the classic studies of Nyman, Lindhe, Karring, and Gottlow and is based on the assumption that only the periodontal ligament cells have the potential for regeneration of the attachment apparatus of the tooth.[42,77,78,140,141] GTR consists of placing barriers of different types (membranes) to cover the bone and periodontal ligament, thus temporarily separating them from the gingival epithelium and connective tissue. Excluding the epithelium and the gingival connective tissue from the root surface during the postsurgical healing phase not only prevents epithelial migration into the wound but also favors repopulation of the area by cells from the periodontal ligament and the bone[33] (Figure 61-9; see also Chapter 35).

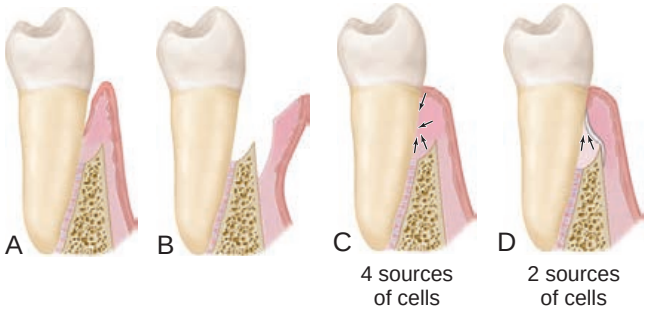

Figure 61-9 **A,** Periodontal pocket with angular bone loss. **B,** Full-thickness flap reflected. **C,** Flap is closed without a membrane. Note four sources of cells can occupy the defect. **D,** Flap closed with membrane in place. Only bone and PDL cells can occupy the defect.

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Initial animal experiments using Millipore filters and Teflon membranes resulted in regeneration of cementum and alveolar bone and a functional periodontal ligament.[32,34,38,129] Clinical case reports presented GTR results in a gain in attachment level that is not necessarily associated with a buildup of alveolar bone.[9,10] Histologic studies in humans provided evidence of periodontal reconstruction in most cases, even with horizontal bone loss.[78,188,192]

The use of polytetrafluoroethylene (PTFE) membranes has been tested in controlled clinical studies in mandibular molar furcations and has shown statistically significant decreases in pocket depths and improvement in attachment levels after 6 months, but bone level measurements have been inconclusive.[107,151] A study on maxillary molar furcations did not result in significant gain in attachment or bone levels.[128]

The initial membranes developed were nonresorbable and required a second surgical procedure to remove them. This second procedure was accomplished after the initial stages of healing, usually 3 to 6 weeks after the first intervention. The second procedure was a significant obstacle in the utilization of the GTR technique, therefore the resorbable membranes were developed.[206]

The ePTFE membrane (nonresorbable) can be obtained in different shapes and sizes to suit proximal spaces and facial/lingual surfaces of furcations (Figure 61-10). The technique for its use is as follows (see Figure 61-21):

1. Raise a mucoperiosteal flap with vertical incisions, extending a minimum of two teeth anteriorly and one tooth distally to the tooth being treated.
2. Debride the osseous defect and thoroughly plane the roots.
3. Trim the membrane to the approximate size of the area being treated. The apical border of the material should extend 3 to 4 mm apical to the margin of the defect and laterally 2 to 3 mm beyond the defect. The occlusal border of the membrane should be placed 2 mm apical to the cementoenamel junction.[10]
4. Suture the membrane tightly around the tooth with a sling suture.
5. Suture the flap back in its original position or slightly coronal to it, using independent sutures interdentally and in the vertical incisions. The flap should cover the membrane completely.
6. The use of periodontal dressings is optional, and the patient is placed on antibiotic therapy for 1 week.

After 4 to 6 weeks, the margin of the membrane may become exposed. The membrane is removed carefully, minimizing trauma to the underlying tissue. If it cannot be removed easily, the tissues are anesthetized, and the material is surgically removed using a small flap.

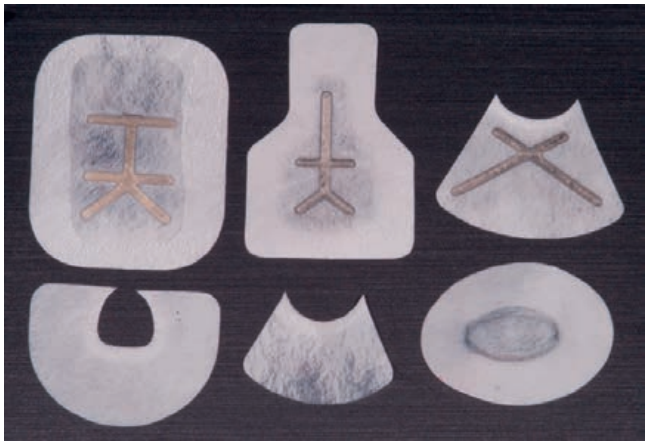

Figure 61-10 Nonresorbable membranes of different shape and size. Note membranes with titanium support.

The results obtained with the GTR technique are enhanced when the technique is combined with grafts placed in the defects (see section on combined techniques).

Biodegradable Membranes. The search for resorbable membranes has included trials and tests with numerous materials and collagens from different species such as bovine, porcine, Cargile membrane derived from the cecum of an ox, polylactic acid, Vicryl (polyglactin 910), synthetic skin (Biobrane), and freeze-dried dura mater.* Clinical studies with a mixture of co-polymers derived from polylactic acid and acetyl tributylcitrate resorbable membranes (Guidor membrane, no longer on the market) and a poly-D,L-lactide-co-glycolide (Resolut membrane, also no longer on the market) have shown significant gains in clinical attachment and bone fill.[43,68,198]

Resorbable membranes marketed in the United States (US) include *OsseoQuest* (Gore), a combination of polyglycolic acid, polylactic acid, and trimethylene carbonate that resorbs at 6 to 14 months; *BioGuide* (Osteohealth), a bilayer porcine-derived collagen; *Atrisorb* (Block Drug), a polylactic acid gel; and *BioMend* (Calcitech), a bovine Achilles tendon collagen that resorbs in 4 to 18 weeks. Of these, BioGuide is the most popular resorbable membrane.

The use of membranes is usually combined with autogenous bone from adjacent areas or other graft materials and root biomodifiers. These combined techniques are discussed at the end of this chapter.

The potential of using autogenous periosteum as a membrane and also to stimulate periodontal regeneration has been explored in two controlled clinical studies, one of grade II furcation involvements in mandibular molars and another of interdental defects.[103,106] The periosteum was obtained from the patient's palate by means of a window flap. Both studies reported that autogenous periosteal grafts can be used in GTR and result in significant gains in clinical attachment and osseous defect fill.

Biomodification of Root Surface. Changes in the tooth surface wall of periodontal pockets (e.g., degenerated remnants of Sharpey's fibers, accumulation of bacteria and their products, disintegration of cementum and dentin) interfere with new attachment. Although these obstacles to new attachment can be eliminated by thorough root planing, the root surface of the pocket can be treated to improve its chances of accepting the new attachment of gingival tissues.

Several substances have been proposed for this purpose, including citric acid, fibronectin, and tetracycline.

For more information on biomodification of root surface, please visit Expert CONSULT website at www.expertconsult.com.

Graft Materials and Procedures

Numerous therapeutic grafting modalities for restoring periodontal osseous defects have been investigated. The following classifications of bone graft material are important to note: (1) autografts are bone obtained from the same individual; (2) allografts are bone obtained

*References 14, 15, 37, 60, 61, 76, 112, 113, 146–148, 206, 215.

from a different individual of the same species; and (3) xenografts are bone from a different species.

Bone graft materials are also evaluated based on their osteogenic, osteoinductive, or osteoconductive potential. *Osteogenesis* refers to the formation or development of new bone by cells contained in the graft. *Osteoinduction* is a chemical process by which molecules contained in the graft (e.g., bone morphogenetic proteins) convert the neighboring cells into osteoblasts, which in turn form bone. *Osteoconduction* is a physical effect by which the matrix of the graft forms a scaffold that favors outside cells to penetrate the graft and form new bone.

Periodontal defects as sites for transplantation differ from osseous cavities surrounded by bony walls. Saliva and bacteria may easily penetrate along the root surface, and epithelial cells may proliferate into the defect, resulting in contamination and possible exfoliation of the grafts. Therefore the principles established to govern transplantation of bone or other materials into closed osseous cavities are not fully applicable to transplantation of bone into periodontal defects.[53]

Schallhorn[174] defined the considerations that govern the selection of a material as follows: biologic acceptability, predictability, clinical feasibility, minimal operative hazards, minimal postoperative sequelae, and patient acceptance. It is difficult to find a material with all these characteristics and to date there is no ideal material or technique.

Graft materials have been developed and tried in many forms. To familiarize the reader with various types of graft material, as defined by either the technique or the material used, a brief discussion of each is provided.

All grafting techniques require presurgical scaling, occlusal adjustment as needed, and exposure of the defect with a full-thickness flap. The flap technique best suited for grafting purposes is the *papilla preservation flap* because it provides complete coverage of the interdental area after suturing (see Chapter 59). The use of antibiotics after the procedure is generally recommended.

Autogenous Bone Grafts

Bone from Intraoral Sites. In 1923, Hegedüs[87] attempted to use bone grafts for the reconstruction of bone defects produced by periodontal disease. The method was revived by Nabers and O'Leary[135] in 1965, and numerous efforts have been made since that time to define its indications and technique.

Sources of bone include bone from healing extraction wounds, bone from edentulous ridges, bone trephined from within the jaw without damaging the roots, newly formed bone in wounds especially created for the purpose, bone removed from tuberosity, and the ramus and bone removed during osteoplasty and ostectomy.[30,81,89,90,164]

Osseous Coagulum. Robinson described a technique using a mixture of bone dust and blood that he termed "osseous coagulum."[164] The technique uses small particles ground from cortical bone. The advantage of the particle size is that it provides additional surface area for the interaction of cellular and vascular elements.

Sources of the graft material include the lingual ridge on the mandible, exostoses, edentulous ridges, the bone distal to a terminal tooth, bone removed by osteoplasty or ostectomy, and the lingual surface of the mandible or maxilla at least 5 mm from the roots. Bone is removed with a carbide bur #6 or #8 at speeds between 5000 and 30,000 rpm, placed in a sterile dappen dish and used to fill the defect (Figure 61-11). The obvious advantage of this technique is the ease of obtaining bone from a area already exposed during surgery. The disadvantages are its relatively low predictability and the inability to procure adequate material for large defects.[54] Although notable success has been reported by many individuals (Figure 61-12), studies documenting the efficacy of the technique are still inconclusive.[44,64,66,163]

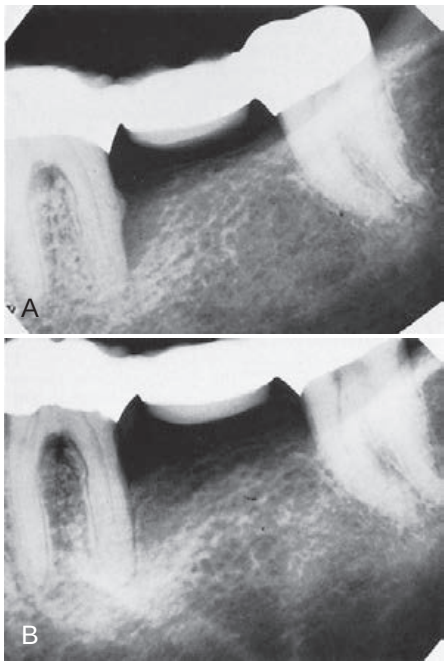

Figure 61-11 Bone defect on the distal root of a first molar treated with osseous coagulum implants. **A,** Before treatment. **B,** One year after treatment, showing bone repair. *(Courtesy Dr. R. Earl Robinson.)*

Bone Blend. Some disadvantages of osseous coagulum derive from the inability to use aspiration during accumulation of the coagulum. Another problem is the unknown quantity and quality of the bone fragments in the collected material. To overcome these problems, the "bone blend technique" has been proposed.[48]

The bone blend technique uses an autoclaved plastic capsule and pestle. Bone is removed from a predetermined site, triturated in the capsule to a workable, plasticlike mass, and packed into bony defects. Froum et al[64–66] found osseous coagulum–bone blend procedures to be at least as effective as iliac autografts and open curettage.

Cancellous Bone Marrow Transplants. Cancellous bone can be obtained from the maxillary tuberosity, edentulous areas, and healing sockets.[86] The maxillary tuberosity frequently contains abundant cancellous bone, particularly if the third molars are not present. After a ridge incision is made distally from the last molar, bone is removed with a curved rongeur. Care should be taken not to extend the incision too far distally to avoid entering the mucosal tissue of the pharyngeal area. Also, the location of the maxillary sinus must be analyzed on the radiograph to avoid entering or disturbing it.

Edentulous ridges can be approached with a flap, and cancellous bone and marrow are removed with curettes, back-action chisels (Figure 61-13), or trephine (Figure 61-14). Extraction sockets are allowed to heal for 8 to 12 weeks before reentering and removing the newly formed bone from the apical portion, which is used as the donor material.

Bone Swaging. The bone swaging technique requires an edentulous area adjacent to the defect, from which the bone is pushed

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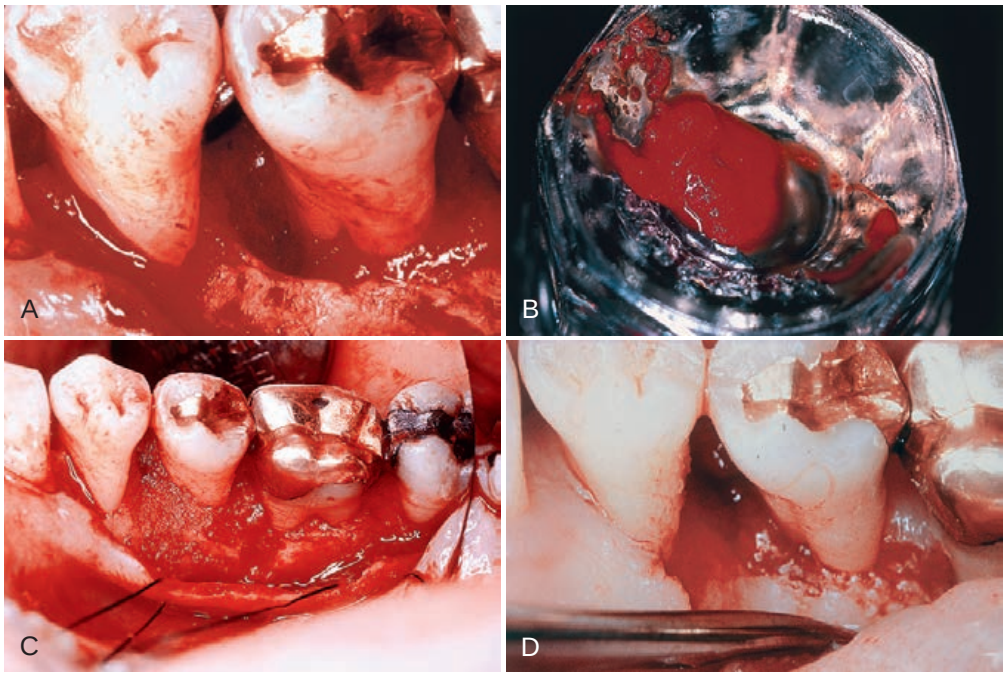

Figure 61-12 **A,** Osseous defect mesial to a second premolar. **B,** Graft material placed in dappen dish before transfer to the graft site. **C,** Material in place. **D,** Reentry 6 months later. *(Courtesy Dr. E. Earl Robinson.)*

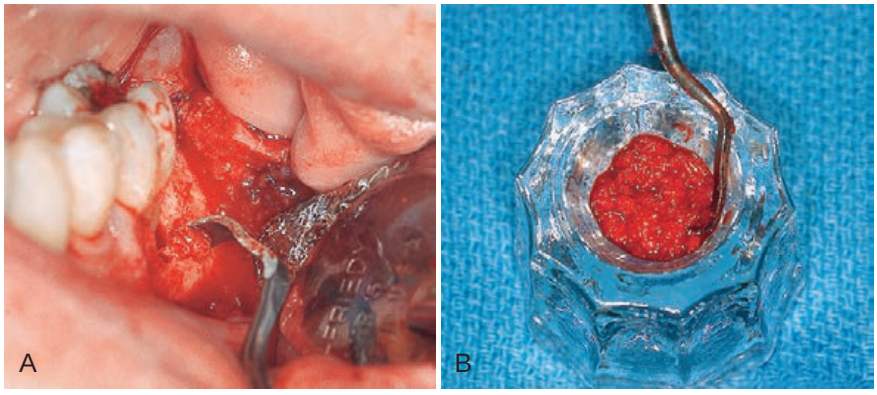

Figure 61-13 **A,** Bone particles obtained by use of back-action chisel, and **B,** placed in dappen dish.

into contact with the root surface without fracturing the bone at its base.[56,166] Bone swaging is technically difficult, and its usefulness is limited.

Bone from Extraoral Sites. In 1923, Hegedüs also pioneered the use of extraoral sites as a source of bone for grafting into periodontal osseous defects, using bone from the tibia. Schallhorn and Hiatt revived this approach in the 1960s using the iliac crest (see next discussion).

Iliac Autografts. The use of fresh or preserved iliac cancellous marrow bone has been extensively investigated. This material has been used by orthopedic surgeons for years. Data from human and animal studies support its use, and the technique has proved successful in osseous defects with various numbers of walls. It has also been successful in furcations and even supracrestally to some extent.* However, because of numerous problems associated with its use, the technique is no longer in use.[172] Some of the problems were postoperative infection, bone exfoliation, sequestration, varying rates of healing, root resorption, and rapid recurrence of the defect (Figure 61-15). Other problems were increased patient expense and difficulty in procuring the donor material.[22,50,173,174]

Allografts. Obtaining donor material for autograft purposes necessitates inflicting surgical trauma on another part of the patient's body. Obviously, it would be to the patient's and therapist's advantage if a suitable substitute could be used for grafting purposes that would offer similar potential for repair and not require the additional surgical removal of donor material from the patient. However, both allografts and xenografts are foreign to the patient and therefore have the potential to provoke an immune response. Attempts have been made to suppress the antigenic potential of allografts and xenografts by radiation, freezing, and chemical treatment.[21]

Bone allografts are commercially available from tissue banks. They are obtained from cortical bone within 12 hours of the death of the donor, defatted, cut in pieces, washed in absolute alcohol, and deep-frozen. The material may then be demineralized, and subsequently ground and sieved to a particle size of 250 to 750 µm and freeze-dried. Finally, it is vacuum-sealed in glass vials.

*References 11, 22, 46, 49, 50, 171, 172, 177.

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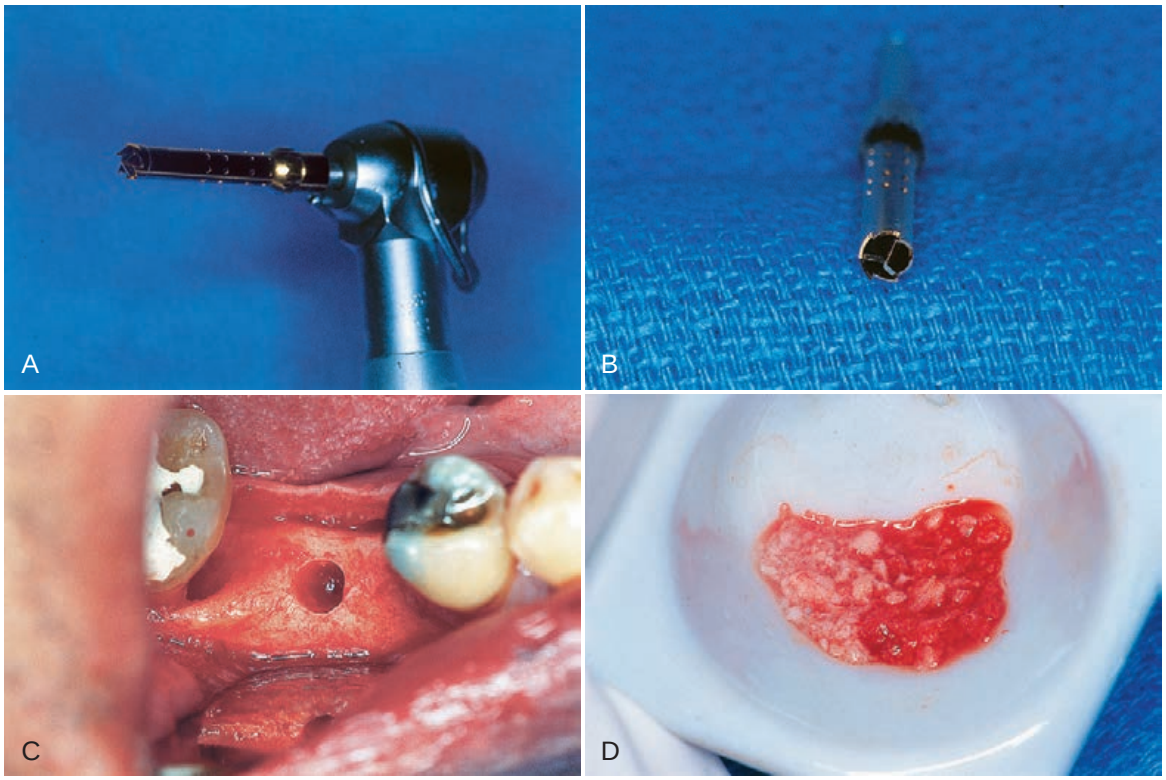

Figure 61-14 **A,** Trephine used for obtaining bone from adjoining jaw area. **B,** Frontal view of trephine. **C,** Donor site after harvesting. **D,** Harvested material placed in dappen dish.

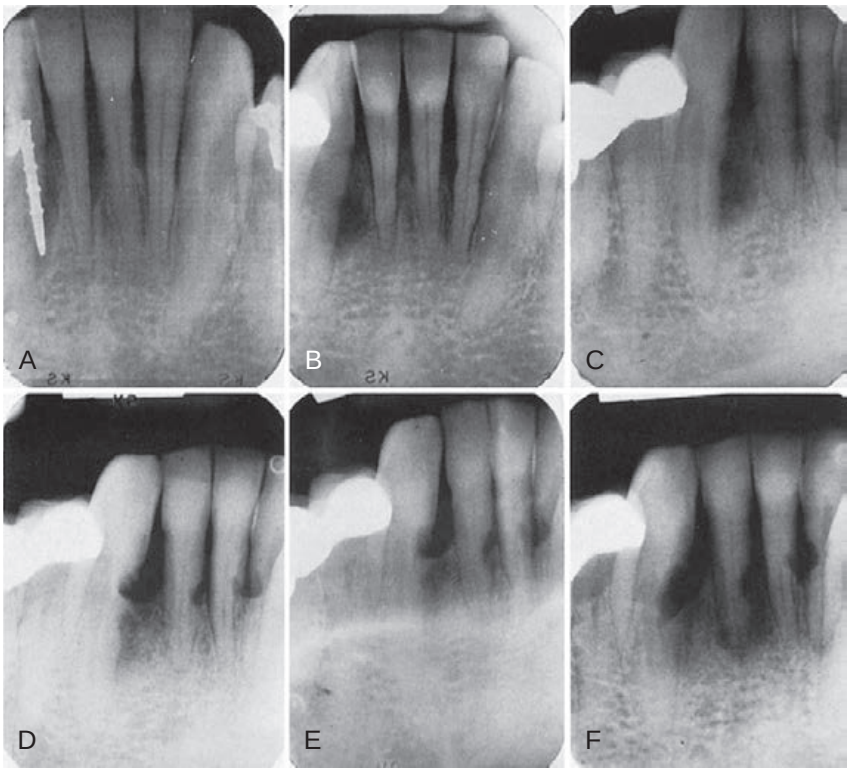

Figure 61-15 **A,** November 1973. Radiograph of a patient immediately before placement of a fresh iliac autograft. **B,** Two months later, bone repair is evident. Note the early radiolucent areas on the mesial aspect of the canine. **C,** After 7 months, "bone fill" is occurring, but obvious root resorption is present. **D,** April 1975. Root resorption is apparent on all grafted teeth. Note the obvious degree of fill of the original bone defects. **E,** February 1976. Further involvement. **F,** October 1977. Four years later, root resorption has progressed into the pulp of the lateral incisor, causing a periosteal-endosteal complication.

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Numerous steps are also taken to eliminate viral infectivity. These include exclusion of donors from known high-risk groups and various tests on the cadaver tissues to exclude individuals with any type of infection or malignant disease. The material is then treated with chemical agents or strong acids to inactivate the virus, if still present. The risk of human immunodeficiency virus (HIV) infection has been calculated as 1 in 1 to 8 million and is therefore characterized as highly remote.[126]

Undecalcified Freeze-Dried Bone Allograft. Several clinical studies by Mellonig, Bowers, and co-workers reported bone fill exceeding 50% in 67% of the defects grafted with freeze-dried bone allograft (FDBA) and in 78% of the defects grafted with FDBA plus autogenous bone.[125,135,167,180] FDBA, however, is considered an osteoconductive material, whereas demineralized FDBA (DFDBA) is considered an osteoinductive graft. Laboratory studies have found that DFDBA has a higher osteogenic potential than FDBA and is therefore preferred.[120,123,124]

Demineralized Freeze-Dried Bone Allograft. Experiments by Urist[201–203] have established the osteogenic potential of DFDBA. Demineralization in cold, diluted hydrochloric acid exposes the components of bone matrix, which are closely associated with collagen fibrils and have been termed *bone morphogenetic proteins* (BMPs).[36,203]

In 1975, Libin et al[110] reported three patients with 4 to 10 mm of bone regeneration in periodontal osseous defects. Subsequent clinical studies were made with cancellous DFDBA and cortical DFDBA.[144,154] The latter resulted in more desirable results (2.4 mm versus 1.38 mm of bone fill).

Bowers et al,[17] in a histologic study in humans, showed new attachment and periodontal regeneration in defects grafted with DFDBA. Mellonig et al[123,124] tested DFDBA against autogenous materials in the calvaria of guinea pigs and showed it to have similar osteogenic potential.

These studies provided strong evidence that DFDBA in periodontal defects results in significant probing depth reduction, attachment level gain, and osseous regeneration (Figure 61-16).

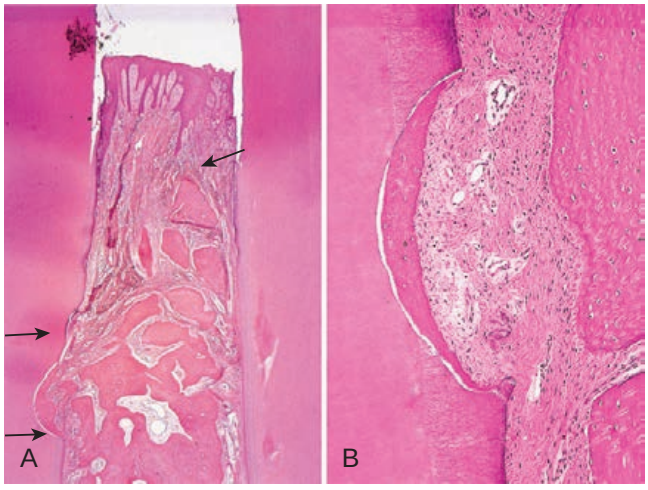

Figure 61-16 A, Six-month postoperative histologic block section of experimental site grafted with decalcified freeze-dried bone, depicting osseous regeneration coronal to the crestal bone notch. **B,** Higher magnification of reference notch through calculus at base of defect, showing new cementum (artifactual split during histologic preparation), new bone, and new periodontal ligament. *(Courtesy Dr. Gerald Bowers, University of Maryland, College Park, MD.)*

The combination of DFDBA and GTR has also proved to be very successful[6,175]; however, limitations of the use of DFDBA include the possible, although remote, potential of disease transfer from the cadaver.

A bone-inductive protein isolated from the extracellular matrix of human bones, termed *osteogenin* or *BMP-3,* has been tested in human periodontal defects and seems to enhance osseous regeneration.[18] This bone inductive protein is discussed later in this chapter.

Xenografts. Bone products from other species have a long history of use in periodontal therapy. A few of these xenograft products are mentioned here for historic purposes but are no longer used today.

Calf bone (Boplant), treated by detergent extraction, sterilized, and freeze-dried, has been used for the treatment of osseous defects.[7,170] Kiel bone is calf or ox bone denatured with 20% hydrogen peroxide, dried with acetone, and sterilized with ethylene oxide. Anorganic bone is ox bone from which the organic material has been extracted by means of ethylenediamine; it is then sterilized by autoclaving.[119] These materials have been tried and discarded for various reasons.

Currently, an anorganic, bovine-derived bone marketed under the brand name Bio-Oss (Osteohealth) has been successfully used both for periodontal defects and in implant surgery. It is an osteoconductive, porous bone mineral matrix from bovine cancellous or cortical bone. The organic components of the bone are removed, but the trabecular architecture and porosity are retained.[26,118] The physical features permit clot stabilization and revascularization to allow for migration of osteoblasts, leading to osteogenesis. Bio-Oss is biocompatible with the adjacent tissues, eliciting no systemic immune response.

Several studies have reported successful bone regeneration and new attachment with Bio-Oss in periodontal defects,[26,122] as well as regeneration around implants and sinus grafting (Figure 61-17) (see Chapter 72).

Periodontally, Bio-Oss has been used as a graft material covered with a resorbable membrane (BioGuide). The membrane prevents the migration of fibroblasts and connective tissues into the pores and between the granules of the graft. Histologic studies of this technique have shown significant osseous regeneration and cementum formation.

Yukna et al[219] have used Bio-Oss in combination with a cell-binding polypeptide (P-15) that is a synthetic analog of a 15–amino acid sequence of type I collagen. marketed as Pepgen P-15 (Dentsply/Ceramed); this combination seems to enhance the bone-regenerative results of the matrix alone in periodontal defects.

Nonbone Graft Materials. In addition to bone graft materials, many nonbone graft materials have been used in an attempt to restore the periodontium. These include sclera, dura, cartilage, cementum, dentin, plaster of Paris, plastic materials, ceramics, and coral-derived materials.* None offers a reliable substitute to bone graft materials; some of these materials are briefly presented here to offer a complete picture of the many attempts that have been made to solve the critical problem of periodontal regeneration.

For more information on nonbone graft materials, please visit Expert CONSULT website at www.expertconsult.com.

*References 23, 55, 108, 109, 137, 169, 179.

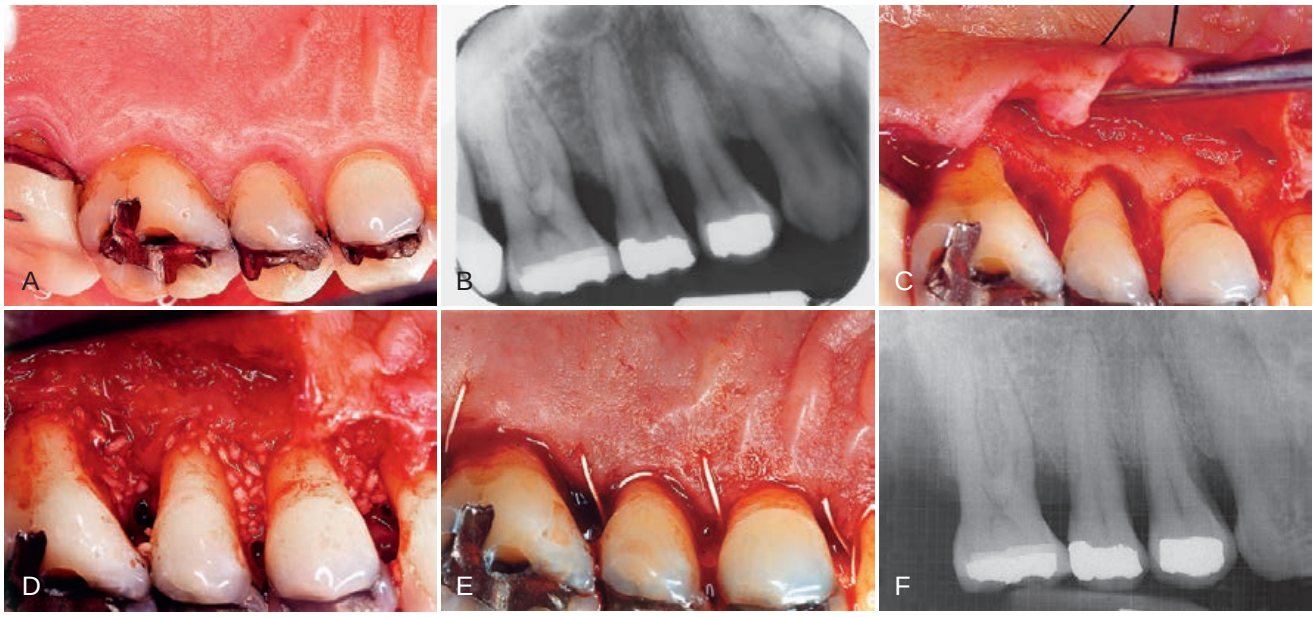

Figure 61-17 **A,** Palatal view of premolar and molar area. **B,** Radiographs show bone loss mesially to first molar and first and second premolars. **C,** After raising a flap, areas of periodontal bone loss are clearly seen. **D,** Teeth undergo careful root planing, and Bio-Oss graft is placed in the defects. **E,** Area is sutured. **F,** Postoperative radiograph at 9 months shows bone repair. *(Courtesy Dr. Philip Melnick, Los Angeles.)*

Biologic Mediators

As indicated in this chapter, clinicians have tried to enhance bone formation in periodontal defects by adding various bone grafting substitutes in the hope that these materials might be sufficient to stimulate the regeneration of cementum, periodontal ligament, and alveolar bone.[161] Such attempts have resulted in some regeneration, but this is usually limited to the base or apical aspect of the defect and the resultant tissue formation is not sufficient in terms of quantity or predictability. For these reasons, new therapeutic approaches for periodontal regeneration have been sought. The recognition and appreciation that new tissues are formed by cell populations have resulted in efforts to stimulate the cells that are located in the periodontal defect.[134] One way to stimulate these cells is to use proteins (growth factors) that can bind to surface receptors on the cell membranes, which in turn trigger a series of events to occur that alter the genetic activity of the cell with the result that cell behavior is stimulated.[40,73,200] Most growth factors stimulate one of two cell behaviors: one is cellular proliferation (cell division and an increase in the number of cells) and the other is cellular differentiation (the cells produce terminal products [e.g., fibroblasts produce collagen and osteoblasts produce bone]). These growth factors, primarily secreted by macrophages, endothelial cells, fibroblasts, and platelets, include platelet-derived growth factor (PDGF), insulinlike growth factor (IGF), basic fibroblast growth factor (bfGF), BMP, and transforming growth factor (TGF). These biologic mediators have been used to stimulate periodontal wound healing (e.g., promoting migration and proliferation of fibroblasts for periodontal ligament formation) or to promote the differentiation of cells to become osteoblasts, thereby favoring bone formation.[39,41,79,94,145]

The addition of PDGF, for example, has been shown to enhance bone formation in periodontal osseous defects. In a two-center, prospective, randomized, double-masked human trial, 38 patients with bilateral defects were treated in a split-mouth design. Control subjects received conventional periodontal flap surgery and similar surgery, including the vehicle (carrier material). No local or systemic safety issues were noted, and no patients developed antibodies to the recombinant human growth factors. Two PDGF doses were tested, and the results demonstrated significant bone fill increases in defects treated with the larger amount. Such trials indicate current attempts to deliver growth factors to periodontal defects to stimulate a cascade that results in periodontal tissue formation. Many questions remain about the best vehicle or carrier material, application of the growth factors, and the release kinetics of the material.

Currently, recombinant human PDGF (rhPDGF) -BB is approved by the US Food and Drug Administration (FDA) only for periodontal regeneration as part of the dental bone filling device GEM 21S. This commercial product is indicated in the treatment of intrabony periodontal defects, furcation periodontal defects, and gingival recession associated with periodontal defects. GEM 21S consists of 0.5 cc of beta-tricalcium phosphate (β-TCP) particles, (0.25 to 1.0 mm), which acts as the carrier, and 0.5 ml rhPDGF-BB (0.3 mg/ml). The effectiveness of GEM 21S has not been established in other regenerative indications such as alveolar bone defects and sinus augmentation. The clinical effectiveness of rhPDGF-BB and β-TCP in the treatment of periodontal intrabony defects has been rigorously tested in an 180 patient clinical trial,[138] and "proof-of-principle" histologic evidence of regeneration has been shown.[127,162] Although this device uses β-TCP as the scaffold/carrier, there has been considerable clinical interest in combining rhPDGF-BB with other bone replacement grafts, particularly bone allografts and xenografts.

Clinical case reports also provide information efficacy series on the clinical of combinatorial approaches using PDGF-BB with bone allografts. Camelo et al[27] and Nevins et al[138] reported human histologic evidence of periodontal regeneration in intraosseous defects and furcation defects treated using a combination of rhPDGF-BB and DFDBA (Figures 61-18 and 61-19).

The other growth factor that has been studied and is of great interest in bone regeneration is BMP. Besides the bone replacement graft materials, including autograft, allograft, xenograft, or alloplast, the formation of bone can be enhanced when growth factors are

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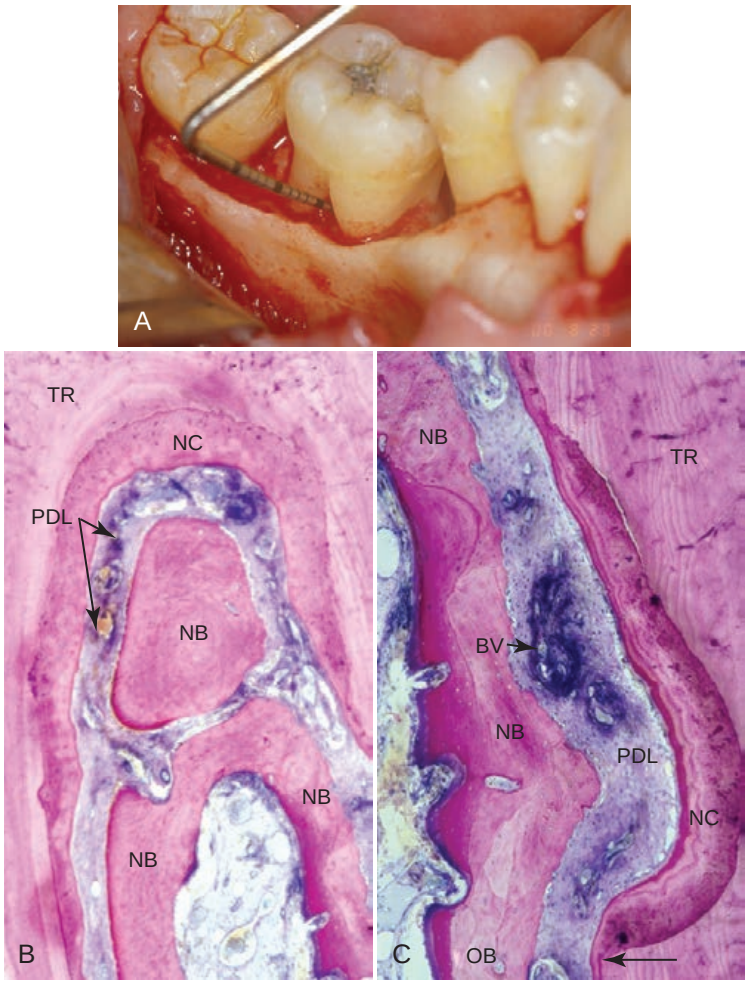

Figure 61-18 **A,** Intraoperative photograph with probe in place showing a 5 mm furcation defect on the lingual of tooth #19. The furcation was treated with recombinant human platelet-derived growth factor (rhPDGF) mixed with demineralized freeze-dried bone allograft (DFDBA). After 9 months of healing, the tooth was resected and submitted for histologic analysis. **B,** Histologic section showing tooth root *(TR),* original bone *(OB),* new bone *(NB),* new cementum *(NC),* new periodontal ligament *(PDL),* and new blood vessel *(BV)* formation. New cementum is shown extending coronally from the notch placed at the apical extent of calculus during the treatment surgery. (Original magnification ×25.) **C,** Area of the fornix of the furcation shows new bone completely filling the original furcation defect, with restoration of a well organized new periodontal ligament *(PDL)* throughout the furcation. (Original magnification ×25.) *(**A,** from Nevins M, et al: Periodontal regeneration in humans using recombinant human platelet-derived growth factor-BB (rhPDGF-BB) and allogenic bone.* J Periodontol *74(9):1282-1292, 2003. **B,** and **C,** from Camelo M, et al: Periodontal regeneration in human class II furcations using purified recombinant human platelet-derived growth factor-BB (rhPDGF-BB) with bone allograft,* Int J Periodontics Restorative Dent *23(3):213-225. 2003.)*

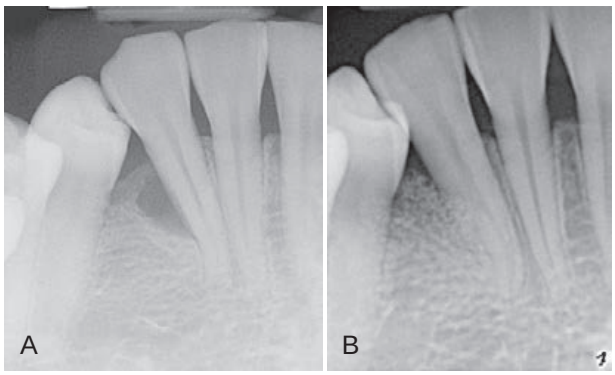

Figure 61-19 **A,** Periapical radiograph of mandibular lateral incisor reveals evidence of vertical periodontal bone loss. The tooth was treated with a regenerative periodontal surgical procedure grafting with mineral collagen bone substitute saturated with recombinant human platelet-derived growth factor (rhPDGF) -BB (0.3 mg/ml). **B,** Periapical radiograph 18 months posttreatment reveals suggestion of bone fill consistent with the noted clinical improvement of 5 mm attachment gain. *(Courtesy of Dr. Marc Nevins. Boston.)*

used to stimulate osteoblast precursor or osteoblast cells and hence to push the bone formation/bone resorption balance in favor of bone formation.

The bone morphogenetic proteins are a group of related proteins that are found in the body and are important for skeletal development.[40,211] Each of the proteins has relatively specific functions, and BMP-2 has been shown to have some of the strongest bone producing activity. BMP-7 (also called *osteogenic protein-1,* or OP-1) and BMP-3 (also know as osteogenin) have also been shown to stimulate bone formation. The BMPs were originally isolated from bovine bone by Marshall Urist [203]. As a growth factor, the BMPs induce the differentiation of mesenchymal stem cells to become bone producing osteoblast cells. Thus unlike proliferation factors that increase the number of cells, the BMPs are differentiation factors, stimulating the cells to produce bone tissue. Many carriers have been tested with the BMPs, but the binding and release kinetics using bovine type I collagen have proven to be the most useful clinically. BMP binds tightly to the collagen within minutes and has been shown to be released over time for 2 to 3 weeks at the defect site. The extended release kinetics likely allow the undifferentiated mesenchymal cells migrating into the wound site to be exposed to the growth factor. These BMP proteins have been extensively studied and have been shown to stimulate bone formation in many animal models and in humans under a variety of conditions and indications.[210] In fact, the same product approved for use in the oral cavity had previously been approved by the FDA for use in single level spine fusion procedures and in open tibial fractures in humans. The commercially available bone growth product contains rhBMP-2 combined with a bovine type I collagen sponge. This product has been cleared for oral cavity use in both sinus augmentation therapy and for ridge augmentation in maxillary anterior extraction sites with bone loss.

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The results of using rhBMP-2 and collagen in patients are remarkable considering that only one bone differentiation agent is being delivered to the bone site, and it is only being delivered at the time of the surgical procedure. Relatively high doses of the protein are often used in some sites, however, because of the need to release effective doses at later times. Future research directions will likely focus on more targeted delivery of the bone growth factor at specified times during the wound healing process.

Enamel Matrix Proteins

One of the early attempts to isolate proteins (growth factors) to stimulate cell growth for periodontal regeneration was based on the knowledge that enamel proteins were present in acellular cementum. Purified enamel matrix proteins have been extracted from porcine developing enamel, and their use for periodontal tissue regeneration has shown mixed results in the numerous studies that have been conducted.

The enamel matrix proteins, mainly amelogenin, are secreted by Hertwig's epithelial root sheath during tooth development and are known to induce acellular cementum formation. Based on these observations, these proteins are believed to favor periodontal regeneration.[82]

One of these enamel matrix protein derivatives obtained from developing porcine teeth has been approved by the FDA and is marketed under the trade name *Emdogain.* The material is a viscous gel consisting of enamel-derived proteins from tooth buds in a polypropylene liquid; 1 ml of a vehicle solution is mixed with a powder and delivered by syringe to the defect site. Ninety percent of the protein in this mixture is amelogenin, with the rest primarily proline-rich non-amelogenins, tuftelin, tuft protein, serum proteins, ameloblastin, and amelin.[20,35,102]

The technique using enamel protein derivatives, as described by Mellonig,[121] is as follows (Figure 61-20):

1. Raise a flap for reconstructive purposes which is to retain as much tissue as possible (see Chapter 59).
2. Remove all granulation tissue and tissue tags, exposing the underlying bone. The root surface should be thoroughly planed.
3. Completely control bleeding within the defect.
4. Demineralize the root surface with citric acid (pH of 1.0), or preferably with 24% ethylenediaminetetraacetic acid (EDTA Biora) (pH of 6.7) for 15 seconds. This removes the smear layer and facilitates adherence of the Emdogain.
5. Rinse the wound with saline, and apply the gel to completely cover the exposed root surface. Avoid contamination with blood or saliva.
6. Complete closure of the wound is necessary, therefore a well-designed flap and suturing technique is mandatory. If adequate closure cannot be obtained, correct the scalloping of the gingival margin or slight osteoplasty may be necessary. A periodontal dressing is preferred to protect the wound.

A systemic antibiotic coverage for 10 to 21 days is recommended.

Heijl et al[88] compared the use of enamel matrix derivatives with a placebo in 33 patients with 34 paired test and control sites, mostly one-wall and two-wall defects, followed for 3 years. They found a statistically significant radiographic bone gain of 2.6 mm. Froum et al[68] reported that use of Emdogain resulted in a reduction in probing depth of 4.94 mm, increase in attachment level of 4.26 mm, and bone fill of 3.83 mm (74% of defects).

In a histologic study of 10 defects in 8 patients, Yukna and Mellonig[216] reported evidence of regeneration (new cementum, bone, and periodontal ligament) in three specimens, new attachment (connective tissue attachment, adhesion only) in three specimens, and a long junctional epithelium in four specimens. No evidence of root resorption or ankylosis was found. Other studies suggest that enamel matrix proteins have characteristics that could enhance periodontal regeneration. For example, the enamel matrix proteins promote bone cell attachment and cell spreading[92] and enhance the proliferation of more immature bone cells while stimulating the differentiation of more mature bone cells.[178] An in vitro wound-healing model demonstrated that the enamel matrix derivative enhanced human periodontal ligament fibroblast cell wound healing.[93] Additionally, the enamel matrix derivative is not osteoinductive, but it is "osteopromotive" in that it stimulates bone formation when combined with demineralized FDBA.[19]

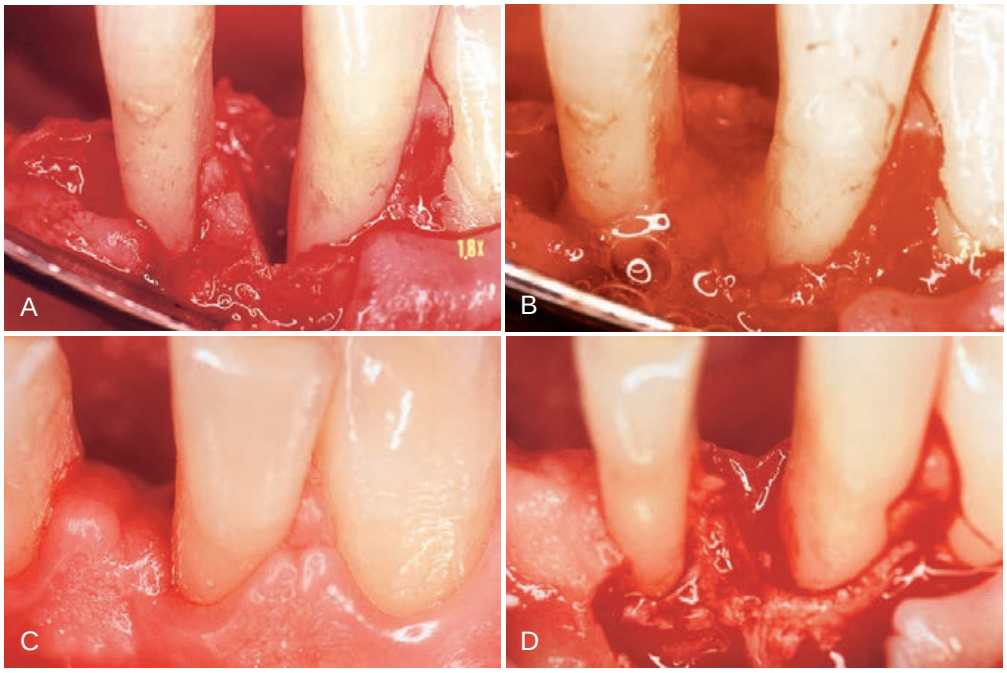

Figure 61-20 A, Deep vertical bone loss distal to lower left central incisor. **B,** Area flapped, root prepared, and defect filled with enamel matrix protein (Emdogain). **C,** Postoperative photo 6 months later. **D,** Reentry surgery showing extensive bone fill. *(Courtesy Dr. Marco Orsini, Aquila, Italy.)*

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As mentioned earlier, the type of attachment achieved during periodontal reconstructive therapy can only be determined histologically. In two studies involving baboons, the use of enamel matrix alone or in combination with autogenous bone grafting resulted in significantly enhanced histologic periodontal reconstruction. This stimulated tissue response occurred in one-wall periodontal defects ranging from 1 to 6 mm. These data and the results from many other studies support the clinical use of enamel matrix derivatives to stimulate periodontal tissue reconstruction around teeth.

Combined Techniques

Periodontal new attachment and bone reconstruction has been a challenge for the clinicians throughout the history of periodontal therapy. To take advantage of the different bone graft materials and biologic mediators, clinicians have combined these graft materials along with the use of membranes in a attempt to find a predictable technique to regenerate bone.

Several clinicians have proposed a combination of the techniques previously described in an attempt to enhance their results.*

A classic paper published by Schallhorn and McClain[175] in 1988 described a combination technique using graft material, root conditioning with citric acid, and coverage with a nonresorbable membrane (the only available one at the time).[176] More recently, with the advent of osteopromotive agents, such as the enamel matrix derivative (Emdogain) and osteoconductive bovine-derived anorganic bone (Bio-Oss) graft materials, other combination techniques have been advocated.[108] The combined use of these products, along with autogenous bone with resorbable membrane coverage, has resulted in an increased percentage of cases with successful new attachment and periodontal reconstruction (Figures 61-21 and 61-22).

Froum et al[68] have analyzed the criteria that should guide the choice of treatment technique. They believe that clinical results depend on (1) the dimension and morphology of the defect (deeper lesions result in greater bone fill than shallower defects), (2) the number of walls in the defect[75] (three-wall defects have greater potential to fill than two-wall or one-wall defects), (3) the amount of root surface exposed and the ability to obtain adequate flap coverage,[90,189] and (4) the angle of the defect to the long axis of the tooth[193] (the smaller the angle, the better chance of success). On the basis of these criteria, Froum et al[64] established the clinical decision tree in Box 61-1.

*References 6, 16, 83, 105, 115, 116, 119, and 205.

Refer to the Expert CONSULT companion website at www.expertconsult.com for clinical cases and further discussion of biologic mediators.

SUMMARY

Based on available information, a clinically significant reconstruction of human supporting periodontal tissues is possible in select sites and patients with the use of the proper graft materials, membranes, and their combination.

BOX 61-1 For Deep, Well-Contained Defect

Use EMD alone, with a coronally advanced flap (see Figure 61-10)

For Moderate to Deep, Noncontained Defects

Use EMD + graft, with a coronally advanced flap (if necessary).

For Supracrestal Defects with a Shallow Vertical Defect

Use EMD + graft + barrier membrane, with a coronally advanced flap.

From Froum S, Lemler J, Horowitz R, et al: *Int J Periodont Restor Dent* 21:437, 2001; Froum SJ, Weinberg MA, Rosenberg E, et al: *J Periodontol* 72:25, 2001.
EMD, Enamel matrix derivative.

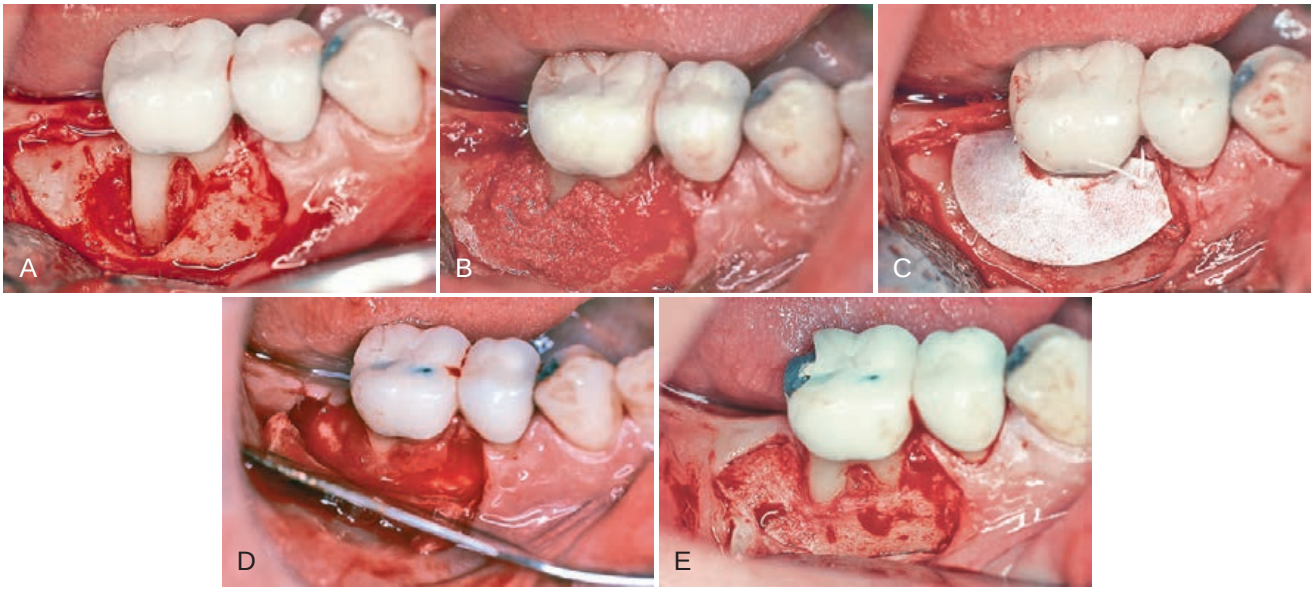

Figure 61-21 **A,** After flap elevation and debridement, mandibular first molar shows distal root with extensive bone loss facially and distally, as well as grade II furcation involvement. **B,** Bone replacement graft (decalcified freeze-dried bone allograft [DFDBA]) in position. **C,** Barrier membrane (expanded polytetrafluoroethylene [ePTFE]) over bone graft. **D,** Appearance of new tissue at time of membrane removal (6 weeks after surgery) suggestive of new alveolar bone. **E,** Reentry after 2 years shows bone reconstruction. *(Courtesy Dr. Thomas J. Han, University of California, Los Angeles.)*

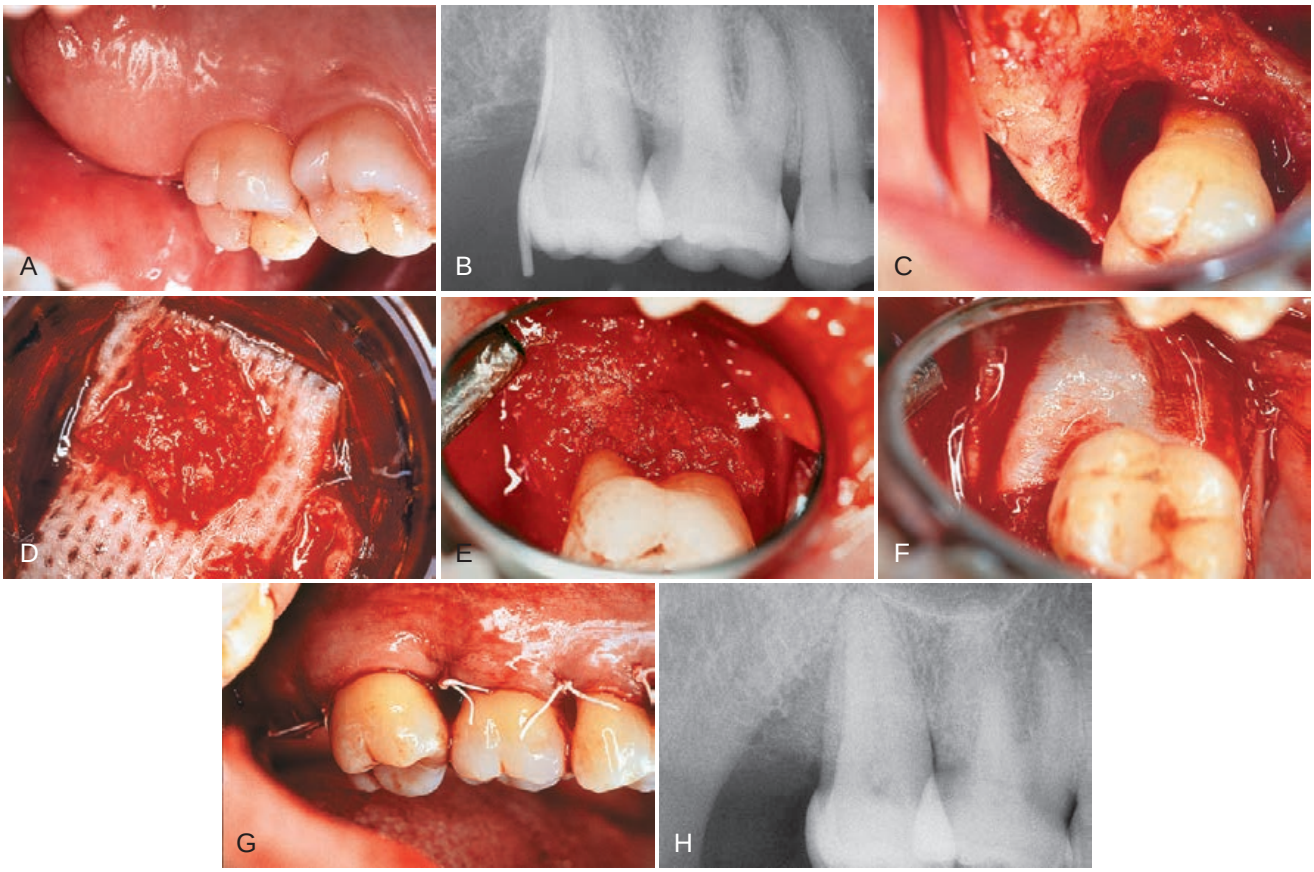

Figure 61-22 **A,** Preoperative view of maxillary first and second molar area. **B,** Preoperative radiograph of second molar showing deep, distal vertical bone loss with a radiopaque pointer to indicate pocket depth. **C,** Clinical view of three-wall osseous defect distal to second molar. **D,** Autogenous bone procured from adjacent area undergoing resective osseous surgery placed in defect. **E,** Autogenous bone placed in defect after scaling and root planing. **F,** Bioresorbable membrane placed over graft to protect it. **G,** Area sutured. **H,** Postoperative radiograph at 6 months showing defect fill.

SCIENCE TRANSFER

Regenerative surgical treatment of intrabony periodontal defects results in dramatic improvements of bone loss attachment level and pocket depths that cannot be matched by other nonsurgical and surgical approaches. These improvements are maintainable over many years if appropriate maintenance care is used. A wide variety of bone graft materials, membranes, and biological active molecules have been successful in regenerative periodontal surgery so the clinician has a wide choice of approaches. The regeneration of periodontal defects associated with uniform horizontal bone loss has not been as dramatically successful as the treatment of vertical bone defects.

Clinicians can combine the use of bone graft materials with protective membranes and biologically active agents such as enamel matrix-derived proteins and platelet-derived growth factors. The combined approach is most useful in large wide defects where bone grafts supply structural functions, membranes provide guided tissue and graft retention functions, and biologic agents give cellular enhancement.

Clinicians need to continually upgrade their surgical skills to get the least traumatic management of tissues and new minimally invasive approaches using microsurgical techniques may give better and better esthetic and regenerative results.

The future of periodontal reconstruction techniques depend on the emergence of new products such as the growth factors that are now being researched and several in clinical use today. The clinician should attempt to differentiate between techniques that have been studied in depth and with acceptable results and those that are still experimental, although promising. Research papers must be critically evaluated for adequacy of controls, selection of cases, methods of evaluation, and long-term postoperative results. In addition, the clinician should remember that he or she is seeking "clinical" success, which is not always similar to "statistical" success. A gain in clinical attachment of half a millimeter may be statistically significant but not clinically significant.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 62

Furcation: Involvement and Treatment

Thomas Sims, Henry H. Takei, William F. Ammons Jr.†, and Gerald W. Harrington

CHAPTER OUTLINE

ETIOLOGIC FACTORS
DIAGNOSIS AND CLASSIFICATION OF FURCATION DEFECTS
LOCAL ANATOMIC FACTORS
Root Trunk Length
Root Length
Root Form
Interradicular Dimension
Anatomy of Furcation
Cervical Enamel Projections
ANATOMY OF THE BONY LESIONS
Pattern of Attachment Loss
Other Dental Findings
INDICES OF FURCATION INVOLVEMENT
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Grade II
Grade III
Grade IV
Other Classification Indices
TREATMENT (online only)
Therapeutic Classes of Furcation Defects
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Oral Hygiene Procedures
Scaling and Root Planing
SURGICAL THERAPY (online only)
Osseous Resection
Regenerative
Root Resection
Hemisection
Root Resection/Hemisection Procedure
Extraction
Dental Implants
PROGNOSIS

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

The progress of inflammatory periodontal disease, if unabated, ultimately results in attachment loss sufficient enough to affect the bifurcation or trifurcation of multirooted teeth. The **furcation** is an area of complex anatomic morphology[5,6,11] that may be difficult or impossible to debride by routine periodontal instrumentation.[29,36] Routine home care methods may not keep the furcation area free of plaque.[17,23]

The presence of furcation involvement is one clinical finding that can lead to a diagnosis of advanced periodontitis and potentially to a less favorable prognosis for the affected tooth or teeth. Furcation involvement therefore presents both diagnostic and therapeutic dilemmas.

ETIOLOGIC FACTORS

The primary etiologic factor in the development of furcation defects is *bacterial plaque* and the inflammatory consequences that result from its long-term presence. The extent of attachment loss required to produce a furcation defect is variable and related to local anatomic factors (e.g., root trunk length, root morphology)[12,27] and local developmental anomalies (e.g., cervical enamel projections).[22,27] Local factors may affect the rate of plaque deposition or complicate the performance of oral hygiene procedures, thereby contributing to the development of periodontitis and attachment loss. Studies indicate that prevalence and severity of furcation involvement increase with age.[21,22,36] Dental caries and pulpal death may also affect a tooth with furcation involvement or even the area of the furcation. All of these factors should be considered during the diagnosis, treatment planning, and therapy of the patient with furcation defects.

DIAGNOSIS AND CLASSIFICATION OF FURCATION DEFECTS

A thorough clinical examination is the key to diagnosis and treatment planning. Careful probing is required to determine the presence and extent of furcation involvement, the position of the attachment relative to the furca, and the extent and configuration of the furcation defect.[38] The Nabors probe may be helpful to enter

†Deceased.

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and measure difficult to access furcal areas (Figure 62-1). Transgingival sounding may further define the anatomy of the furcation defect.[29] The goal of this examination is to identify and classify the extent of furcation involvement and to identify factors that may have contributed to the development of the furcation defect or that could affect treatment outcome. These factors include (1) the morphology of the affected tooth, (2) the position of the tooth relative to adjacent teeth, (3) the local anatomy of the alveolar bone, (4) the configuration of any bony defects, and (5) the presence and extent of other dental diseases (e.g., caries, pulpal necrosis).

The dimension of the furcation entrance is variable but usually quite small; 81% of furcations have an orifice of 1 mm or less, and 58% are 0.75 mm or less.[5,6] The clinician should consider these dimensions, along with the local anatomy of the furcation area,[11–13] when selecting instruments for probing. A probe of small cross-sectional dimension is required if the clinician is to detect early furcation involvement.

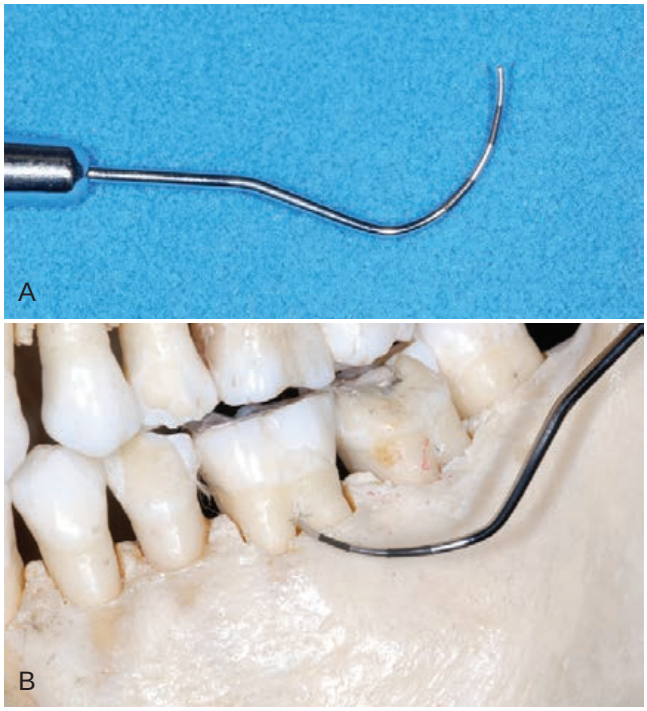

Figure 62-1 **A,** The Nabors probe is designed to probe into the furcation. **B,** The probe placed into a Class II furcation of a dried skull.

LOCAL ANATOMIC FACTORS

Clinical examination of the patient should allow the therapist to identify not only furcation defects but also many of the local anatomic factors that may affect the result of therapy (prognosis). Well-made dental radiographs, although not allowing a definitive classification of furcation involvement, provide additional information vital for treatment planning (Figure 62-2). Important local factors include anatomic features of the affected teeth, as described next.

Root Trunk Length

A key factor in both the development and the treatment of furcation involvement is the root trunk length. The distance from the cementoenamel junction to the entrance of the furcation can vary extensively. Teeth may have very short root trunks, moderate root trunk length, or roots that may be fused to a point near the apex (Figure 62-3). The combination of root trunk length with the number and configuration of the roots affects the ease and success of therapy. The shorter the root trunk, the less attachment needs to be lost before the furcation is involved. Once the furcation is exposed, teeth with short root trunks may be more accessible to maintenance procedures, and the short root trunks may facilitate some surgical procedures. Alternatively, teeth with unusually long root trunks or fused roots may not be appropriate candidates for treatment once the furcation has been affected.

Root Length

Root length is directly related to the quantity of attachment supporting the tooth. Teeth with long root trunks and short roots may have lost a majority of their support by the time that the furcation becomes affected.[13,20] Teeth with long roots and short-to-moderate

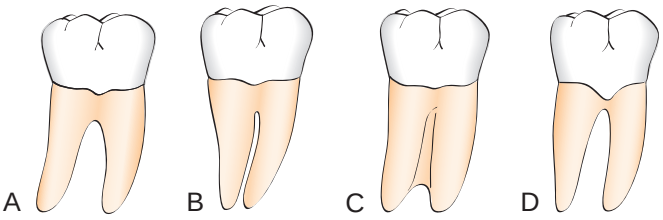

Figure 62-3 Different anatomic features that may be important in prognosis and treatment of furcation involvement. **A,** Widely separated roots. **B,** Roots are separated but close. **C,** Fused roots separated only in their apical portion. **D,** Presence of enamel projection that may be conducive to early furcation involvement.

Figure 62-2 Different degrees of furcation involvement in radiographs. **A,** Grade I furcation on the mandibular first molar and a grade III furcation on the mandibular second molar. The root approximation on the second molar may be sufficient to impede accurate probing of this defect. **B,** Multiple furcation defects on a maxillary first molar. Grade I buccal furcation involvement and grade II mesiopalatal and distopalatal furcations are present. Deep developmental grooves on the maxillary second molar simulate furcation involvement in this molar with fused roots. **C,** Grade III and IV furcations on mandibular molars.

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BOX 62-1 Classification of Cervical Enamel Projections

Grade I: The enamel projection extends from the cementoenamel junction of the tooth toward the furcation entrance.
Grade II: The enamel projection approaches the entrance to the furcation. It does not enter the furcation, and therefore no horizontal component is present.
Grade III: The enamel projection extends horizontally into the furcation.

From Masters DH, Hoskins SW: *J Periodontol* 35:49, 1964.

root trunk length are more readily treated because sufficient attachment remains to meet functional demands.

Root Form

The mesial root of most mandibular first and second molars and the mesiofacial root of the maxillary first molar are typically curved to the distal side in the apical third. In addition, the distal aspect of this root is usually heavily fluted. The curvature and fluting may increase the potential for root perforation during endodontic therapy or complicate post placement during restoration.[1,25] These anatomic features may also result in an increased incidence of vertical root fracture. The size of the mesial radicular pulp may result in removal of most of this portion of the tooth during preparation.

Interradicular Dimension

The degree of separation of the roots is also an important factor in treatment planning. Closely approximated or fused roots can preclude adequate instrumentation during scaling, root planing, and surgery. Teeth with widely separated roots present more treatment options and are more readily treated.

Anatomy of Furcation

The anatomy of the furcation is complex. The presence of bifurcational ridges, a concavity in the dome,[11] and possible accessory canals[16] complicates not only scaling, root planing, and surgical therapy[28] but also periodontal maintenance. Odontoplasty to reduce or eliminate these ridges may be required during surgical therapy for an optimal result.

Cervical Enamel Projections

Cervical enamel projections (CEPs) are reported to occur on 8.6% to 28.6% of molars.[26,27,35] The prevalence is highest for mandibular and maxillary second molars. The extent of CEPs was classified by Masters and Hoskins[27] in 1964 (Box 62-1). Figure 62-4 provides an example of a grade III CEP. These projections can affect plaque removal, can complicate scaling and root planing, and may be a local factor in the development of gingivitis and periodontitis. CEPs should be removed to facilitate maintenance.

ANATOMY OF THE BONY LESIONS

Pattern of Attachment Loss

The form of the bony lesions associated with the furcation can vary significantly. Horizontal bone loss can expose the furcation as thin facial/lingual plates of bone that may be totally lost during resorption. Alternatively, areas with thickened bony ledges may persist and predispose to the development of furcations with deep vertical components. The pattern of bone loss on other surfaces of the affected tooth and adjacent teeth must also be considered during treatment planning. The treatment response in deep, multiwalled bony defects is different from that in areas of horizontal bone loss. Complex multiwalled defects with deep, interradicular vertical components may be candidates for regenerative therapies. Alternatively, molars with advanced attachment loss on only one root may be treated by resective procedures.

Figure 62-4 Furcation involvement by grade III cervical enamel projections.

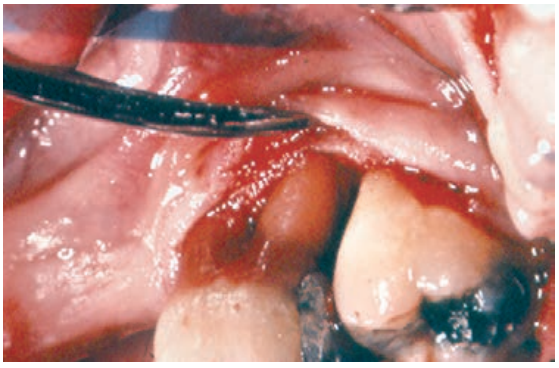

Figure 62-5 Advanced bone loss, furcation involvement, and root approximation. Note the buccal furcation, which communicates with the distal furcation of a maxillary first molar that also displays advanced attachment loss on the distal root and approximation with the mesial root of the maxillary second molar. The patient with such teeth may benefit from root resection of the distobuccal root of the first molar or extraction of the molar.

Other Dental Findings

The dental and periodontal condition of the adjacent teeth must be considered during treatment planning for furcation involvement. The combination of furcation involvement and root approximation with an adjacent tooth represents the same problem that exists in furcations without adequate root separation. Such a finding may dictate the removal of the most severely affected tooth or the removal of a root or roots (Figure 62-5).

The presence of an adequate band of gingiva and a moderate to deep vestibule will facilitate the performance of a surgical procedure, if indicated.

INDICES OF FURCATION INVOLVEMENT

The extent and configuration of the furcation defect are factors in both diagnosis and treatment planning. This has led to the development of a number of indices to record furcation involvement. These indices are based on the horizontal measurement of attachment loss in the furcation,[14,17] on a combination of horizontal and vertical measurements,[37] or a combination of these findings with the localized configuration of the bony deformity.[10]

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Figure 62-6 Glickman's classification of furcation involvement. **A,** Grade I furcation involvement. Although a space is visible at the entrance to the furcation, no horizontal component of the furcation is evident on probing. **B,** Grade II furcation in a dried skull. Note both the horizontal and the vertical component of this cul-de-sac. **C,** Grade III furcations on maxillary molars. Probing confirms that the buccal furcation connects with the distal furcation of both these molars, yet the furcation is filled with soft tissue. **D,** Grade IV furcation. The soft tissues have receded sufficiently to allow direct vision into the furcation of this maxillary molar.

Glickman[14] classified furcation involvement into four grades (Figure 62-6).

Grade I

A grade I furcation involvement is the incipient or early stage of furcation involvement (see Figure 62-6, *A*). The pocket is suprabony and primarily affects the soft tissues. Early bone loss may have occurred with an increase in probing depth, but radiographic changes are not usually found.

Grade II

A grade II furcation can affect one or more of the furcations of the same tooth. The furcation lesion is essentially a cul-de-sac (see Figure 62-6, *B*) with a definite horizontal component. If multiple defects are present, they do not communicate with each other because a portion of the alveolar bone remains attached to the tooth. The extent of the horizontal probing of the furcation determines whether the defect is early or advanced. Vertical bone loss may be present and represents a therapeutic complication. Radiographs may or may not depict the furcation involvement, particularly with maxillary molars because of the radiographic overlap of the roots. In some views, however, the presence of furcation "arrows" indicates possible furcation involvement (see Chapter 31).

Grade III

In grade III furcations, the bone is not attached to the dome of the furcation. In early grade III involvement, the opening may be filled with soft tissue and may not be visible. The clinician may not even be able to pass a periodontal probe completely through the furcation because of interference with the bifurcational ridges or facial/lingual bony margins. However, if the clinician adds the buccal and lingual probing dimensions and obtains a cumulative probing measurement that is equal to or greater than the buccal/lingual dimension of the tooth at the furcation orifice, the clinician must conclude that a grade III furcation exists (see Figure 62-6, *C*). Properly exposed and angled radiographs of early Class III furcations display the defect as a radiolucent area in the crotch of the tooth (see Chapter 31).

Grade IV

In grade IV furcations, the interdental bone is destroyed, and the soft tissues have receded apically so that the furcation opening is clinically visible. A tunnel therefore exists between the roots of such an affected tooth. Thus the periodontal probe passes readily from one aspect of the tooth to another (see Figure 62-6, *D*).

Other Classification Indices

Hamp et al[17] modified a three-stage classification system by attaching a millimeter measurement to separate the extent of horizontal involvement. Easley and Drennan[10] and Tarnow and Fletcher[37] have described classification systems that consider both horizontal and vertical attachment loss in classifying the extent of furcation involvement. The Tarnow and Fletcher article utilizes a subclassification that measures the probeable vertical depth from the roof of the furca apically. The subclasses being proposed are: A, B, and C. "A" indicates a probeable vertical depth of 1 to 3 mm, "B" indicates 4 to 6 mm, and "C" indicates 7 or more mm of probeable depth from the roof of the furca apically. Furcations would thus be classified as IA, IB, and IC; IIA, IIB, and IIC; and IIIA, IIIB, and IIIC.

Consideration of defect configuration and the vertical component of the defect provides additional information that is useful in planning therapy.

For more information on furcation therapy, please visit the companion Expert CONSULT website at www.expertconsult.com.

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NONSURGICAL THERAPY

Oral Hygiene Procedures

Furcal management is difficult at best. Therapeutic modalities for the treatment and maintenance of furcations have long been a dilemma amongst periodontists and restorative dentists. Nonsurgical therapy is a very effective way of producing a satisfactory stable result. Ideal results with furcations are impossible to obtain. Once furcation breakdown has begun, there is always a somewhat compromised result clinically. Both surgical and nonsurgical therapies have been shown to work effectively over time. Nonsurgical therapy, a combination of oral hygiene instruction and scaling and root planing, has provided excellent results in some patients. The earlier the furcation is detected and treated the more likely a good long-term result can be obtained. Nonetheless, even advanced furcation lesions can have successful long-term treatment.[34] Several oral hygiene procedures have been utilized over time. All include access to the furcation. Obtaining access to the furcation requires a combination of the awareness of the furcation by the patient and an oral hygiene tool that facilitates that access. Many tools, including rubber tips; periodontal aids; toothbrushes, both specific and general; and other aids have been used over time for access to the patient (Figure 62-8).

Scaling and Root Planing

Nonsurgical maintenance by the clinician has also improved over time as instrumentation has improved. In recent decades, instruments beyond simple curettes have been used to instrument the furcation. The frustration of instrumentation of the furcation was illustrated beautifully by Bower in 1979 in his articles[5,6] illustrating that only 58% of furcations could be entered by typically utilizing curettes (see Chapter 33). Subsequent to this time, other instrumentation has evolved, including DeMarco curettes, diamond files, Quetin furcation curettes, and mini Five Gracey Curettes. See Chapter 45 for a detailed discussion on this subject.

Svärdström and Wennström[34] illustrated that in the long term, furcations could be maintained over a 10-year period in patients utilizing nonaggressive techniques and who were participants in consistent maintenance. Other studies have also illustrated that maintenance therapy has been useful for patients to facilitate furcation cleanliness. Chemotherapy has proven disappointing. Ribeiro et al[32] found that nonsurgical therapy can effectively treat Class II furcation involvements, but using povidone-iodine did not provide additional benefits to subgingival instrumentation.

The area most critical in furcation management is maintaining a relatively plaque-free status to the furcation. Attaining access is a problem in this regard, but with the previously mentioned instruments and an effective nonsurgical approach, much can be accomplished. The most critical component of multirooted tooth maintenance is always the successful reduction or elimination of plaque retention areas from the furcation area; meticulous oral hygiene by the patient and an effective nonsurgical therapy can play a major role in attaining this goal.[21,33]

PROGNOSIS

For many years the presence of significant furcation involvement meant a hopeless long-term prognosis for the tooth. Clinical research, however, has indicated that furcation problems are not as severe a complication as originally suspected if one can prevent the development of caries in the furcation. Relatively simple

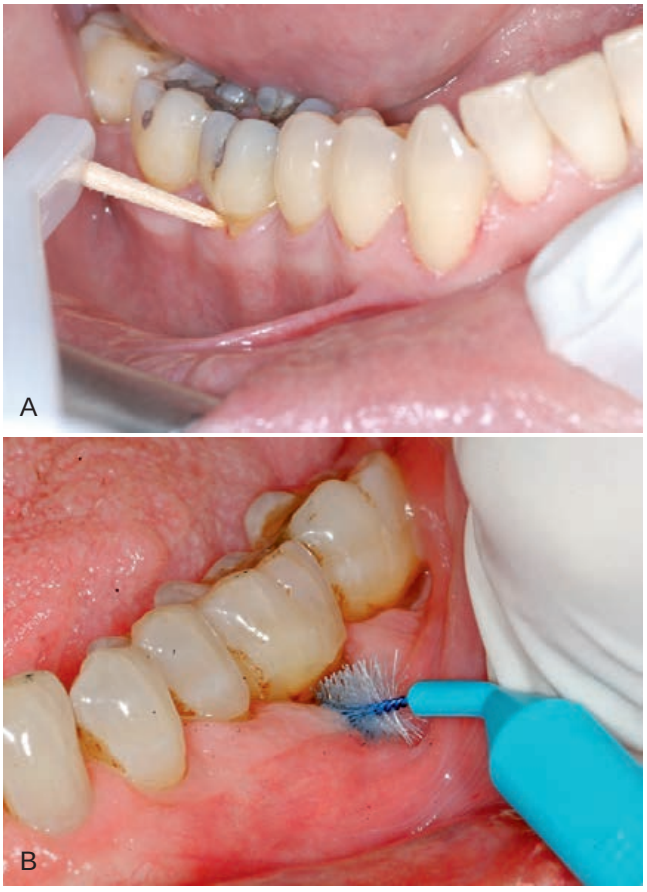

Figure 62-8 **A,** The utilization of a Perio-Aid into the furcation for plaque removal. **B,** Proxy brush is used for plaque removal into the furcation lesion. *(Courtesy Karen DeYoung, RDH, and Janet Shigekawa, RDH.)*

SCIENCE TRANSFER

Furcations can be classified into grade I, grade II, grade III, and grade IV categories with increasing severity and increasing poor prognosis associated with the higher number of the category. Nonsurgical therapy may be applicable to some grade I furcations, but clinicians must closely monitor all furcation defects as progression of bone loss makes treatment more complex and less predictable; so early surgical intervention with flap and osseous surgery or flaps with regenerative materials should be implemented. This will generally provide excellent treatment outcomes but when a furcation has advanced bone loss and is a grade III or grade IV classification, the prognosis may not be good and so clinicians need to consider extraction and regenerative surgical treatment of the socket followed in 4 to 6 months by implant replacement of the tooth. This use of predictable implant treatment has reduced the utilization of the root resective and hemisection approaches.

periodontal therapy is sufficient to maintain these teeth in function for long periods.[21,33] Other investigators have defined the reasons for clinical failure of root-resected or hemisected teeth.[2,25] Their data indicate that recurrent periodontal disease is not a major cause of the failure of these teeth. Investigations of root-resected or hemisected teeth have shown that such teeth can function successfully for long periods.[2,8,25] The keys to long-term success appear to be (1) thorough diagnosis, (2) selection of patients with good oral hygiene, (3) excellence in nonsurgical therapy, and (4) careful surgical and restorative management.

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CHAPTER 63

Periodontal Plastic and Esthetic Surgery

Henry H. Takei, E. Todd Scheyer, Robert R. Azzi, Edward P. Allen, and Thomas J. Han

CHAPTER OUTLINE

TERMINOLOGY
OBJECTIVES
Problems Associated with Attached Gingiva
Problems Associated with Shallow Vestibule
Problems Associated with Aberrant Frenum
Esthetic Surgical Therapy
Tissue Engineering
ETIOLOGY OF MARGINAL TISSUE RECESSION
FACTORS THAT AFFECT SURGICAL OUTCOME
Irregularity of Teeth
Mucogingival Line (Junction)
TECHNIQUES TO INCREASE ATTACHED GINGIVA (online only)
Gingival Augmentation Apical to Recession
Gingival Augmentation Coronal to Recession (Root Coverage)
TECHNIQUES TO DEEPEN THE VESTIBULE (online only)
TECHNIQUES TO REMOVE THE FRENUM (online only)
Frenectomy and Frenotomy Procedure
TECHNIQUES TO IMPROVE ESTHETICS (online only)
Root Coverage
Papilla Reconstruction
Excessive Gingival Display
TISSUE ENGINEERING
Passive Engineering
Active Engineering
CRITERIA FOR SELECTION OF TECHNIQUES
Surgical Site Free of Plaque, Calculus, and Inflammation
Adequate Blood Supply
Anatomy of the Recipient and Donor Sites
Stability of the Grafted Tissue to the Recipient Site
Minimal Trauma to the Surgical Site
CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content and supplements to this chapter. Some figures may be out of numeric order in this printed chapter.

TERMINOLOGY

The term *mucogingival surgery* was initially introduced in the literature by Friedman[38] to describe surgical procedures for the correction of relationships between the gingiva and the oral mucous membrane with reference to three specific problem areas: attached gingiva, shallow vestibules, and a frenum interfering with the marginal gingiva. With the advancement of periodontal surgical techniques, the scope of nonpocket surgical procedures has increased, now encompassing a multitude of areas that were not addressed in the past. Recognizing this, the 1996 World Workshop in Clinical Periodontics renamed mucogingival surgery as "periodontal plastic surgery,"[3] a term originally proposed by Miller in 1993 and broadened to include the following areas[2,3]:

- Periodontal-prosthetic corrections
- Crown lengthening
- Ridge augmentation
- Esthetic surgical corrections
- Coverage of the denuded root surface
- Reconstruction of papillae
- Esthetic surgical correction around implants
- Surgical exposure of unerupted teeth for orthodontics

Periodontal plastic surgery is defined as the surgical procedures performed to correct or eliminate anatomic, developmental, or traumatic deformities of the gingiva or alveolar mucosa.[2,3] Mucogingival therapy is a broader term that includes nonsurgical procedures such as papilla reconstruction by means of orthodontic or restorative therapy. Periodontal plastic surgery includes only the surgical procedures of mucogingival therapy.

This chapter discusses the periodontal plastic surgical techniques included in the traditional definition of mucogingival surgery: (1) widening of attached gingiva (2) deepening of shallow vestibules, and (3) resection of the aberrant frena. In addition, esthetic surgical therapy around the natural dentition and tissue engineering (biologic mediator) are included in this chapter. Other aspects of periodontal plastic surgery, such as periodontal-prosthetic surgery, esthetic surgery around implants, and surgical exposure of

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teeth for orthodontic therapy, are covered in Chapters 50, 65, and 74.

OBJECTIVES

The five objectives of periodontal plastic surgery addressed in this chapter are as follows:

1. Problems associated with attached gingiva
2. Problems associated with a shallow vestibule
3. Problems associated with an aberrant frenum
4. Esthetic surgical therapy
5. Tissue engineering

Problems Associated with Attached Gingiva

The ultimate goal of mucogingival surgical procedures is the creation or widening of attached gingiva around teeth and implants.[3] The width of the attached gingiva varies in different individuals and on different teeth of the same individual (see Chapter 2). Attached gingiva is not synonymous with "keratinized gingiva" because the latter also includes the free gingival margin.

The width of the attached gingiva is determined by subtracting the depth of the sulcus or pocket from the distance between the crest of the gingival margin and the mucogingival junction.

The original rationale for mucogingival surgery was predicated on the assumption that a minimal width of attached gingiva was required to maintain optimal gingival health. However, several studies have challenged the view that a wide, attached gingiva is more protective against the accumulation of plaque than a narrow or a nonexistent zone. No minimum width of attached gingiva has been established as a standard necessary for gingival health. People who practice good, atraumatic oral hygiene may maintain excellent gingival health with almost no attached gingiva.

However, those individuals whose oral hygiene practices are less than optimal can be helped by the presence of keratinized gingiva and vestibular depth. Vestibular depth provides space for easier placement of the toothbrush and prevents brushing on mucosal tissue. To improve esthetics, the objective is the coverage of the denuded root surface. The maxillary anterior area, especially the facial aspect of the canine, often presents extensive gingival recession. In such cases, the covering of the denuded root surface not only widens the zone of attached gingiva but also creates a improved esthetic result. This recession and the resultant denuded root surface have a special esthetic concerns for individuals with a high smile line. A wider zone of attached gingiva is also needed around teeth that serve as abutments for fixed or removable partial dentures, as well as in the ridge areas bearing a denture. Teeth with subgingival restorations and narrow zones of keratinized gingiva have higher gingival inflammation scores than teeth with similar restorations and wide zones of attached gingiva.[90,91] Therefore, in such cases, techniques for widening the attached gingiva are considered preprosthetic periodontal surgical procedures. Chapter 65 discusses this subject in detail.

Widening the attached gingiva accomplishes the following four objectives:

1. Enhances plaque removal around the gingival margin.
2. Improves esthetics.
3. Reduces inflammation around restored teeth.
4. Gingival margin binds better around teeth and implants with attached gingiva.

Problems Associated with Shallow Vestibule

Another objective of periodontal plastic surgery is the creation of vestibular depth when it is lacking. Gingival recession displaces the gingival margin apically, thus reducing vestibular depth, which is measured from the gingival margin to the bottom of the vestibule. As indicated previously, with minimal vestibular depth, proper hygiene procedures are jeopardized. The sulcular brushing technique requires the placement of the toothbrush at the gingival margin, which may not be possible with reduced vestibular depth.

Minimal attached gingiva with adequate vestibular depth may not require surgical correction if proper atraumatic hygiene is practiced with a soft brush. Minimal amounts of keratinized attached gingiva with no vestibular depth benefit from mucogingival correction. Adequate vestibular depth is also necessary for the proper placement of removable prostheses.

Problems Associated with Aberrant Frenum

Still another important objective of periodontal plastic surgery is to correct frenal or muscle attachments that may extend coronal to the mucogingival junction. If adequate keratinized, attached gingiva is present coronal to the frenum, it may not be necessary to remove the frenum. A frenum that encroaches on the margin of the gingiva may interfere with plaque removal, and the tension on the frenum may tend to open the sulcus. In such cases, surgical removal of the frenum is indicated.

Esthetic Surgical Therapy

As indicated earlier, the recession of the facial, gingival margin will alter the proper gingival symmetry and result in an esthetic problem. The presence of the interdental papilla is also important to satisfy the esthetic goals of the patient. A missing papilla creates a space which many address as a "black hole." The regeneration of the lost or reduced papilla is one of the most difficult goals in esthetic periodontal plastic surgery. Another area of concern is the patient who presents an excessive amount of gingiva in the visible area. This condition is often addressed as a "gummy smile" and may be corrected surgically by crown lengthening. The correction of these anatomic defects has become an important part of periodontal plastic surgery.

Tissue Engineering

The future of periodontal plastic surgery will encompass the use of tissue-engineered products at the recipient site to reduce the donor site morbidity. Currently, there are numerous studies, both clinically and in the laboratories, to allow the clinician to utilize this minimally invasive approach to periodontal plastic surgery.

ETIOLOGY OF MARGINAL TISSUE RECESSION

The most common cause of gingival recession and the loss of attached gingiva is abrasive and traumatic toothbrushing habits. The bone and soft tissue anatomy of the buccal, radicular surface of the dentition is usually thin, especially around the anterior area. Teeth positioned buccally may have a even thinner bone and gingiva. In many instances, such areas may have a complete absence of bone beneath the thin overlying gingival tissue. Such defect in the bone is called a *dehiscence*. This anatomic status combined with external trauma from overzealous brushing can lead to the loss of gingival tissue. Recession of the gingival tissue and bone exposes the cemental surface of the root, which results in abrasion and "ditching" of the cemental surface apical to the cementoenamel junction (CEJ). The cementum is softer than enamel and will be destroyed before the enamel surface of the crown.

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Figure 63-1 High frenum attachments. **A,** Frenum between maxillary central incisors. **B,** Frenum attached to facial surface of maxillary lateral incisors. **C,** Frenum attached to facial surface of mandibular incisor. **D,** Frenum attached to facial surface of an incisor.

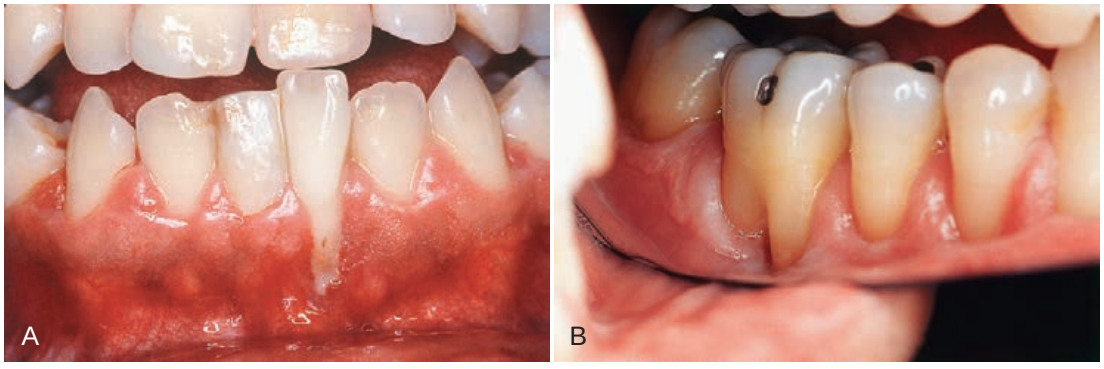

Figure 63-2 **A,** Gingival recession and extreme inflammation around a lower central incisor. **B,** Advanced recession of mesial root of a first lower molar.

Another cause for gingival recession is periodontal disease and chronic marginal inflammation. The loss of attachment caused by the inflammation is followed by the loss of bone and gingiva. Advanced periodontal involvement in areas of minimal attached gingiva results in the base of the pocket extending close to, or apical to, the mucogingival junction. Periodontal therapy of these areas also results in gingival recession caused by the loss of gingiva and bone.

Frenal and muscle attachments that encroach on the marginal gingiva can distend the gingival sulcus, which creates an environment for plaque accumulation. This condition increases the rate periodontal recession and will contribute to the recurrence of the recession even after treatment (Figure 63-1). These problems are more common on facial surfaces, but may also occur on the lingual surface.[11]

Orthodontic tooth movement through a thin buccal osseous plate may lead to a dehiscence beneath a thin gingiva. This also can lead to the recession of the gingiva[45,106] (Figure 63-2).

FACTORS THAT AFFECT SURGICAL OUTCOME

Irregularity of Teeth

Abnormal tooth alignment is an important cause of gingival deformities that require corrective surgery and also an important factor in determining the outcome of treatment. The location of the gingival margin, width of the attached gingiva, and alveolar bone height and thickness are all affected by tooth alignment. On teeth that are tilted or rotated labially, the labial bony plate is thinner and located farther apically than on the adjacent teeth; therefore the gingiva is recessed so that the root is exposed.[106] On the lingual surface of such teeth, the gingiva is bulbous, and the bone margins are closer to the CEJ. The level of gingival attachment on root surfaces and the width of the attached gingiva after mucogingival surgery are affected as much by tooth alignment as by variations in treatment procedures.

Orthodontic correction is indicated when mucogingival surgery is performed on malposed teeth in an attempt to widen the attached gingiva or to restore the gingiva over denuded roots. If orthodontic treatment is not feasible, the prominent tooth should be reduced to within the borders of the alveolar bone, with special care taken to avoid pulp injury.

Roots covered with thin bony plates present a hazard in mucogingival surgery. Even the most protective type of flap, a partial-thickness flap, creates the risk of bone resorption on the periosteal surface.[48] Resorption in amounts that ordinarily are not significant may cause loss of bone height when the bone plate is thin or tapered at the crest.

Mucogingival Line (Junction)

Normally, the mucogingival line in the incisor and canine areas is located approximately 3 mm apical to the crest of the alveolar bone on the radicular surfaces and 5 mm interdentally.[92] In periodontal disease and on malposed disease free teeth, the bone margin is located farther apically and may extend beyond the mucogingival line. The distance between the mucogingival line and the CEJ before and after periodontal surgery is not necessarily constant. After inflammation is eliminated, the tissue tends to contract and draw the mucogingival line in the direction of the crown.[31]

For more information on numerous surgical techniques to increase attached gingiva, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on alternative donor tissue and supplements to this chapter, please visit the companion Expert CONSULT website at www.expertconsult.com.

For more information on techniques to deepen the vestibule and frenum is, please visit the companion Expert CONSULT website at www.expertconsult.com.

TISSUE ENGINEERING

Tissue engineering and the use of biologic mediators in periodontal plastic surgery has become a reality in recent years as the result of research and the demand for noninvasive surgical procedures by both the patients and clinicians. Current periodontal plastic surgery is based on the use of the palatal site for donor tissue (subepithelial connective tissue graft), which entails a second surgical wound. Many patients are fearful of this donor wound and resist the surgery needed to correct a mucogingival problem. For these patients who desire to avoid the palatal donor site, morbidity associated with the subepithelial connective tissue graft (SECTG), and free gingival graft (FGG), tissue engineering methods are a excellent alternative. Periodontal therapy has been involved with tissue engineering for decades, beginning with GTR, a form of passive tissue engineering that excluded certain cell types and created an engineered wound left to heal with the appropriate cell types (see Chapter 61). The following is a brief summary of the materials that have been used for procedures based on tissue engineering and is divided into passive and active categories to recognize the role the cells play in tissue engineering.

Passive Engineering

1. Therapies based on guided tissue replacement (GTR) based therapies; barrier membranes
2. Biologically based acellular dermal matrix (ADM)

Active Engineering

1. Enamel matrix derivative (EMD)
2. Growth factors: Recombinant human platelet-derived growth factor-BB (rhPDGF-BB) + beta-tricalcium phosphate (β-TCP) + collagen wound dressing
3. Cell therapy
   - Autologous fibroblast: Isolagen
   - Bilayered cell therapy (BLCT): Celltx
   - Human fibroblast-derived dermal substitute (HFDDS): Dermagraft

GTR (see Chapter 61) and ADM are both considered passive cell manipulation in which the cells themselves are not biologic mediators. The active portion will be discussed in this section. In 2003, McGuire and Nunn[62,63] published results of a single-center randomized controlled clinical trial comparing a coronally advanced flap with enamel matrix derivative (EMD) to subepithelial connective tissue graft (SECTG). The results showed no statistical difference in the percentage of root coverage between test and control. It was concluded that within the limitations of this single-center trial that EMD with a coronally advanced flap is a valid alternative to the subepithelial connective tissue.[62,63]

Currently, the rhPDGF-BB has been studied and marketed under the brand name GEM21S. Recently, McGuire and Scheyer published a case series using this growth factor rhPDGF, β-TCP, and a collagen wound dressing to obtain root coverage comparable to SECTG, which led to a controlled clinical trial. This randomized controlled clinical trial used the growth factor–mediated procedure to obtain clinical and histologic evidence of root coverage and regeneration when compared to the SECTG was published.[68,69] These studies provided "proof of principle" that in four of four human histology block sections, true periodontal regeneration (alveolar bone, periodontal ligament, and new cementum) was possible with rhPDGF + β-TCP + a collagen wound dressing (see Supplement B Figure 63-13). Histologic evidence of root coverage verifies that this tissue engineering method offers true periodontal regeneration, which is not expected with the SECTG.

The reconstruction of the open interproximal space remains one of the greatest challenges in esthetic periodontal therapy. In a landmark study by McGuire and Scheyer,[66] autologous fibroblasts were injected into the interdental papilla in a method to atraumatically augment the deficient gingival papilla. Although this method has not been thoroughly validated, treatment outcomes reduced open interproximal spaces and improved esthetics in the maxillary anterior region (Figure 63-24). No long-term evidence exists for tissue stability with this method, but this pilot study shows promise for an innovative study design for a tissue engineering application in dentistry.

In 2008, McGuire and Scheyer[67] published a pilot study comparing a tissue-engineered BLCT (Apligraf, Organogenesis, Inc, Canton, MA) to an free gingival graft (FGG) with promising enough results to warrant a multicentered controlled clinical trial. From the pilot study, there is evidence of de novo formation of attached and keratinized gingiva with the placement of a live cell therapy device without donor site surgery. In this randomized, controlled clinical trial, the test material was capable of generating up to 2.72 mm of keratinized gingiva, and in more than 75% of the subjects, greater than 2 mm of keratinized tissue was developed after a 6-month follow-up visit. This pilot study supports further

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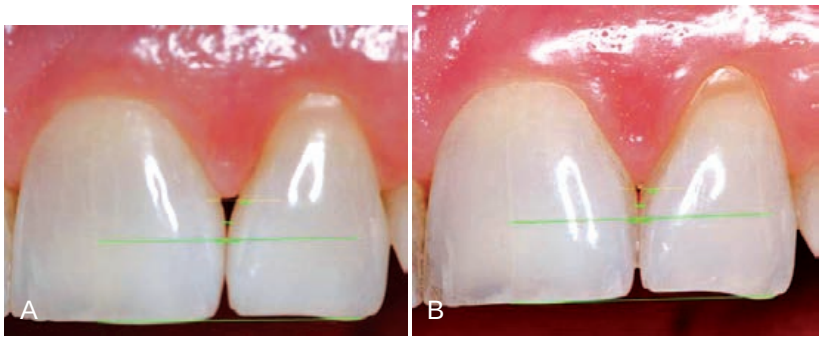

Figure 63-24 **A,** Preoperative view of open interproximal space. **B,** Four months after three injections into the papilla with the patient's own expanded and concentrated fibroblast. Note the improved papillary form. Also note green horizontal lines used for image analysis. *(From McGuire MK, Scheyer ET: A randomized double blind placebo-controlled study to determine the safety and efficacy of cultured and expanded autologous fibroblast injection for the treatment of interdental papillary insufficiency associated with the papilla priming procedure* J Periodont *78: 4-17, 2007).*

research of the BLCT to produce de novo keratinized gingiva without the use of a traditional autograft. More data are being analyzed that will allow for the commercial use of this material.

In 2005, McGuire and Nunn[64] published a pilot study evaluating the safety and efficacy of a living tissue-engineered HFDDS (Dermagraft, Advanced Tissue Science, La Jolla, CA) compared to a gingival autograft. Although the autograft produced a greater band of keratinized tissue, the test group represented the first attempt to use an "off-the-shelf" tissue-engineered material capable of generating attached and keratinized gingival.[105]

For more information on tissue-engineering, see Chapter 63 Supplement B on the companion Expert CONSULT website at www.expertconsult.com.

In the quest for new technologies to reduce trauma during surgery, further research is needed to investigate treatment methods such as ADM and biologic factors, as well as materials that could carry growth factors to the surgical site and maintain their action. At some point, recombinant growth factors could eliminate both the need for donor sites and the use of exogenous materials inserted into the human body. Future research should focus on obtaining additional evidence when using growth factors and live-cell technology during periodontal therapy to eliminate donor site morbidity.

CRITERIA FOR SELECTION OF TECHNIQUES

Different techniques are presented for solving mucogingival problems outlined in this chapter. The proper selection of the numerous techniques must be based on the predictability of success, which in turn is based on the criteria described next.

Criteria for selection of mucogingival techniques are as follows:

1. Surgical site free of plaque, calculus, and inflammation.
2. Adequate blood supply to the donor tissue.
3. Anatomy of the recipient and donor sites.
4. Stability of the grafted tissue to the recipient site.
5. Minimal trauma to the surgical site.

Surgical Site Free of Plaque, Calculus, and Inflammation

Periodontal plastic surgical procedures should be undertaken in a plaque-free and inflammation-free environment to enable the clinician to manage gingival tissue that is firm. Meticulous, precise incisions and flap reflection cannot be achieved when the tissue is inflamed and edematous. Thorough scaling and root planing, as well as meticulous plaque removal by the patient, must be accomplished before any surgical procedure.

Adequate Blood Supply

To obtain the maximum amount of blood supply to the donor tissue, gingival augmentation apical to the area of recession provides a better blood supply than coronal augmentation, since the recipient site is entirely periosteal tissue. Root coverage procedures present a portion of the recipient site (denuded root surface) without blood supply. Therefore, if esthetics is not a factor, gingival augmentation apical to the recession may be more predictable. A pedicle-displaced flap has a better blood supply than a free graft, with the base of the flap intact. In root coverage, therefore, if the anatomy is favorable, the pedicle flap or any of its variants may be the best procedure.

The subepithelial connective tissue graft (Langer) and the pouch and tunnel techniques use a split flap with the connective tissue sandwiched between the flap. This flap design maximizes the blood supply to the donor tissue. If large areas require root coverage, these sandwich-type recipient sites provide the best flap design for blood supply.

Anatomy of the Recipient and Donor Sites

The presence or absence of vestibular depth is an important anatomic criterion at the recipient site for gingival augmentation. If gingival augmentation is indicated apical to the area of recession, there must be adequate vestibular depth apical to the recessed gingival margin to provide space for either a free or a pedicle graft. If a vestibule is necessary, only a free graft can accomplish this objective apical to the recession.

Mucogingival techniques, such as free gingival grafts and free connective tissue grafts, can be used to create vestibular depth and widen the zone of attached gingiva. Other techniques require vestibular depth to be present before the surgery, including pedicle grafts (lateral and coronal), subepithelial connective tissue graft (Langer), and pouch and tunnel procedures.

The availability of donor tissue is another anatomic factor that must be considered. Pedicle displacement of tissue necessitates the presence of an adjacent donor site that presents gingival thickness and width. Palatal tissue thickness is also necessary for the connective tissue donor autograft. Gingival thickness is required at the recipient site for techniques using split-thickness, sandwich-type flap or the pouch and tunnel techniques.

Stability of the Grafted Tissue to the Recipient Site

Good communication of the blood vessels from the grafted donor tissue to the recipient site requires a stable environment. This necessitates sutures that stabilize the donor tissue firmly against the recipient site. The least amount of sutures and maximum stability should be achieved.

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SCIENCE TRANSFER

As defined in this chapter, perioplastic surgery refers to soft tissue relationships and manipulations. *In all these procedures, blood supply is the most significant concern and must be the underlying issue for all decisions regarding the individual surgical procedure. A major complicating factor is the avascular root surface, and many modifications to existing techniques are used to overcome this. Diffusion of fluids is short term and of limited benefit as tissue size increases. Thus the formation of a circulation through anastomosis and angiogenesis is crucial to the survival of these therapeutic procedures.* The formation of vascularity is based on growth molecules, such as vascular endothelial growth factor (VEGF), and cellular migration, proliferation, and differentiation. As tissue-engineering techniques improve, the success and predictability of mucogingival surgery should dramatically increase. Undoubtedly, however, all advancements will have adequate circulation and blood supply as their basis.

Surgical techniques used solely to increase the width and thickness of the keratinized tissue are among the most predictable periodontal procedures. Periodontal plastic surgical procedures for root coverage have less predictability because of the absence of a foundation for blood supply over the root surface. The literature shows that the most predictable root coverage procedure is the use of connective tissue grafts in combination with split-thickness flaps. In cases where it is not possible to obtain a connective tissue graft because of the thinness of the palatal tissue, alternative procedures include coronally displaced flaps with membranes or Emdogain. In cases requiring root coverage where the gingival recession is 2 mm or less, a semilunar pedicle flap can be used. *Cigarette smoking is a contraindication for root coverage techniques because the compromised gingival blood supply results in many failures.*

Minimal Trauma to the Surgical Site

As with all surgical procedures, periodontal plastic surgery is based on the meticulous, delicate, and precise management of the oral tissues. Unnecessary tissue trauma caused by poor incisions, flap perforations, tears, or traumatic and excessive placement of sutures can lead to tissue necrosis. The selection of proper instruments, needles, and sutures is mandatory to minimize tissue trauma; sharp contoured blades (see Figure 63-17 online), smaller-diameter needles, and resorbable monofilament sutures all are important factors in achieving atraumatic surgery.

CONCLUSION

New techniques are constantly being developed and are slowly being incorporated into periodontal practice. The practitioner should be aware that, at times, new methods are published without adequate clinical research to ensure the predictability of the results and the extent to which the techniques may benefit the patient. Critical analysis of newly presented techniques should guide our constant evolution toward better clinical methods.

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 64

Recent Advances in Surgical Technology

*Dennis A. Shanelec, Leonard S. Tibbetts, Isao Ishikawa, Bobby Butler, Akira Aoki, Adriana McGregor, and J. David Cross**

CHAPTER OUTLINE

MICROSURGERY
MAGNIFICATION SYSTEMS
Magnifying Loupes
Surgical Microscope
PERIODONTAL MICROSURGERY
Root Preparation
Surgery under Magnification
Microsurgical Instruments
Ergonomics
Conclusion
LASERS IN PERIODONTICS
Application for Periodontal Therapy
Advantages and Disadvantages

Refer to the Expert CONSULT companion website at www.expertconsult.com for clinical cases in the use of lasers and microsurgery in the supplements to this chapter.

This chapter discusses surgical advances in the following two areas:

1. Recent developments in the use of magnification systems, especially periodontal microsurgery, which are part of a broad movement in medicine and dentistry toward minimally invasive approaches to replace procedures that previously required extensive surgical incisions.
2. Advances in the use of laser technology in periodontal surgery and other areas of periodontal therapy.

MICROSURGERY

Refer to the Expert CONSULT companion website at www.expertconsult.com for a supplement to this chapter with additional content on the use of microsurgery in periodontology.

Microsurgery is defined as a refinement in surgical technique by which visual acuity is increased using a microscope at magnifications exceeding 10×. Although loupes improve normal vision, they do not increase visual acuity to the degree required for true microsurgery. Microsurgery is also an *ergonomic* methodology in which surgical manipulations are improved through better motor coordination. In addition to increasing clinical accuracy, the microscope is important for diagnostic and nonsurgical procedures in periodontics.[3,5,7,30,31]

*The following doctors contributed to this chapter: Bryan S. Pearson, Scott O. Kissel, Leslie Broline, and Robert Henshaw.

MAGNIFICATION SYSTEMS

A variety of simple and complex magnification systems is available to dentists, ranging from simple loupes to prism telescopic loupes and surgical microscopes. Each magnification system has its specific advantages and limitations. Although magnification improves the accuracy of clinical and diagnostic skills, it requires an understanding of optical principles that govern all magnification systems. The assumption that "more magnification is better" must always be weighed against the decrease in field of view and depth of focus that can occur as magnification increases, which is a problem more common with dental loupes than operating microscopes.

Magnifying Loupes

Dental loupes are the most common system of optical magnification used in periodontics. *Loupes* are fundamentally dual monocular telescopes with side-by-side lenses convergent to focus on the operative field. The magnified image formed has stereoscopic properties by virtue of their convergence. A convergent lens optical system is called a *Keplerian optical system.*

Although dental loupes are widely used, they have disadvantages compared with the microscope. The clinician's eyes must converge to view the operative field. This can result in eyestrain, fatigue, and even pathologic vision changes, especially after prolonged use.

Three types of Keplerian loupes are typically used in periodontics: simple or single-element loupes, compound loupes, and prism telescopic loupes. Each type may differ widely in optical sophistication and individual design.

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Simple Loupes. Simple loupes consist of a pair of single meniscus lenses (Figure 64-1). Simple loupes are primitive magnifiers with limited capabilities. Each lens is limited to only two refracting surfaces. Their magnification can only increase by increasing lens diameter and thickness. Size and weight constraints make simple loupes impractical for magnification beyond 1.5×. Another disadvantage of simple loupes is that they are greatly affected by spherical and chromatic aberration. This distorts the image shape and color of objects being viewed.

Compound Loupes. Compound loupes use multi-element lenses with intervening air spaces to gain additional refracting surfaces (Figure 64-2). This allows increased magnification with more favorable working distance and depth of field. Magnification of compound loupes can be increased by lengthening the distance between lenses, thereby avoiding excessive size and weight.

In addition to offering improved optical performance, compound lenses can be *achromatic,* which is an optical feature that clinicians should always choose when selecting magnifying loupes. Achromatic lenses consist of two glass lenses joined together with clear resin. The specific density of each lens counteracts the chromatic aberration of its paired lens to produce a color-correct image. However, multi-element compound loupes become optically inefficient at magnifications above 3×.

Prism Telescopic Loupes. The most advanced loupe optical magnification currently available is the prism telescopic loupe. Such loupes employ Schmidt or "rooftop" prisms to lengthen the light path through a series of switchback mirrors between the lenses. This arrangement folds the light so that the barrel of the loupes can be shortened. Prism loupes produce better magnification, wider depths of field, longer working distances, and larger fields of view than other types of loupes. The barrels of prism loupes are short enough to be mounted on either eyeglass frames (Figure 64-3) or headbands. However, increased weight of prism telescopic loupes with magnification above 4× makes headband mounting more comfortable and stable than eyeglass frame mounting. Recent innovations in prism telescopic loupes include coaxial fiberoptic lighting incorporated in the lens elements to improve illumination (Figure 64-4).

Magnification Range of Surgical Loupes. Dental loupes provide a limited range of magnification, 1.5× to 6×. Loupes delivering magnification of less than 3× are usually inadequate for the visual acuity necessary for clinical periodontics. Surgical loupes providing magnification of more than 4× are impractical because of their small field of view, shallow depth of focus, and excessive weight. Excessively heavy loupes can make it difficult to maintain a stable visual field.

For some periodontal procedures, prism telescopic loupes with magnification of 4× provide an adequate combination of magnification, field of view, and depth of focus. However, the surgical microscope offers much higher magnification and superior optics compared with any of the loupe optical systems mentioned.

Surgical Microscope

The operating microscope provides higher magnification and superior optical performance compared with dental loupes (Figure 64-5). When one considers that a microscope lasts for a clinician's entire career, its expense is minimal. A microscope requires training and practice to gain proficiency but offers better performance and versatility than loupes. Surgical microscopes designed for dentistry employ Galilean optics with binocular eyepieces joined by

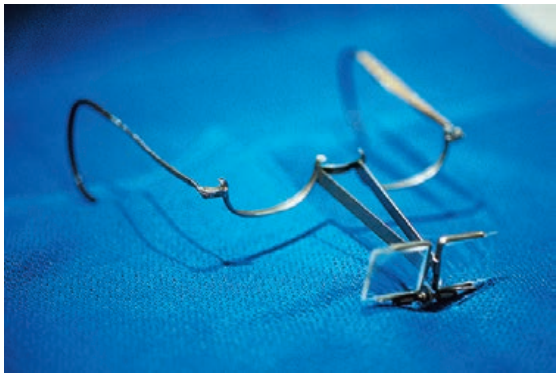

Figure 64-1 Simple loupes.

Figure 64-3 Eyeglass-mounted prism loupes.

Figure 64-2 Compound loupes.

Figure 64-4 Coaxial lighted prism loupes.

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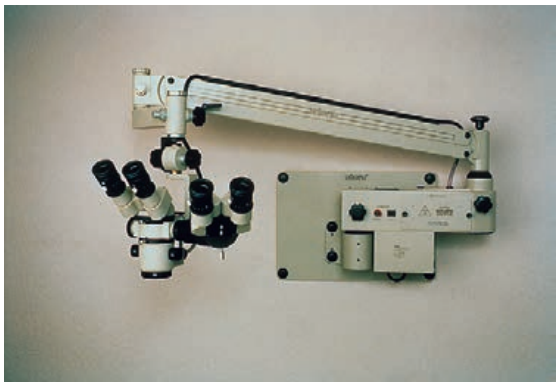

Figure 64-5 Surgical microscope.

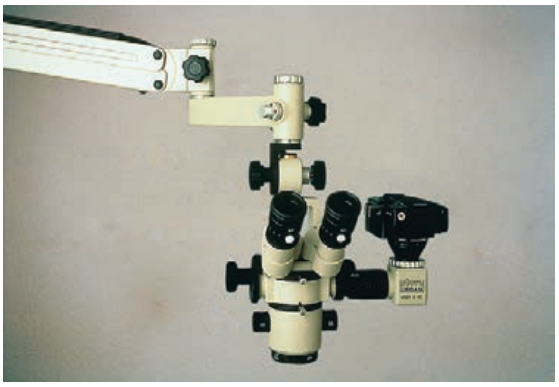

Figure 64-6 Microscope camera and beam splitter.

offsetting prisms to establish parallel optical axes. Galilean optics permits stereoscopic vision without eye convergence, which aligns the eyes as if they were focused on infinity and permits a relaxed vision without eyestrain or fatigue. Operating microscopes incorporate coated optics with achromatic lenses to provide the best optical resolution and the most efficient illumination.

Perhaps the greatest advantage of the surgical microscope is that it allows the dentist to change working magnification easily to a value appropriate for the clinical task at hand.[12] Operating microscopes have a rotating variable-magnification element that changes magnification to match surgical needs. Some operating microscopes incorporate electronic foot-controlled focus and magnification for further convenience. Because the optical elements of surgical microscopes are more advanced than those found in loupes, depth-of-focus and field-of-view characteristics are enhanced.

The periodontal surgeon must establish adequate working distance between the surgical field and the objective microscope lens. This distance permits the surgical assistant to retract tissues and irrigate or evacuate the surgical site. Such assistant-aided control of surgical access is essential for microsurgical visibility. Assistant eyepiece attachments are available for surgical microscopes and can greatly aid the progress of microsurgical procedures. Surgical microscopes have objective lenses with various working distances. A useful range in dentistry is 250 to 350 mm. Because operating with indirect mirror vision adds 100 to 150 mm to the working distance, a ready means of changing working distances is valuable. Quick-change objective lenses are available for many surgical microscopes.

For practical use in periodontics, the surgical microscope must have both *maneuverability* and *stability*. Microscope mountings are available for ceiling, wall, or floor. Inclining eyepieces lend great postural flexibility to clinical use of the microscope in periodontics. Maneuverability must be sufficient to provide visual access to the posterior region of the mouth and to all anatomic structures addressed during periodontal treatment. The optical quality of various microscopes is comparable. Maneuverability therefore becomes more important than optics in choosing an appropriate microscope for periodontics.

Illumination of the microsurgical field is an important consideration. Periodontists are accustomed to lateral illumination from side-mounted dental lights. Clinicians who work with loupes often require a headlamp to compensate for the decreased illumination of dental loupes. Fiberoptic coaxial illumination is a major advantage of the operating microscope over surgical loupes. Coaxial lighting focuses the light parallel to the microscope's optical axis. With coaxial lighting, no shadows are produced. The surgeon can view perfectly the deepest reaches of the oral cavity, including into subgingival pockets and angular bony defects. Definitive visualization of root surface deposits and irregularities is only possible at magnification and resolution provided by a surgical microscope. Using the microscope, surgeons can view periodontal anatomy that previously could not be seen. Clinical decisions can be made based on certain knowledge of pathologic anatomy rather than decisions based on blind, educated guesses.

Documentation of periodontal procedures has become important for dental-legal reasons, as well as for patient and professional education. The surgical microscope is ideal for documenting periodontal pathology and procedures of all types; digital or 35-mm images can be produced using a beam-splitter camera attachment (Figure 64-6). With a foot-operated shutter control, the surgeon can compose the photographic field as the procedure unfolds without interrupting surgery. In addition, the microphotographic image represents the surgical field exactly as the surgeon sees it, as opposed to a camera view over the surgeon's shoulder. High-quality video documentation is also possible through the operating microscope using a video beam-splitter attachment. Digital cameras providing motion and still images are currently replacing 35-mm cameras for documentation in many microsurgical applications. Such high-resolution cameras bring new capabilities for live or recorded video of periodontal procedures for educational purposes.

PERIODONTAL MICROSURGERY

Refer to the Expert CONSULT companion website at www.expertconsult.com for a supplement to this chapter with additional content on periodontal microsurgery.

In recent years, periodontics has seen increasing application of procedures requiring progressively more intricate surgical skills. Regenerative and resective surgical procedures, periodontal plastic surgery, and dental implants all demand clinical performance levels that challenge the technical and motor skills of periodontal surgeons beyond a range possible with unassisted vision.

Periodontal microsurgery introduces the potential for a less invasive surgical approach in periodontics. This is exemplified by a decreased need for vertical releasing incisions and greater use of smaller surgical sites. Periodontal surgeons, as with other microsurgeons, continue to notice the extent to which reduced incision size and surgical retraction are directly related to decreased postoperative pain and rapid healing.[24,34]

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Root Preparation

The importance of root debridement is recognized universally as an essential component of periodontal therapy.[11,18,23,27] Research in clinical dentistry has shown that microscope-enhanced vision more readily accomplishes the long-established clinical goals of endodontic and restorative dentistry. In periodontics, studies demonstrated that root debridement performed without magnification was incomplete. When debrided roots were examined with the aid of a microscope, substantial deposits remained. Even in the absence of clinical studies, it may be inferred that microscope-enhanced vision in periodontics permits more definitive root debridement.

The primary goals of periodontal surgery include visual access to the root surface for plaque and calculus removal and for removing pathologically altered tooth structure. Magnification greatly improves the surgeon's ability to create a clean, smooth root surface (Figure 64-7). The root surface represents one opposing edge of the periodontal wound. Root planing is therefore analogous to establishing a clean soft tissue incision. Magnification permits preparation of both hard and soft tissue wound surfaces so that they may be joined together according to the accepted microsurgical principle of butt-joint wound approximation. This encourages primary wound healing and enhanced periodontal reconstruction. Wound-healing studies show epithelial anastomosis of microsurgically joined surgical wounds in animals within 48 hours.[5,17,34]

Surgery under Magnification

Viewing periodontal surgery under magnification impresses the periodontal surgeon with the coarseness of conventional surgical manipulation. What appears to the unaided eye as gentle surgery is revealed under magnification to be gross crushing and tearing of delicate tissues. Periodontists have long advocated atraumatic surgery to achieve primary wound closure. However, the limits of normal vision made this goal impossible. Periodontal microsurgery is the natural transition from conventional surgical principles to a surgical ethic in which the microscope is employed to permit the most accurate and atraumatic handling of tissue to enhance wound healing.

Microsurgical Instruments

In addition to the use of magnification and reliance on atraumatic technique, microsurgery requires specially constructed instruments designed specifically to minimize trauma. An important characteristic of microsurgical instruments is their ability to create clean incisions that prepare wounds for healing by primary intention. Microsurgical incisions are established at a 90-degree angle to the surface using ophthalmic microsurgical scalpels (Figure 64-8). Microscopy permits easy identification of ragged wound edges for trimming and freshening. For primary wound closure, microsutures in the range of 6-0 to 9-0 are needed to approximate the wound edges accurately (Figure 64-9). Microsurgical wound apposition minimizes gaps or voids at the wound edges and encourages rapid healing with less postoperative inflammation and less pain.

Figures 64-10 and 64-11 illustrate periodontal surgery cases using microsurgical techniques.

Ergonomics

The ergonomics of hand position and body posture are closely related to improved motor skills made possible by a microsurgical approach to therapy. Studies show that motor coordination is greatly improved when surgeons use microsurgical instruments specifically designed to employ a precision grip of the hand.

Microsurgical instruments are circular in cross-section to permit precise rotational movements. They are manufactured of titanium

Figure 64-7 Magnified root planing.

Figure 64-8 Castroviejo microsurgical scalpel.

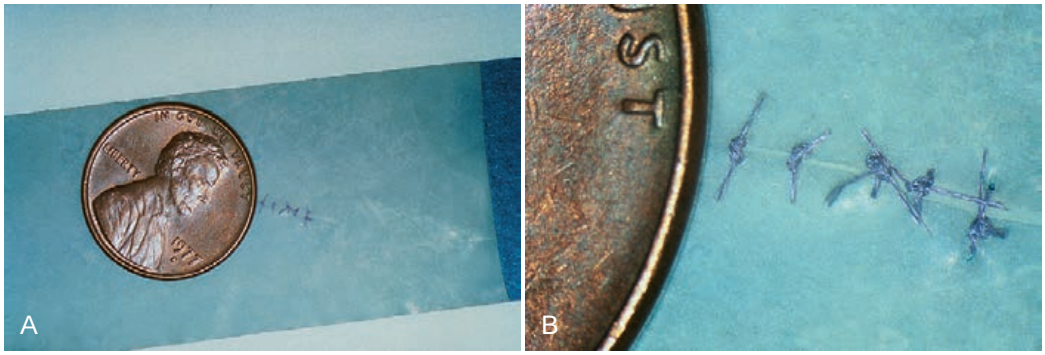

Figure 64-9 Microsurgical suturing.

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Figure 64-10 Microsurgical extraction. **A,** Before surgery. **B,** Microsurgical view. **C,** One week after surgery.

Figure 64-11 Papilla reconstruction. **A,** Before surgery. **B,** Microsurgical view. **C,** After surgery.

because of its strength, lightness, and nonmagnetic characteristics. The various postural and ergonomic methods of reducing unwanted hand movements result in more precise surgeries. These methods also greatly reduce surgical fatigue, as well as the spinal and occupational pathology common in periodontics. In the long term, the beneficial ergonomic aspects of microscopy may be the most influential factors in its adoption by the dental profession at large.

Conclusion

Microsurgery offers new opportunities for periodontal surgery that can enhance the therapeutic results for a variety of procedures. Its benefits include improved cosmetics, rapid healing, minimal discomfort, and enhanced patient acceptance. Periodontics of the future will see increasing use of magnification in all areas of practice, including implantology.

LASERS IN PERIODONTICS

Refer to the Expert CONSULT companion website at www.expertconsult.com for a supplement to this chapter with additional content on the use of lasers in periodontology.

Laser is an acronym for *l*ight *a*mplification by *s*timulated *e*mission of *r*adiation. The stimulated emission of a photon by an excited atom, which triggers the release of a subsequent photon, is responsible for generation of a coherent, monochromatic, and collimated form of light, or laser. Lasers can concentrate light energy and exert a strong effect, targeting tissue at an energy level much lower than natural light. The wavelength of a laser determines its characteristics (Figure 64-12).

Maiman[19] invented the first laser device in 1960, based on theories derived by Einstein in the early 1900s. Since then, lasers have been used for various purposes in medicine and surgery. Once in contact with tissue, laser energy is reflected, scattered, absorbed, or transmitted to the neighboring tissues (Figure 64-13). The water molecules, proteins, pigments, and other macromolecules present in biologic tissue are responsible for absorption, but the absorption coefficient actually depends on the wavelength of the incoming laser irradiation.[22]

Application for Periodontal Therapy

The neodymium:yttrium-aluminum-garnet (Nd:YAG), carbon dioxide (CO_2), diode, erbium:YAG (Er:YAG), erbium, chromium: yttrium (Er,Cr:YSGG), and argon are the lasers most often used and studied in dentistry (Table 64-1). In dentistry, lasers were first used in the field of operative dentistry for caries removal and cavity preparation.[9] The effects of newer lasers (e.g., argon, CO_2, Nd:YAG, diode) on dental hard tissue and caries have been investigated[15,33] but were reported to be unsuccessful because of thermal damage.

The use of lasers for periodontal treatment becomes more complex because the periodontium consists of both hard and soft tissues. Among the many lasers available, high-power lasers, such as CO_2, Nd:YAG, and diode lasers, can be used in periodontics. Because of their excellent soft tissue ablation and hemostatic characteristics, the use of these lasers has been approved for soft tissue management in periodontal and oral surgery.[6,10,26,28] However, when applied to the root surface or alveolar bone, carbonization and major thermal damage have been reported on the target and adjacent tissues.[28] Therefore the use of these lasers is limited to gingivectomy, frenectomy, and similar soft tissue procedures,[6,10,26] including the removal of melanin pigmentation[4,32,37] and metal tattoos of the gingiva.[28]

The use of lasers also has been investigated for other indications in periodontal therapy, including subgingival debridement and curettage,[2] removal of granulation tissue during flap surgery,[35] and osseous recontouring,[21] as well as in implant surgery,[8] maintenance of implants, and management of periimplantitis.[16,20]

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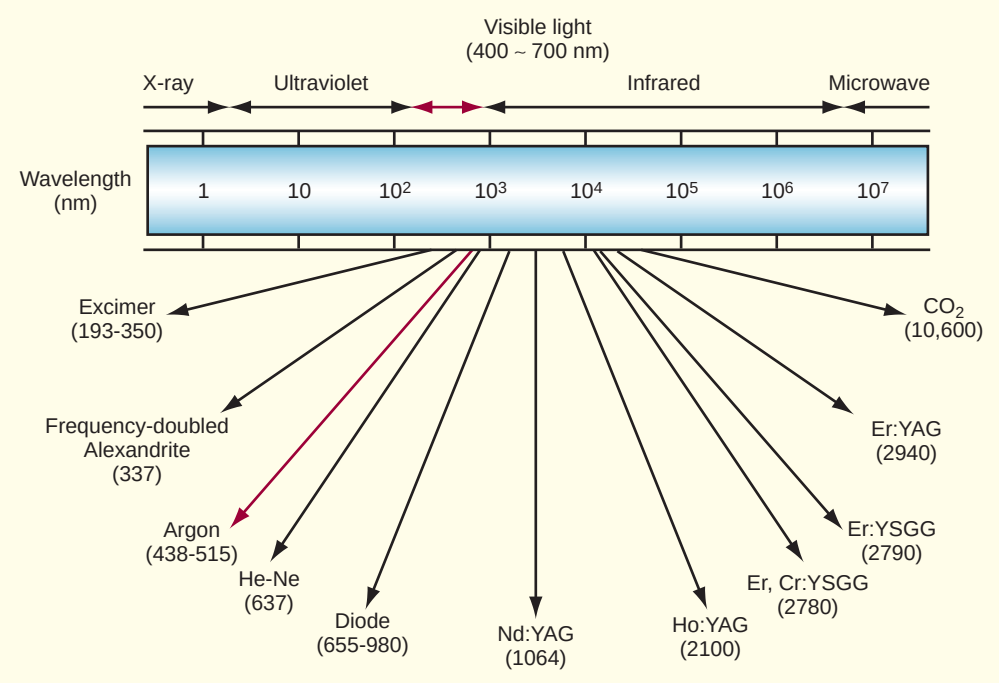

Figure 64-12 Wavelengths of various lasers.

TABLE 64-1 Current and Potential Applications of Lasers in Dentistry

Type	Active Medium	Dental Applications
Excimer lasers	Argon-fluoride (ArF) Xenon-chloride (XeCl)	Hard tissue ablation, dental calculus removal
Gas lasers	Argon (Ar)	Curing of composite materials, tooth whitening, intraoral soft tissue surgery, sulcular debridement (subgingival curettage in periodontitis and periimplantitis)
	Helium-neon (HeNe)	Analgesia, treatment of dentin hypersensitivity, aphthous ulcer treatment
	Carbon dioxide (CO_2)	Intraoral soft tissue and soft tissue surgery, aphthous ulcer treatment, removal of gingival melanin pigmentation, treatment of dentin hypersensitivity, analgesia
Diode lasers	Indium-gallium-arsenide-phosphorus (InGaAsP) Galium-aluminum-arsenide (GaAlAs) Galium-arsenide (GaAs)	Caries and calculus detection Intraoral general and implant soft tissue surgery, sulcular debridement (subgingival curettage in periodontitis and periimplantitis), analgesia, treatment of dentin hypersensitivity, pulpotomy, root canal disinfection, aphthous ulcer treatment, removal of gingival melanin pigmentation
Solid-state lasers	Frequency-doubled alexandrite Neodymium:yttrium-aluminum-garnet (Nd:YAG)	Selective ablation of dental plaque and calculus Intraoral soft tissue surgery, sulcular debridement (subgingival curettage in periodontitis), analgesia, treatment of dentin hypersensitivity, pulpotomy, root canal disinfection, removal of enamel caries, aphthous ulcer treatment, removal of gingival melanin pigmentation
	Erbium group Erbium:YAG (Er:YAG) Erbium:yttrium (Er:YSGG) Erbium, chromium:YSGG (Er,Cr:YSGG)	Caries removal and cavity preparation, modification of enamel and dentin surfaces, intraoral general and implant soft tissue surgery, sulcular debridement (subgingival curettage in periodontitis and periimplantitis), scaling of root surfaces, osseous surgery, treatment of dentin hypersensitivity, analgesia, pulpotomy, root canal treatment and disinfection, aphthous ulcer treatment, removal of gingival melanin and metal-tattoo pigmentation

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Microsurgical techniques using magnification procedures give clinicians a methodology to carry out the most precise and atraumatic surgical procedures possible. These techniques require special training, instrumentation, and procedures. Surgeons need to be dedicated to excellence and have outstanding technical and clinical skills. Patients benefit from the microsurgical approach because healing is optimized and treatment outcomes may be improved.

Light amplification by stimulated emission of radiation (laser) therapy has been applied to periodontal treatment using a number of different lasers and different procedures. It is essential that thorough root planing is done and plaque control is maintained. Treatment results have been variable, but recent reports of success using the Neodymium:yttrium-aluminum-garnet (Nd:YAG) laser with the excisional new attachment procedure (ENAP) technique show promise of wider applications.

With both microsurgical techniques and laser procedures, there is a need for more controlled comparative clinical trials to establish a foundation for evidence-based evaluation of the place of these therapies in the overall spectrum of periodontal therapy.

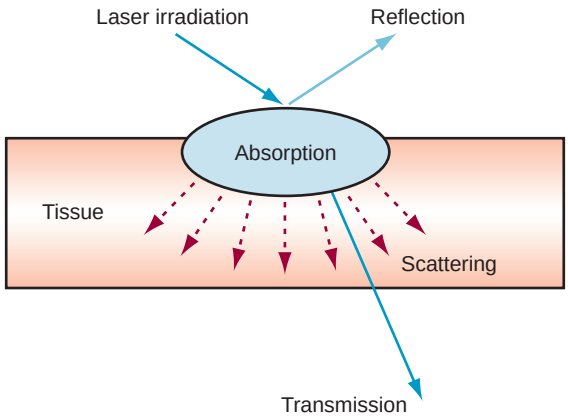

Figure 64-13 Interaction of human tissue and laser irradiation.

BOX 64-1 Precautions and Risks Associated with Clinical Use of Lasers

Precautions before and during Irradiation

- Use glasses for eye protection (patient, operator, and assistants).
- Prevent inadvertent irradiation (action in noncontact mode).
- Protect the patient's eyes, throat, and oral tissues outside the target site.
- Use wet gauze packs to avoid reflection from shiny metal surfaces.
- Ensure adequate high-speed evacuation to capture the laser plume.

Potential Risks

- Excessive tissue destruction by direct ablation and thermal side effects.
- Destruction of the attachment apparatus at the bottom of pockets.
- Excessive ablation of root surface and gingival tissue within periodontal pockets.
- Thermal injury to the root surface, gingival tissue, pulp, and bone tissue.

Advantages and Disadvantages

Advantages of laser treatment are greater hemostasis, bactericidal effect, and minimal wound contraction.[1,16,36] However, previous laser systems have strong thermal side effects, leading to melting, cracking, and carbonization of hard tissues.

Recently, Er:YAG and Er,Cr:YSGG lasers have been developed for dental applications. Of all dental lasers available, the absorption of the Er:YAG and Er,Cr:YSGG lasers in water is the highest. These lasers effectively ablate all biologic tissues that contain water molecules[1,16,36] and are applicable to both soft and hard tissues.[2,8,21] The erbium laser group has emerged as a promising laser system for periodontal indications.[2,14,29] Further research and development are needed, however, before they can be recommended for these purposes.[2,13,14]

The use of lasers also has disadvantages that require precautions to be taken during clinical application (Box 64-1).[6] Laser irradiation can interact with tissues even in the noncontact mode, which means that laser beams may reach the patient's eyes and other tissues surrounding the target in the oral cavity. Clinicians should be careful to prevent inadvertent irradiation to these tissues, especially to the eyes. Protective eyewear specific for the wavelength of the laser in use must be worn by the patient, operator, and assistant.[6] Laser beams can be reflected by shiny surfaces of metal dental instruments, causing irradiation to other tissues, which should be avoided by using wet gauze packs over the area surrounding the target.[25]

During CO_2 or Nd:YAG irradiation for periodontal treatment, caution must be taken not to contact enamel surfaces because of the risk of melting. Improper irradiation in periodontal pockets could cause thermal damage to the root surface and pocket walls and destruction of the intact attachment apparatus at the base of the pocket, affecting the normal cell attachment during healing. Damage to the underlying bone and pulp tissue is another concern. Therefore proper irradiation techniques and conditions need to be followed when using lasers.

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CHAPTER 65

Preparation of the Periodontium for Restorative Dentistry

Philip R. Melnick

CHAPTER OUTLINE

RATIONALE FOR THERAPY
SEQUENCE OF TREATMENT
CONTROL OF ACTIVE DISEASE (online only)
- Emergency Treatment
- Extraction of Hopeless Teeth
- Oral Hygiene Measures
- Scaling and Root Planing
- Reevaluation
- Periodontal Surgery
- Adjunctive Orthodontic Therapy

PREPROSTHETIC SURGERY (online only)
- Management of Mucogingival Problems
- Preservation of Ridge Morphology after Tooth Extraction
- Crown-Lengthening Procedures
- Alveolar Ridge Reconstruction

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Periodontal health is the "sine qua non," a prerequisite, of successful comprehensive dentistry.[23] To achieve the long-term therapeutic targets of comfort, good function, treatment predictability, longevity, and ease of restorative and maintenance care, active periodontal infection must be treated and controlled before the initiation of restorative, esthetic, and implant dentistry. In addition, the residual effects of periodontal disease or anatomic aberrations inconsistent with realizing and maintaining long-term stability must be addressed. More recently, this phase of treatment includes techniques performed in anticipation of esthetic or implant dentistry, such as clinical crown lengthening, covering denuded roots, alveolar ridge retention or augmentation, and implant site development.

RATIONALE FOR THERAPY

The many reasons for establishing periodontal health before performing restorative dentistry include the following[45]:

1. Periodontal treatment is undertaken to ensure the establishment of stable gingival margins before tooth preparation. Noninflamed, healthy tissues are less likely to change (e.g., shrink) as a result of subgingival restorative treatment or postrestoration periodontal care.[26,27] In addition, tissues that do not bleed during restorative manipulation allow for a more predictable restorative and esthetic result.[20,21]
2. Certain periodontal procedures are designed to provide for adequate tooth length for retention, access for tooth preparation, impression making, tooth preparation, and finishing of restorative margins in anticipation of restorative dentistry.[20,41] Failure to complete these procedures before restorative care can add to the complexity of treatment and introduce unnecessary risk of failure.[20]
3. Periodontal therapy should antecede restorative care because the resolution of inflammation may result in the repositioning of teeth[40] or in soft tissue and mucosal changes. Failure to anticipate these changes may interfere with prosthetic designs planned or constructed before periodontal treatment.
4. Traumatic forces placed on teeth with ongoing periodontitis may increase tooth mobility, discomfort, and possibly the rate of attachment loss.[8] Restorations constructed on teeth free of periodontal inflammation, synchronous with a functionally appropriate occlusion, are more compatible with long-term periodontal stability and comfort (see Chapters 15 and 49).
5. Quality, quantity, and topography of the periodontium may play important roles as structural defense factors in

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During phase I therapy, clinicians should correct any faulty restorative problems and position malposed teeth for optimization of periodontal health. In phase II therapy, crown lengthening procedures are utilized to allow proper retention of crowns. Generally this will involve flap surgery combined with osseous surgery, but in a few cases with pockets 5 mm or deeper and a wide band of keratinized gingiva, gingivectomy may be used. Clinicians should establish the level of the subgingival restorative margin and then remove bone so that it is 2 to 3 mm apical to this margin. Mucogingival procedures to correct inadequate attached keratinized gingival tissue should be completed before restorative treatments in that region. The use of regenerative materials during extractions to maintain alveolar ridge dimensions provides optimal forms of edentulous regions for idealized implant placement and pontic form.

BOX 65-1 Sequence of Treatment in Preparing Periodontium for Restorative Dentistry

Control of Active Disease
Emergency treatment
Extraction of hopeless teeth
Oral hygiene instructions
Scaling and root planing
Reevaluation
Periodontal surgery
Adjunctive orthodontic therapy

Preprosthetic Surgery
Management of mucogingival problems
Preservation of ridge morphology after tooth extraction
Crown-lengthening procedures
Alveolar ridge reconstruction

maintaining periodontal health. Orthodontic tooth movement and restorations completed without the benefit of periodontal treatment designed for this purpose may be subject to negative changes that complicate construction and future maintenance.[47]

6. Successful esthetic and implant procedures may be difficult or impossible without the specialized periodontal procedures developed for this purpose.

SEQUENCE OF TREATMENT

Treatment sequencing should be based on logical and evidenced-based methodologies, taking into account not only the disease state encountered but also the psychologic and esthetic concerns of the patient. Because periodontal and restorative therapy is situational and specific to each patient, a plan must be adaptable to change depending on the variables encountered during the course of treatment. For example, teeth initially determined to be salvageable may be judged "hopeless," thus altering the established treatment scheme.

Generally, the preparation of the periodontium for restorative dentistry can be divided into two phases: (1) control of periodontal inflammation with nonsurgical and surgical approaches and (2) preprosthetic periodontal surgery (Box 65-1).

For more information on control of active disease and preprosthetic surgery, please visit the companion Expert CONSULT website at www.expertconsult.com.

CONCLUSION

As described in this and other sections of this textbook, the therapeutic goals of patient comfort, function, esthetics, predictability, longevity, and ease of restorative and maintenance care are attainable only by a carefully constructed interdisciplinary approach, with accurate diagnosis and comprehensive treatment planning serving as cornerstones. The complex interaction between periodontal therapy and successful restorative dentistry only serves to underscore this premise.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 66

Restorative Interrelationships

Frank M. Spear and Joseph P. Cooney

CHAPTER OUTLINE

BIOLOGIC CONSIDERATIONS
- Margin Placement and Biologic Width
- Biologic Width Evaluation
- Correcting Biologic Width Violations
- Margin Placement Guidelines
- Clinical Procedures in Margin Placement
- Tissue Retraction
- Provisional Restorations
- Marginal Fit
- Crown Contour
- Subgingival Debris
- Hypersensitivity to Dental Materials

ESTHETIC TISSUE MANAGEMENT (online only)
- Managing Interproximal Embrasures
- Correcting Open Gingival Embrasures Restoratively
- Managing Gingival Embrasure Form for Patients with Gingival Recession
- Pontic Design

OCCLUSAL CONSIDERATIONS IN RESTORATIVE THERAPY (online only)

SPECIAL RESTORATIVE CONSIDERATIONS (online only)
- Root-Resected Teeth
- Splinting
- Anterior Esthetic Surgery

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

The relationship between periodontal health and the restoration of teeth is intimate and inseparable. For restorations to survive long term, the periodontium must remain healthy so that the teeth are maintained. For the periodontium to remain healthy, restorations must be critically managed in several areas so that they are in harmony with their surrounding periodontal tissues. To maintain or enhance the patient's esthetic appearance, the tooth/tissue interface must present a healthy natural appearance, with gingival tissues framing the restored teeth in a harmonious manner. This chapter reviews the key areas of restorative management necessary to optimize periodontal health, with a focus on the esthetics and function of restorations.

BIOLOGIC CONSIDERATIONS

Margin Placement and Biologic Width

One of the most important aspects of understanding the periodontal-restorative relationship is the location of the restorative margin to the adjacent gingival tissue. Restorative clinicians must understand the role of biologic width in preserving healthy gingival tissues and controlling the gingival form around restorations. They must also apply this information in the positioning of restoration margins, especially in the esthetic zone, where a primary treatment goal is to mask the junction of the margin with the tooth.

A clinician is presented with three options for margin placement: supragingival, equigingival (even with the tissue), and subgingival.[70] The *supragingival margin* has the least impact on the periodontium. Classically, this margin location has been applied in unesthetic areas because of the marked contrast in color and opacity of traditional restorative materials against the tooth. With the advent of more translucent restorative materials, adhesive dentistry, and resin cements, the ability to place supragingival margins in esthetic areas is now a reality (Figures 66-1 and 66-2). Therefore, whenever possible, these restorations should be chosen not only for their esthetic advantages but also for their favorable periodontal impact.

The use of *equigingival margins* traditionally was not desirable because they were thought to retain more plaque than supragingival or subgingival margins and therefore result in greater gingival inflammation. There was also the concern that any minor gingival recession would create an unsightly margin display. These concerns are not valid today not only because the restoration margins can be esthetically blended with the tooth but also because restorations can be finished easily to provide a smooth, polished interface at the gingival margin. From a periodontal viewpoint, both supragingival and equigingival margins are well tolerated.

The greatest biologic risk occurs when placing *subgingival margins.*[43] These margins are not as accessible as supragingival or equigingival margins for finishing procedures. In addition, if the

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Figure 66-1 With the advent of adhesive dentistry and ultrathin ceramic veneers, it now is possible to prepare restorations equigingival without visible margins. The preparations for six porcelain veneers with the margins placed at the level of tissue are shown.

Figure 66-2 The completed veneers from Figure 66-1. Note the invisible gingival finish line, even though the margin has not been carried below tissue.

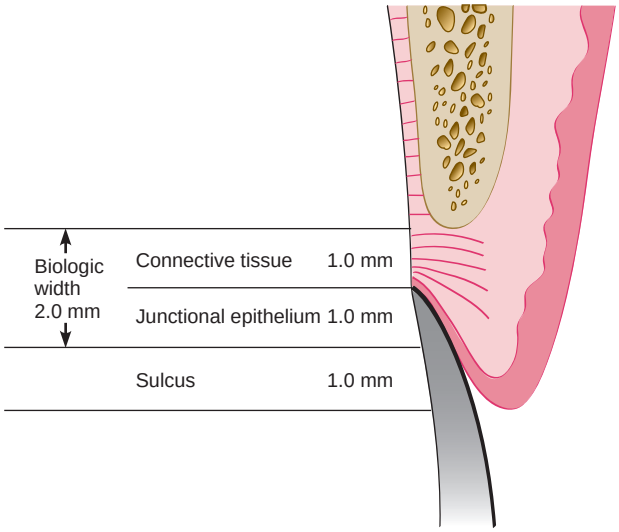

Figure 66-3 Average human biologic width: Connective tissue attachment 1 mm in height; junctional epithelial attachment 1 mm in height; sulcus depth of approximately 1 mm. The combined connective tissue attachment and junctional epithelial attachment, or biologic width, equals 2 mm.

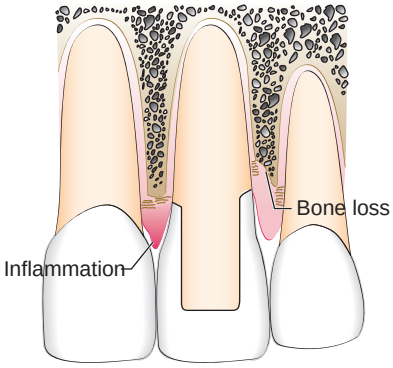

Figure 66-4 Ramifications of a biologic width violation if a restorative margin is placed within the zone of the attachment. On the mesial surface of the left central incisor, bone has not been lost, but gingival inflammation occurs. On the distal surface of the left central incisor, bone loss has occurred, and a normal biologic width has been reestablished.

margin is placed too far below the gingival tissue crest, it violates the gingival attachment apparatus.

As described in Chapter 2, the dimension of space that the healthy gingival tissues occupy between the base of the sulcus and the underlying alveolar bone is comprised of the junctional epithelial attachment and the connective tissue attachment. The combined attachment width is now identified as the *biologic width.* Most authors credit Gargiulo, Wentz, and Orban's study in 1961[19] on cadavers with the initial research establishing the dimensions of space required by the gingival tissues. They found that, in the average human, the connective tissue attachment occupies 1.07 mm of space above the crest of the alveolar bone and that the junctional epithelial attachment below the base of the gingival sulcus occupies another 0.97 mm of space above the connective tissue attachment. The combination of these two measurements, averaging approximately 1 mm each, constitutes the biologic width (Figure 66-3). Clinically, this information is applied to diagnose biologic width violations when the restoration margin is placed 2 mm or less away from the alveolar bone and the gingival tissues are inflamed with no other etiologic factors evident.

Restorative considerations frequently dictate the placement of restoration margins beneath the gingival tissue crest. Restorations may need to be extended gingivally (1) to create adequate resistance and retentive form in the preparation, (2) to make significant contour alterations because of caries or other tooth deficiencies, or (3) to mask the tooth/restoration interface by locating it subgingivally. When the restoration margin is placed too far below the gingival tissue crest, it impinges on the gingival attachment apparatus and create a violation of biologic width.[50] Two different responses can be observed from the involved gingival tissues (Figure 66-4).

One possibility is that bone loss of an unpredictable nature along with gingival tissue recession occurs as the body attempts to recreate room between the alveolar bone and the margin to allow space for tissue reattachment. This is more likely to occur in areas in which the alveolar bone surrounding the tooth is very thin in width. Trauma from restorative procedures can play a major role in causing this fragile tissue to recede. Other factors that may impact the likelihood of recession include (1) whether the gingiva is thick and fibrotic or thin and fragile and (2) whether the periodontium is highly scalloped or flat in its gingival form. It has been found

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that highly scalloped, thin gingiva is more prone to recession than a flat periodontium with thick fibrous tissue.[48]

The more common finding with deep margin placement is that the bone level appears to remain unchanged, but gingival inflammation develops and persists. To restore gingival tissue health, it is necessary to establish space clinically between the alveolar bone and the margin. This can be accomplished either by surgery to alter the bone level or by orthodontic extrusion to move the restoration margin farther away from the bone level.

Biologic Width Evaluation

Radiographic interpretation can identify interproximal violations of biologic width. However, with the more common locations on the mesiofacial and distofacial line angles of teeth, radiographs are not diagnostic because of tooth superimposition. If a patient experiences tissue discomfort when the restoration margin levels are being assessed with a periodontal probe, it is a good indication that the margin extends into the attachment and that a biologic width violation has occurred.

A more positive assessment can be made clinically by measuring the distance between the bone and the restoration margin using a sterile periodontal probe. The probe is pushed through the anesthetized attachment tissues from the sulcus to the underlying bone. If this distance is less than 2 mm at one or more locations, a diagnosis of biologic width violation can be confirmed. This assessment is completed circumferentially around the tooth to evaluate the extent of the problem. However, biologic width violations can occur in some patients in whom the margins are located more than 2 mm above the alveolar bone level.[23] In 1994, Vacek et al[71] also investigated the biologic width phenomenon. Although their average width finding of 2 mm was the same as that previously presented by Gargiulo et al,[19] they also reported a range of different biologic widths that were patient specific. They reported biologic widths as narrow as 0.75 mm in some individuals, whereas others had biologic widths as tall as 4.3 mm (Figure 66-5).

This information dictates that specific biologic width assessment should be performed for each patient to determine if the patient needs additional biologic width, in excess of 2 mm, for restorations to be in harmony with the gingival tissues. The biologic, or attachment, width can be identified for the individual patient by probing to the bone level (referred to as "sounding to bone") and subtracting the sulcus depth from the resulting measurement. This measurement must be done on teeth with healthy gingival tissues and should be repeated on more than one tooth to ensure an accurate assessment. The technique allows the variations in sulcus depths found in individual patients to be assessed and factored into the diagnostic evaluation. The information obtained is then used for definitive diagnosis of biologic width violations, the extent of correction needed, and the parameters for placement of future restorations.

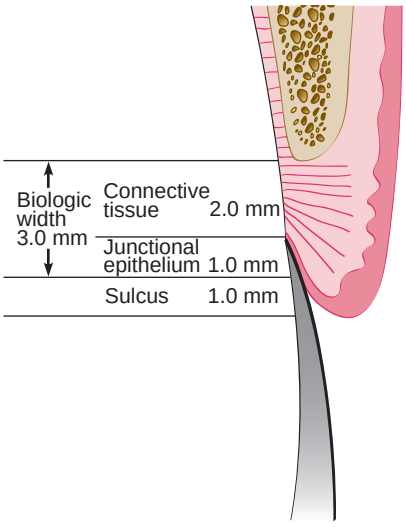

Figure 66-5 Possible variations exist in biologic width. Connective tissue attachments and junctional epithelial attachments may be variable. In this example the connective tissue attachment is 2 mm in height, the junctional epithelial attachment 1 mm in height, and the sulcus depth 1 mm, for a combined total tissue height above bone of 4 mm. However, the biologic width is 3 mm. This is just one variation that can occur from the average depicted in Figure 66-3.

Correcting Biologic Width Violations

Biologic width violations can be corrected either by surgically removing bone away from proximity to the restoration margin or by orthodontically extruding the tooth and thus moving the margin away from the bone. Surgery is the more rapid of the two treatment options.[58] It is also preferred if the resulting crown lengthening creates a more pleasing tooth length. Indications and contraindications for surgical crown lengthening are presented in Chapter 65 and illustrated with clinical examples. In these situations the bone should be moved away from the margin by the measured distance of the ideal biologic width for that patient, with an additional 0.5 mm of bone removed as a safety zone.

There is a potential risk of gingival recession after removal of bone.[7] If interproximal bone is removed, there is a high likelihood of papillary recession and the creation of an unesthetic triangle of space below the interproximal contacts. If the biologic width violation is on the interproximal side or if the violation is across the facial surface and the gingival tissue level is correct, orthodontic extrusion is indicated[28] (Figures 66-6 to 66-9). The extrusion can be performed in two ways. By applying low orthodontic extrusion force, the tooth will erupt slowly, bringing the alveolar bone and gingival tissue with it. The tooth is extruded until the bone level has been carried coronal to the ideal level by the amount that needs to be removed surgically to correct the attachment violation. The tooth is stabilized in this new position and then is treated with surgery to correct the bone and gingival tissue levels. Another option is to perform rapid orthodontic extrusion where the tooth is erupted to the desired amount over several weeks.[32] During this

Figure 66-6 The left central incisor was fractured in an accident 12 months ago and restored at that time. The patient is unhappy with the appearance of the tissue surrounding the restoration (See Figures 66-7, 66-8, and 66-9).

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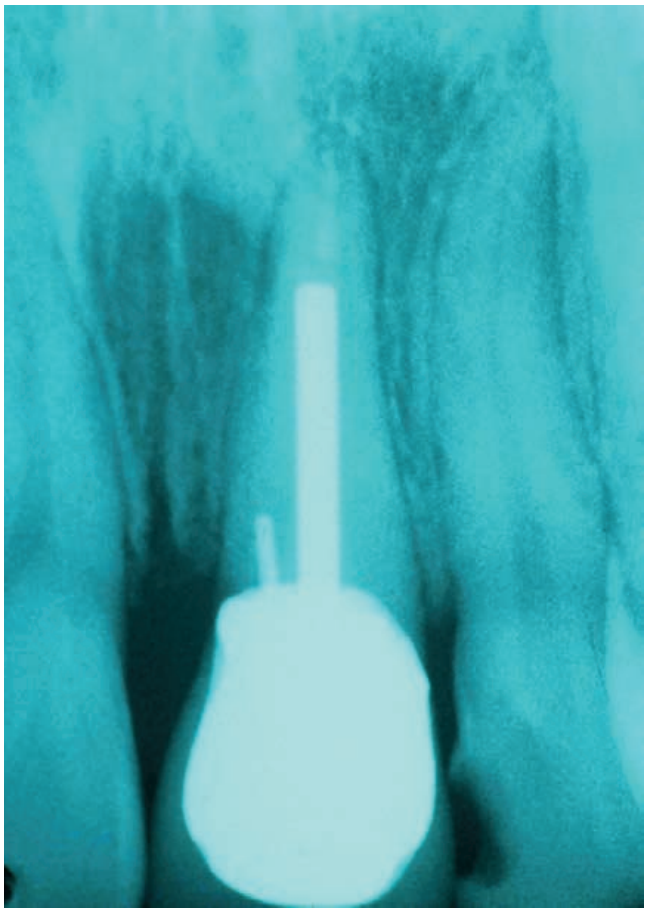

Figure 66-7 Radiograph reveals a biologic width violation on the mesial surface interproximally. Removal of interproximal bone would create an esthetic deformity. This patient is better treated with orthodontic extrusion (see Figures 66-6 and 66-8).

Figure 66-8 After orthodontic eruption. The tooth has been erupted 3 mm to move the bone and gingiva coronally 3 mm on the left central incisor. It is now possible to reposition the bone surgically to the correct level and position the gingiva to the correct level, reestablishing normal biologic width.

period, a supracrestal fiberotomy is performed circumferentially around the tooth weekly in an effort to prevent the tissue and bone from following the tooth. The tooth is then stabilized for at least 12 weeks to confirm the position of the tissue and bone, and any coronal creep can be corrected surgically.

Figure 66-9 One-year recall photograph after orthodontic extrusion, osseous surgery, and placement of a new restoration for patient in Figure 66-6. Note the excellent tissue health after the reestablishment of biologic width.

Margin Placement Guidelines

When determining where to place restorative margins relative to the periodontal attachment, it is recommended that the patient's existing sulcular depth be used as a guideline in assessing the biologic width requirement for that patient. The base of the sulcus can be viewed as the top of the attachment, and therefore the clinician accounts for variations in attachment height by ensuring that the margin is placed in the sulcus and not in the attachment.[4,37,38,57] The variations in sulcular probing depth are then used to predict how deep the margin can safely be placed below the gingival crest. With shallow probing depths (1.0 to 1.5 mm), extending the preparation more than 0.5 mm subgingivally risks violating the attachment. This assumes that the periodontal probe will penetrate into the junctional epithelial attachment in healthy gingiva an average of 0.5 mm. With shallow probing depths, future recession is unlikely because the free gingival margin is located close to the top of the attachment. Deeper sulcular probing depths provide more freedom in locating restoration margins farther below the gingival crest. In most circumstances, however, the deeper the gingival sulcus, the greater is the risk of gingival recession.

The first step in using sulcus depth as a guide in margin placement is to manage gingival health. Once the tissue is healthy, the following three rules can be used to place intracrevicular margins:

- **Rule 1**: If the sulcus probes 1.5 mm or less, place the restoration margin 0.5 mm below the gingival tissue crest. This is especially important on the facial aspect and will prevent a biologic width violation in a patient who is at high risk in that regard.
- **Rule 2**: If the sulcus probes more than 1.5 mm, place the margin half the depth of the sulcus below the tissue crest. This places the margin far enough below tissue, so that it will still be covered if the patient is at higher risk of recession.
- **Rule 3**: If a sulcus greater than 2 mm is found, especially on the facial aspect of the tooth, evaluate to see if a gingivectomy could be performed to lengthen the teeth and create a 1.5-mm sulcus. Then the patient can be treated using Rule 1.

The rationale for Rule 3 is that deep margin placement is more difficult and the stability of the free gingival margin is less predictable when a deep sulcus exists. Reducing the sulcus depth creates

a more predictable situation in which to place an intracrevicular margin. The clinician cannot be sure that the tissue will remain at the corrected level, however, because some gingival rebound can occur after gingivectomy. However, sulcular depth reduction ensures that the restorative margins will not be exposed and visible in the patient's mouth (Figures 66-10 to 66-14).

Clinical Procedures in Margin Placement

The placement of supragingival or equigingival margins is simple because it requires no tissue manipulation. With regard to overall tooth preparation, the amount reduced incisally or occlusally, facially, lingually, and interproximally is dictated by the choice of restorative materials. Before extending subgingivally, the preparation should be completed to the free gingival margin facially and interproximally. This allows the margin of the tooth preparation to be used as a reference for subgingival extension once the tissue is retracted (Figure 66-15).

Tissue Retraction

Once the supragingival portion of the preparation is completed, it is necessary to extend below the tissue.[6,24] The preparation margin must now be extended to the appropriate depth in the sulcus,

Figure 66-12 Two options were available to manage treatment appropriately: (1) place the original margins to half the depth of the sulcus, in which case the recession that occurred would not have exposed them, or (2) perform a gingivectomy, creating a 1-mm to 1.5-mm sulcus. The second option was chosen when the restorations were redone. The margins were then placed 0.5 mm below the tissue after the gingivectomy (see Figures 66-11 and 66-13).

Figure 66-10 A 78-year-old woman presents with the maxillary anterior restorations placed 6 months earlier. She is unhappy with the exposed margins and notes that the margins were covered the day the restorations were placed (see Figures 66-11 to 66-14).

Figure 66-13 At 6 weeks after the gingivectomy and preparation of the teeth. Note the tissue level and that the tissue is rebounding coronally over the margins. This is a common finding when a pure gingivectomy is done.

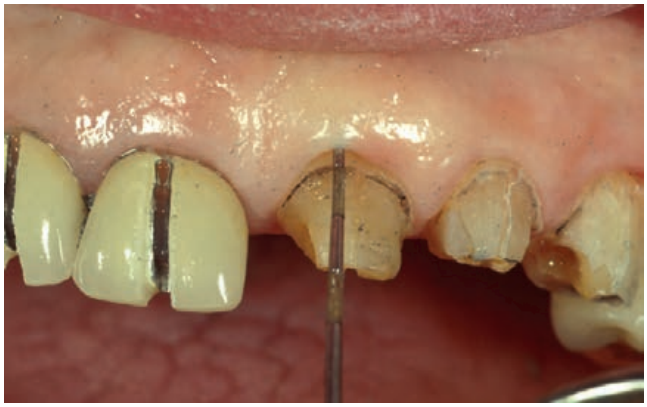

Figure 66-11 Depth from the attachment to the level of the preparation margin is greater than 3 mm. This patient in Figure 66-10 had an altered eruption pattern and a sulcus depth of more than 3 mm when these restorations were placed.

Figure 66-14 Four-year recall photograph after placement of the final restorations for patient in Figure 66-10. Note the tissue level has been maintained, with a sulcus depth of 2 mm on the facial surface.

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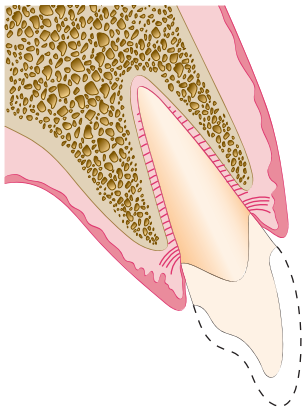

Figure 66-15 To provide a reference position for margin placement after tissue retraction, the margin of the tooth preparation is initially established level with the free gingival margin.

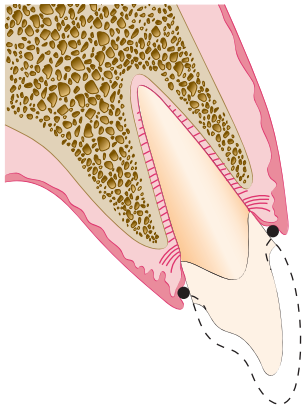

Figure 66-16 Second step in margin placement is to place a single layer of deflection cord below the previously prepared margin to the desired final margin level. Here, a single cord has been placed 0.5 mm below the previously prepared margin.

applying the guidelines presented previously. In this process the tissue must be protected from abrasion, which will cause hemorrhage and can adversely affect the stability of the tissue level around the tooth. Access to the margin is also required for the final impression, with a clean, fluid-controlled environment. Tissue management is achieved with gingival retraction cords using the appropriate size to achieve the displacement required. Thin, fragile gingival tissues and shallow sulcus situations usually dictate that smaller diameter cords be chosen to achieve the desired tissue displacement.

For a Rule 1 margin (sulcus depth 1.5 mm or less), the cord should be placed so that the top of the cord is located in the sulcus at the level in which the final margin is to be established, which will be 0.5 mm below the previously prepared margin (Figure 66-16). On the interproximal aspects of the tooth, the cord is usually 1.0 to 1.5 mm below the tissue height because the interproximal sulcus is often 2.5 to 3.0 mm in depth. With this initial cord in place, the preparation is extended to the top of the cord, with the bur angled to the tooth so that it does not abrade the tissue (Figure 66-17). This process protects the tissue, creates the correct axial reduction, and establishes the margin at the desired subgingival level. To create space and allow access for a final impression, it is now necessary to pack a second retraction cord. The second cord is pushed so that it displaces the first cord apically

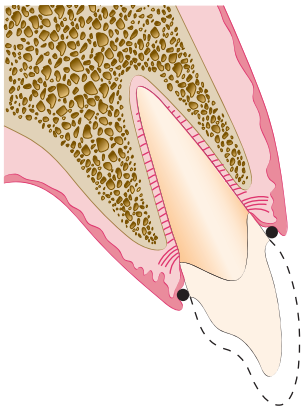

Figure 66-17 Margin of the preparation is now extended apically to the top of the retraction cord; this represents the correct placement of the margin below the previously nonreflected, free gingival margin.

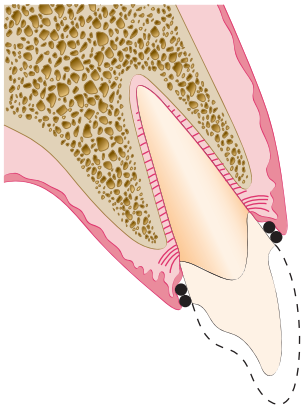

Figure 66-18 To provide space for impression material, a second impression cord is now placed on top of the first deflection cord. This impression cord is placed so that it is between the margin of the preparation and the gingiva to create adequate space for impression material after removal of the cord.

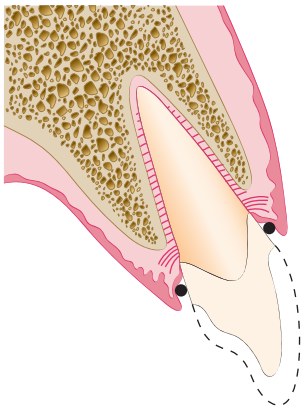

Figure 66-19 Ideal situation after removal of impression cord. The deflection cord is still in place maintaining the open sulcus but has been displaced apically another 0.5 mm by the placement of the impression cord, exposing tooth structure apical to the margin so that it can be captured in the impression.

and sits between the margin and the tissue (Figure 66-18). For the final impression, only the top cord is removed, leaving the margins visible and accessible to be recorded with the impression material (Figure 66-19). The initial cord remains in place in the sulcus until the provisional restoration is completed.

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As an alternative to additional retraction cords, electrosurgery can be used to remove any overlying tissue in the retraction process. A fine-wire electrode tip is held parallel to the tooth and against the margin in the sulcus and moved through the overhanging tissue, opening up the margin and the retraction cord to visual access (Figures 66-20 to 66-23). The electrosurgery tip sits on top of the retraction cord in place in the sulcus. This controls the vertical position of the tip and results in the removal of the least tissue needed for access.

For Rule 2 situations in which the sulcus is deeper, two larger-diameter cords are used to deflect the tissue before extending the margin apically (Figures 66-24 to 66-26). The top of the second cord is placed to identify the final margin location at the correct distance below the previously prepared margin, which was at the gingival tissue crest level. The margin is lowered to the top of the second cord (Figure 66-27), then a third cord is placed in preparation for the impression (Figures 66-28 and 66-29). In the patient with a deep sulcus in which the margin may be 1.5 to 2.0 mm below the tissue crest, electrosurgery is often required to remove

Figure 66-22 Using electrosurgery, the fine-wire electrode tip is held parallel to the tooth preparation and rests on the cord as the tip is moved around the tooth.

Figure 66-20 Deflection cord and impression cord are in place. The soft tissue is falling over the margins of the preparation. In this situation, if the impression cord were removed, the impression would not capture the margins in the areas in which the tissue is overhanging.

Figure 66-23 After removal of the impression cord, an adequate space is created for the impression material, with no soft tissue overhanging the margins to trap or tear the impression material. Note the first cord, or deflection cord, is still in place.

Figure 66-21 Overhanging tissue has been removed and space created for the impression material with electrosurgery. Note that the deflection cord and the impression cord are still in place. The impression cord is now visible completely around the tooth, allowing easy access for the impression material to the margin after removal of the impression cord.

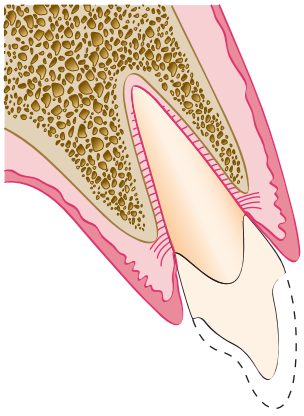

Figure 66-24 First step in margin placement for the patient with altered eruption or a deep sulcus is to prepare to the existing free gingival margin, as in the "Rule 1" patient (see text).

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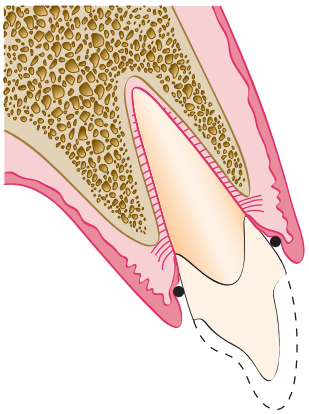

Figure 66-25 Second step for the patient with altered eruption is to place the deflection cord. Note that the placement of a single deflection cord does not provide adequate deflection of the tissue to allow the margin to be carried below tissue without abrading the gingiva with the bur.

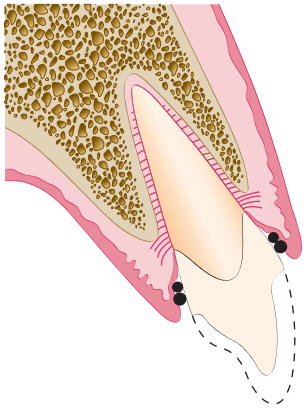

Figure 66-26 Third step for the patient with altered eruption and a deep sulcus is to place a second, larger-diameter deflection cord on top of the first deflection cord. Combined, these two cords allow adequate deflection to open up the sulcus so that the margin can be carried below tissue without abrading the gingiva.

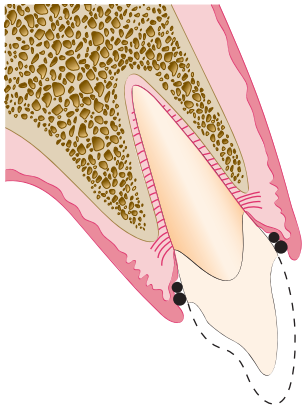

Figure 66-27 Preparation is now extended to the top of the second deflection cord, finalizing margin location.

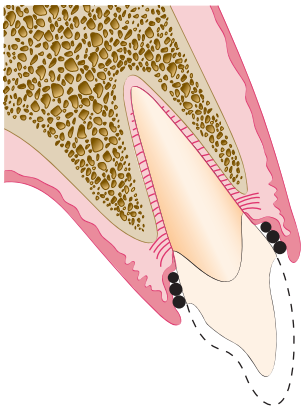

Figure 66-28 After extension of the margin to the top of the deflection cord, a third layer of cord is applied that will act as the impression cord. This impression cord should be placed so that it fits between the free gingival margin and the margin of the preparation. Its placement will also apically displace the two previously positioned deflection cords.

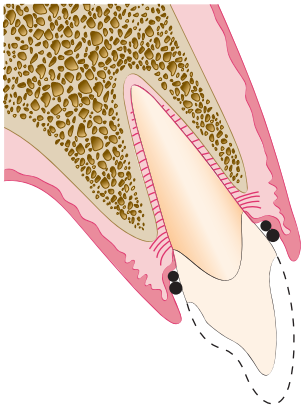

Figure 66-29 Removal of the impression cord creates an adequate space for the impression material to capture the margin and 0.5 mm of tooth structure below the margin in which the impression cord had displaced the first two cords.

overhanging tissue. To avoid altering the gingival tissue height, it is important to hold the electrosurgery tip parallel to the preparation (Figure 66-30).

Provisional Restorations

Three critical areas must be effectively managed to produce a favorable biologic response to provisional restorations.[3,75] The marginal fit, crown contour, and surface finish of the interim restorations must be appropriate to maintain the health and position of the gingival tissues during the interval until the final restorations are delivered. Provisional restorations that are poorly adapted at the margins, that are overcontoured or undercontoured, and that have rough or porous surfaces can cause inflammation, overgrowth, or recession of gingival tissues. The outcome can be unpredictable, and unfavorable changes in the tissue architecture can compromise the success of the final restoration.

Marginal Fit

Marginal fit has clearly been implicated in producing an inflammatory response in the periodontium. It has been shown that the level of gingival inflammation can increase corresponding with the level of marginal opening.[16] Margins that are significantly open (several tenths of a millimeter) are capable of harboring large

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Clinicians need to combine their pursuit of technical excellence of restorative preparations and crowns with a biologic sense of reducing the deleterious effects of restorative dentistry on the periodontium. Restorative margins on both teeth and implants provide an enhancement of the formation of the bacterial biofilm that causes periodontal disease and so the further away the crown margin is from supporting bone, the better expectation there is for periodontal health. Tooth restorative margins should be placed at least 2 mm distance from the crest of the alveolar bone process, and patients need to be instructed in the best approaches to oral hygiene.

To maintain periodontal health, overcontouring of restorations should be avoided. Undercontoured dental restorations rarely cause periodontal complications. A distance of 5 mm or greater between the contact point and the bone margin usually results in an unesthetic black triangle. Occlusal schemes need to be harmonized, and the use of a repeatable condylar position means that a stable centric relation is important, with avoidance of posterior guidance for protrusion. Lateral excursions should be smooth and free of balancing interferences.

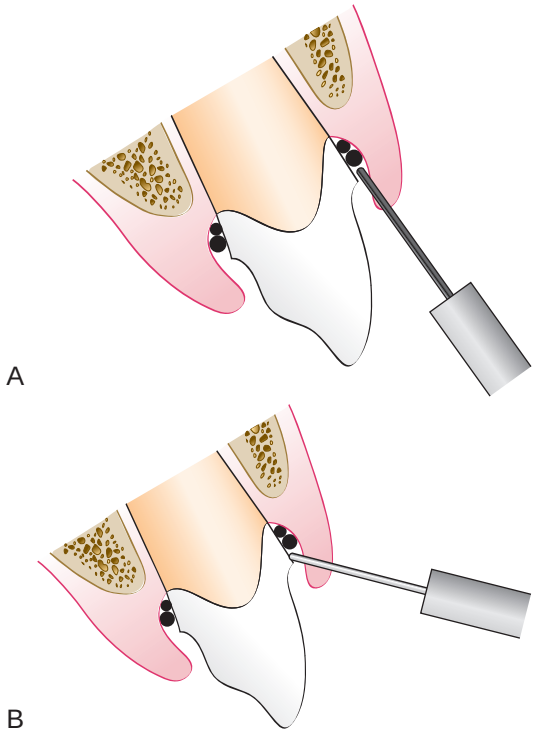

Figure 66-30 If it is necessary to use electrosurgery, either in the normal or altered-eruption patient, the correct inclination of the electrosurgery tip is important. **A,** Electrosurgery tip being held parallel to the preparation and resting on the previously placed retraction cord. This removes a minimal amount of tissue, and the presence of the retraction cord protects the attachment from the electrosurgery. **B,** Incorrect inclination of electrosurgery tip. The tip is leaning away from the preparation. This inclination results in excess tissue removal.

numbers of bacteria and may be responsible for the inflammatory response seen. However, the quality of marginal finish and the margin location relative to the attachment are much more critical to the periodontium than the difference between a 20-µm fit and a 100-µm fit.[43,47,60]

Crown Contour

Restoration contour has been described as extremely important to the maintenance of periodontal health.[27,76] Ideal contour provides access for hygiene, has the fullness to create the desired gingival form, and has a pleasing visual tooth contour in esthetic areas. Evidence from human and animal studies clearly demonstrates a relationship between overcontouring and gingival inflammation, whereas undercontouring produces no adverse periodontal effect.[49,52] The most frequent cause of overcontoured restorations is inadequate tooth preparation by the dentist, which forces the technician to produce a bulky restoration to provide room for the restorative material. In areas of the mouth in which esthetic considerations are not critical, a flatter contour is always acceptable.

Subgingival Debris

Leaving debris below the tissue during restorative procedures can create an adverse periodontal response. The cause can be retraction cord, impression material, provisional material, or either temporary or permanent cement.[56] The diagnosis of debris as the cause of gingival inflammation can be confirmed by examining the sulcus surrounding the restoration with an explorer, removing any foreign bodies, and then monitoring the tissue response. It may be necessary to provide tissue anesthesia for patient comfort during the procedure.

Hypersensitivity to Dental Materials

Inflammatory gingival responses have been reported related to the use of nonprecious alloys in dental restorations.[53] Typically, the responses have occurred to alloys containing nickel, although the frequency of these occurrences is controversial.[51] Hypersensitivity responses to precious alloys are extremely rare, and these alloys provide an easy solution to the problems encountered with the nonprecious alloys. More importantly, tissues respond more to the differences in surface roughness of the material rather than to the composition of the material.[1,67] The rougher the surface of the restoration subgingivally, the greater are the plaque accumulation and gingival inflammation. In clinical research, porcelain, highly polished gold, and highly polished resin all show similar plaque accumulation. Regardless of the restorative material selected, a smooth surface is essential on all materials subgingivally.

For more information on esthetic management of the gingival tissues, occlusal considerations in restorative therapy, and special restorative considerations, please visit the companion Expert CONSULT website at www.expertconsult.com.

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 67

Multidisciplinary Approach to Dental and Periodontal Problems

Dennis Tarnow and Mitchell Bloom

CHAPTER OUTLINE

EDUCATIONAL TRENDS TOWARD MULTIDISCIPLINARY SPECIALIST EDUCATION IN IMPLANT TREATMENT

THE FUTURE

Refer to Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures may be out of numeric order in this printed chapter.

Traditionally, periodontal treatment has been delivered using an interdisciplinary model of therapy, with general dentists and specialists providing their respective aspects of care to the same patient per a comprehensive plan of therapy (Figures 67-1 to 67-3, Figures 67-4 to 67-19 online, and Figures 67-20 to 67-22). This system has worked well because the patient benefits from the best mix of talent from a "team" of dentists. For periodontal aspects of such care to be most effective, it is critical that the primary provider, often the general practitioner, have a thorough understanding of the signs, symptoms, risk factors, and pathophysiology of disease processes along with their related local and systemic risk factors. Additionally, they must also possess a thorough understanding of the available treatment options along with their related indications, contraindications, benefits, and liabilities to effectively formulate a proper treatment plan. The dentist then decides whether he or she has the requisite knowledge, expertise, and experience to meet the patient's needs or whether to refer the patient to a specialist for care at a more advanced level. Conversely, when a single provider (general dentist or specialist) delivers all aspects of the patient's care, rather than a team of dentists, it is best described as *multidisciplinary treatment* as opposed to the term *interdisciplinary treatment.*

Many of the early innovators in the field of implant dentistry were general practitioners. Subsequently, their early accomplishments were built on by the predictable outcomes achieved using the surgical and prosthetic tenets put forth by Dr. P.I. Brånemark. His namesake implant design allowed the clinician to achieve predictable treatment outcomes through rigid case selection, strict surgical protocols, specialized armamentarium, and a narrow range of treatment options. The initial offering of training in the Brånemark method was limited to specialist prosthodontists and oral surgeons. The former group being limited to providing only the restorative aspect of care and the latter group the surgical phase of therapy. However, as implant dentistry continued to evolve, periodontists become increasingly more active in the field, ultimately sharing the same role and stature as that of their oral surgeon colleagues in this arena. The same was true for general practitioners with respect to their prosthodontist colleagues as it relates to the delivery of implant restorative care.

The range of indications for the use of dental implants grew beyond the mandibular full arch case type to include partial edentulism, single teeth, and even orthodontic and maxillofacial applications. Regenerative techniques were developed to address hard and soft tissue deficiencies for many patients that were previously deemed unsuitable candidates for dental implant therapy. Autogenous intraoral block grafting, guided bone regeneration, maxillary sinus grafting, transposition of the inferior alveolar nerve, ridge splitting, and distraction osteogenesis are just a few and have emerged as solutions to increase access for patients to implant therapy in less than optimal sites.

Early implant designs and materials were subject to limitations and even prone to problems. Those with machined surfaces suffered from a significantly higher failure rate in sites with poor quality bone, whereas those with rough surfaces, coupled with other design flaws, were prone to late failures resulting from inflammatory periimplant disease or prosthetic complications. With all of these variables in play and emerging so rapidly during the formative years, implant dentistry was relegated largely to the specialty care arena.

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Through innovative implant designs, advances in material science, opportunities for simplified surgical techniques, and systematic treatment strategies confirmed via studies that examined success and risk factors, predictable outcomes have become readily achievable. The widespread emergence and acceptance of implant dentistry in its contemporary iteration leads us to where we are today and where we are poised to go.

Figure 67-1 Complex interdisciplinary implant care scenario. Extraoral preoperative condition. Esthetic compromise is evident in this challenging treatment scenario in which there is significant gingival display.

It is suggested that in addition to learning all of the standard periodontal procedures of the past, the contemporary periodontist should also be able to *restore* simple implant cases such as those located outside of the esthetic zone (Figures 67-23, 67-25, 67-30, and 67-32; Figures 67-24, 67-26 to 67-29, and 67-31 online). It will continue to be the case that the periodontist will be trained to manage hard and soft tissues and perform all of the latest periodontal–plastic surgical procedures for preserving and reconstructing pleasing gingival architecture in the esthetic zone to the highest level of sophistication and complexity. However, as the definition of what is deemed a successful outcome continues to evolve and the bar for the definition is raised, the surgeon must remain acutely aware of the restorative aspects of care and abreast of related advances. In other words, it is not enough for the future periodontist to limit care to the treatment of periodontal disease alone.

Surgical specialists (i.e., periodontists) who are trained according to a multidisciplinary approach will provide even greater benefit to their patients from this evolved philosophy. For the implant surgeon to provide optimal outcomes in implant dentistry, even if their role is limited solely to the surgical aspect of care, they must have a thorough understanding of the realities and intricacies related to the fabrication and delivery of the planned prosthesis. Without such insight, achieving this on a predictable basis will

Figure 67-2 Intraoral preoperative condition. A concave soft tissue profile that contributes to a dark shadow and esthetic compromise is evident bilaterally *(arrows)*.

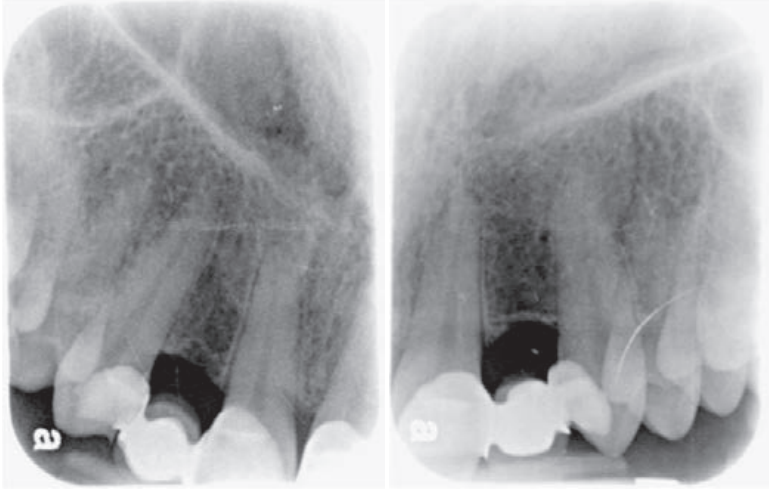

Figure 67-3 Preoperative radiographs. The roots of adjacent teeth do not converge to interfere with proper orientation of the positions of the planned dental implants. However, there is limited space between the adjacent tooth roots.

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Figure 67-20 The final crowns are seen here on the date of insertion. The crown contours dictate the gingival contours.

Figure 67-21 Note the customized emergence profile of the prosthetic components on the dental implants.

Figure 67-22 The final esthetic outcome from both an intraoral and extraoral perspective.

continue to remain elusive. This "restorative" knowledge and experience will help them to place implants in optimal orientation in all three spatial dimensions and to avoid such common errors as excessive implant angulation through greater understanding of the resultant restorative challenges. Another benefit is that the prosthetically well-versed surgical specialist will be positioned to educate and guide their restorative colleagues with whom they work through the nonsurgical aspects of care. This will enhance the relationship between restorative and surgical colleagues by becoming a source of knowledge with respect to the restorative phase of implant therapy and ultimately raise the level of care available and provided to patients.

EDUCATIONAL TRENDS TOWARD MULTIDISCIPLINARY SPECIALIST EDUCATION IN IMPLANT TREATMENT

For the multidisciplinary model of practice to be able to deliver care at the level presently available using an interdisciplinary approach, the provider (general practitioner or specialist) would need to have

Figure 67-23 Multidisciplinary simple implant care scenario: favorable soft tissue parameters combined with a low smile line. Preoperative view of the maxillary right first premolar. With the exception of a small degree of gingival recession, all other aspects of the surrounding periodontium are intact.

Figure 67-30 The healed periimplant sulcus demonstrates the three-dimensionally generated result achieved using a provisional crown for soft tissue sculpting. Note the re-creation of the interdental papilla, the result of a favorable relationship between the interproximal bone height on the adjacent teeth and reestablishment of contact areas between the natural teeth and the provisional restoration.

Figure 67-25 Healed ridge 3 months after tooth extraction. Note the wide zone of keratinized tissue present along with favorable maintenance of the height of the adjacent interdental papillae.

Figure 67-32 Final implant supported restoration in place.

been more comprehensively trained with a broader scope and depth of expertise than typical contemporary norms deliver.

The surgical aspect of implant dentistry has risen to center stage within many contemporary postgraduate specialty and continuing education programs. Continuing education programs span the gamut, with some being limited to didactic teaching with laboratory simulation and others take the form of clinical fellowship-style programs that encompass a clinical patient care component spanning one or more years in duration. As such, some general practitioners and prosthodontists who have sought out advanced postgraduate training might add some additional aspects of surgical care (commensurate with the scope and level of their respective training) to the range of services they personally provide. Conversely, members of the surgical specialties (e.g., periodontists and oral surgeons) would have received training to a level that would enable them to recommend, guide, and if necessary, provide a range of restorative treatments. Indeed, rigidly defined accreditation standards for some postgraduate specialties have already been revised and reflect this trend toward encouraging multidisciplinary training. Traditionally, nonsurgical restorative training programs have included basic implant surgical training into their curricula. Advanced education specialty programs in prosthodontics, among others, now include an increased allocation of time in their didactic and clinical curricula in the area of diagnosis, as well as training to the level of competency with respect to simple single-tooth implant scenarios in healed ridges of favorable dimension and in sites outside the esthetic zone.

In the United States, postgraduate periodontology training programs have been driven by the Accreditation Standards for Advanced Specialty Education Programs in Periodontics, which is regulated by the American Dental Association's Council on Dental Education (CODA). These requirements are based in part on input from the American Academy of Periodontology, whose major emphasis is on ensuring competency as a periodontal specialist. Multidisciplinary training and education represents a new era of training for periodontists that is beginning to emerge.

THE FUTURE

In the future, it will not be the accepted norm for all surgery to be done by a periodontist or oral surgeon nor will it likely be for all restorative work to be done by a general dentist or prosthodontist. Instead, simple cases that require surgery and restoration will probably be performed entirely by either a well-trained general dentist or a specialist. In fact, many periodontists have already begun working with their restorative colleagues by making the final impression or index of the implant at the time of surgery and forwarding it to them. In such scenarios, the restorative dentist may

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SCIENCE TRANSFER

The trend in the application of dental implant therapy is for simple cases to be carried out by well-trained general dentists, as well as by specialists. Specialists in the surgical approaches are also able to complete straight-forward prosthodontic cases, and some prosthodontic specialists may surgically place implants in noncomplex cases.

The interdisciplinary approach involving surgical specialists, prosthodontists, and general dentists is best applied to complex cases. Patients requiring advanced bone and/or soft tissue surgeries to optimize the implant site should be treated by surgical specialists. Patients with special occlusal or esthetic needs are best managed with a team approach. Those patients with systemic disease complexity are also best managed with a team lead by a specialist.

now only need to insert the final restoration when it comes back from the laboratory, thus expediting treatment and enhancing the experience for both the patient and doctor. As with most trends over time, it is conceivable that simple implant cases might be treated in a multidisciplinary fashion, whereas an interdisciplinary approach is more likely to be associated with advanced treatment scenarios.

The periodontists of the future will be multidisciplinary in the way they provide patient care. They will continue to provide all of the specialty services that "classically" trained periodontists have done for decades, but they will also be well suited to better support their restorative colleagues.

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PART 7

Oral Implantology

Perry R. Klokkevold

The routine use of dental implants to replace missing and compromised teeth has dramatically enhanced the practice and philosophy of dental practice. This is particularly true for the specialty of periodontics. Dental implants offer predictable, long-lasting tooth replacement outcomes for patients while preserving the natural tooth structure of adjacent teeth. As we gain experience, develop new techniques, and apply evolving treatment approaches, it is important to understand and appreciate the fundamentals of implant therapy. All health care professionals, regardless of specialty, are compelled to become knowledgeable in all aspects of dental implant therapy and to continue their education as new information and evidence becomes available.

The chapters in Part 7 comprehensively cover the several important implant-related topics including biology, examination, diagnosis, surgical techniques, biomechanics, treatment planning, prosthetic considerations, and complications. A new chapter presents esthetic management of difficult cases and new material has been added to expand the foundation of knowledge in all areas.

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CHAPTER 68

Periimplant Anatomy, Biology, and Function

Joseph Fiorellini, Daniel W. K. Kao, Keisuke Wada, and Perry R. Klokkevold

CHAPTER OUTLINE

IMPLANT GEOMETRY (MACRODESIGN) (online only)
- Endosseous Implants
- Root Form Implants
- Transmandibular Implants
- Subperiosteal Implants

IMPLANT SURFACE CHARACTERISTICS (MICRODESIGN) (online only)
- Additive Processes
- Subtractive Processes
- Implant Surface Chemical Composition
- Implant Surface Free Energy and Micro-Roughness

HARD TISSUE INTERFACE
- Initial Bone Healing
- Bone Remodeling and Function

SOFT TISSUE INTERFACE
- Epithelium
- Connective Tissue
- Keratinized Tissue
- Vascular Supply and Inflammation

CLINICAL COMPARISON OF TEETH AND IMPLANTS

CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Attempts to replace lost dentition by means of implanted materials can be traced back to the ancient Egyptians, who hammered shaped seashells directly into the jaws for the purpose of replacing teeth.[41] Over the last few centuries, a variety of materials have been implanted into jaws in an attempt replace missing teeth. The success of these early implants was extremely poor primarily because they never achieved a stable state of integration with the supporting tissues.[85] The typical outcome, regardless of material or design, was healing with a soft tissue layer interposed between the implant and bone (i.e., fibrous encapsulation). Consequently, implants became mobile, infected, and painful, which led to failure.

The history of modern implant dentistry began with the introduction of titanium implants.[41] In the 1950s, Per-Ingvar Brånemark, a Swedish professor of anatomy, had a serendipitous finding while studying blood circulation in bone that became a historical breakthrough in medicine. He discovered an intimate bone-to-implant apposition with titanium that offered sufficient strength to cope with load transfer. He called the phenomenon "osseointegration" and developed an implant system with a specific protocol to predictably achieve it. The implants were used to anchor prosthetic replacement teeth in the edentulous jaw,[29] and the first patient was successfully treated in 1965.[28,70] Subsequent clinical studies proved that commercially pure (CP) titanium implants, placed with a strict protocol, including an unloaded healing period, could predictably achieve osseointegration and retain a full-arch prosthesis in function with long-term success (15 years).[9] Since that time, millions of patients worldwide have had missing teeth replaced with implants based on this original concept. Over the years, implant systems with variations in design (geometric and surface characteristics) and modified protocols have been developed and used with equal or better long-term success.

Today, implant designs, surgical placement techniques, healing times, and restorative protocols continue to evolve with the goal of improving outcomes. It is important for clinicians to know periimplant anatomy, to understand the biology, and to appreciate the functional capacity of osseointegrated implants. This chapter reviews implant geometry and surface characteristics, as well as the anatomical and biological relationship of periimplant tissues.

For more information on implant geometry and implant surface characteristics, please visit the companion Expert CONSULT website at www.expertconsult.com.

HARD TISSUE INTERFACE

The primary goal of implant installation is to achieve and maintain a stable bone-to-implant connection (i.e., osseointegration).[26,28] Histologically, osseointegration is defined as the direct structural and functional connection between ordered, living bone and the surface of a load-bearing implant without intervening soft tissues

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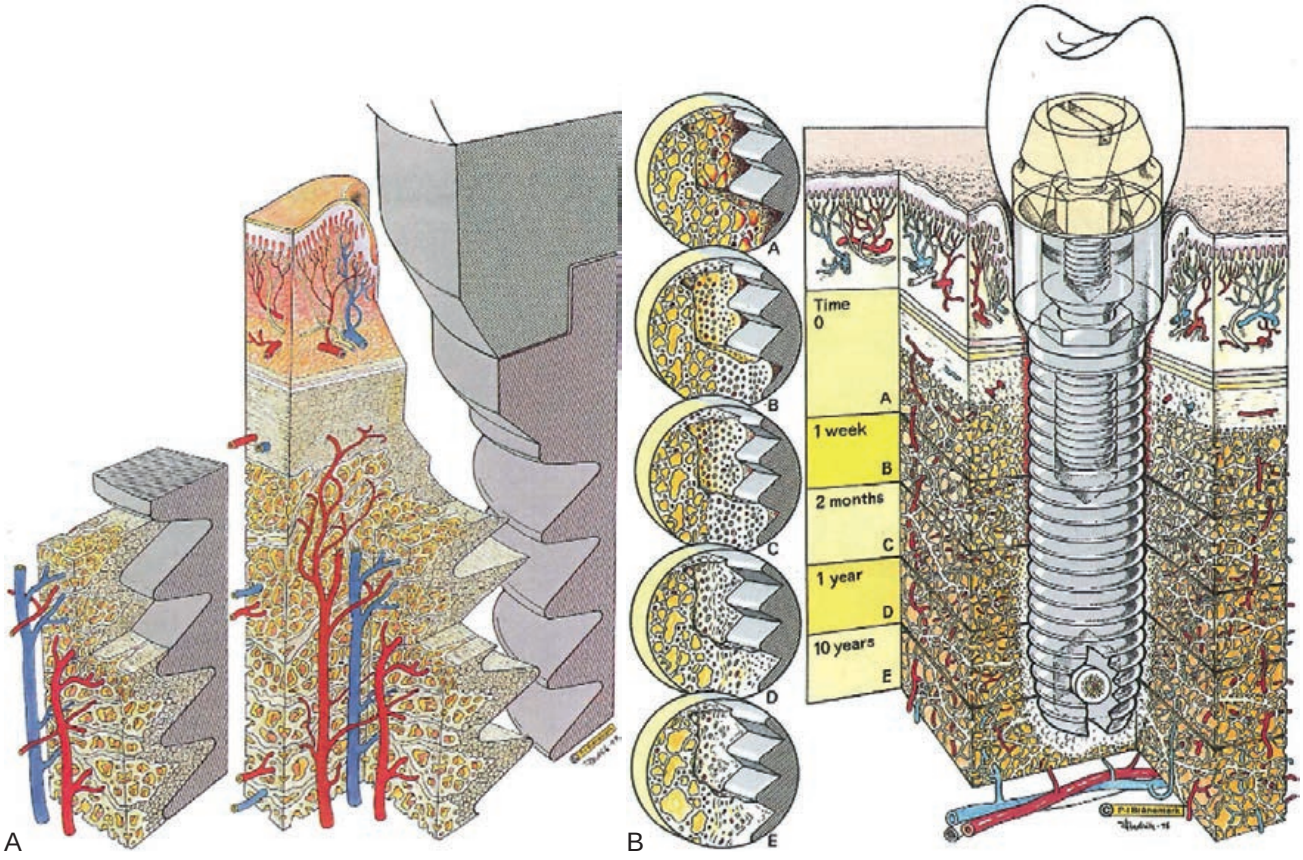

Figure 68-12 **A,** Three-dimensional diagram of the tissue and titanium interrelationship showing an overall view of the intact interfacial zone around the osseointegrated implant. **B,** Physiologic evolution of the biology of the interface over time.

(Figure 68-12).[27,29] Clinically, osseointegration is the asymptomatic rigid fixation of an alloplastic material (implant) in bone with the ability to withstand occlusal forces.[13,119] The hard tissue interface is a fundamental requirement for and an essential component of implant success.

Initial Bone Healing

The osseointegration process observed after implant insertion can be compared to bone fracture healing. Implant site osteotomy preparation (bone wounding) initiates a sequence of events, including an inflammatory reaction, bone resorption, release of growth factors, and attraction by chemotaxis of osteoprogenitor cells to the site. Differentiation of osteoprogenitor cells into osteoblasts leads to bone formation at the implant surface. Extracellular matrix proteins, such as osteocalcin, modulate apatite crystal growth.[116] Specific conditions, optimal for bone formation, must be maintained at the healing site to achieve osseointegration.

Immobility of the implant relative to the bone must be maintained for bone formation at the surface. A mild inflammatory response enhances the bone healing, but moderate inflammation or movement above a certain threshold is detrimental.[7] When micromovements at the interface exceed 150 µm, the movement will impair differentiation of osteoblasts and fibrous scar tissue will form between the bone and implant surface.[84] Therefore it is important to avoid excessive forces, such as occlusal loading, during the early healing period.

Bone tissue damage and debris created by the osteotomy site preparation must be cleared up by osteoclasts for normal bone healing. These multinuclear cells, originating from the blood, can resorb bone at a pace of 50 to 100 µm per day. There is a coupling between bone apposition and bone resorption (Figure 68-13). Preosteoblasts, derived from primary mesenchymal cells, depend on a favorable oxidation-reduction (redox) potential of the environment. Thus a proper vascular supply and oxygen tension are needed. If oxygen tension is poor, the primary stem cells may differentiate into fibroblasts, form scar tissue, and lead to implant failure (nonintegration).

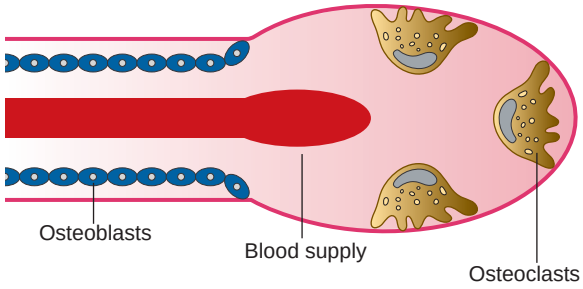

Figure 68-13 The basic multicellular unit is the basic remodeling process for bone renewal. Osteoclasts are imported by the vascular supply, and the resorption lacunae are soon filled by the lining osteoblasts.

If bone is overheated or crushed during preparation, it will become necrotic and may lead to nonmineralized (soft tissue) scar formation or be sequestered. The critical temperature for bone cells that should not be exceeded is 47°C at an exposure time of one minute.[7] Thus preparation of implant osteotomy sites require profuse irrigation (cooling) along with gentle, intermittent, moderate-speed drilling using sharp drills. Another complicating

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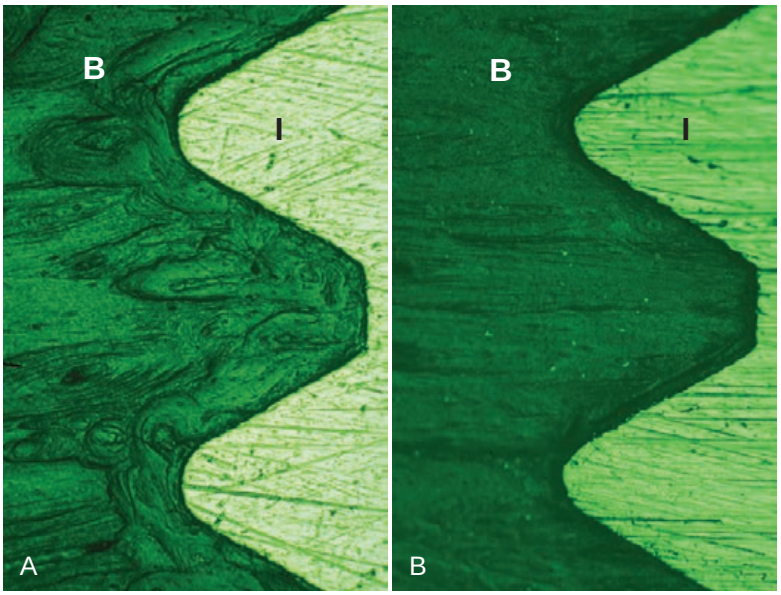

Figure 68-14 **A,** After initial healing, woven bone, as characterized by its irregular pattern, is laid down. **B,** After weeks or months, progressively a lamellar bone is laid down, with regular concentric lamellae. *B,* Bone; *I,* implant. *(Courtesy Professor T. Albrektsson, Gothenburg, Sweden.)*

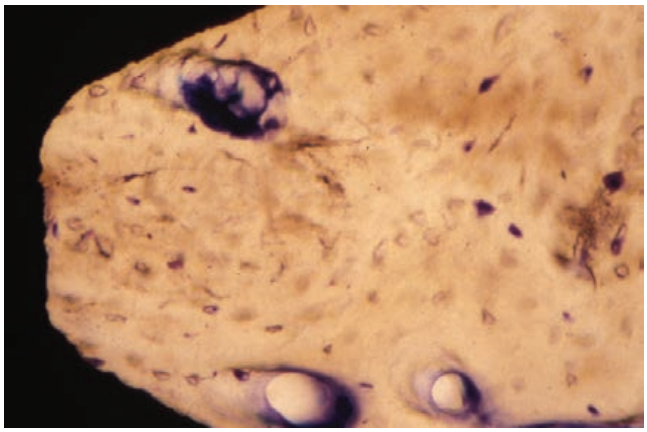

Figure 68-15 Once a steady state has been achieved at the bone-to-implant interface, an intimate contact can be observed, with some marrow spaces seen in between at the light microscopic level.

factor, well recognized from open wound fractures, is that microbial contamination jeopardizes the normal bone healing. Accordingly, strict aseptic techniques should be maintained.

New bone formation follows a specific sequence of events. Woven bone is quickly formed in the gap between the implant and the bone; it grows fast, up to 100 µm per day, and in all directions. Characterized by a random orientation of its collagen fibrils, high cellularity, and limited degree of mineralization, the biomechanical capacity of woven bone is poor (Figure 68-14, *A*). Thus any occlusal load should be well controlled or avoided in the early phase of healing. After several months, woven bone is progressively replaced by lamellar bone with organized, parallel layers of collagen fibrils and dense mineralization. Contrary to the fast-growing woven bone, lamellar bone formation occurs at a slow pace (only a few microns per day). Ultimately, after 18 months of healing, a steady state is reached where lamellar bone is continuously resorbed and replaced (Figure 68-14, *B*).[29] At the light microscopic level, an intimate bone-to-implant contact has been extensively reported (Figure 68-15).[90] Once the bone-to-implant interface has reached a steady state, it can maintain itself over decades, as ascertained by human histology from implants retrieved because of hardware fractures.[11]

Bone Remodeling and Function

Clinically, both primary and secondary stability of an implant are critical to success. Primary stability, achieved at the time of surgical placement, depends on the implant geometry (macrodesign), as well as the quality and quantity of bone available for implant anchorage at a specific site. Secondary stability, achieved over time with healing, depends on the implant surface (microdesign), as well as the quality and quantity of adjacent bone, which will determine the percentage of contacts between the implant and bone.[18,47,88,106] For example, areas such as the anterior mandible have dense cortical bone and provide rigid primary stabilization and good support throughout the healing process. Conversely, areas such as the posterior maxilla have thin cortical bone and large marrow spaces provide less primary stability. For this reason, the posterior maxilla has been associated with lower success rates compared to other sites with greater bone density and support.[17,58]

Once osseointegration is achieved, implants can resist and function under the forces of occlusion for many years. Longitudinal biomechanical assessments seem to indicate that during the first weeks after placement of one-stage implants, decreased rigidity is observed.[45] This may be indicative of bone resorption during the initial phase of healing. Subsequently, rigidity increases and continues to increase for years.[105] Thus, when a prosthesis is installed immediately (in 1 day) or early (in 1 to 2 weeks), care must be taken to control against overload. It is important to recognize that sites with limited primary stability or less bone-to-implant contact (e.g., posterior maxilla) will likely go through a period of even less bone support in the early stages of bone healing due to the initial phase of bone resorption.

SOFT TISSUE INTERFACE

Not surprisingly, for two decades, research and clinical interest focused on the bone-to-implant interface of osseointegrated implants and the overlying soft tissues were largely ignored. Except for a few descriptive sentences, the classic handbook by Brånemark et al[29] presented no data or information about the soft tissue

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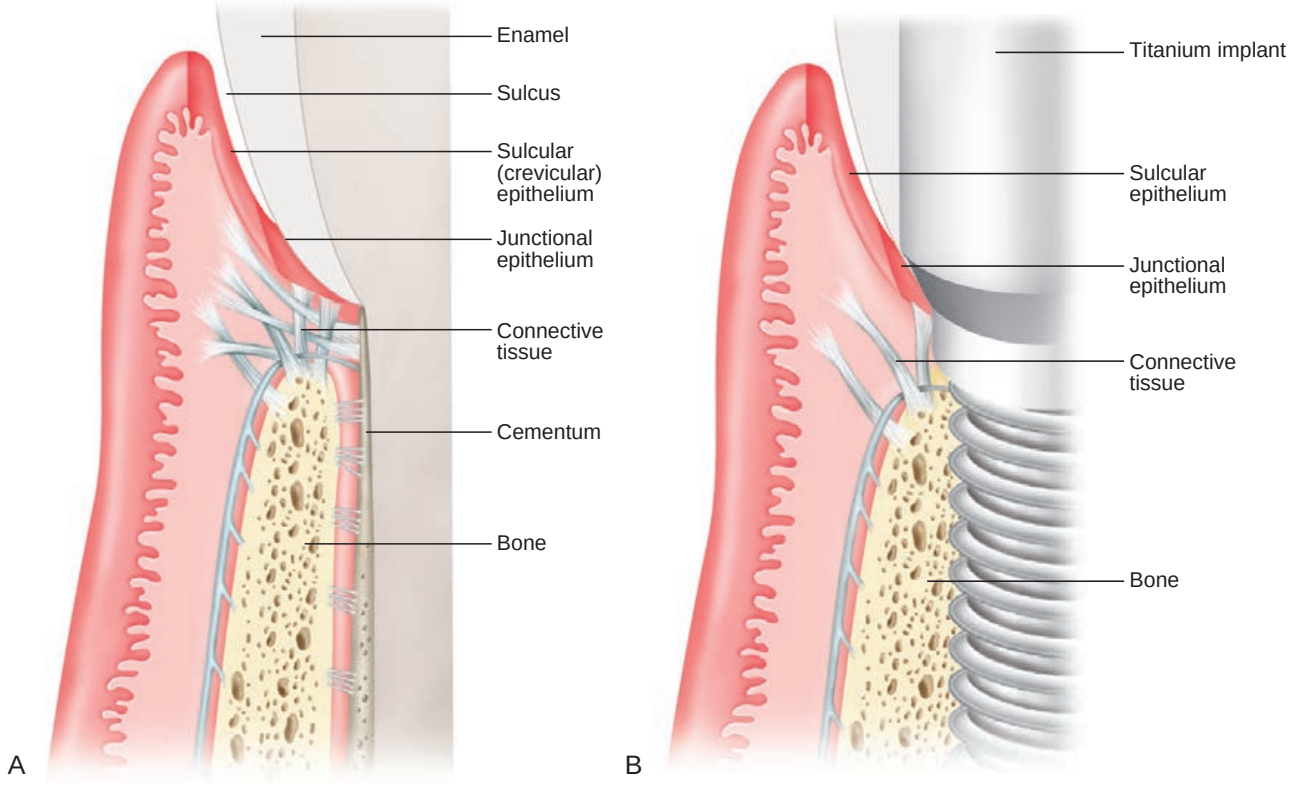

Figure 68-16 Schematic illustration of hard and soft tissue around a tooth and an implant. **A,** Hard and soft tissue anatomy around a natural tooth demonstrates bone support with a periodontal ligament, a connective tissue zone above the crest of bone with connective tissue fibers (Sharpey's) inserting into dentin, a long junctional epithelial attachment, a gingival sulcus lined with sulcular epithelium, and oral gingival epithelium (outer surface of gingiva). **B,** Hard and soft tissue anatomy around an implant demonstrates some similarities and some distinct differences. There is supporting bone in direct approximation to the implant surface without any intervening soft tissues (i.e., no periodontal ligament). A connective tissue zone is present above the level of bone with fibers running parallel to the implant surface and no inserting fibers. There is a long junctional epithelial attachment, a gingival/mucosal sulcus lined with sulcular epithelium and oral gingival/mucosal epithelium (outer surface of soft tissue). *(From Rose LF, Mealey BL:* Periodontics: medicine, surgery, and implants, *St. Louis, 2004, Mosby.)*

interface. This may be due in part to the fact that most patients were fully edentulous and the Brånemark system implants had turned (machined) surfaces, which are less likely to be associated with soft tissue inflammatory problems.[6] Today, there is greater interest in and appreciation for periimplant soft tissues and the soft tissue-to-implant interface as a function of esthetics and maintenance of a seal or barrier against microbial invasion.

Periimplant soft tissues are similar in appearance and structure to periodontal soft tissues (see Chapter 2). Clearly, both implants and teeth emerge through the soft tissues on the alveolar ridge. The soft tissues consist of connective tissue covered by epithelium. There is a gingival/mucosal sulcus, a long junctional epithelial attachment, and a zone of connective tissue above the supporting bone (Figure 68-16). Despite the apparent similarities in soft tissues around teeth and implants, the presence of a periodontal ligament around teeth and not around implants is an important, distinct difference. There are no inserting collagen fibers anywhere along the interface of osseointegrated implants. Bone is in direct contact with the implant surface without intervening soft tissues. Whereas natural teeth have a periodontal ligament with connective tissue fibers suspending them in the alveolar bone, osseointegrated implants do not.

Epithelium

As in the natural dentition, the oral epithelium around implants is continuous with a sulcular epithelium that lines inner surface of gingival sulcus; the apical part of the gingival sulcus is lined with long junctional epithelium.[69] Ultrastructural examination of the long junctional epithelial attachment adjacent to dental implants has demonstrated that epithelial cells attach with a basal lamina and hemidesmosomes[3,5,48,60,98] (Figure 68-17). Histologic studies indicate that these epithelial structures and the surrounding lamina propria cannot be distinguished from those structures around teeth.[32] In health, the sulcular epithelium has a thickness of about 0.5 mm.[87]

The apical edge of the epithelial attachment is about 1.5 to 2.0 mm above the bone margin.[82] In healthy periimplant tissues, progressive epithelial downgrowth does not occur, indicating that factors other than inserted collagen fiber bundles (i.e., Sharpey's fibers in natural dentition) prevent it.

Connective Tissue

Periimplant connective tissue morphology closely resembles that of the natural dentition except that it lacks a periodontal ligament, cementum, and inserting fibers (Figure 68-18). No significant differences were found at the biochemical level between the periimplant and the periodontal soft tissues.[33] Clinically, the thickness of the periimplant soft tissues varies from two to several millimeters (Figure 68-19). An animal study determined the total height of the periimplant "biologic width" to be approximately 3 to 4 mm, where about 2 mm is the epithelial attachment and about 1 to 2 mm is the supracrestal connective tissue zone.[19] Consistent with this

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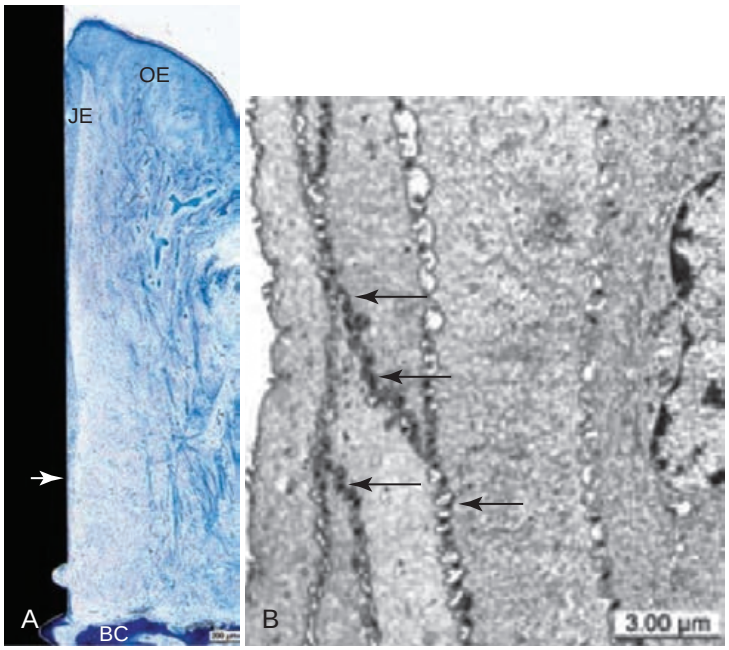

Figure 68-17 **A,** Overview of ground section showing peri-implant tissues covered with keratinizing oral epithelium *(OE).* Junctional epithelium *(JE)* is interposed between connective tissue and alveolar bone crest *(BC).* Apical end of junctional epithelium *(arrow).* Toluidine blue stain. (Bar = 200 µm.) **B,** Transmission electron microscopic view of sulcular epithelium showing tightly sealed intercellular spaces by numerous spot desmosomes *(arrows),* contributing to low permeability of this portion of periimplant mucosa. Bar = 3 µm.[1]

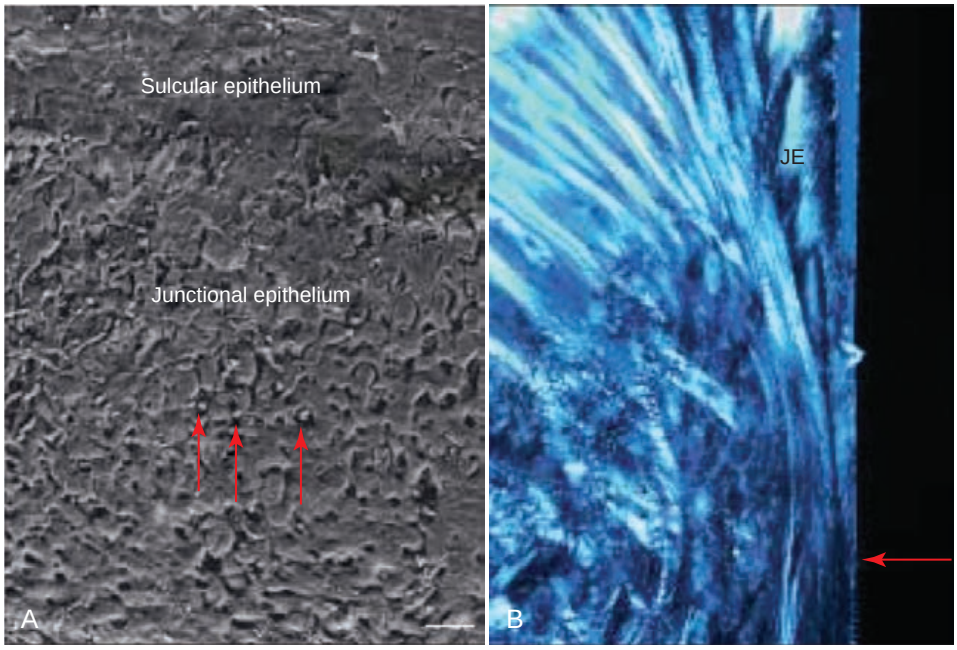

Figure 68-18 **A,** SEM of the junctional epithelium. Note the neutrophils located between the cells *(red arrows).* Bar = 40 µm. **B,** Higher magnification of Figure 68-17 with polarized light showing the apical extent *(red arrow)* of the junctional epithelium *(JE).* Note the dense collagen fibers running apicocoronal (i.e., parallel to the implant surface).

finding, a human histologic study determined the height of the periimplant "biologic width," consisting of an epithelial attachment and supracrestal connective tissue, to be about 4 to 4.5 mm[46] (Figure 68-20).

Between the epithelial attachment and the marginal bone is a zone of dense connective tissue. This zone of supracrestal connective tissue has an important function in the maintenance of a stable soft tissue–implant interface and as a seal or barrier to the "outside" oral environment. The orientation of connective tissue fibers adjacent to an implant differs from that of periodontal connective tissue fibers. In the absence of cementum and inserting connective tissue fibers (i.e., as in a natural tooth), most periimplant connective tissue fibers run in a direction more or less parallel to the implant surface. Even when the fiber bundles are oriented perpendicularly, which occurs more often in the gingiva than in the mucosa surrounding implants, the bundles are never embedded in the implant surface.

The fiber bundles can also have a "cuff-like" circular orientation.[20] The role of these fibers remains unknown, but it appears that their presence helps to create a soft tissue "seal" around the implant. The adaptation of the connective tissue to an implant surface may also be affected by the mobility of the soft tissue around the

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Figure 68-19 Clinical appearance of normal, healthy periimplant tissue with implant restoration removed. Soft tissue thickness varies from site-to-site, depending on quantity and quality of tissue, as well as the anatomy of the surrounding area (e.g., adjacent to natural teeth with healthy periodontal attachment versus adjacent to a space). Note that the intrasulcular tissue appears more erythematous as the result of the thin, nonkeratinized layer of epithelium overlying the connective tissue. *(Courtesy Dr. Stuart Froum, New York, NY.)*

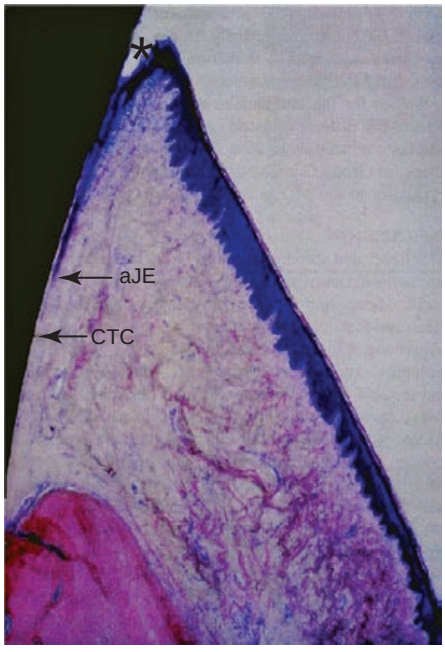

Figure 68-20 Buccolingual section (basic fuchsin stain; original magnification ×12.5; 1-part SLA implant, 3 months unloaded) showing the gingiva and the most coronal part of alveolar bone. Rete peg formation is only apparent in the area of the keratinized oral gingival epithelium. The oral sulcular epithelium exhibits no keratinization. In the area of the most coronal point of the junctional epithelium (cJE), the soft tissues are slightly torn away (artifact) because of non-decalcified histologic processing. The most apical point of the junctional epithelium is indicated *(aJE).* No rete peg formation is evident adjacent to the basal cell layer of the junctional epithelium (JE), all showing healthy and physiologic soft tissue structures. In addition, the area of connective tissue contact *(CTC)* adjacent to the machined titanium surface is marked. A slight round cell infiltrate in the connective tissue is indicating a mild inflammation. Note bone remodeling/new bone formation in the crestal bone region indicated by saturated, dark-red stained areas.

implant. The connective tissue in direct contact with the implant surface is characterized by an absence of blood vessels and an abundance of fibroblasts interposed between collagen fibers.[66] Several animal and human studies have shown that the alignments of connective fibers were circular and horizontal around the implants* (Figure 68-21).

This connective tissue interface has been examined by clinical attachment level measurements in patients. Probing attachment levels were consistently found coronal to the alveolar crest in patients with periimplant tissue health, indicating the presence of a zone of direct connective tissue contact to the implant surface. This means that the probing depth measurement performed with a periodontal probe may be about 1.5 mm higher above the bone level in healthy tissues. At inflamed sites, the probe may penetrate to the bone with probing depth measurement reflecting the total soft tissue thickness above bone. In cases with inflammatory periimplant tissue disease, increasing probing depth and reduced attachment levels have been reported.[4,39,68,79,110]

Keratinized Tissue

Questions emerged decades ago, as it did for the natural dentition, about the need for keratinized tissue to surround implants. Prospective and cross-sectional studies, evaluating screw-shaped implants with a machined surface, suggest that the presence or absence of keratinized gingiva is not a prerequisite for long-term stability.[93] However, it has been suggested that implants surrounded by mucosa only (i.e., nonkeratinized) are more susceptible to periimplant problems. An animal study observed that ligature-induced periimplantitis occurs more frequently when alveolar mucosa surrounds the implant as compared to when keratinized mucosa surrounds the implant.[109]

Keratinized mucosa tends to be more firmly anchored by collagen fibers to the underlying periosteum than nonkeratinized mucosa, which has more elastic fibers and tends to be moveable relative to the underlying bone. In clinical studies evaluating intraoral implants, with or without periimplant keratinized mucosa, no clinically significant difference in implant success was reported.[64,114] However, when there is a lack of keratinized tissue, patients tend to complain about pain and discomfort while performing oral hygiene procedures or other functions in the area. The symptoms are alleviated by increasing the amount of keratinized (firmly bound) tissue around the implant(s) via soft tissue grafting (see Figures 63-5, *A-F* and 63-5, *G-L*). Finally, although it may not be comparable to intraoral implants, mobility of soft tissues surrounding extraoral implants is associated with a higher incidence of implant failure.[10]

Vascular Supply and Inflammation

The vascular supply of the periimplant gingival or alveolar mucosa may be limited, as compared to periodontal gingiva, due to the lack of a periodontal ligament (Figure 68-22).[21] This is especially true in the tissue immediately adjacent to the implant surface. However, capillary loops in the connective tissue under the junctional and sulcular epithelium around implants appear to be anatomically similar to those found in the normal periodontium (Figure 68-23).[97]

Emerging knowledge indicates that the periimplant gingival or alveolar mucosa has the same morphology as the corresponding tissues around teeth. These soft tissues also react the same way to plaque accumulation. Studies investigating the histology (light microscopic and ultrastructural) of healthy and inflamed tissues

*References 2, 15, 34, 47, 50, 51, and 91.

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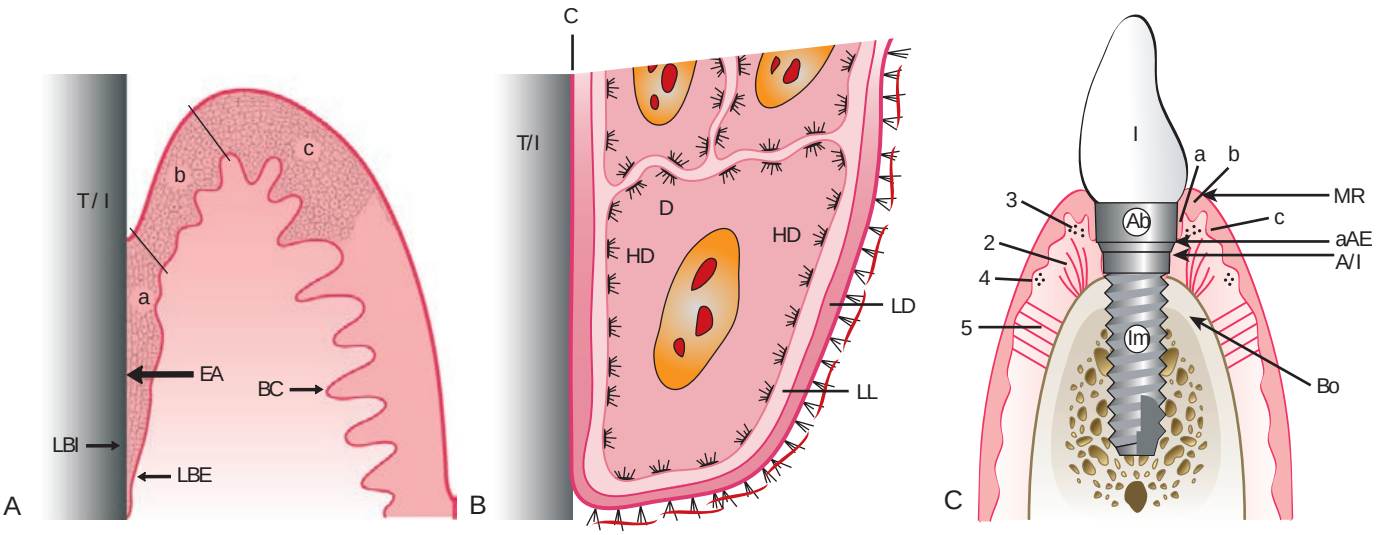

Figure 68-21 **A,** Histologic scheme of epithelial attachment *(EA)* (identical for tooth and implant). *T/I,* Titanium implant; *BC,* basal complex; *LBI,* lamina basalis interna; *LBE,* lamina basalis externa (only location where cell divisions occur); *a,* long junctional epithelial attachment zone; *b,* sulcular epithelial zone; *c,* oral epithelial zone. **B,** At electron microscopic level, basal complex at epithelial attachment (three most apical cells) and connection with stroma. *HD,* Hemidesmosomes; *D,* desmosome; *LL,* lamina lucida; *LD,* lamina densa; *C,* cuticle. **C,** Implant, abutment *(Ab)*, and crown within alveolar bone and soft tissues. *Im,* Endosseous part of implant; *MR,* margin of gingiva/alveolar mucosa; *Bo,* marginal bone level; *1,* implant crown; *2,* vertical alveolar-gingival connective tissue fibers; *3,* circular gingival connective tissue fibers; *4,* circular gingival connective tissue fibers; *5,* periosteal-gingival connective tissue fibers; *a,* junctional epithelium; *b,* sulcular epithelium; *c,* oral epithelium; *A/I,* abutment/implant junction; *aAE,* apical (point) of attached epithelium.

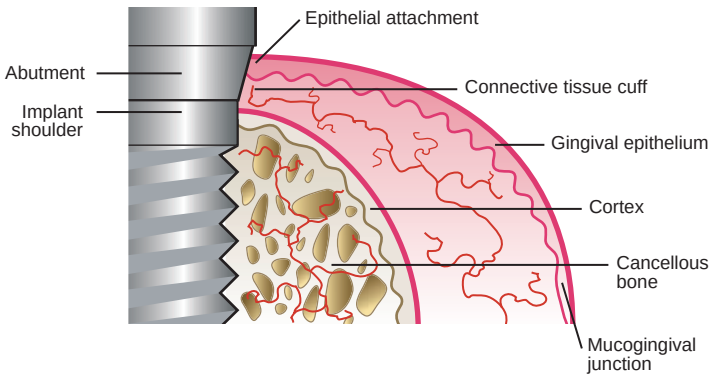

Figure 68-22 Schematic illustration of the blood supply in the connective tissue cuff surrounding the implant/abutment is scarcer than in the gingival complex around teeth because none originates from a periodontal ligament.

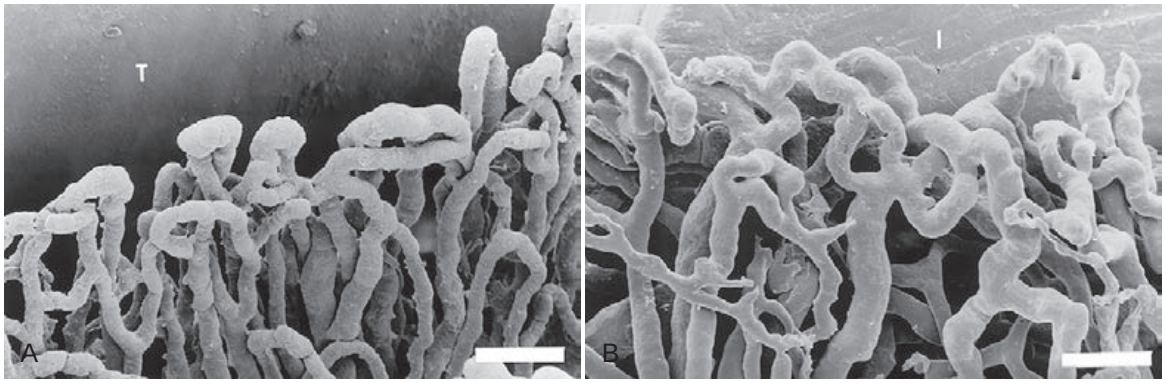

Figure 68-23 **A,** Microvascular topography surrounding an implant. **B,** Microvascular topography surrounding a tooth. Bar = 5 µm. *(Courtesy Drs. N. Selliseth and K. Selvig, Bergen, Norway.)*

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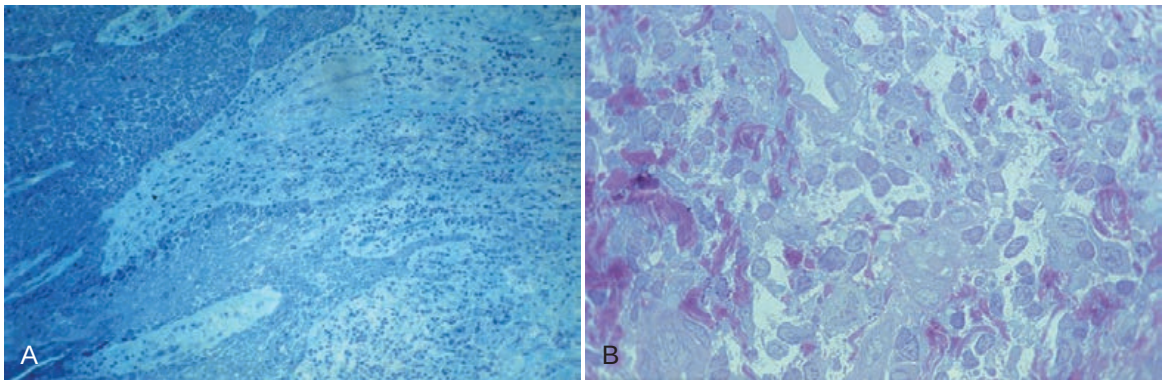

Figure 68-24 **A,** Histologic slide from healthy gingiva surrounding a well-functioning implant in human patient. No morphologic characteristics differentiate tissue around implant from that around teeth. **B,** When gingivitis occurs, a profuse migration of inflammatory cells through the pocket epithelium can be observed. *(Courtesy Professor Mariano Sanz, Madrid.)*

SCIENCE TRANSFER

Most current implants are screw-retained cylinders or tapers and are made of titanium, which results in a layer of titanium oxide that has the capacity to enable osseointegration. Roughening of the surfaces resulted in improved blood clot adhesion, osteoprogenitor cell differentiation, and early bone formation, as well as the mechanical advantage of increased surface area. Thus clinicians should utilize any of the variety of new surface modifications available on different implant brands. The use of surface liquids to enhance bone formation, such as fluoride and saline, may accelerate early bone formation.

The soft tissue surrounding implants is not as firmly attached as it is in teeth that have cementum. Probing depths around healthy implants may reach within 1 to 2 mm of the bone margin, but in inflamed tissues the probe will go to the bone level. Clinicians should use light and gentle procedures when measuring pocket depths in the periimplant environment to avoid damage to the fragile soft tissue adhesion to the implant surface. Implants do have healthier soft tissue around them when there is an adequate amount of keratinized gingiva, and in some cases, mucogingival surgery is needed to stabilize gingival health. Plaque control around implants is just as vital to long-term success as it is around teeth, so patients need close supervision of plaque levels during maintenance visits, which should be every 3 or 4 months.

surrounding implants in humans have indicated that the inflammatory response to plaque is similar to that observed in periodontal tissues.[87] Polymorphonuclear cells and mononuclear cells transmigrate normally through the periimplant sulcular epithelium (Figure 68-24).[87]

CLINICAL COMPARISON OF TEETH AND IMPLANTS

Although the soft tissue-to-implant (abutment) interface offers striking similarities with tissue surrounding the natural dentition, some differences should be considered. At the bone level, the lack of a periodontal ligament is the most striking difference. The following discussion elaborates on the clinical perspectives of these similarities and differences.

At the bone level the absence of the periodontal ligament surrounding an implant has important clinical consequences. This means that no resilient connection exists between implants and supporting bone. Implants cannot intrude or migrate to compensate for the presence of a premature occlusal contact (as teeth can). Implants and the rigidly attached implant restorations do not move. Thus any occlusal disharmony will have repercussions at either the restoration-to-implant connection, the bone-to-implant interface, or both.

Proprioception in the natural dentition comes from the periodontal ligament. The absence of a periodontal ligament around implants reduces tactile sensitivity[56] and reflex function.[23] This can become even more challenging when osseointegrated, implant-supported, fixed prostheses are present in both jaws.

The lack of a periodontal ligament and inability of implants to move contraindicates their use in growing individuals. Natural teeth continue to erupt and migrate during growth while implants do not. Implants placed in individuals prior to the completion of growth can lead to occlusal disharmonies with implants.[77] Likewise, it may be problematic to place one or more implants in a location adjacent to teeth that are very mobile from the loss of periodontal support because as the teeth move in response to or away from the occlusal forces, the implant(s) will bear the entire load.

Overload, because of improper superstructure design, parafunctional habits, or excessive occlusal load, may cause microstrains and microfractures in the bone, which will lead to bone loss and a fibrous inflammatory tissue at the implant interface.[107]

CONCLUSION

Appreciating the osseointegration process is facilitated by a good knowledge and understanding of bone healing. Many factors can

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interfere with the predictable establishment of a permanent rigid connection between an implant surface and the surrounding bone that is able to sustain occlusal loads. The bone-to-implant interface and its rigidity are a predominant biomechanical aspect of coping with the time and intensity of loading. The soft tissue-to-implant interface also plays an important role in the long-term maintenance of a stable marginal bone levels around implants. Clinicians must familiarize themselves with the underlying molecular and cellular events to evaluate the future evolution of implant design and implant protocols, including surgical placement, restoration, and maintenance.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 69

Clinical Evaluation of the Implant Patient

Perry R. Klokkevold and David L. Cochran

CHAPTER OUTLINE

CASE TYPES AND INDICATIONS
- Edentulous Patients
- Partially Edentulous Patients

PRETREATMENT EVALUATION
- Chief Complaint
- Medical History
- Dental History
- Intraoral Examination

RISK FACTORS AND CONTRAINDICATIONS
- Medical and Systemic Health–Related Issues
- Psychologic and Mental Conditions
- Habits and Behavioral Considerations

POSTTREATMENT EVALUATION
- Clinical Examination
- Radiographic Examination
- Oral Hygiene and Implant Maintenance

CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures may be out of numeric order in this chapter.

Endosseous dental implants and their retained prostheses have had great success over the past few decades following the landmark research and development of osseointegrated implants by Brånemark et al.[18–20] Initially, most prosthetic reconstructions with osseointegrated implants were limited to use in the edentulous patient, with many reports documenting excellent long-term success of implant-retained prostheses for edentulous patients.[1,2,32]

Following much success with implants in edentulous patients, the original implant treatment protocols were adapted for use in partially edentulous patients. Although some transitional problems were associated with the early use of dental implants in the partially edentulous patient, successes were achieved for this population as well. Subsequently, modifications in implant design, procedural techniques, and treatment planning greatly improved implant therapy for the partially edentulous patient. Currently, the long-term success of dental implants used to replace single and multiple missing teeth in the partially edentulous patient is very good[37,51,53,63,70] (see Chapter 80). Additionally, with the implementation of bone augmentation procedures, even patients with inadequate bone volume have a good opportunity to be successfully restored with implant-retained prostheses.[34,43,72] Virtually any patient with an edentulous space is a candidate for endosseous implants, and studies suggest that greater than 90% to 95% success rates can be expected in healthy patients with good bone and normal healing capacity.[30]

The ultimate goal of dental implant therapy is to satisfy the patient's desire to replace one or more missing teeth in an esthetic, secure, functional, and long-lasting manner. To achieve this goal, clinicians must accurately diagnose the current dentoalveolar condition, as well as the overall mental and physical well-being of the patient to determine whether implant therapy is possible or practical and perhaps most importantly, whether it is indicated for a particular patient. Local evaluation of potential jaw sites for implant placement (e.g., measuring available alveolar bone height, width, and spatial relationship) and prosthetic restorability are an essential part of determining whether an implant(s) is possible. However, determining whether the patient is a good candidate for implants is an equally important aspect of the evaluation process. This aspect of the patient evaluation includes identifying factors that might increase the risk of failure or complications, as well as determining whether the patient's expectations are reasonable.

This chapter presents an overview of the clinical aspects of dental implant therapy, including an assessment of possible risk factors and contraindications. It also provides guidelines for the pretreatment evaluation of potential implant patients, posttreatment evaluation of patients with implants, and implant maintenance.

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Figure 69-1 Clinical photograph of patient with a complete maxillary denture opposing a full-arch implant-supported fixed prosthesis in the mandibular arch.

CASE TYPES AND INDICATIONS

Edentulous Patients

The patients who seem to benefit most from dental implants are those with fully edentulous arches. These patients can be effectively restored, both esthetically and functionally, with an implant-assisted removable prosthesis or an implant-supported fixed prosthesis.

The original design for the edentulous arch was a fixed-bone–anchored bridge that used five to six implants in the anterior area of the mandible or the maxilla to support a fixed, hybrid prosthesis. The design is a denture-like complete arch of teeth attached to a substructure (metal framework), which in turn is attached to the implants with cylindrical titanium abutments (Figure 69-1). The prosthesis is fabricated without flange extensions and does not rely on any soft tissue support. It is entirely implant supported (see Figure 76-13). Usually, the prosthesis includes bilateral distal cantilevers, which extend to replace posterior teeth (back to premolars or first molars).

Another implant-supported design used to restore an edentulous arch is the ceramic-metal fixed bridge (Figure 69-2). Some patients prefer this design because the ceramic restoration emerges directly from the gingival tissues in a manner similar to the appearance of natural teeth. One limitation of both hybrid and ceramometal implant-supported fixed prostheses is that they provide very little lip support and thus may not be indicated for patients who have lost significant alveolar dimension. This is often more problematic for maxillary reconstructions because lip support is more critical in the upper arch. For some patients, the lack of a complete seal (i.e., spaces under the framework) allows air to escape during speech, thus creating phonetic problems.

Depending on the volume of existing bone, the jaw relationship, the amount of lip support, and phonetics, some patients may not be able to be rehabilitated with an implant-supported fixed prosthesis. For these patients, a removable, complete-denture type of prosthesis is a better choice because it provides a flange extension that can be adjusted and contoured to support the lip, and there are no spaces for unwanted air escape during speech. This type of prosthesis can be retained and stabilized by two or more implants

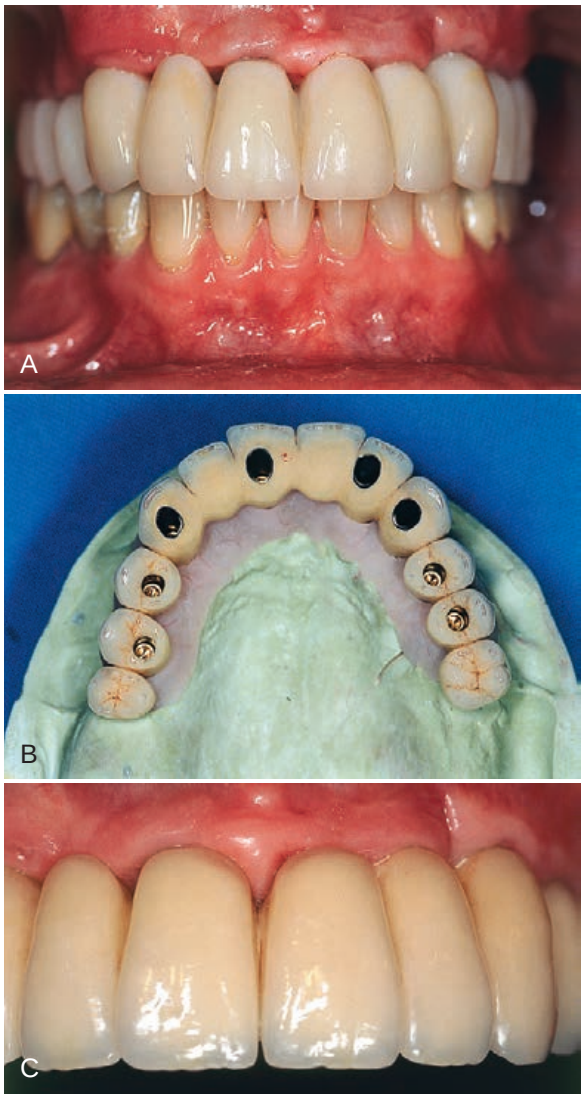

Figure 69-2 **A,** Clinical photograph of acrylic provisional fixed full-arch prosthesis in the maxilla. **B,** Clinical photograph of the final ceramometal restoration, anterior view. **C,** Occlusal view of final restoration on master cast. *(Courtesy Dr. Russell Nishimura, Westlake Village, CA.)*

placed in the anterior region of the maxilla or mandible. Methods used to secure the denture to the implants vary from separate attachments on each individual implant to clips or other attachments that connect to a bar, which splints the implants together (Figure 69-3). Advantages and disadvantages of these attachment designs are discussed in Chapter 76.

Although the stability of the implant-retained overdenture does not compare to the rigidly attached, implant-supported fixed prosthesis, the increased retention and stability over conventional complete dentures is an important advantage for denture wearers.[74] Additionally, implant-assisted and implant-supported prostheses are thought to protect alveolar bone from additional bone loss caused by long-term use of removable prostheses that are bearing directly on the alveolar ridges.

Partially Edentulous Patients

Multiple Teeth. Partially edentulous patients with multiple missing teeth represent another viable treatment population for osseointegrated implants, but the remaining natural dentition (occlusal schemes, periodontal health status, spatial relationships,

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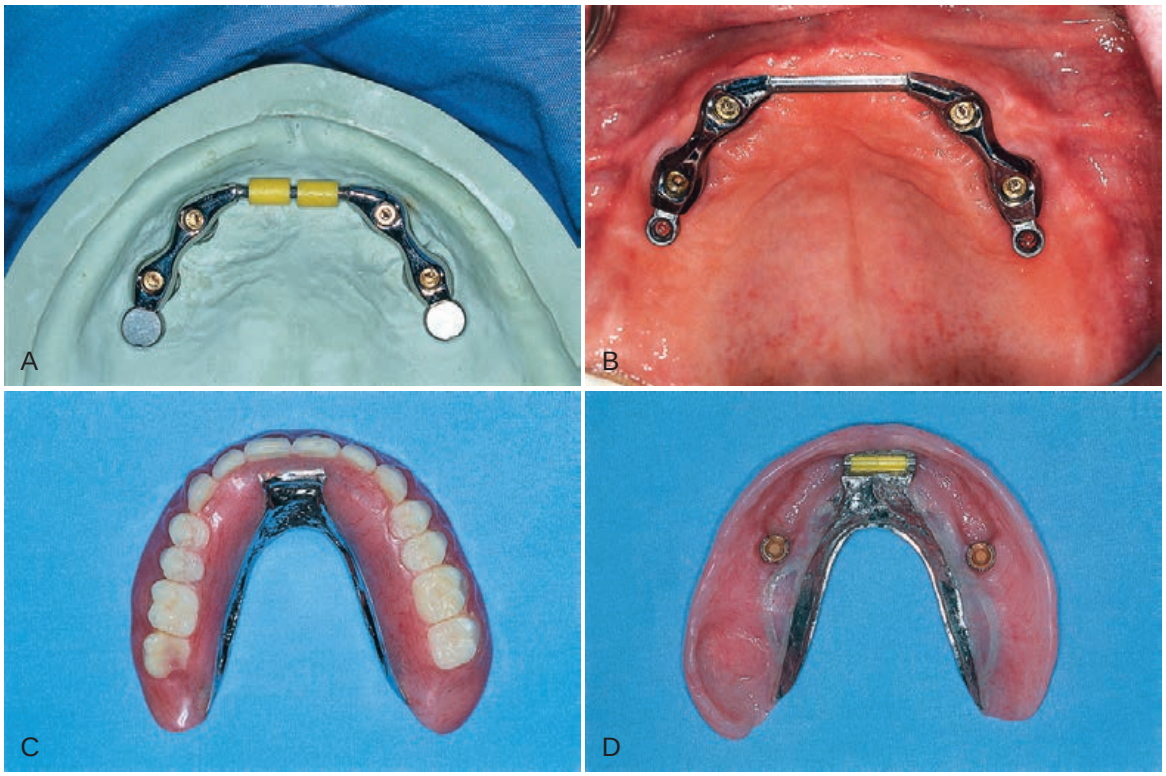

Figure 69-3 **A,** Laboratory view of maxillary overdenture bar attached to four implants with anterior clips and posterior extracoronal resilient attachments (ERAs). **B,** Clinical view of maxillary overdenture bar. **C,** Palateless maxillary complete overdenture. **D,** Tissue surface of same maxillary implant-assisted overdenture showing clips and ERAs. *(Courtesy Dr. John Beumer, UCLA Maxillofacial Prosthodontics, Los Angeles.)*

and esthetics) introduces additional challenges for successful rehabilitation.[52] The juxtaposition of implants with natural teeth in the partially edentulous patient presents the clinician with challenges not encountered with implants in the edentulous patient. As a result of distinct differences in the biology and function of implants compared with natural teeth, clinicians must educate themselves and use a prescribed approach to the evaluation and treatment planning of implants for partially edentulous patients (see Chapter 76). In general, endosseous dental implants can support a free-standing fixed partial denture. Adjacent natural teeth are not necessary for support, but their close proximity requires special attention and planning.[12] The major advantage of implant-supported restorations in partially edentulous patients is that they replace missing teeth without invasion or alteration of adjacent teeth. Preparation of natural teeth becomes unnecessary, and larger edentulous spans can be restored with implant-supported fixed bridges.[65] Moreover, patients who previously did not have a fixed option, such as those with Kennedy Class I and II partially edentulous situations, can be restored with an implant-supported fixed restoration (Figure 69-4).

Early attempts to use endosseous implants to replace missing teeth in the partially edentulous patient were a challenge partly because the implants and armamentarium were designed for the edentulous patient and did not have much flexibility for adaptation and use in the partially edentulous patient (i.e., one standard-diameter implant, one type of abutment, and one surgical kit with instrumentation that was difficult to use adjacent to teeth). As the demand for implants in the partially edentulous patient increased, implant manufacturers responded with the development of wide-diameter and narrow-diameter implants and a variety of abutment choices. They also developed instrumentation that was better suited for the placement of implants adjacent to natural teeth. Currently, clinicians have many choices in terms of implant length, diameter, and abutment connection to choose for the optimal replacement of any missing tooth, large or small (Figure 69-5).

Another difficulty with partially edentulous cases is an underestimation of the importance of treatment planning for implant-retained restorations with an adequate number of implants to withstand occlusal loads. For example, one problem that required correction was the misconception that two implants could be used to support a multiunit fixed bridge in the posterior area. Multiunit fixed restorations in the posterior jaw are more likely to experience complications or failures (mechanical or biologic) when they are inadequately supported either in terms of the number of implants, quality of bone, or strength of the implant material (see Chapter 76). The use of stronger implants and better treatment planning (more implants used to support more restorative units), particularly in areas of poor-quality bone, has solved many of these problems.

Single Tooth. Patients with a missing single tooth (anterior or posterior) represent another type of patient who benefits greatly from the success and predictability of endosseous dental implants. Replacement of a single missing tooth with an implant-supported crown is a much more conservative approach than preparing two adjacent teeth for the fabrication of a tooth-supported fixed partial denture. It is no longer necessary to "cut" healthy or minimally restored adjacent teeth to replace a missing tooth with a nonremovable prosthetic replacement (Figure 69-6). Reported success rates for single-tooth implants are excellent.[29]

Replacement of an individual missing posterior tooth with an implant-supported restoration has been successful as well. The greatest challenges to overcome with the single-tooth implant restorations were screw loosening and implant or component fracture.

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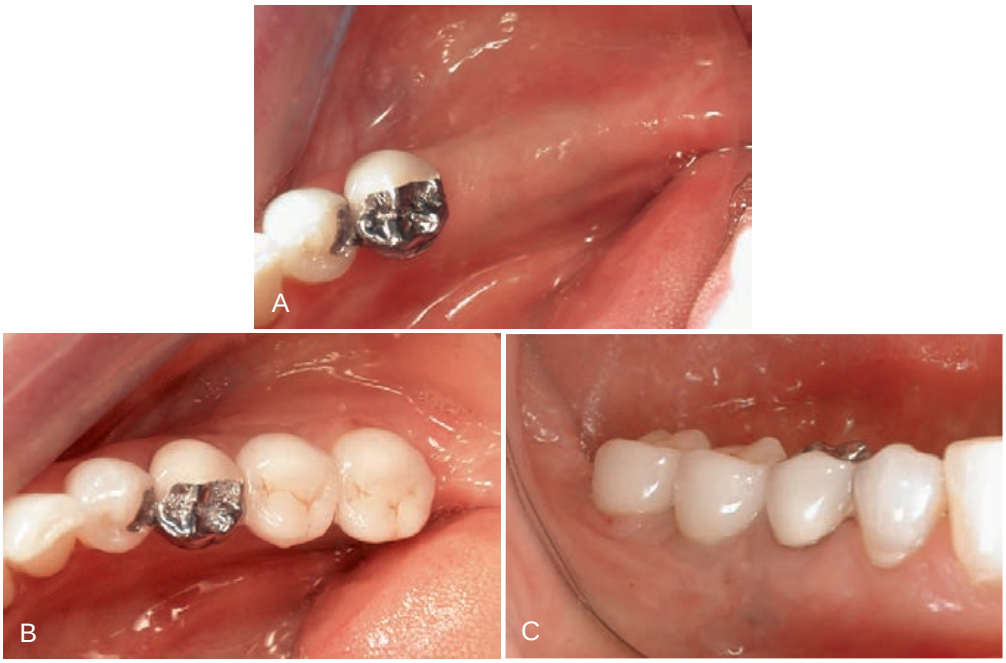

Figure 69-4 **A,** Clinical view of partially edentulous posterior mandible (Kennedy Class II distal extension). **B,** Occlusal view of same patient in **A** restored with implant-supported fixed restoration replacing teeth #18 and 19. Notice the dimensions of the crowns are smaller than typical mandibular molars (i.e., closer to bicuspid size). **C,** Buccal view of same restorations.

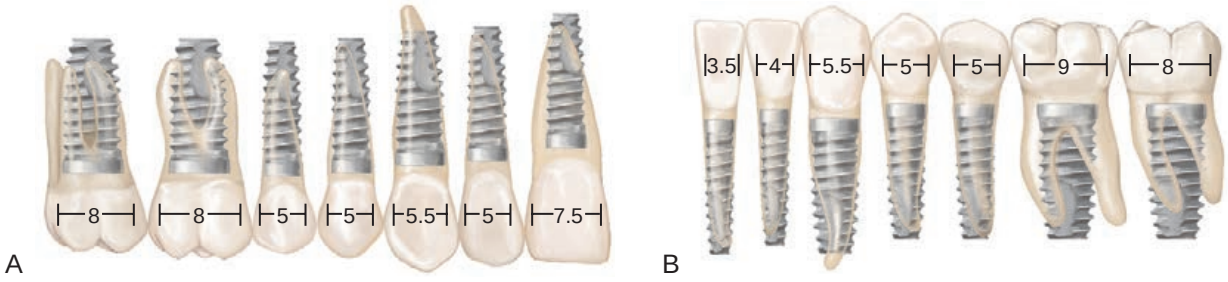

Figure 69-5 Diagram representing the use of wide-, narrow-, and standard-diameter implants for molars, mandibular incisors, and other teeth (different-sized implants superimposed over various teeth). **A,** Maxillary teeth. **B,** Mandibular teeth.

Because of increased potential to generate forces in the posterior area, the implants, components, and screws often failed. Both these problems have been addressed with the use of wider-diameter implants and internal fixation of components (Figure 69-7). Wide-diameter implants often have a wider platform (restorative interface) that resists tipping forces and thus reduces screw loosening. The wide-diameter implant also provides greater strength and resistance to fracture as a result of increased wall thickness (the thickness of the implant between the inner screw thread and the outer screw thread). Implants with an internal connection are inherently more resistant to screw loosening and thus have an added advantage for single-tooth applications. Most dental implant manufacturers now sell implants with internal component fixation.

Esthetic Considerations. Anterior single-tooth implants present some of the same challenges as the single posterior tooth supported by an implant, but they also are an esthetic concern for patients. Some cases are more esthetically challenging than others because of the nature of each individual's smile and display of teeth. The prominence and occlusal relationship of existing teeth, the thickness and health of periodontal tissues, and the patient's own psychologic perception of esthetics all play a role in the esthetic challenge of the case. Cases with good bone volume, bone height, and tissue thickness can be predictable in terms of achieving satisfactory esthetic results (see Figure 69-6). However, achieving esthetic results for patients with less-than-ideal tissue qualities poses difficult challenges for the restorative and surgical team.[12] Replacing a single tooth with an implant-supported crown in a patient with a high smile line, compromised or thin periodontium, inadequate hard or soft tissues, and high expectations is probably one of the most difficult challenges in implant dentistry and should not be attempted by novice clinicians.

PRETREATMENT EVALUATION

A comprehensive evaluation is indicated for any patient who is being considered for dental implant therapy. The evaluation should assess all aspects of the patient's current health status, including a review the patient's past medical history, medications, and medical treatments. Patients should be questioned about parafunctional habits, such as clenching or grinding teeth, as well as any substance

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use or abuse, including tobacco, alcohol, and drugs. The assessment should also include an evaluation of the patient's motivations, level of understanding, compliance, and overall behavior. For most patients, this involves simply observing their demeanor and listening to their comments for an impression of their overall sensibility and coherence with other patient norms. for some individuals with questionable behavior, however, a professional psychologic assessment of their mental health status may be indicated.

Figure 69-6 Single-tooth replacement. **A,** Implant in place. **B,** Metallo-ceramic crown.

An intraoral and radiographic examination must be done to determine whether it is possible to place implant(s) in the desired location(s). Properly mounted diagnostic study models and intraoral clinical photographs are a useful part of the clinical examination and treatment-planning process to aid in assessment of spatial and occlusal relationships. Once the data collection is completed, the clinician will be able to determine whether implant therapy is possible, practical, and indicated for the patient.

Conducting an organized, systematic history and examination is essential to obtaining an accurate diagnosis and creating a treatment plan that is appropriate for the patient. Each treatment plan should be comprehensive and provide several treatment options for the patient, including periodontal and restorative therapies. Then, in consultation, the clinician can agree on the final treatment plan with the patient. Information gathered throughout the process will help the clinician's decision making and determination of whether a patient is a good candidate for dental implants. A thoughtful and well-executed evaluation can also reveal deficiencies and indicate what additional surgical procedures may be necessary to accomplish the desired goals of therapy (e.g., localized ridge augmentation, sinus bone augmentation). Each part of the pretreatment evaluation is briefly discussed here.

Chief Complaint

What is the problem or concern in the patient's own words? What is the patient's goal of treatment, and how realistic are the patient's expectations? The patient's chief concern, desires for treatment, and vision of the successful outcome must be taken into consideration. The patient will measure implant success according to personal criteria. The overall comfort and function of the implant restoration are often the most important factors, but satisfaction with the appearance of the final restoration will also influence the patient's perception of success. Furthermore, patient satisfaction may be influenced simply by the impact that the treatment has on the patient's perceived quality of life. Whether the clinician inquires about quality-of-life changes or not, the patient most likely will measure the success of treatment by comparison to the pretreatment condition. Patients will evaluate for themselves whether the treatment helped them to eat better, look better, or feel better about themselves.

The clinician could consider an implant(s) and the retained prosthesis a success using standard criteria of symptom-free implant function, implant stability, and lack of periimplant infection or bone loss. at the same time, however, the patient who does not like

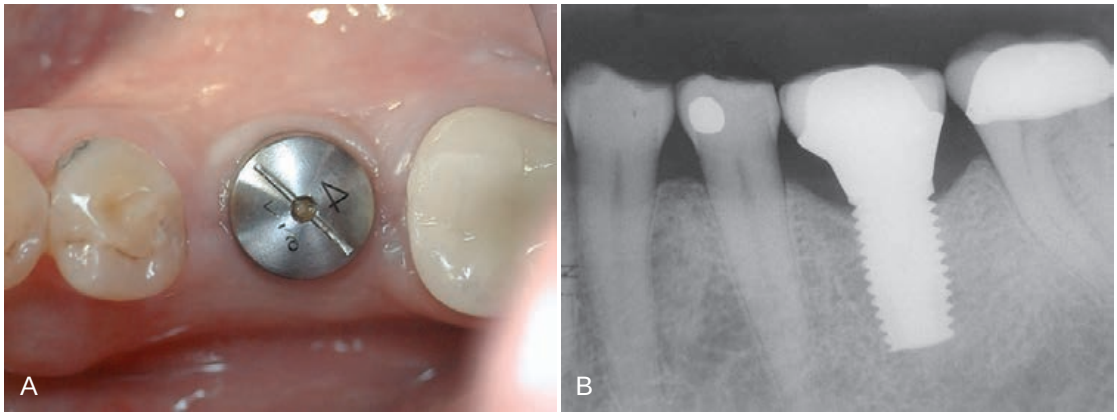

Figure 69-7 **A,** Occlusal view of healing abutment, which is attached to a wide-diameter implant used to replace a single missing molar. **B,** Radiograph of same patient depicted in **A,** showing the wide-diameter implant supporting the final restoration (molar replaced with a single-tooth implant-supported crown).

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the esthetic result or does not think the condition has improved could consider the treatment a failure. Therefore it is critical to inquire, as specifically as possible, about the patient's expectations before initiating implant therapy and to appreciate the patient's desires and values. With this goal in mind, it is often helpful and advisable to invite patients to bring their spouse or a family member to the consultation and treatment-planning visits to add an independent "trusted" observer to the discussion of treatment options. Ultimately, it is the clinician's responsibility to determine if the patient has realistic expectations for the outcome of therapy and to educate the patient about realistic outcomes for each treatment option.

Medical History

A thorough medical history is required for any patient in need of dental treatment, regardless of whether implants are part of the plan. This history should be documented in writing by the patient's completion of a standard health history form and verbally through an interview with the treating clinician. The patient's health history should be reviewed for any condition that might put the patient at risk for adverse reactions or complications.

Patients must be in reasonably good health to undergo surgical therapy for the placement of dental implants. Any disorder that may impair the normal wound-healing process, especially as it relates to bone metabolism, should be carefully considered as a possible risk factor or contraindication to implant therapy (see later discussion).

A thorough physical examination is warranted if any questions arise about the health status of the patient.[18] Appropriate laboratory tests (e.g., coagulation tests for a patient receiving anticoagulant therapy) should be requested to evaluate further any conditions that may affect the patient's ability to undergo the planned surgical and restorative procedures safely and effectively. If any questions remain about the patient's health status, a medical clearance for surgery should be obtained from the patient's treating physician.

Dental History

A review of a patient's past dental experiences can be a valuable part of the overall evaluation. Does the patient report a history of recurrent or frequent abscesses, which may indicate a susceptibility to infections or diabetes? Does the patient have many restorations? How compliant has the patient been with previous dental recommendations? What are the patient's current oral hygiene practices?

The individual's previous experiences with surgery and prosthetics should be discussed. If a patient reports numerous problems and difficulties with past dental care, including a history of dissatisfaction with past treatment, the patient may have similar difficulties with implant therapy. It is essential to identify past problems and to elucidate any contributing factors. The clinician must also assess the patient's dental knowledge and understanding of the proposed treatment, as well as the patient's attitude and motivation toward implants.

Intraoral Examination

The oral examination is performed to assess the current health and condition of existing teeth, as well as to evaluate the condition of the oral hard and soft tissues. It is imperative that no pathologic conditions are present in any of the hard or soft tissues in the maxillofacial region. All oral lesions, especially infections, should be diagnosed and appropriately treated before implant therapy. Additional criteria to consider include the patient's habits, level of oral hygiene, overall dental and periodontal health, occlusion, jaw relationship, temporomandibular joint condition, and ability to open wide.

After a thorough intraoral examination, the clinician can evaluate potential implant sites. All sites should be clinically evaluated to measure the available space in the bone for the placement of implants and in the dental space for prosthetic tooth replacement (Box 69-1). The mesial-distal and buccal-lingual dimensions of edentulous spaces can be approximated with a periodontal probe or other measuring instrument. The orientation or tilt of adjacent teeth and their roots should be noted as well. There may be enough space in the coronal area for the restoration but not enough space in the apical region for the implant if roots are directed into the area of interest (Figure 69-8). Conversely, there may be adequate space between roots, but the coronal aspects of the teeth may be too close for emergence and restoration of the implant. If either of these conditions is discovered, orthodontic tooth movement may

BOX 69-1 How Much Space Is Required for Placement of One or More Implants?*

Alveolar Bone

Assuming an implant that is 4 mm in diameter and 10 mm long, the minimal width of the jawbone needs to be 6 to 7 mm, and the minimal height should be 10 mm (minimum of 12 mm in the posterior mandible, where an additional margin of safety is required over the mandibular nerve). This dimension is desired to maintain at least 1.0 to 1.5 mm of bone around all surfaces of the implant after preparation and placement.

Interdental Space

Edentulous spaces need to be measured to determine whether enough space exists for the placement and restoration with one or more implant crowns. The minimal space requirements for the placement of one, two, or more implants are illustrated diagrammatically in Figure 69-9. The minimal mesial-distal space for an implant placed between two teeth is 7 mm. The minimal mesial-distal space required for the placement of two standard-diameter implants (4.0-mm diameter) between teeth is 14 mm. The required minimal dimensions for wide-diameter or narrow-diameter implants will increase or decrease incrementally according to the size of the implant. For example, the minimal space needed for the placement of an implant 6 mm in diameter is 9 mm (= 7 mm + 2 mm). Whenever the available space between teeth is greater than 7 mm and less than 14 mm, only one implant, such as placement of a wide-diameter implant, should be considered. The placement of a wide-diameter implant should be considered. Two narrow-diameter implants could be positioned in a space that is 12 mm. However, the smaller implant may be more vulnerable to implant fracture.

Interocclusal Space

The restoration consists of the abutment, the abutment screw, and the crown (it may also include a screw to secure the crown to the abutment if it is not cemented). This restorative "stack" is the total of all the components used to attach the crown to the implant. The dimensions of the restorative stack vary slightly depending on the type of abutment and the implant-restorative interface (i.e., internal or external connection). The minimum amount of interocclusal space required for the restorative "stack" on an external hex-type implant is 7 mm.

*All the minimal space requirements discussed here are generic averages. The actual space limitations for any particular implant system must be determined according to the manufacturer's specifications.

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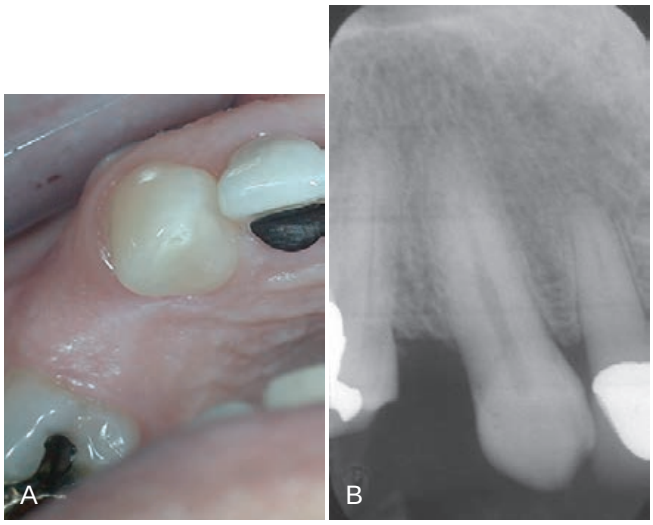

Figure 69-8 **A,** Clinical photograph of maxillary premolar space with apparently adequate space between the remaining teeth for an implant-supported crown. **B,** Radiograph clearly shows a lack of space between the roots of the adjacent teeth as a result of convergence into the space (same patient as in **A**).

BOX 69-2 How Much Keratinized Tissue Is Required for Health and Maintenance of Implants?

Debate continues about whether it is necessary to have a zone of keratinized tissue surrounding implants. Despite strong opinions and beliefs about the need for keratinized mucosa around implants versus this mucosa being unnecessary, neither argument has been proved.

Some studies have concluded that, in the presence of good oral hygiene, a lack of keratinized tissue does not impair the health or function of implants.[73] Others strongly believe that keratinized mucosa has better functional and esthetic results for implant restorations. Keratinized mucosa is typically thicker and denser than alveolar mucosa (nonkeratinized). It forms a strong seal around the implant with a cuff of circular (parallel) fibers around the implant, abutment, or restoration that is resistant to retracting with mastication forces and oral hygiene procedures. Implants with coated surfaces (i.e., hydroxyapatite [H] or titanium plasma spray [TPS] coating) demonstrate greater periimplant bone loss and failures in the absence of keratinized mucosa.[15,47]

be indicated. Ultimately, edentulous areas need to be precisely measured using diagnostic study models and imaging techniques to determine whether space is available and whether adequate bone volume exists to replace missing teeth with implants and implant restorations. Figure 69-9 diagrams the minimal space requirements for standard-, wide-, and narrow-diameter implants placed between natural teeth, and the minimal interocclusal space needed to restore implants.

Diagnostic Study Models. Mounted study models are an excellent means of assessing potential sites for dental implants. Properly articulated models with diagnostic wax-up of the proposed restorations allow the clinician to evaluate the available space and to determine potential limitations of the planned treatment (Figure 69-10 online). This is particularly useful when multiple teeth are to be replaced with implants or when a malocclusion is present.

To view Figure 69-10, please visit the companion Expert CONSULT website at www.expertconsult.com.

Hard Tissue Evaluation. The amount of available bone is the next criterion to evaluate. Wide variations in jaw anatomy are encountered, and it is therefore important to analyze the anatomy of the dentoalveolar region of interest both clinically and radiographically.

A visual examination can immediately identify deficient areas (Figure 69-11), whereas other areas that appear to have good ridge width will require further evaluation (Figure 69-12). Clinical examination of the jawbone consists of palpation to feel for anatomic defects and variations in the jaw anatomy, such as concavities and undercuts. If desired, it is possible with local anesthesia to probe through the soft tissue (intraoral bone mapping) to assess the thickness of the soft tissues and measure the bone dimensions at the proposed surgical site.

The spatial relationship of the bone must be evaluated in a three-dimensional view because the implant must be placed in the appropriate position relative to the prosthesis. it is possible that an adequate dimension of bone is available in the anticipated implant site (see Box 69-1), but that the bone and thus the implant placement might be located too lingual or too buccal for the desired prosthetic tooth replacement.[38] Bone augmentation procedures may be necessary to facilitate the placement of an implant in an acceptable prosthetic position despite the availability of an adequate quantity of bone (i.e., the bone is in the wrong location). Indications for the use of bone augmentation procedures are discussed in Chapter 72.

Radiographic Examination. Radiographic assessment of the quantity, quality, and location of available alveolar bone in potential implant sites ultimately determines whether a patient is a candidate for implants and if a particular implant site needs bone augmentation. Appropriate radiographic procedures, including periapical radiographs, panoramic projections, and tomographic cross-sectional imaging, can help identify vital structures such as the floor of the nasal cavity, maxillary sinus, mandibular canal, and mental foramen (see Chapter 70). In addition to the absolute dimensional measurement of the alveolar bone, it is important to determine whether the volume of bone radiographically (as well as clinically) is located in a position to allow for the proper position of the implant to facilitate restoration of the tooth/teeth in proper esthetic and functional relationship with the adjacent and opposing dentition. The best way to evaluate the relationship of available bone to the dentition is to image the patient with a diagnostically accurate guide using radiopaque markers that accurately represent the proposed prosthetic contours (see Figure 70-3).

Soft Tissue Evaluation. Evaluation of the quality, quantity, and location of soft tissue present in the anticipated implant site helps to anticipate the type of tissue that will surround the implant(s) after treatment is completed (keratinized versus nonkeratinized mucosa). For some cases, depending on the clinician's view of keratinized tissue, evaluation may reveal a need for soft tissue augmentation (Box 69-2). Areas with minimal or no existing keratinized mucosa may be augmented with gingival or connective tissue grafts. Additionally, any mucogingival concerns, such as frenum attachments or pulls, should be thoroughly evaluated.

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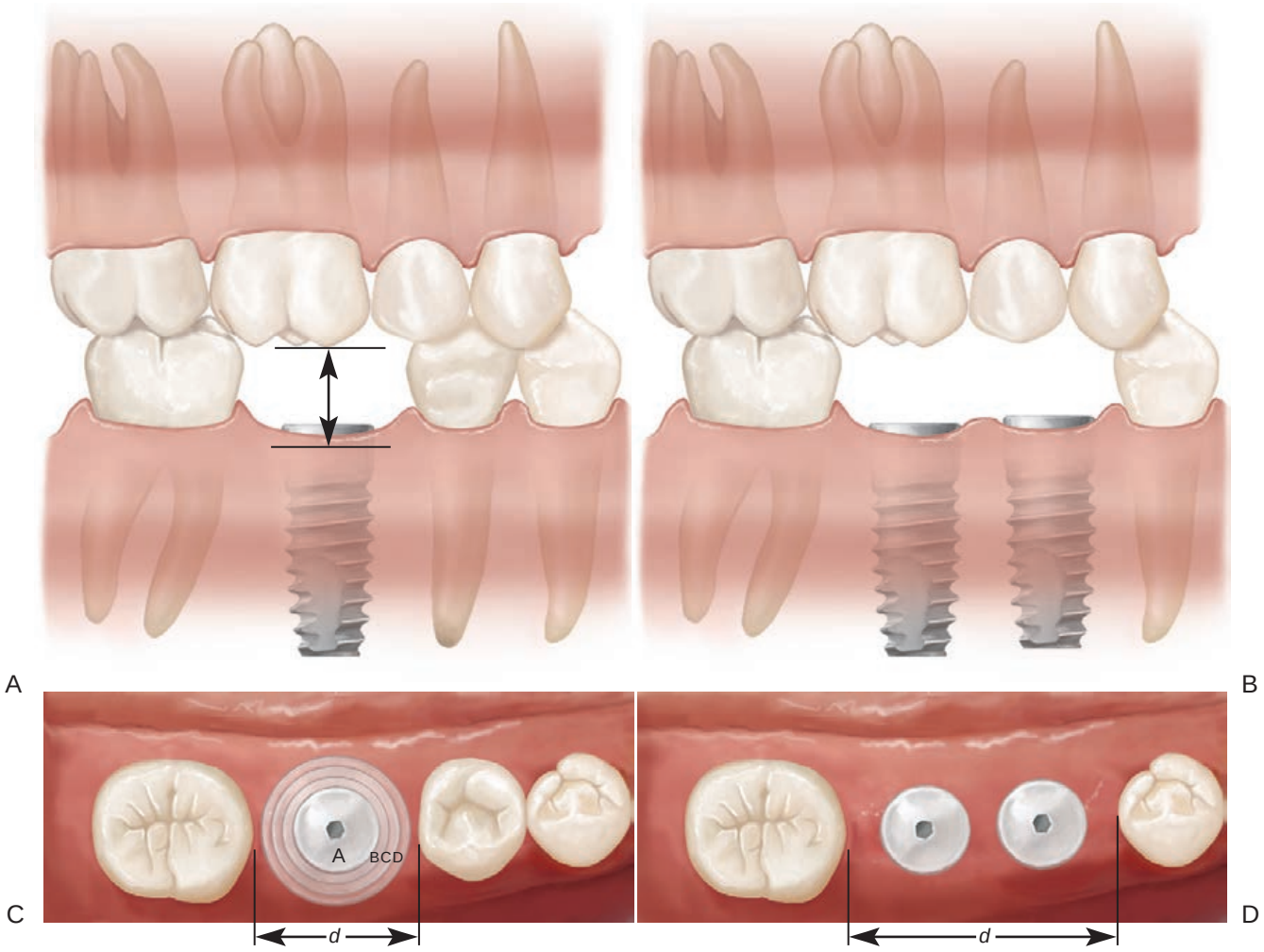

The minimum mesial-distal (*d*) required for a:
A. Narrow diameter implant (e.g., 3.25 mm) is 6 mm.
B. Standard diameter implant (e.g., 4.1 mm) is 7 mm.
C. Wide diameter implant (e.g., 5.0 mm) is 8 mm.
D. Wide diameter implant (e.g., 6.0 mm) is 9 mm.

The minimum mesial-distal (*d*) required for two standard diameter implants is 14 mm wide.

Figure 69-9 **A,** Minimum amount of mesial-distal space *(d)* required for placement of single-tooth implant between natural teeth: **A,** 6 mm for narrow-diameter implant (3.25 mm); **B,** 7 mm for standard-diameter implant (4.1 mm); **C** and **D,** 8 mm and 9 mm, respectively, for wide-diameter implants (5.0 mm and 6.0 mm). **B,** minimum amount of mesial-distal space *(d)* required for placement of two standard-diameter (4.1 mm) implants between natural teeth is 14 mm. This allows approximately 2 mm between teeth/implants and between implant/implant. Minimum amount of space required between implant/restoration interface and opposing occlusal surfaces for restoration of an implant. This dimension will vary, depending on implant design and manufacturer component dimensions. The minimal dimension of 7 mm is based on an externally hexed implant and UCLA abutment.

RISK FACTORS AND CONTRAINDICATIONS

Clearly, there are numerous indications for the use of endosseous dental implants to replace missing teeth. Most patients who are missing one or more teeth can benefit from the application of an implant-retained prosthesis provided they meet the requirements for surgical and prosthetic rehabilitation. Edentulous patients who are unable to function with complete dentures and who have adequate bone for the placement of dental implants can be especially good dental implant candidates. More and more partially edentulous patients are also being treated with dental implant restorations. Many patients, whether they are missing one, several, or all of their teeth, can be predictably restored with implant-retained prostheses.

In this era of high implant success and predictability and thus possible complacency, it is imperative for clinicians to recognize risk factors and contraindications to implant therapy so that problems can be minimized and patients can be accurately informed about risks. As such, the clinician must be knowledgeable in this area and inform patients about risk factors and contraindications before initiating treatment. Contraindications for the use of dental implants, although relatively few and often not well defined, do exist. Some conditions are probably best described as "risk factors" rather than "contraindications" to treatment because implants can be successful in almost all patients; implants may be less *predictable* in some situations, and this distinction should be recognized. Ultimately, it is the clinician's responsibility with the patient to make decisions as to when implant therapy is not indicated.

Table 69-1 lists some conditions and factors that are thought to increase the risk for implant failure or otherwise deem the patient a poor candidate for implant therapy. Some of these conditions are briefly discussed here.

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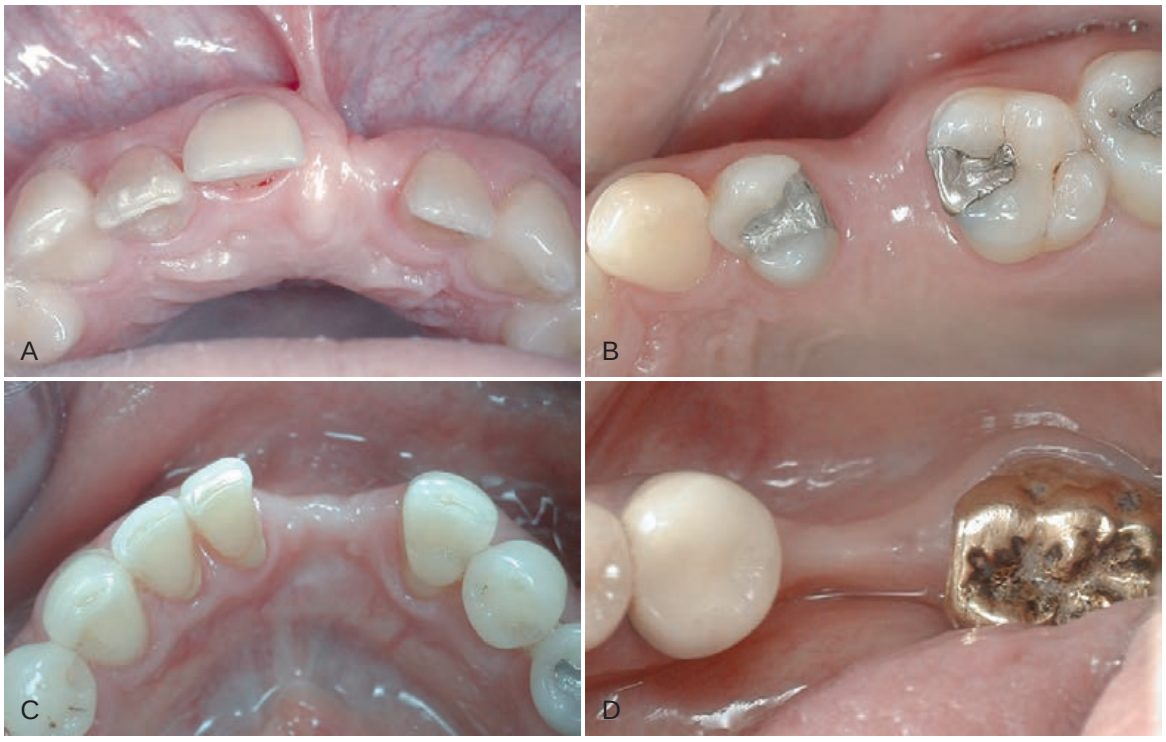

Figure 69-11 Clinical photographs of edentulous areas with obvious deficient areas of alveolar dimension noted on visual examination: **A,** anterior maxilla; **B,** posterior maxilla; **C,** anterior mandible; **D,** posterior mandible. These clinical images all represent buccal-lingual deficiencies in the alveolar dimensions.

Figure 69-12 Clinical photographs of edentulous areas with apparent good alveolar dimension noted on visual examination: **A,** anterior maxilla; **B,** posterior maxilla; **C,** anterior mandible; **D,** posterior mandible. It is likely that these sites have adequate bone volume for implant placement. However, it is also possible to find alveolar deficiencies despite the appearance of wide ridges.

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TABLE 69-1 Risk Factors and Contraindications for Implant Therapy

	Risk Factor	Contraindication
Medical and Systemic Health–Related Issues		
Diabetes (poorly controlled)	??—Possibly	Relative
Bone metabolic disease (e.g., osteoporosis)	??—Probably	Relative
Radiation therapy (head and neck)	Yes	Relative/Absolute
Bisphosphonate therapy (intravenous)	??—Probably	Relative/Absolute
Bisphosphonate therapy (oral)	??—Possibly	Relative
Immunosuppressive medication	??—Probably	Relative
Immunocompromising disease (e.g., HIV, AIDS)	??—Possibly	Relative
Psychologic and Mental Conditions		
Psychiatric syndromes (e.g., schizophrenia, paranoia)	No	Absolute
Mental instability (e.g., neurotic, hysteric)	No	Absolute
Mentally impaired; uncooperative	No	Absolute
Irrational fears; phobias	No	Absolute
Unrealistic expectations	No	Absolute
Habits and Behavioral Considerations		
Smoking; tobacco use	Yes	Relative
Parafunctional habits	Yes	Relative
Substance abuse (e.g., alcohol, drugs)	??—Possibly	Absolute
Intraoral Examination Findings		
Atrophic maxilla	Yes	Relative
Current infection (e.g., endodontic)	Yes	Relative
Periodontal disease	??—Possibly	Relative

HIV, Human immunodeficiency virus; *AIDS,* acquired immunodeficiency syndrome.

Medical and Systemic Health–Related Issues

Although few absolute medical contraindications to implant therapy exist, some relative contraindications are important to consider. The clinician must consider medical and health-related conditions that affect bone metabolism or any aspect of the patient's capacity to heal normally.[11] This category includes conditions such as diabetes, osteoporosis, and immune compromise, medications, and medical treatments such as chemotherapy and irradiation.

Diabetes Mellitus. Diabetes is a metabolic disease that can have significant effects on the patient's ability to heal normally and resist infections. This is particularly true for patients whose diabetes is not well controlled. Poorly controlled diabetics often have impaired wound healing and a predisposition to infections, whereas diabetic patients whose disease is well controlled experience few, if any, problems (see Chapter 27).

There is concern about the success and predictability of implants in patients with diabetes. Several studies have reported moderate failure rates in diabetic patients, with implant success ranging from 85.6% to 94.3%.[9,33,45,48] A prospective study demonstrated 2.2% early failures and 7.3% late failures in diabetic patients.[69] After 5 years, the overall success rate for this group of diabetic patients was 90%.[62] None of these studies was able to correlate gender, age, smoking, diabetes type, or level of diabetic control with implant failure. In a metaanalytical review of implant failures in patients who were not diabetics, the early implant failure rate was 3.2% and the late implant failure rate 5.2%.[28] The finding that diabetic patients experience slightly more late failures may be related to less tissue integrity caused by reduced tissue turnover and impaired tissue perfusion. These results suggest that diabetes may be a risk factor for implants, particularly for late failures. However, the risk does not appear to be particularly high.

Bone Metabolic Disease. Osteoporosis is a skeletal condition characterized by decreased mineral density. The two main classifications are primary (three types) and secondary (many types) osteoporosis. *Primary osteoporosis* has been attributed to menopausal changes (type I), age-related changes (type II), or idiopathic causes (type III). *Secondary osteoporosis* has been attributed to many different diseases and conditions, including diabetes, alcoholism, malnutrition, and smoking.[39]

All the various types of osteoporosis share the same fundamental problem of decreased bone mineral density, and the concern that this condition may impair the patient's ability to achieve and maintain implant osseointegration. The premise that implants will not perform as well in a patient with osteoporosis is reasonable given that osseointegration depends on bone formation adjacent to the implant surface and that success rates are highest in dense bone and lowest in poor-quality, loose trabecular bone. However, to date, there is no clear evidence to suggest that implants will not be successful in patients with osteoporosis, so the issue continues to be debated.[10,24] On the positive side, although the evidence is weak, case reports have demonstrated successful implant treatment in patients with osteoporosis.[38] Some investigators advocate the use of longer healing times for osseointegration to occur before loading the implants in patients with osteoporosis.[35] Conversely, in a retrospective analysis of 49 patients who received sinus bone augmentation, individuals (11 patients) with lower bone mass density had significantly lower implant success rates as compared to age- and sex-matched controls.[16] Other parameters evaluated in this study did not demonstrate any significant differences.

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Interestingly, there is a trend in aging adults (men over 50 years and postmenopausal women) for bone mass to decrease progressively through bone demineralization at a rate of 1% to 2% per year and in some individuals as much as 5% to 8% per year throughout their later life.[26,44] If one considers this decline in bone mass with aging along with a continually increasing life expectancy in the population, the number of individuals with osteopenia or osteoporosis will continue to increase, and the concern about this condition's influence on implant success will become increasingly important for clinicians.

Medications. Some prescribed medications, including steroids and bisphosphonates, may be cause for concern relative to the potential implant patient. Corticosteroid therapy, whether used for hormone replacement, cancer treatment, immune suppression or other chronic condition may suppress the immune response, impair wound healing or compromise the normal adrenal response to stress. See the later section on immune compromise and immune suppression, as well as Chapters 27 and 37 for more information on the treatment of patients taking corticosteroids and bisphosphonate medications. Only a brief statement regarding the risk of bisphosphonate therapy is offered here. Readers are encouraged to review more detailed explanations in Chapters 27 and 37 and to consult online information, as well as other resources to get updated information about this important subject as more is learned and recommendations are developed.

Although there is heightened awareness and great concern about risk of bisphosphonate-related osteonecrosis of the jaw (BRONJ), the causal relationship and pathogenesis of the problem has not been defined. A review of available literature offers information that will help to guide clinicians in their decision-making but it is far from definitive. The prevalence and incidence remains uncertain. In general, the risk of BRONJ is between 1 in 10,000 and 1 in 100,000 but may increase to 1 in 300 after an oral surgical procedure. The great majority of BRONJ cases will likely remain in the intravenous population. Co-factors, such as smoking, steroid use, anemia, hypoxemia, diabetes, infection, and immune deficiency, have not been firmly established but may be important.[56] Rarely does BRONJ in the oral bisphosphonate patient appear to progress beyond stage 2, and many cases reverse with discontinuation of oral medication. Procedures reported to have contributed to the development of BRONJ include extractions, periodontal surgery, root canal treatment, and dental implant surgery.[57] Dental implant therapy, as well as other surgical procedures, should be avoided in individuals who have been treated with intravenous (IV) bisphosphonate therapy and carefully considered with caution in patients treated with oral bisphosphonate therapy, particularly those with a history of more than 3 years of use.[4]

Immune Compromise and Immune Suppression. Individuals undergoing chemotherapy or taking medications that impair healing potential (e.g., steroids) are probably not good candidates for implant therapy because of the effects these agents have on normal healing. This is especially true for cancer chemotherapy. A lowered resistance to infection may also be problematic for these patients. Patients with very low or undetectable viral loads and normal (T cell counts) immune function may be candidates for implant therapy (see Chapter 19). Past history of chemotherapy or immunosuppressive therapy may not be problematic if the patient has recovered from the side effects of treatment.

Patients with an immunocompromising disease, such as human immunodeficiency virus (HIV) infection or acquired immunodeficiency syndrome (AIDS), are probably not good candidates for implants, especially when their immune system is seriously impaired.

Radiation Therapy. Patients with a history of radiation treatment to the head and neck region may not heal well after surgery. Soft tissue dehiscence may follow surgical manipulation, which may lead to osteoradionecrosis (ORN), a serious condition of nonhealing exposure and infection of bone. This is especially problematic for patients who have received radiation dosages greater than 60 Gy. Surgical procedures, or any procedure that may initiate a wound, are generally avoided in patients with a history of radiation therapy. If deemed necessary, surgical procedures can be done in conjunction with hyperbaric oxygen (HBO) therapy to reduce the risk of ORN.

Several studies have documented poor success rates for implants in patients with a history of radiation therapy.[40,41,55] In a literature review, Sennerby and Roos[68] found irradiation to be associated with high failure rates, as did Esposito et al[30] in their review. Beumer et al[14] reported success rates as low as 60.4% in the irradiated maxilla. Granstrom et al[40] reported a significant improvement in survival rates for implants in patients treated with HBO. However, in a systematic review, Coulthard et al[23] concluded that the evidence is lacking to support the clinical effectiveness of HBO in irradiated patients receiving implants. The application of implants in patients with a history of irradiation, with or without the use of HBO, is not resolved and continues to be debated. Clearly, irradiation is a risk factor for implant success and may be a contraindication.

Psychologic and Mental Conditions

In general, any type of psychologic abnormality can be considered a contraindication to dental implant treatment because of the patient's uncooperativeness, lack of understanding, or behavioral problems. Physiologically, there is no reason to suspect that implants could not become osseointegrated in these patients. However, the patient's ability to tolerate the number and type of treatment appointments required for implant placement, restoration, and maintenance could be problematic. All psychologic conditions have the potential to be absolute contraindications to implant treatment depending on the severity of the condition. The exception might be individuals who demonstrate good cooperative behavior with only mild psychologic or mental impairment. The clinician should take great care before accepting a mentally or psychologically impaired individual for treatment with implants.

Habits and Behavioral Considerations

Patients have a variety of habits and behaviors that may increase the risk of failure for dental implants. Smoking, clenching or grinding of teeth, and drug or alcohol abuse are among the most well-known habits that should be identified because of the increased risk for implant failure or complications.

Smoking and Tobacco Use. Moderate to heavy smoking has been documented to result in higher rates of early implant failure and adversely affect the long-term prognosis of dental implant restorations.[7,25,54] This is particularly true for implants placed in poor quality bone such as the posterior maxilla.[48] The mechanisms of action responsible for higher implant failures associated with smoking are not understood. Plausible explanations include the effect of smoking on white blood cells, vasoconstriction, wound healing, and osteoporosis.[5,46] Smoking is a known risk factor for osteoporosis and thus may adversely affect implant success through its effect on bone metabolism. Smoking cessation may

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improve the success rate of implants.[6] In a metaanalytical review, Bain et al[8] found that implants with an altered surface microtopography (Osseotite, acid-etched surface) seemed to significantly lessen the adverse affects of smoking on implant success.

Parafunctional Habits. Parafunctional habits, such as clenching or grinding of teeth (consciously or unconsciously), has been associated with an increased rate of implant failure (e.g., failure to integrate, loss of integration, implant fracture). Repeated lateral forces (i.e., parafunctional habits) applied to implants can be detrimental to the osseointegration process, especially during the early healing period. Patients with known parafunctional habits should be advised of an increased risk of complications or failures as a result of their clenching or grinding. Many consider bruxism to be a contraindication to implant treatment, especially in the case of a short-span, fixed partial denture or a single-tooth implant. If implants are planned for a patient with parafunctional habits, protective measures should be employed, such as creating a narrow occlusal table with flat cusp angles, protected occlusion, and the regular use of occlusal guards (see Chapter 76).

Substance Abuse. Drug and alcohol abuse should be considered a contraindication for implant therapy for reasons similar to the psychologic problems discussed earlier. Patients with drug or alcohol addictions can be irresponsible and noncompliant with treatment recommendations. Depending on the severity and duration of an individual's addiction, some patients may be malnourished or may even have impaired organ function and therefore may not be a good surgical candidate because of poor healing capacity. All elective treatments, including implant therapy, should be refused until addictions are treated and controlled.

POSTTREATMENT EVALUATION

Periodic posttreatment examination of implants, the retained prosthesis, and the condition of the surrounding periimplant tissue is an important part of successful treatment. Aberrations and complications can often be treated if discovered early, but many problems will go unnoticed by the patient. Thus periodic examination is essential to discovering problems early. Several parameters are available to evaluate the condition of the prosthesis, the stability of the implant(s), and the health of surrounding periimplant tissues after implant integration and prosthetic restoration. Many of these clinical measures are adaptations from dental and periodontal examination methods, such as clinical inspection, probing, and radiographic examination.

Clinical Examination

The clinical examination includes visual inspection and probing. Visual evaluation of the tissue color contour and consistency, periimplant probing, and radiographic images are some of the ways to evaluate implants in the posttreatment phase. Soft tissues can be visually inspected for signs of inflammation or swelling. They can also be palpated to detect areas of edema, tenderness, exudate, or suppuration. Periimplant probing can be used to assess the condition and level of hard and soft tissues surrounding implants.

Periimplant Probing. Periodontal probing around natural teeth is very useful to assess the health of periodontal tissues, the sulcus or pocket depth, and the level of attachment. However, using a periodontal probe around implants may not provide comparable results.[17] Clinicians should use caution when evaluating periimplant probing because these measures cannot be interpreted the same as probing depths around teeth. Because of distinct differences in the surrounding tissues that support teeth compared to those that support implants, the probe inserts and penetrates differently. Around teeth, the periodontal probe is resisted by the health of the periodontal tissues and, perhaps most importantly, by the insertion of supracrestal connective tissue fibers into the cementum of the root surface.

These fibers, unique to teeth, are the primary source of resistance to the probe.[3] There is no equivalent fiber attachment around implants. Connective tissue fibers around implants generally run parallel to the implant or restorative surface and do not have perpendicular or inserting fibers (see Chapter 68). The primary source of resistance to the probe around an implant will differ, depending on the conditions surrounding the implant.[27,50] At noninflamed sites, the probe will be resisted by the most coronal aspect of connective tissue adhesion to the implant. At inflamed sites, the probe tip consistently penetrates farther into the connective tissue until less inflamed connective tissue is encountered, which is often close to or at the level of bone.

The value of periimplant probing is different than periodontal probing and offers very limited information by comparison. Probing around implants can measure the level of the mucosal margin relative to a fixed position on the implant or restoration and can also measure the depth of tissue around the implant. The periimplant probing depth is often a measure of the thickness of the surrounding connective tissues and correlates most consistently with the level of surrounding bone. However, periimplant probing is affected by several conditions, including the size of the probe, the force and direction of insertion, the health and resistance of periimplant tissues, the level of bone support, and the features of the implant, abutment, and prosthesis design. In other words, the probe can be an accurate measure of soft tissue thickness around an implant (i.e., periimplant soft tissue above the bone level), but in some cases or sites, it may not be an accurate assessment of soft tissue thickness caused by an inability to properly angle and direct the probe alongside of the implant. In these situations, the area should not be measured or recorded since the as a result will be erroneous. Furthermore, probing around implants is likely to be more variable than around teeth; studies have shown that a change in probing force around implants results in more dramatic changes than a similar change in probing force around teeth.[60] The probing depth around implants presumed to be "healthy" (and without bleeding) has been documented to be about 3 mm around all surfaces.[2,21] The absence of bleeding on probing around teeth has been established as an indicator of health and a predictor of periodontal stability.[49] Studies comparing bleeding on probing around teeth and implants in the same patient have reported that bleeding around implants occurs more frequently. However, the ability to use bleeding as an indicator of assessing diseased versus healthy sites around implants has not been established. Microbiologic studies suggest that greater probing depth or "pockets" around implants harbor higher levels of pathogenic microorganisms.[61,66,67]

Microbial Testing. Studies in animals and humans have demonstrated the development of periimplant mucosal inflammation in response to the accumulation of bacterial plaque.[13,64] Studies have also documented similarities in the microbial composition of plaque in healthy periodontal sites compared with healthy periimplant sites.[59] Likewise, evidence indicates that the microbiota of inflamed periimplant sites (periimplantitis) harbors the same periodontal pathogenic microorganisms as those observed in diseased periodontal pockets.[59,67] However, there is no evidence to prove that

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periodontal pathogens cause periimplant disease, and the pathogenesis of inflammatory disease around implants has not been defined.[22] No convincing evidence indicates that laboratory tests for the identification of suspected periodontal pathogens are of any use in the evaluation of implants.[30] The usefulness of microbial testing may be limited to the evaluation of periimplant sites that are showing signs of infection and bone loss, so the clinician can prescribe appropriate antibiotics.

Stability Measures. The assessment of implant stability (or mobility) is an important measure for determining whether osseointegration is being maintained. Importantly, however, this measure has extremely low sensitivity but high specificity. That is, a large amount of bone loss can occur around an implant, but the implant remains stable (stability measure in this case has a low sensitivity for the detection of an implant that has lost much of its bone support). On the other hand, if significant mobility is detected, the implant has likely failed (mobility is highly specificity for the detection of implant failure). There is great interest in evaluating the stability of the bone-to-implant contact in a non invasive manner. Two techniques that have been used as non invasive ways of evaluating implant stability are impact resistance (e.g., Periotest) and resonance frequency analysis (RFA).

Originally designed to evaluate tooth mobility quantitatively, the Periotest (Gulden, Bensheim, Germany) is a noninvasive, electronic device that provides an objective measurement of the reaction of the periodontium to a defined impact load applied to the tooth crown. The Periotest value depends to some extent on tooth mobility but mainly on the damping characteristics of the periodontium. Despite the dependence on the periodontium, the Periotest has been used to evaluate implant stability as well. However, unlike teeth, the movement of implants and the surrounding bone is minuscule, and therefore the Periotest values fall within a much smaller range compared to the range found with teeth. Detection of horizontal mobility may be a significant advantage for the use of the Periotest because it is much more sensitive to horizontal movement than similar detection by other means, such as manual assessment.[30] Additionally, many variables have been associated with the use of the Periotest related to positioning of the device.

Resonance frequency analysis (RFA) is another noninvasive method used to measure the stability of implants.[58] This method uses a transducer that is attached to the implant or abutment. A steady-state signal is applied to the implant through the transducer, and a response is measured. The RFA value is a function of the stiffness of the implant in the surrounding tissues. The stiffness is influenced by the implant, the interface between the implant and bone, and soft tissues as well as the surrounding bone itself. Additionally, the height of the implant or abutment above the bone will influence the RFA value. Unlike the Periotest, however, the RFA is not dependent on movement in only one direction. Thus the absolute RFA values vary from one implant design to another and from one site to another, but there is high consistency for any one implant or location. The value of RFA is most appreciated with repeated measures of the same implant over time because it is very sensitive to changes in the bone-implant interface. Small changes in tissue support can be detected using RFA. An increase in RFA value indicates increased implant stability, whereas a decrease indicates loss of stability. However, this is a relative measure and it has not been determined whether RFA is capable of detecting impending failure before the implant actually fails. Currently, much interest and research have focused on the use of noninvasive methods to evaluate implant stability. Mobility remains the cardinal sign of implant failure, and detecting mobility is therefore an important parameter.

Radiographic Examination

Intraoral radiographs should be taken at the time of placement (baseline), at the time of abutment connection (confirm seating and serve as another baseline), at the time of final restoration delivery (loading), and subsequently to monitor marginal or periimplant bone changes. Periapical radiographs have excellent resolution and provide adequate details for evaluating bone support around implants if taken at a perpendicular direction. The limitation of periapical radiographs is that they are difficult to standardize, and great variability is inherent in the acquisition process. However, periapical films are relatively simple, inexpensive, and readily available in the dental office.

The objective of the radiographic examination is to measure the height of bone adjacent to the implant(s) and to evaluate the presence and quality of bone along the length of the implant. Finally, the periimplant areas are assessed for any radiolucent lesions around the implant. Although the predictive value of assessing implant stability with radiographs is low, films do offer a reasonable method to measure changes in bone levels.[71] The predictive value of detecting implant failure or loss of stability is good when radiolucent lesions are discovered with periapical radiographs. Radiographic identification of unstable implants is reliable when performed as part of annual examinations and when examining patients on a routine, long-term basis.[42]

The radiographic examination remains one of the primary tools for detection of failed implants in routine clinical evaluation, even though it is not as accurate as mobility tests. In one study designed to evaluate the accuracy and precision of radiographic diagnosis of mobility, the probability of predicting implant mobility in a population with a low prevalence of implant failures was found to be low.[71] Other studies, however, have demonstrated much higher predictive value for radiographic diagnosis of implant mobility.[36,42] The authors concluded that the most important factors for making an accurate radiographic diagnosis are the quality of the radiograph and the experience of the clinician.[42,71]

Oral Hygiene and Implant Maintenance

The long-term success of dental implants likely requires the maintenance of healthy periimplant tissues because the soft tissue "seal" around implants is best when the surrounding mucosa is not inflamed. For this reason, good oral hygiene and regular professional care are essential to maintaining implants. The importance of good oral hygiene should be stressed even before implants are placed, and oral hygiene instructions for plaque control should begin as early as possible. The patient's ability to maintain good oral hygiene should be monitored and reinforced at each visit, and the patient should be given instructions specific to individual needs. Interestingly, despite many years of experience with implants, a recent systematic review revealed that no well-designed studies with a high level of evidence (i.e., randomized controlled trials) have defined the most effective regimens for long-term implant maintenance.[31]

Professionally, several actions can be taken to enhance the patient's ability to perform good oral hygiene. For example, implant superstructures, frameworks, and restorations should be fabricated to accommodate and facilitate oral hygiene (e.g., embrasure spaces should be made to allow the passage of a proxy brush). Specific oral hygiene procedures with appropriate hygiene aids should be demonstrated for each implant area for every patient. Initially, for the first year after treatment is completed, recall maintenance visits

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should be scheduled at 3-month intervals and then adjusted to suit the patient's individual needs. Some patients, with good oral hygiene and minimal deposits, will require infrequent professional hygiene maintenance, whereas others, with poor oral hygiene and heavy deposits, will require more frequent follow-up care.

The use of plastic and gold-coated curettes has been advocated to protect the titanium implant surface and the titanium abutment from contamination by other metals. These curettes were also used to reduce the likelihood of scratching the surface. Unfortunately, plastic curettes do not work very well, and gold-coated curettes cannot be sharpened. Most current implant prostheses are made with gold alloys or ceramic materials, which are usually identical to the materials used in restorations for the natural dentition. Furthermore, the location of the connection between these restorative materials and the implant is typically below the mucosa and often near the crest of bone; most calculus removal will be above this level. Thus the fear of contaminating the titanium implant is unwarranted. The gold alloy or ceramic surfaces can be debrided with most scalers and curettes (plastic, gold coated, stainless steel) without damaging the surface. Rotary instruments (e.g., prophy cup) can be used to remove plaque or biofilms and polish surfaces. The use and sonic instruments (e.g., Cavitron) should be avoided because of irregularities that can easily be created in the surface, which can contribute to plaque and calculus accumulation. One advantage of using screw-retained implant restorations (e.g., super structures, overdenture bars, fixed prosthesis, etc.) is that they can be removed, cleaned outside the mouth, and replaced.

Recall maintenance visits should include an evaluation of soft and hard tissue health, the patient's level of oral hygiene compliance and plaque control, prosthesis integrity and stability, and implant stability. Implant stability can be evaluated with a combination of mobility testing and radiographic assessment.

CONCLUSION

Dental clinicians can now predictably replace missing teeth with endosseous dental implants. Most patients, whether missing a single tooth, several teeth, or all their teeth, can be candidates for dental implant therapy. However, many factors influence the outcome; the clinician must consider the quantity, quality, and location of available bone; the patient's mental and physical health; and risk factors and contraindications. Patients should be advised about risk factors and provided treatment options both with and without dental implants. Periodic evaluation, good oral hygiene, and regular maintenance are important aspects of care for the long-term success and the prevention of complications with dental implants. Radiographic examination and mobility tests appear to be some of the most reliable parameters in the assessment of endosseous dental implants.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

All implant patients need to have a comprehensive evaluation before treatment planning. This includes a thorough medical history and documented detailed clinical examinations. Three-dimensional radiographs should be used for all patients. Edentulous patients can be restored to function with either implant supported fixed restorations or implant supported removable overdentures. If the clinician is concerned with lip support for esthetics then the overdenture is advised. Also, this approach prevents problems of saliva and air passage through fixed restorations that can affect speech and cause uncontrolled spitting of saliva.

Some patients have definite contraindications to implant treatment. These include patients with a history of 60 Gy or more therapeutic radiation exposure, patients who are immunocompromised (e.g., undergoing immunosuppressive therapy, patients with active HIV infections with lowered T cell counts), patients who are unable to maintain adequate levels of oral hygiene (e.g., some physically or mentally compromised patients), or patients with substance abuse who are oral hygiene incompetent. Patients with uncontrolled diabetes should not have implant surgery nor should patients who have undergone intravenous bisphosphonate therapy. Controlled diabetic patients can be treated and may have a small risk of increased implant complications. Patients who have a history of treatment with oral bisphosphonate therapy are at risk for bisphosphonate-related osteonecrosis, particularly if they have 3 or more years of treatment and have associated corticosteroid usage. These patients can be assessed using a blood test for C-terminal cross linked teleopeptide type I collagen (CTX), which measures the ability of bone to heal. Levels of 150 pg/ml or higher are generally not at risk and so can be successfully treated with implant surgery.

Smokers and patients with parafunctional occlusal abnormalities have increased risk of complications and failure following implant surgery. Osteoporotic patients can be successfully treated with implants, but it is wise to lengthen the time for osseointegration before functional loading. HIV-positive patients with undetectable viral loads and normal T cell counts can be treated with implants as well as patients with a history of chemotherapy who currently have normal immunological function.

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CHAPTER 70

Diagnostic Imaging for the Implant Patient

Sotirios Tetradis and Perry R. Klokkevold

CHAPTER OUTLINE

STANDARD PROJECTIONS
- Periapical Radiographs
- Occlusal Radiographs
- Panoramic Radiographs
- Lateral Cephalometric Radiographs

CROSS-SECTIONAL IMAGING
- Conventional X-ray Tomography
- Computed Tomography
- Cone-Beam Computed Tomography

INTERACTIVE "SIMULATION" SOFTWARE PROGRAMS

PATIENT EVALUATION
- Exclude Pathology
- Identify Anatomic Structures
- Assess Bone Quantity, Quality, and Volume
- Evaluate Relation of Alveolar Ridge with Existing Teeth and Desired Implant Position

CLINICAL SELECTION OF DIAGNOSTIC IMAGING
- Clinical Examination
- Screening Films
- Fabrication of Radiographic and Surgical Guides
- Cross-Sectional Tomography
- Intraoperative and Postoperative Radiographic Assessment

CONCLUSION

Several radiographic imaging options are available for diagnosis and treatment planning of patients receiving dental implants.[2,21] Options range from standard projections routinely available in the dental office to more complex radiographic techniques typically available only in radiology centers. Standard projections include intraoral (periapical, occlusal) and extraoral (panoramic, lateral cephalometric) radiographs. More complex imaging techniques include conventional x-ray tomography, computed tomography (CT), and cone-beam CT (CBCT). The CT and CBCT image data files can be reformatted and viewed on a personal computer, using simulation software, making the diagnosis and treatment-planning process interactive and visually more meaningful. Often, combinations of various modalities are used because no single modality can provide all information pertinent to the radiographic evaluation of the implant patient. Familiarity with the benefits and limitations of various techniques and awareness of the specific clinical questions that need to be answered should guide the decision-making process and selection of radiographic examinations for individual patients.

Multiple factors influence the selection of radiographic technique(s) for a particular case, including cost, availability, radiation exposure, and case type. The decision is a balance between these factors and the desire to minimize risk of complications to the patient. Accurately identifying vital anatomic structures and being able to perform implant placement surgery without injury to these structures are critical to treatment success. Diagnostic imaging techniques must always be interpreted in conjunction with a good clinical examination.

This chapter discusses common imaging techniques used for evaluation of the implant patient. Indications for each technique are outlined, along with the advantages and limitations.

STANDARD PROJECTIONS

Standard diagnostic imaging modalities include periapical, panoramic, lateral cephalometric, and occlusal radiographs. The advantages and disadvantages of each modality are summarized in Table 70-1.

Periapical Radiographs

Periapical radiographs offer great advantages during the evaluation of the implant patient.[22,24] They provide an overall assessment of the quantity and quality of the edentulous alveolar ridge and the adjacent teeth. They are easy to obtain in the dental office, are inexpensive, and deliver low radiation to the patient (Table 70-2). Dentists are familiar with the depicted anatomy and possible pathology. Because these direct-exposure projections do not use intensifying screens, intraoral radiographs offer the highest detail and spatial resolution of all radiographic modalities (Figure 70-1). Thus, these films are the projection of choice when subtle pathology, such as a retained root tip, needs to be detected and evaluated.

The most significant disadvantage of periapical radiographs is their susceptibility to unpredictable magnification of anatomic structures, which does not allow reliable measurements.[19] Foreshortening or elongation can be minimized by the use of paralleling

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TABLE 70-1 Advantages and Disadvantages of the Various Radiographic Projections

Modality	Advantages	Disadvantages
Periapical and occlusal radiography	High resolution and detail, easy acquisition, low exposure, inexpensive.	Unpredictable magnification, small imaged area, 2D representation of anatomy.
Panoramic radiography	Easy to acquire, images whole ridge, low exposure, inexpensive.	Unpredictable magnification, 2D representation of anatomy, not detailed.
Lateral cephalometric radiography	Easy to acquire, predictable magnification, low exposure, inexpensive.	Limited use in area of midline, 2D representation of anatomy.
Tomography	3D representation, predictable magnification, sufficient detail, low exposure, images area of interest only.	Requires special equipment, for evaluation of multiple sites can be a lengthy procedure because the patient must be repositioned for each site, expensive.
Computed tomography (CT)	3D representation, predictable magnification, sufficient detail, digital format, images whole arch.	Requires special equipment, expensive, high-exposure dose.
Cone-beam computed tomography (CBCT)	3D representation, predictable magnification, sufficient detail, digital format, images whole arch, low dose.	Requires special equipment, expensive.

2D, Two-dimensional; *3D,* three-dimensional.

TABLE 70-2 Radiation Dose (Effective Dose in μSv) Received from Common Projections during Evaluation of the Implant Patient

Modality	Effective Dose (μSv)
Full-mouth x-ray (FMX) series	35–388
Panoramic	9–26
Conventional tomography	26–187
CT of the head	2000
Large field-of-view CBCT	
NewTom 3G	68
i-CAT: next generation portrait mode	74
Small field-of-view CBCT	
Accuitomo 3DX	20
Galileos default exposure	70

Data from Ludlow JB, Davies-Ludlow LE, White SC: *J Am Dent Assoc* 139:1237, 2008.
CT, Computed tomography; *CBCT,* cone-beam computed tomography.

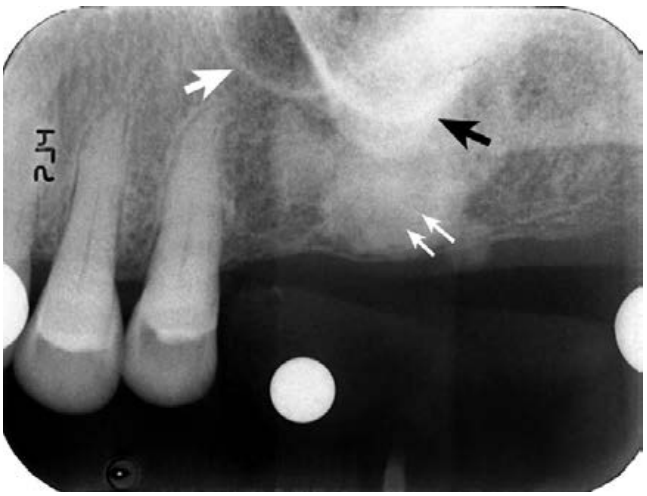

Figure 70-1 The periapical radiograph offers a high-resolution, detailed image of the edentulous area. Healing of the extraction socket with dense bone (socket sclerosis) can be seen *(small white arrows).* Some anatomic structures, such as the maxillary sinus *(large white arrow)* and the zygomatic process of the maxilla *(black arrow),* can also be visualized.

technique. However, distortion is particularly accentuated in edentulous areas, where missing teeth and resorption of the alveolus necessitate film placement at significant angulation in relation to the long axis of the teeth and alveolar bone. Additionally, periapical radiographs are two-dimensional representations of three-dimensional objects and do not provide any information of the buccal-lingual dimension of the alveolar ridge. Structures that are distinctly separated in the buccal-lingual dimension appear to be overlapping. Also, the periapical image is limited by the size of film being used. Often, it is not possible to image the entire height of the remaining alveolar ridge, and when extensive mesial-distal areas need to be evaluated, multiple periapical films are required.

In summary, periapical radiographs are useful screening images that offer a detailed view of a small area of the alveolar arch. Limitations that must be considered include the possibility of distortion and the two-dimensional representation of anatomic structures.

Occlusal Radiographs

Occlusal radiographs are intraoral projections that offer easy, economic, low-dose, and high-resolution images covering a larger area than periapical films.[24] Depending on film placement and the angulation of the x-ray tube, occlusal films can provide a cross-sectional image of the mandible or can depict an extended area of the edentulous ridge. Cross-sectional occlusal films allow the measurement of the buccal-lingual dimension of the mandible. This can be an important consideration when planning implants in the severely resorbed mandible. Occlusal films have the same limitations of distortion and overlapping anatomy as periapical films.

In summary, occlusal film projections are good screening images that can provide an overview of the mandibular width or can visualize larger areas of alveolar ridge compared with periapical film projections.

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Panoramic Radiographs

Panoramic radiographs are often used in the evaluation of the implant patient because they offer several advantages over other modalities.[20] Panoramic films deliver low radiation (see Table 70-2) to provide a broad picture of both arches and thus allow assessment of extensive edentulous areas, angulation of existing teeth and occlusal plane, and important anatomy in implant treatment planning such as the maxillary sinus, nasal cavity, mental foramen, and mandibular canal (Figure 70-2). Panoramic units are widely available and easy to operate, and dentists are familiar with the anatomy and pathology depicted by the images. Similar to intraoral projections, panoramic images are two-dimensional and thus do not offer diagnostic information for the buccal-lingual width of the alveolar arch.

Panoramic images appear intuitively familiar. However, they combine characteristic physical and radiographic principles that make them distinct from other intraoral and extraoral radiographs. Although outside the scope of this chapter, familiarity with the principles underlying panoramic radiography is central in understanding, and thus compensating for, the limitations and constraints of the images. The reader is referred to other textbooks for detailed discussion of this topic.[9,14] Briefly, the existence of ghost shadows, unpredictable horizontal and vertical magnification, distortion of structures outside the focal trough, projection geometry generated by the negative vertical angulation of the x-ray beam, and propensity to patient-positioning errors do not allow consistently detailed and accurate measurements to be generated. As a result, panoramic radiographs do not provide the highly detailed images that are generated by intraoral radiographs.

Measurement distortion is more prevalent and varies across the radiographic image. On average, panoramic radiographs are 25% magnifications of actual size. Implant manufacturers often provide transparency sheets with implant size outlines of 25% magnification. However, it is important to appreciate that the 25% magnification is an estimate. The actual magnification may range from 10% to 30% in different areas within the same film and depends greatly on patient positioning during panoramic radiography. For this reason, precise measurements on panoramic projections are not possible. Nonetheless, panoramic radiographs offer an overall view of the maxilla and mandible that can be used to estimate bone measurements and evaluate the approximate relationships between teeth and other anatomic structures. More precise diagnostic imaging should be used to measure proximity of critical anatomic structures, such as the maxillary sinus or the mandibular canal, to proposed implant positions.

In summary, panoramic projections are useful screening images of the jaws for approximations and relative special relationships. However, because of magnification and distortion errors, panoramic films should not be used for detailed measurements of proposed implant sites.

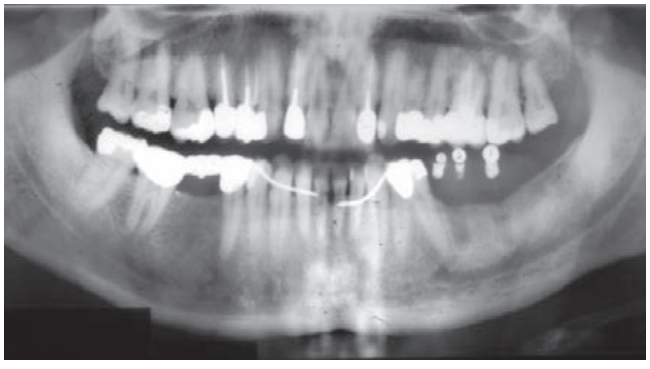

Figure 70-2 Panoramic radiograph. Both jaws are visualized on the same film. An overall assessment of superoinferior and mesial-distal dimensions of the alveolar ridge can be formulated. Tooth and root positions relative to planned implant sites can be evaluated. Important anatomic structures, such as the maxillary sinus and mandibular canal, can be identified.

Lateral Cephalometric Radiographs

Lateral cephalometric radiographs are occasionally used to evaluate potential implant patients.[21] These films provide useful information for the cortical thickness, height, and width of the alveolar ridge at the midline, as well as the skeletal relationship between maxilla and mandible and facial profile. Lateral cephalometric radiographs are low in cost, readily available, and easy to interpret and have a predictable magnification of the depicted structures. However, their use for the implant patient is limited to structures at the midline, with minimal usefulness for other areas of the jaws.

In summary, lateral cephalometric radiographs have a limited use in implant treatment planning.

CROSS-SECTIONAL IMAGING

Cross-sectional diagnostic imaging modalities include conventional x-ray tomography, CT, and CBCT.

Conventional X-ray Tomography

In conventional tomography the x-ray source and the film are connected and rotate around a fixed point (fulcrum), usually performing simple (linear) or complex (elliptic or hypocycloidal) tomographic motions.[4] Structures that are in the plane (focal area) of rotation are depicted in sharp focus, while structures outside the plane of rotation are blurred. The resulting image is a true cross section of the structures within the imaged plane, which is perpendicular to the x-ray beam (Figure 70-3).

Proper patient positioning is essential to generate true cross sections of the jaws.[5,7] The curvature of the alveolar ridge is usually evaluated from scout films, such as submentovertex (SMV) or occlusal projections, or from dental models. The imaged structures are always magnified. However, the magnification is uniform, predictable, and specific to the equipment used for the acquisition of the image. Overlay templates with magnification values that match the magnification of the equipment can be used to measure dimensions on the film reliably.

Conventional x-ray tomography offers many advantages in evaluation of the implant patient.[10,11] With proper patient positioning, it can generate true cross sections of the alveolar ridge and provide diagnostic information of the cortical thickness, trabecular density, height and width of the alveolus, and location of vital anatomic structures. The generated images usually provide adequate resolution.

The specialized equipment used for conventional x-ray tomography requires familiarity with the image acquisition. Interpretation of the images is sometimes challenging, especially when the anatomy of the jaws has been altered because of traumatic extractions, alveolar ridge resorption, or other conditions. Although images are true cross-sections, the progressive blurring of structures outside the focal plane does not allow sharply defined tomographic slices, and prominent opaque structures can cast "ghost" shadows and complicate the images. Another drawback of conventional tomography is that the procedure can be rather lengthy when multiple edentulous areas are evaluated. Finally, the cost of

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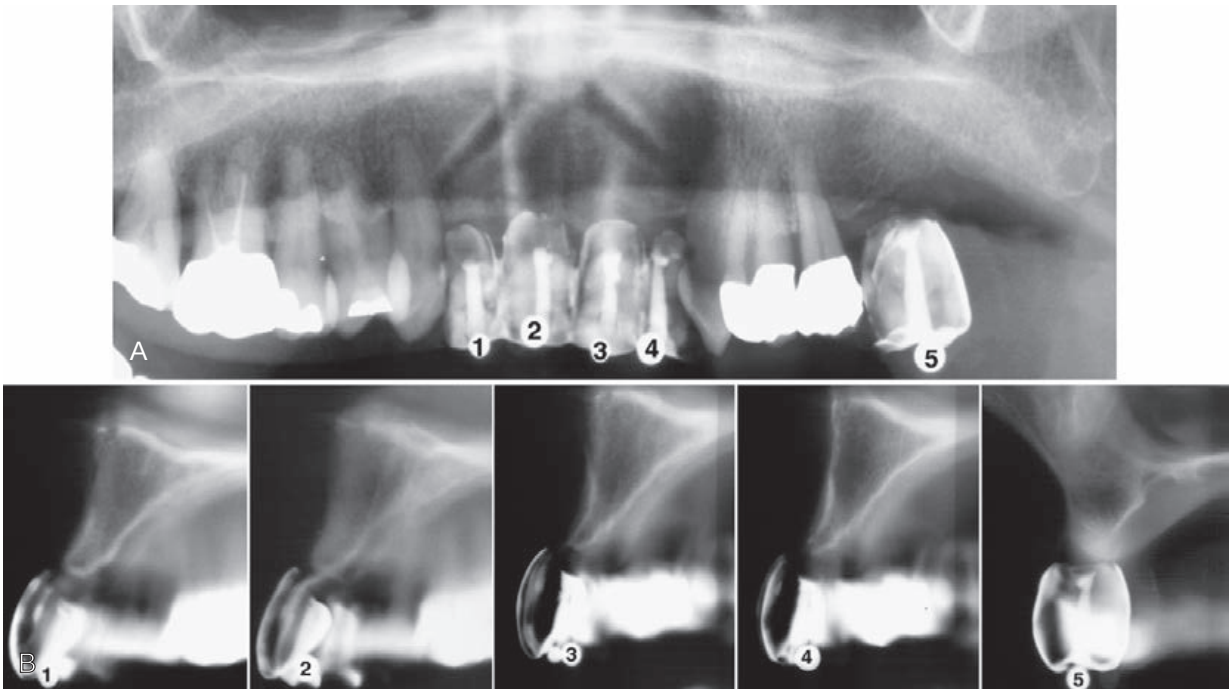

Figure 70-3 **A,** Panoramic radiograph with markers indicating the five prospective implant sites. **B,** Conventional tomography of the same five prospective implant sites in the anterior and posterior maxilla. Note the tooth-shaped markers used that allow the clinician to evaluate bone dimensions in the jaw relative to the planned prosthetic tooth position. The cortical outline of the alveolar ridge and the floor of the nasal cavity or maxillary sinus can be seen. Accurate measurements of the height and buccolingual width can be made. Although conventional x-ray tomographic images focus on a specific area, the image is often difficult to read because of the blurring of structures on either side of the plane.

conventional x-ray tomography is higher than standard intraoral and extraoral radiographs.

In summary, conventional tomography offers true cross-sectional images of the alveolar ridge and is particularly useful during placement of single or few implants. Limitations of conventional tomography include blurring of images by structures on either side of the focal plane and the time required to reposition the patient for each image.

Computed Tomography

CT scanning is widely used in the evaluation of the implant patient.[3,8] The detailed physics behind image acquisition during CT scanning are beyond the scope of this chapter.[4] In general, a thin fan-beam of x-rays rotates around the patient to generate in one revolution a thin (0.5 to 1.0 mm wide) axial slice of the area of interest (Figure 70-4, *A*). Multiple overlapping axial slices are obtained by several revolutions of the x-ray beam until the whole area of interest is covered. These slices are then used to generate, with the help of a computer and sophisticated algorithms, a digital volume of the imaged object. The construction of this three-dimensional digital map of the jaws is the advantage of CT during evaluation of the implant patient. Specialized software can be used to generate appropriate views that best depict the dimensions of the jaws and the location of important anatomic structures.

Typical dental views obtained from a CT scan include axial (Figure 70-4, *B*), panoramic (Figure 70-4, *C*), and cross-sectional (Figure 70-4, *D*) views of the jaws. Appropriate axial slices through the alveolar ridge of interest are selected as scout views. The curvature of the maxillary or mandibular ridge is then drawn on the axial slices, and panoramic images along the drawn line are created. Finally, cross-sectional slices, every 1 to 2 mm and perpendicular to the drawn curvature, are created. In addition to these flat, two-dimensional views, complex, three-dimensional images with surface rendering can also be generated from the CT data (Figure 70-4, *E*). These images can provide useful information about the alveolar ridge defects that are easy to comprehend. Images can be printed on photographic paper or transparent film or can be displayed on the computer screen.

CT scans offer several advantages for evaluation of the implant patient. True cross-sections offer a precise and detailed evaluation of the height and width of the alveolar ridge. The images can be adjusted and printed without magnification, facilitating measurements directly on the prints or films with standard rulers (i.e., not magnified). Vertical and horizontal rulers adjacent to each section allow the clinician to check for magnification and make direct measurements. The digital format allows for image enhancement tools, rapid communication between the radiologist and the surgeon, and generation of multiple copies of the images. Various anatomic structures can be visualized and analyzed at all three coordinate axes, so that their superoinferior, anteroposterior, and buccolingual location can be identified with precision. The process of CT scanning images the entire arch (usually one arch per scan), so several edentulous areas can be visualized with a single examination. The bone and soft tissue contrast and resolution are excellent for the diagnostic task.

CT scanning requires specialized equipment and setting. Radiologists and technicians need to be knowledgeable of the anatomy, anatomic variants, and pathology of the jaws, as well as considerations pertinent to implant treatment planning, so that optimal views will be provided. A CT scan delivers much higher radiation dose to the patient compared with the other modalities used during implant treatment planning[17] (see Table 70-2). Because a CT scan images the whole arch, radiation is delivered to the entire imaged area, regardless of how many or few sites are actually needed.

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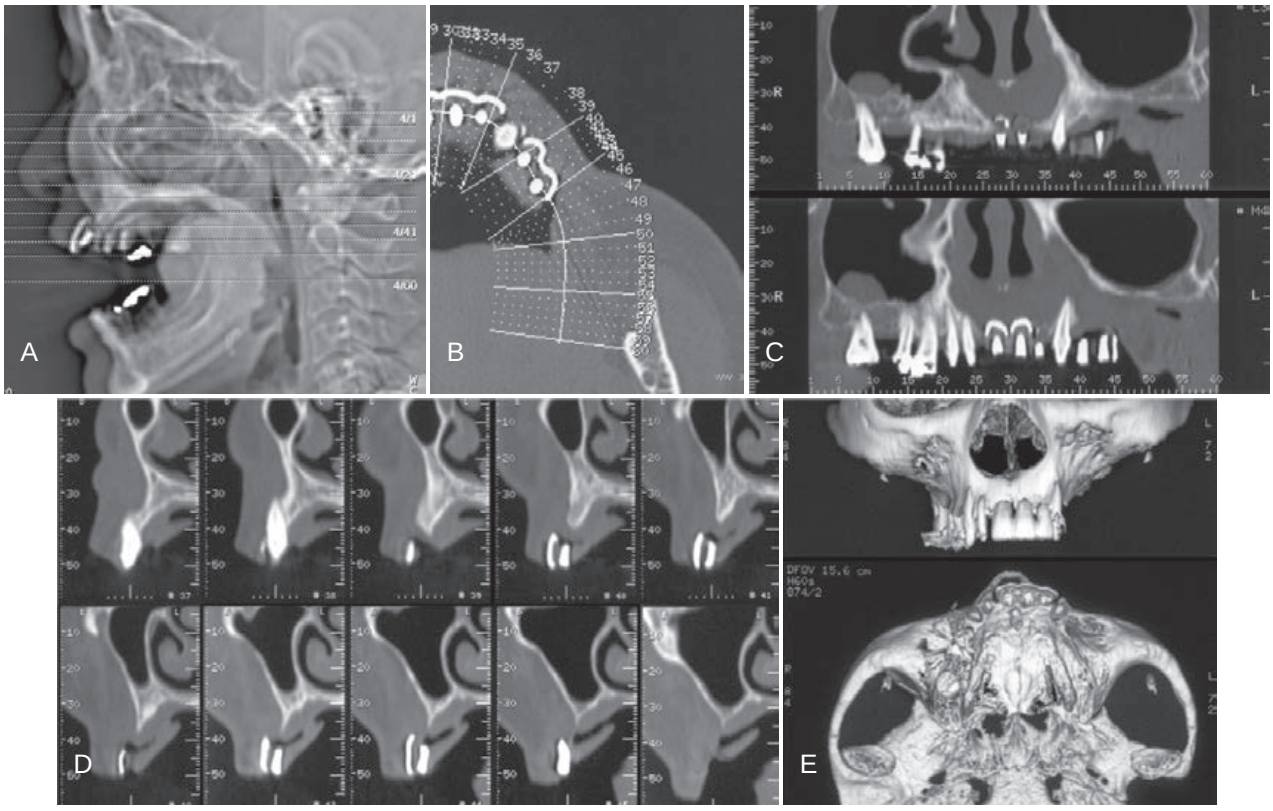

Figure 70-4 Computed tomography (CT) examination for evaluation of edentulous maxilla before implant placement. **A,** Scout view of the patient's head; axial sections through the area of interest are indicated. **B,** Axial slice through the markers is used to display the orientation of the panoramic and cross-sectional images through the alveolar ridge. **C,** Panoramic views through the alveolar ridge demonstrate the relation of the markers to adjacent teeth. **D,** Cross-sectional slices through the area of the markers reveal the height and buccolingual dimension of the alveolar ridge, as well as the relation of the markers to the ridge. **E,** Three-dimensional reconstructions provide an overall impression of the bone contours and shape of the alveolar ridge.

Metallic restorations can cause ring artifacts that impair the diagnostic quality of the images. This is particularly challenging in patients with heavily restored dentition. In general, the cost of CT is significantly higher than that of conventional tomography or the other standard intraoral and extraoral projections.

In summary, CT scanning offers many advantages during implant treatment planning, including accurate cross-sectional imaging and three-dimensional visualization of anatomic structures. High dose to the patient and ring artifacts caused by metallic restorations are concerns that should be considered.

Cone-Beam Computed Tomography

CBCT is a newer imaging modality that offers significant advantages for the evaluation of implant patients.[6,18] CBCT was introduced to dentistry in the late 1990s,[1,16] and currently several CBCT units are commercially available for imaging of the craniofacial complex. Similar to CT scanners, the x-ray source and the detector are diametrically positioned and make a 180 to 360 degree rotation around the patient's head within the gantry. In contrast to the fan-beam generated by CT scanners, however, the CBCT scanner generates a cone-shaped x-ray beam, which images a larger area. Thus, at the end of a single complete rotation, 180 to 500 images of the area are generated. The computer uses these images to generate a digital, three-dimensional map of the face. Once this map is generated, multiplanar reconstructions, as well as axial, coronal, sagittal, or oblique sections of various thicknesses, can be reconstructed from the data, similar to the CT images.

An important feature of the various CBCT units is the field of view (FOV) describing the extent of the imaged volume. CBCT units can be distinguished in large-FOV (6 to 12 inches or 15 to 30.5 cm) and limited-FOV systems (1.6 to 3.1 inches or 4 to 8 cm). In general, large FOV units image a more extensive anatomic area, deliver a higher radiation exposure to the patient, and produce lower resolution images (Figure 70-5). Conversely, limited FOV units image a small area of the face, delivering less radiation and producing a higher resolution image (Figure 70-6).

In general, CBCT offers the same advantages and disadvantages as CT. However, the two modalities have basic differences that result from the different physical principles used during image acquisition. CT scan offers a greater *contrast resolution,* or the ability to distinguish two objects with small density differences. CBCT scans have a limited capacity to separate muscle from fat or connective tissue compared with CT scans. Fortunately, contrast resolution is not a significant concern in implant evaluation. Because bone has a much higher density than surrounding soft tissues, both CBCT and CT can clearly depict bone shape and architecture. One of the most significant advantages of CBCT scanning versus CT scanning is the reduced amount of radiation dose delivered to the patient[13,23] (see Table 70-2).

In summary, CBCT scanning is a valuable imaging modality for three-dimensional and cross-sectional evaluation of the implant patient. It has similar advantages and disadvantages as CT scanning. The most significant difference is that CBCT imaging delivers much less radiation exposure to the patient.

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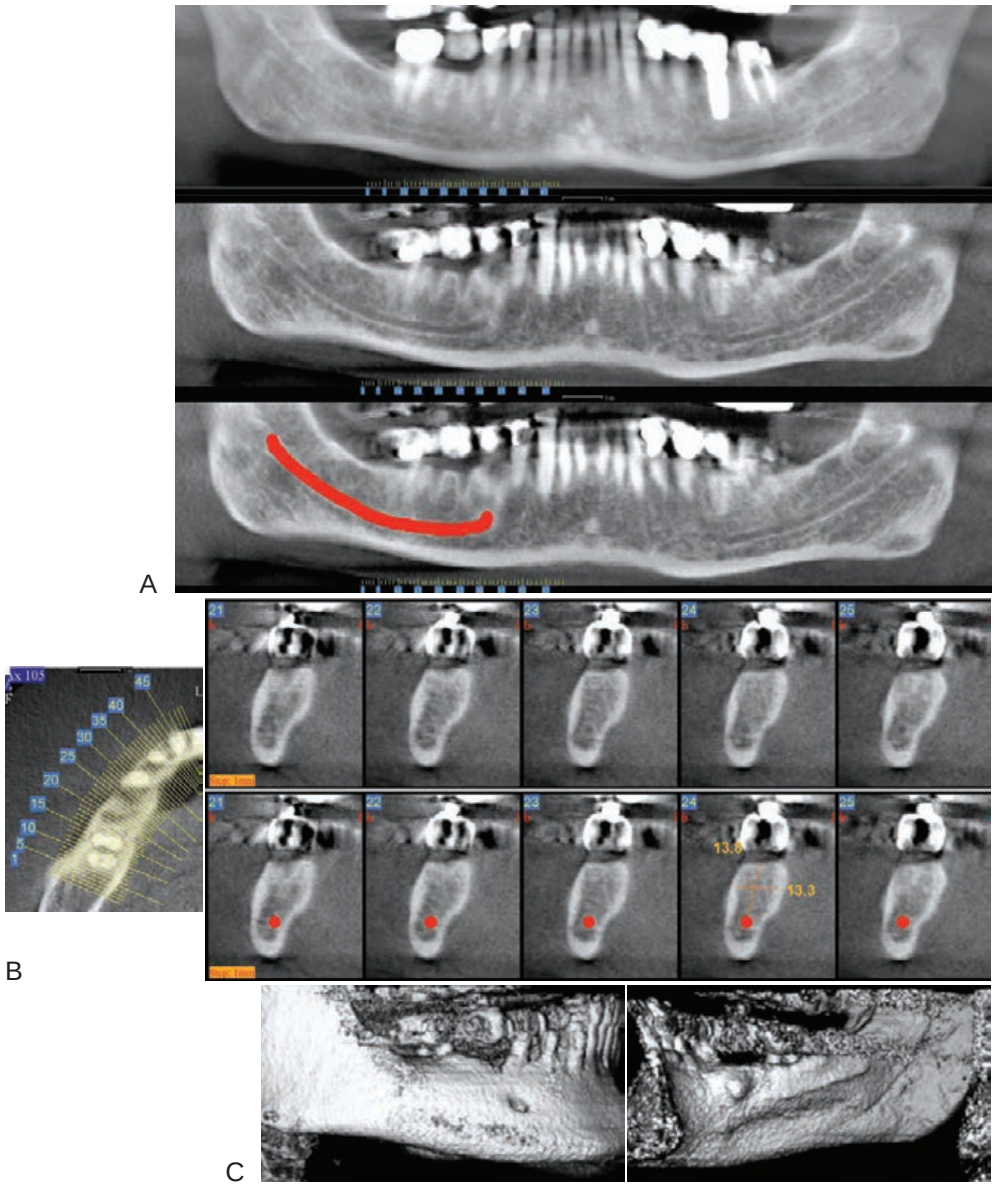

Figure 70-5 Cone-beam computed tomography (CBCT) images for the evaluation of the edentulous space at the area of missing tooth #30 before implant placement utilizing a large field-of-view FOV unit (NewTom 3G, Verona, Italy distributed by AFP Imaging, Elmsford, NY). Note the tooth-shaped marker used. **A,** Series of "panoramic" reconstructions through the alveolar ridge reveals the relationship of the marker to the adjacent teeth. The top "panoramic" view is 12 mm thick to depict most of the extent of the alveolar ridge and adjacent teeth. The middle "panoramic" image is 1 mm thick through the area of the mandibular canal. Note that adjacent teeth are out of the plane of the section and thus not depicted on the image. The bottom "panoramic" view is the same as the middle one, but the position of the mandibular canal has been depicted by the red line. **B,** Scout axial view and series of cross-sections through the area of the marker. The bottom row shows the same axial slices as the top row. However, the position of the red line drawn on the panoramic view is also depicted to help localization of the mandibular canal. The height and width of the alveolar ridge have been measured in a selected section. **C,** Three-dimensional reconstructions provide an overall impression of the bone contours and shape of the alveolar ridge. Note the small exostosis on the lingual surface of the alveolar ridge.

INTERACTIVE "SIMULATION" SOFTWARE PROGRAMS

In many challenging cases, implant treatment planning can be greatly enhanced by the use of specialized software. These programs utilize data from the CT or CBCT scans and allow the simulation of implant placement and restoration on the computer. The quantity and quality of bone can be evaluated. A database of popular implant (commercial size and design) images is usually available to use with the three-dimensional images. The length, width, angulation, and position of implants can be "simulated" in the desired positions. In cases of alveolar ridge deficiency or defects or when sinus bone augmentation is indicated, the additional bone volume needed can be evaluated and quantified. The restoration of the implants can also be simulated and the distribution of mechanical forces onto the implant and adjacent bone predicted.

Software programs specialized in implant treatment planning, such as SIM/Plant (Materialise/Columbia Scientific, Glen Burnie, MD), can reformat CBCT or CT scan data. The clinician can use the reformatted images on a personal computer in an interactive manner to identify anatomic structures, simulate implant placement positions and better appreciate relationships between

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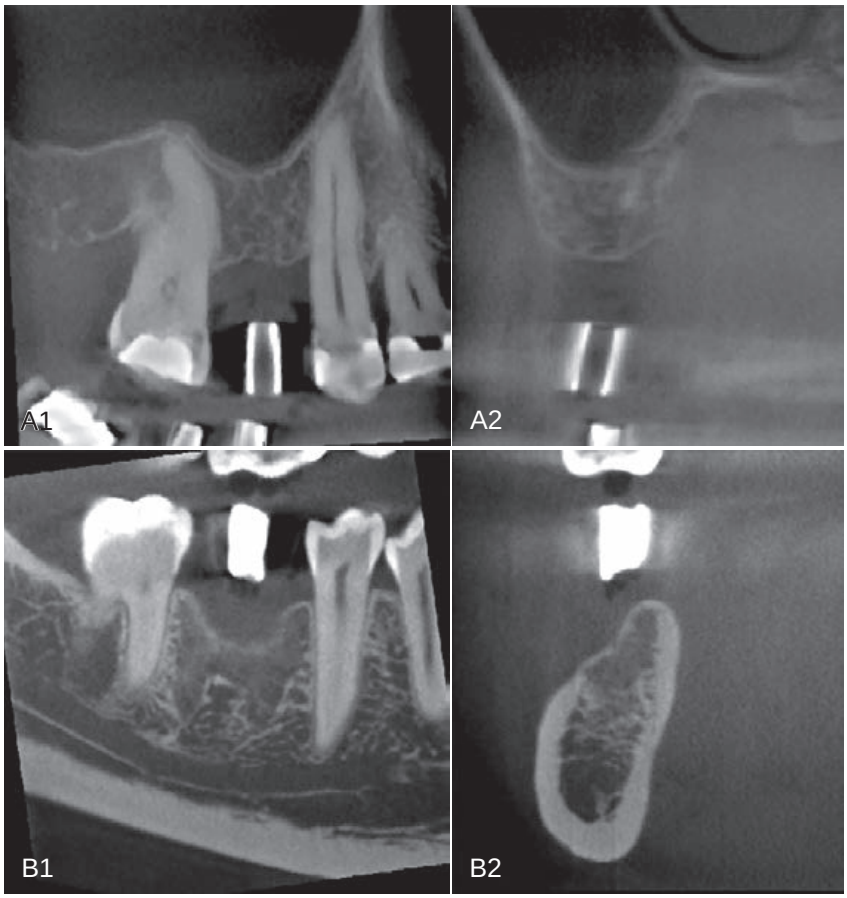

Figure 70-6 Cone-beam computed tomography (CBCT) images for the evaluation of the edentulous space at the area of missing tooth #4 **(A)** and missing tooth #30 **(B)** utilizing a small field-of-view (FOV) unit (Accuitomo 3D x-ray, J. Morita Corporation, Suita City, Osaka, Japan, distributed by J. Morita USA, Inc., Irvine, CA). Sagittal and cross-sectional slices are shown. Although the anatomic area imaged is limited, the resolution of the images are high. **A,** Implant site #4 (maxillary right second premolar) in anterior-posterior and buccal-lingual cross-sectional views. **B,** Implant site #30 (mandibular right first molar) in anterior-posterior and buccolingual cross-sectional views.

planned implant positions and teeth or anatomic structures (Figure 70-7).

PATIENT EVALUATION

Evaluation of the implant patient should be disciplined and objective. Specific questions that can affect implant placement and outcome should be considered and examined carefully and explicitly. The advantages and disadvantages of various radiographic projections should be considered and radiographic modalities chosen based on necessary information for the particular patient. The objectives for any radiographic evaluation, regardless of imaging technique used, should include an evaluation to (1) exclude pathology, (2) identify anatomic structures, and (3) measure the quantity, quality, and location of available bone.

Exclude Pathology

Healthy bone is prerequisite for successful osseointegration and long-term implant success. The first step in the radiographic evaluation of the implant site is to establish the health of the alveolar bone and other tissues imaged within a particular projection. Local and systemic diseases that affect bone homeostasis can preclude, modify, or alter placement of implants. Retained root fragments, residual periodontal disease, cysts, and tumors should be identified and resolved before implant placement. Systemic diseases, such as osteoporosis and hyperparathyroidism, alter bone metabolism and might affect implant osseointegration. Areas of poor bone quality should be identified and if indicated, adjustments to the treatment plan incorporated. Maxillary sinusitis, polyps, or other sinus pathology should be diagnosed and treated when implants are considered in the posterior maxilla, especially if sinus bone augmentation procedures are planned (Figure 70-8).

BOX 70-1 Anatomic Structures Pertinent to Treatment Planning of the Implant Patient

Maxilla
Maxillary sinus (floor and anterior wall)
Nasal cavity (floor and lateral wall)
Incisive foramen
Canine fossa

Mandible
Mandibular canal
Anterior loop of the mandibular canal
Anterior extension of the mandibular canal
Mental foramen
Submandibular fossa
Lingual inclination of the alveolar ridge

Identify Anatomic Structures

Several important anatomic structures are found close to desired areas of implant placement in the maxilla and mandible (Box 70-1). Familiarity with the radiographic appearance of these structures is important during treatment planning and implant placement. Their exact localization is central to prevent unwanted complication and unnecessary morbidity. Important anatomic structures in the maxilla include the floor and anterior wall of the maxillary sinus,

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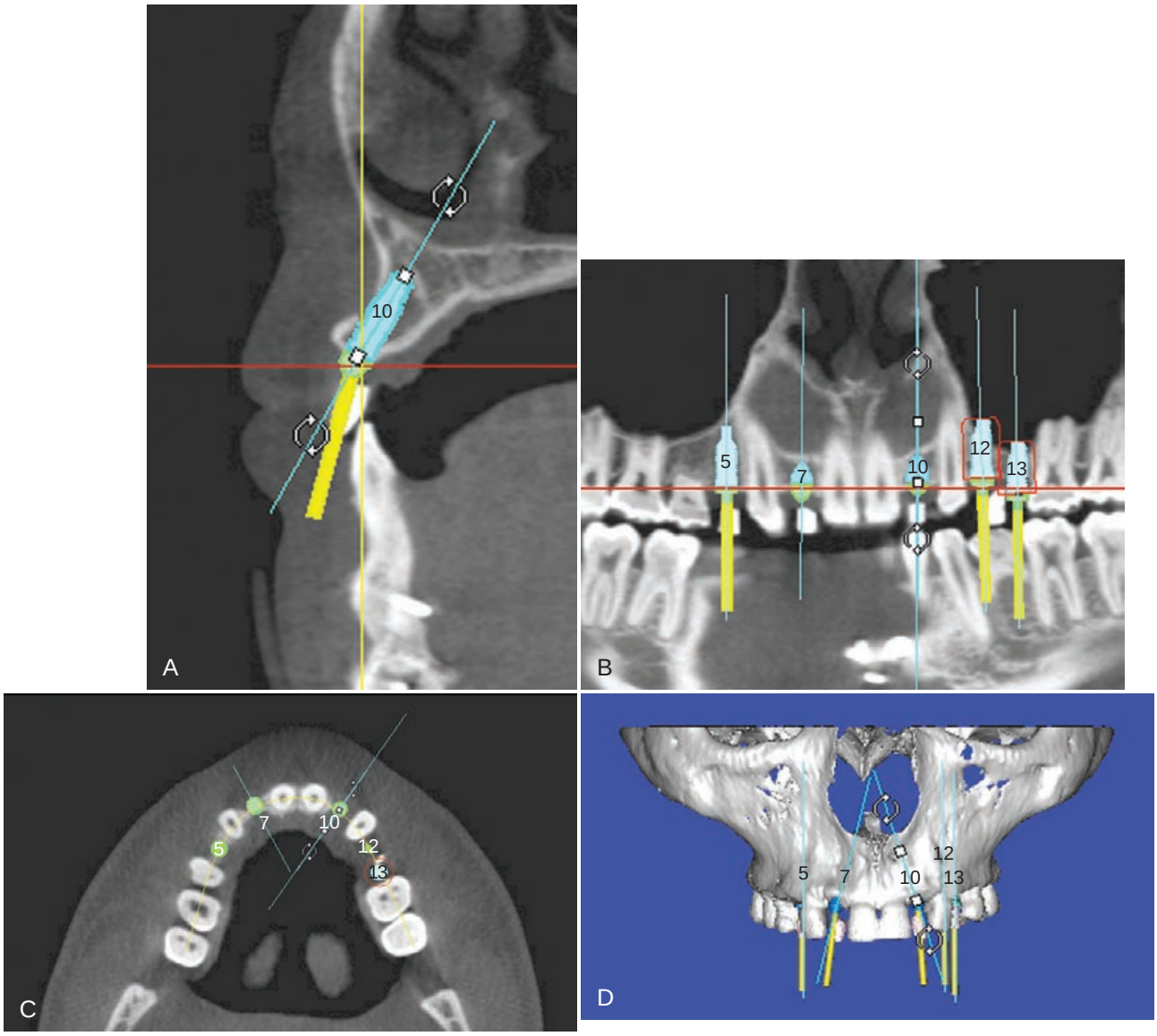

Figure 70-7 SIM/Plant images. The SIM/Plant software program allows clinicians to measure bone height, width, density, and volume on a personal computer. Scan data are reformatted for interactive evaluation and manipulation. Implant positions can be simulated on the patient's scan data before surgery, allowing the surgeon to anticipate areas of deficiency.

incisive foramen, floor and lateral wall of the nasal cavity, and canine fossa. Important anatomic structures in the mandible that should be recognized include the mandibular canal, anterior loop of the mandibular canal, mental foramen, anterior extension of the canal, and submandibular fossa. The existence of anatomic variants, such as incomplete healing of an extraction site, sinus loculation, division of mandibular canal (Figure 70-9), or absence of a well-defined corticated canal, should also be recognized.

Assess Bone Quantity, Quality, and Volume

The primary goal of diagnostic imaging for potential implant patients is to evaluate the available bone volume for implant placement in desired anatomic locations. The clinician wants to estimate and verify exact adequate height, width, and density to the recipient bone while avoiding damage to critical anatomic structures. Failure to assess accurately the location of important anatomic structures can lead to unnecessary complications. For example, inadvertent penetration and damage to the inferior alveolar nerve can result in serious immediate-term (profuse bleeding), short-term, and long-term (nerve paresthesia/anesthesia) complications. The height and width of the alveolar bone should be accurately detailed. depending on the technique, diagnostic imaging can estimate or measure the coronal-apical height, the buccal-lingual width, and the mesial-distal spacing available for implants that will be placed in proximity to teeth or relative to other planned implants.

This task can be simple in cases with good bone quality and sufficient bone volume in the desired implant location(s). However, in cases with moderate-severe bone resorption, alveolar defects, or recent extraction sites, obtaining a clear and accurate diagnostic image can be more challenging. The diagnostic imaging may reveal inadequate bone volume for the proposed implant(s) and indicate a need for bone augmentation or depending on the severity of the deficiency, preclude the patient from the possibility of implant therapy (Figure 70-10).

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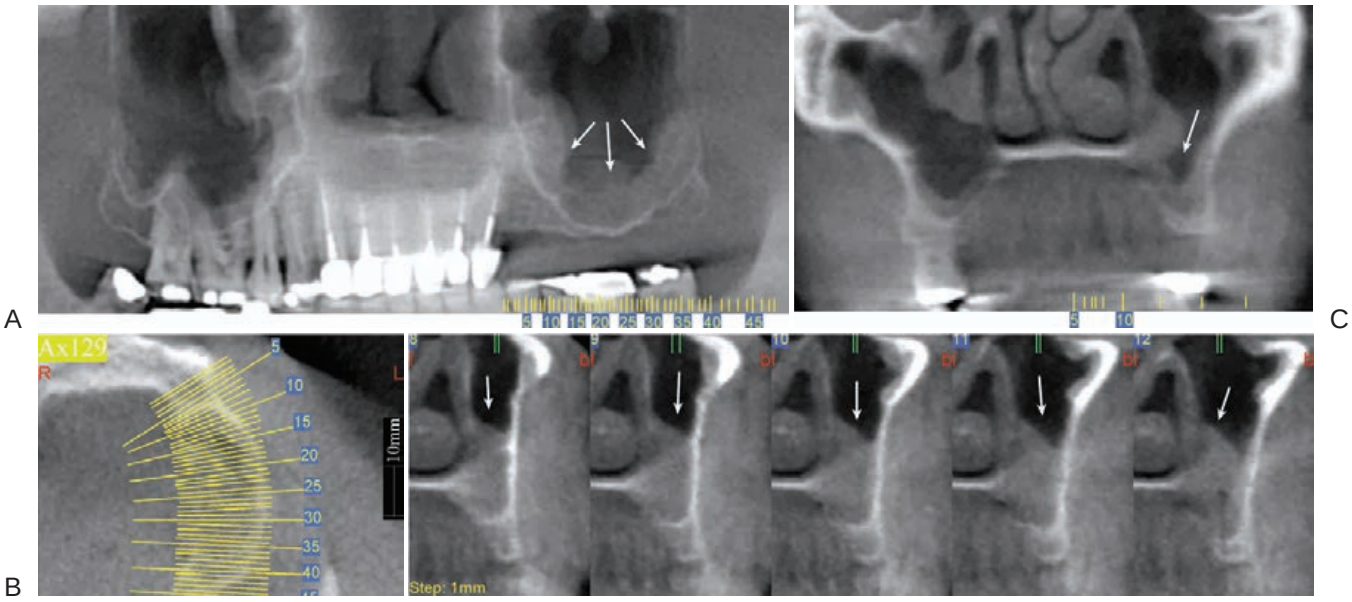

Figure 70-8 Cone-beam computed tomography (CBCT) examination of the posterior left maxilla. "Panoramic" **(A),** coronal **(B),** and axial and cross-sectional **(C)** views of the alveolar ridge. Note the thickened mucoperiosteal lining of the floor of the left maxillary sinus *(white arrow)*. The patient has chronic maxillary sinusitis.

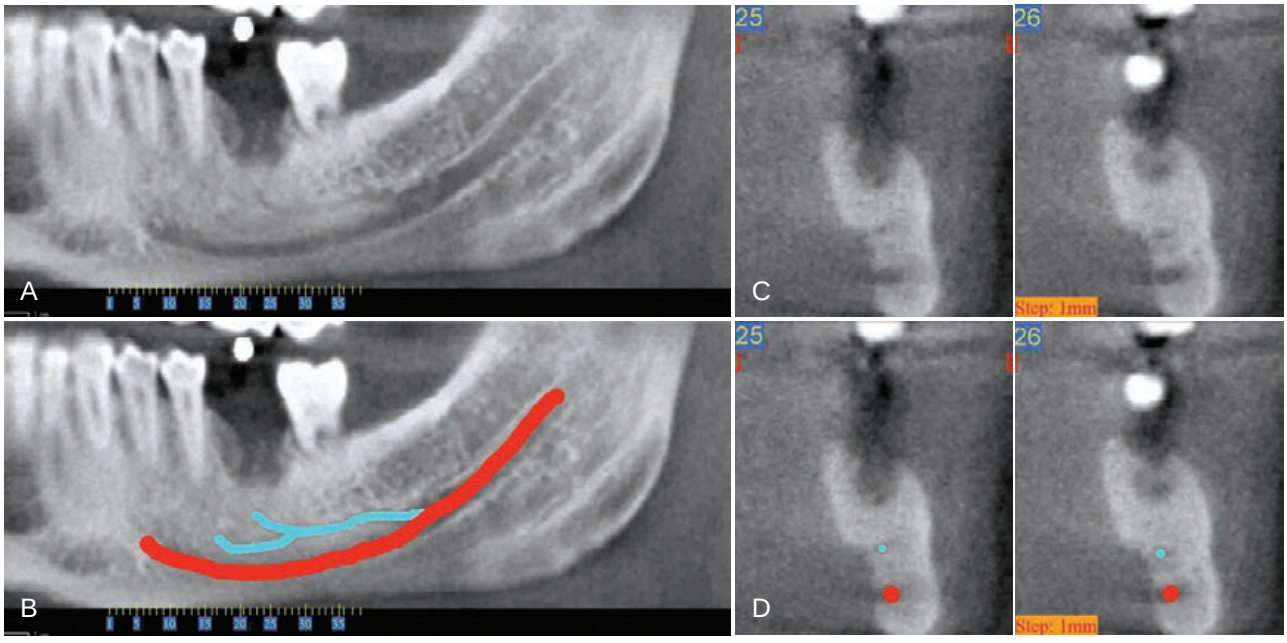

Figure 70-9 Cone-beam computed tomography (CBCT) examination of the area of missing tooth #19 before implant placement. **A,** Panoramic view of the area of interest depicts an accessory mandibular canal. **B,** Same panoramic view with the accessory mandibular canal colored blue and the main canal red. **C,** Cross-sectional views through the area of missing tooth #19. **D,** Same cross-sectional images depicting the blue and red markings. Note that the position of the markings coincides with the position of the accessory and main mandibular canals (compare **C** and **D**).

In addition to the amount, the quality of the available bone should also be evaluated. A uniform, continuous cortical outline and a lacy, well-defined trabecular core reflect the normal bone homeostasis necessary for appropriate bone response around the implant. Thin or discontinuous cortex, sparse trabeculation, large marrow spaces, and altered trabecular architecture should be noted because they might predict poor implant stabilization and less desirable response of the bone. Poor bone quality may necessitate modifications of the treatment planning, such as waiting longer for healing (osseointegration) to maximize bone-to-implant contact before loading.

Evaluate Relation of Alveolar Ridge with Existing Teeth and Desired Implant Position

Accurate placement (spatial position and angulation relative to adjacent teeth and occlusal plane) will greatly affect the restorative success and long-term prognosis of the implant (see Chapter 76). A significant variable during the preimplant evaluation is the

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relation of the desired implant position relative to the existing teeth, alveolar crest, and occlusal plane. Angled or custom abutments can accommodate slight variations in implant position and implant inclination. However, more significant deviations should be avoided.

Prolonged tooth loss is usually associated with atrophy of the alveolar ridge and in the case of the maxilla, with pneumatization of the sinus floor toward the alveolar crest. Traumatic extractions can compromise the buccal or lingual cortex and alter the shape and buccolingual ridge dimension. Anatomic variants, such as lingual inclination of the alveolus or narrow ridges, should be considered during treatment planning of the implant patient (Figure 70-11).

An important part of diagnostic imaging must include an evaluation of the available bone relative to the "prosthetically driven" implant position. This aspect of the patient evaluation is best accomplished with diagnostic models, wax-up of planned tooth replacement, and radiographic markers in the desired tooth positions during imaging. Steel balls, brass tubes, and gutta percha have all been used to establish the proposed tooth positions relative to the existing alveolar bone. The use of these nonanatomic markers is helpful for evaluating bone height and width in specific anatomic locations. However, they do not accurately represent the tooth contours and do not allow the clinician to estimate variations in implant position and angulation relative to the position and emergence of the planned tooth replacement. Therefore, it is more desirable and beneficial to use radiopaque "tooth-shaped" markers so that the existing alveolar bone can be evaluated relative to the entire tooth position/contours (Figure 70-12; see also Figures 70-3 and 70-5). This is particularly important for anterior, esthetic implant cases. Patients should always be imaged with radiographic guides (markers).

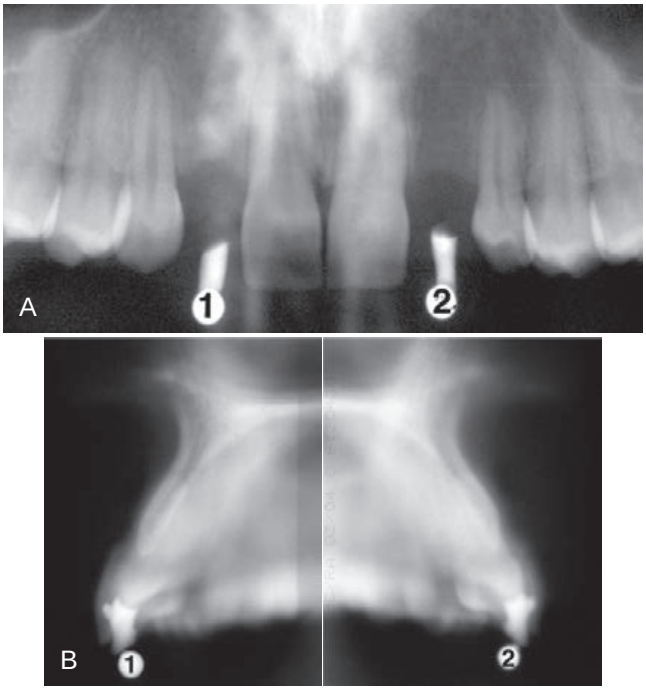

Figure 70-10 Radiographic evaluation of a patient with congenitally missing maxillary lateral incisors before implant placement. **A,** Panoramic radiograph reveals sufficient height and mesial-lateral width of the alveolar ridge. **B,** Cross-sectional conventional tomography of the edentulous areas reveals a narrow (<4 mm) buccal-lingual width of the alveolar ridge that needs to be addressed by modifications in the treatment planning, such as bone augmentation.

CLINICAL SELECTION OF DIAGNOSTIC IMAGING

Radiography is an important diagnostic tool for the evaluation of the implant patient. However, radiographic imaging alone is not sufficient. It is important to correlate diagnostic information with a good clinical examination. Conversely, a clinical examination is insufficient to provide the information needed to plan implant treatment for a patient without some radiographic imaging.

Clinical Examination

Before taking any radiographs, a complete clinical examination of the implant patient is required. This should include the etiology and duration of tooth loss, any history of traumatic extraction, and a review of records and radiographs, if available. Clinical assessment of the edentulous area, covering mucosa, adjacent and opposing teeth, and occlusal plane should be performed. Temporomandibular function, mandibular maximal opening, and protrusive and lateral movements should be evaluated (see Chapter 69).

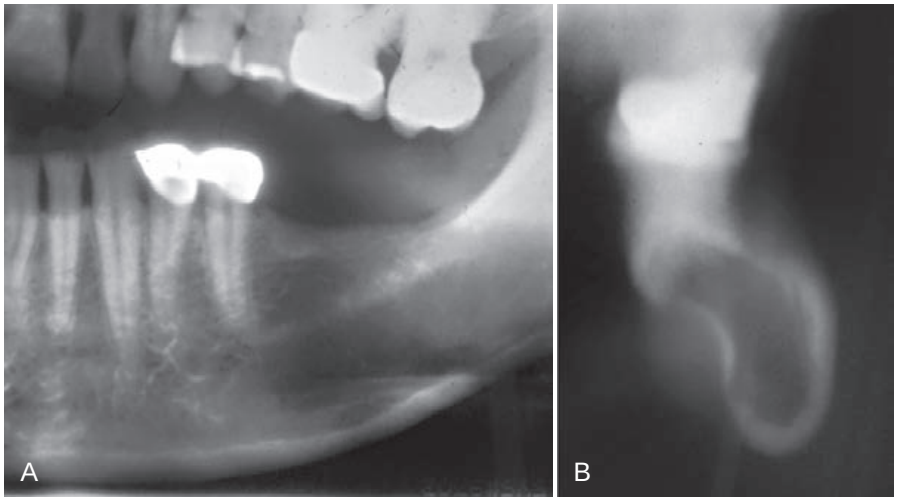

Figure 70-11 Radiographic evaluation of a patient with edentulous posterior left mandible before implant placement. **A,** Panoramic radiograph demonstrates sufficient height of the alveolar ridge with little or no resorption. **B,** Cross-sectional conventional tomography reveals significant lingual inclination of the alveolar ridge with lingual concavity that is not depicted on the panoramic radiograph.

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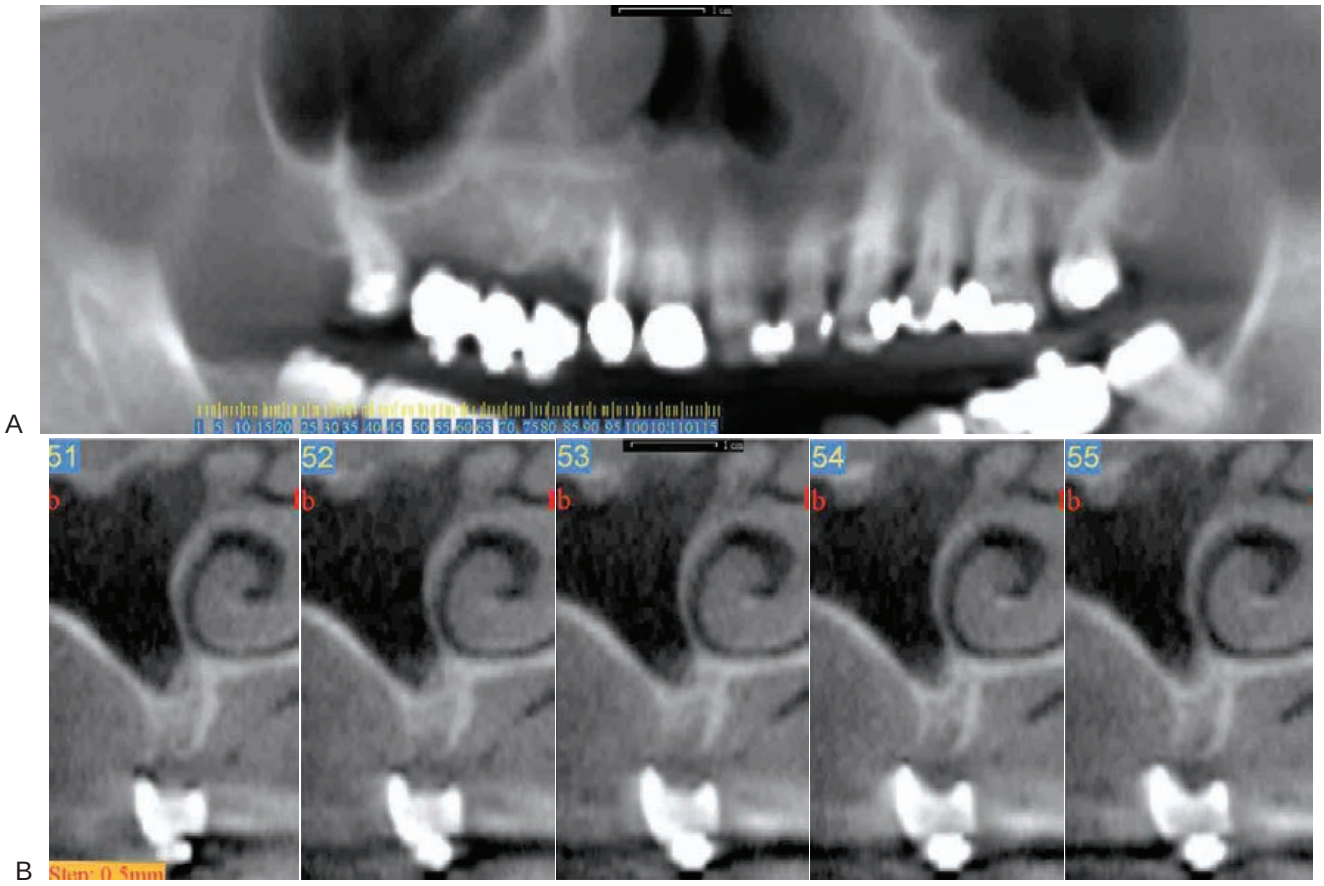

Figure 70-12 **A,** Panoramic view of partially edentulous maxilla with tooth shaped markers in areas of missing teeth (potential implant sites). **B,** Cross-sectional views from a cone-beam computed tomography (CBCT) examination before implant placement in the right maxilla. Appropriately sized and shaped tooth markers placed in the prosthetically desired locations of the planned restorations for the missing teeth help evaluate the existing alveolar ridge relative to the prospective tooth positions and contours.

Screening Films

At this point, an overall assessment of the health of the jaws should be performed. Periapical films provide a high-resolution image of the alveolus and the surrounding structures, including adjacent teeth. For extended edentulous areas, panoramic, lateral cephalometric, and occlusal films can be used to estimate bone height and width. Any pathology of the bone at the prospective implant site, as well as of the surrounding structures, should be identified and treated as indicated.

Fabrication of Radiographic and Surgical Guides

Once the health of the soft and hard tissues is established, casts should be taken and detailed analysis performed. The clinician should decide on the number of implants and their desired location. Next, a radiographic guide should be fabricated, usually with clear acrylic. The position of the desired implants is indicated by the use of radiopaque objects such as metallic balls, cylinders, or rods; gutta percha; or composite resin. If CT imaging might be performed, the use of metallic markers should be avoided. The design of such a guide greatly enhances the diagnostic information provided by the radiographs because it correlates the radiographic anatomy with the exact position of the proposed implant location.

Cross-Sectional Tomography

Some type of cross-sectional imaging, such as conventional tomography, CT, or CBCT, should be performed before implant placement in any site of the jaws.[21] Plain (two-dimensional) films might be sufficient in select implant cases. For example, a single anterior maxillary site with good interdental space and bone volume and no significant anatomic structures at risk might not require cross-sectional imaging. On the other hand, the potential morbidity of a compromised anatomic structure and the poor performance and potential failure of a misplaced implant, combined with the wide availability of tomographic facilities, favor the use of cross-sectional imaging in most cases of implant treatment planning. It is crucial that the cross sections are perpendicular to the curvature of the mandible and parallel to the planned implant. Improper patient positioning can lead to an overestimation of the height and width of the available bone. If the surgeon believes that sections were performed at the wrong angulation, new images should be requested. This might necessitate reexposure of the patient.

Intraoperative and Postoperative Radiographic Assessment

Various radiographic modalities can provide valuable information during implant placement. Because of the ease of acquisition and high resolution, periapical radiographs are most commonly used.

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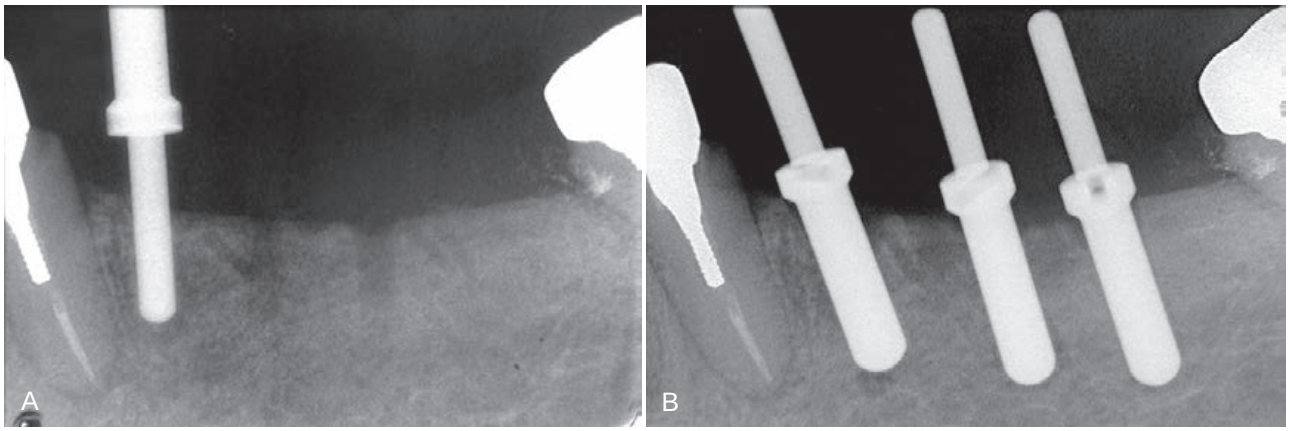

Figure 70-13 Intraoperative periapical radiographs are valuable in assessing the proximity of adjacent teeth. **A,** The 2-mm guide pin is used to determine direction of the osteotomy site and its proximity to the adjacent root. **B,** After angle correction, the osteotomy sites are completed to length with the final drill. Here the 3-mm guide pins confirm the correct angulation and spacing of the final osteotomy site preparation before implant placement.

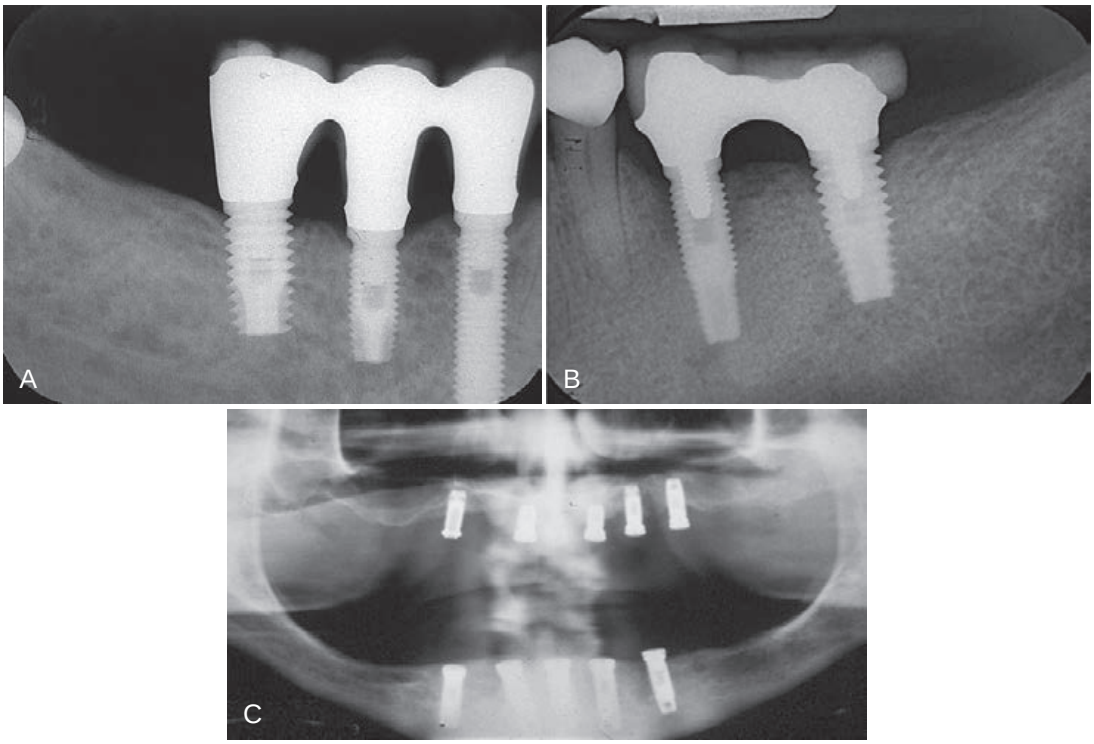

Figure 70-14 Radiographic follow-up after implant placement in three different patients. **A,** Periapical radiograph of three implants in the posterior right mandible. "Normal" bone remodeling around anterior two implants and slight horizontal bone loss/remodeling around the molar/posterior implant is present. **B,** Periapical radiograph of two implants in the left posterior mandible. Severe bone loss (50% of implant length) is seen around anterior implant, while mild bone loss/bone remodeling is observed around posterior implant. A moderate buccal cantilever in the restoration likely contributed to an adverse occlusal load and the resultant bone loss observed in this case. **C,** Panoramic radiograph of maxillary and mandibular implants in an edentulous patient prior to implant loading. The mandibular implants do not show signs of bone loss and appear to be osseointegrated. All maxillary show signs of moderate-to-severe periimplant bone loss and the success of osseointegration is questionable.

Intraoperative radiographs can be taken during surgery to evaluate proximity to important anatomic structures. Sequential periapical films guide the clinician to visualize changes in direction and depth of the drilling procedure and parallelism to adjacent teeth and other implants (Figure 70-13). Digital radiographs are particularly advantageous during intraoperative assessment of implant placement; images appear on the screen almost instantaneously and can be manipulated to extract the most pertinent diagnostic information (see Chapter 31).

Implant osseointegration and the level of periimplant alveolar bone are major determinants of implant prognosis. Panoramic and periapical radiographs offer a fast, easy, and low-radiation depiction of the implant and surrounding tissues and aid in assessment of implant success. To obtain an accurate assessment of periimplant bone height, the x-ray beam should be directed perpendicular to the implant. In the case of threaded implants, the implant threads should be distinguishable and not overlapping (Figure 70-14, *A*).

A 1.2-mm marginal bone loss during the first year after implant placement, and 0.1 mm per year afterwards is expected, while further bone loss is considered abnormal.[2] Pathologic bone loss could be localized along the full extend of the implant

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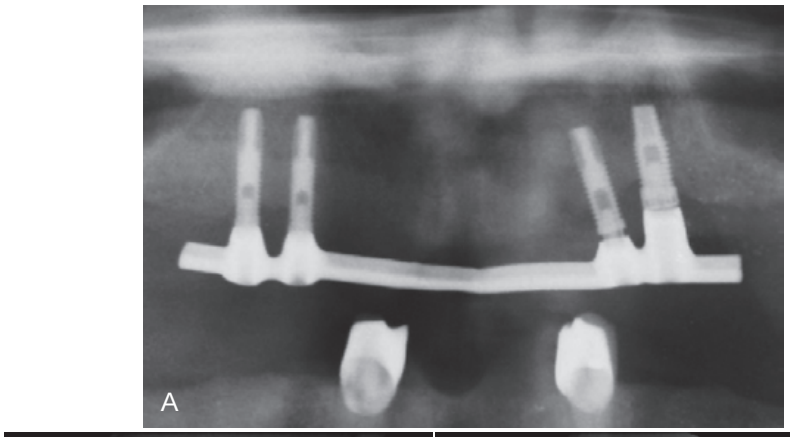

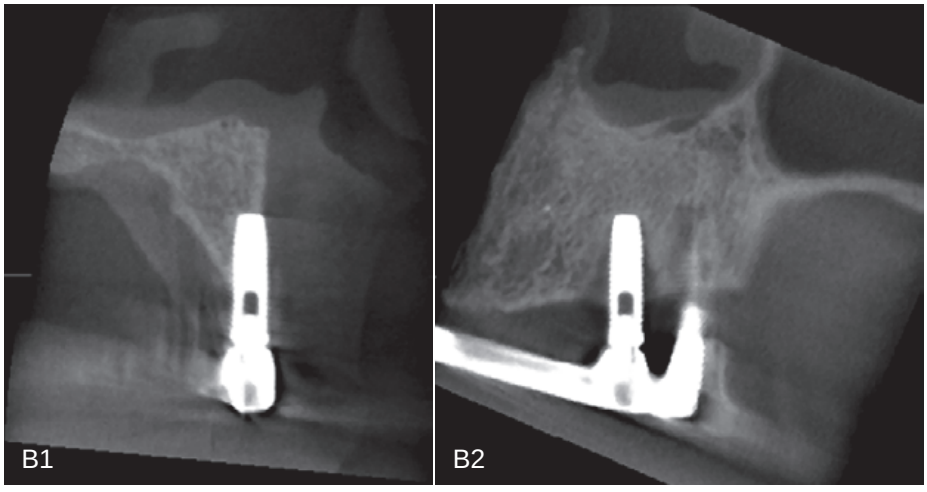

Figure 70-15 Radiographic follow-up after implant placement. **A,** Panoramic radiograph suggests mild-to-moderate bone loss around the neck of all implants. This is especially true for the implants in the left maxilla. These implants appear to be angled and the distal implant is positioned more apical. The overdenture bar is not completely seated on the left implants. Note superimposed overlapping anatomic structures in this panoramic radiograph impair the ability to clearly visualize and assess bone loss around implants. **B,** Cross-sectional and sagittal cone-beam computed tomography (CBCT) images of the anterior implant in the left maxilla. Poor implant placement beyond the buccal cortex of the alveolar ridge (cross-section) and periimplant bone loss (sagittal) are revealed.

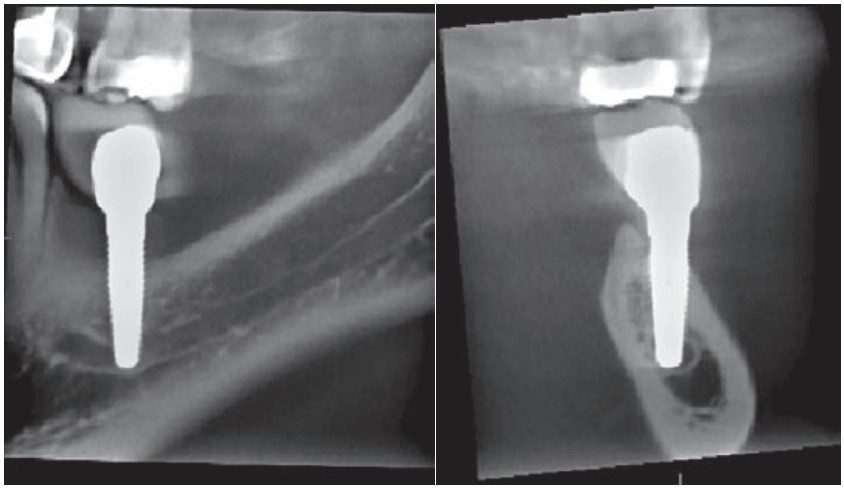

Figure 70-16 Cone-beam computed tomography (CBCT) sagittal and cross-sectional images clearly demonstrate penetration of the implant into the mandibular canal.

(perifixtural bone loss) or around the crestal part of the implant ("saucerization") and could reflect poor osseointegration, periimplantitis and/or unfavorable stress distribution (Figure 70-14, *B* and *C*).

In select cases, when poor implant placement (Figure 70-15) or compromise of vital anatomic structures (Figure 70-16) are suspected, advanced imaging (CBCT, CT, or conventional tomography) provides a three-dimensional evaluation of the oral structures in relation to the implants. Such information can be very important for proper assessment and treatment planning. The treating dentist should recognize relevant signs and symptoms and order appropriate imaging as soon after implant placement as possible. Implant removal, if necessary, would be less complicated before advanced osseointegration.

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SCIENCE TRANSFER

The use of three-dimensional radiographic diagnostics is necessary for almost every patient requiring dental implants. These techniques, combined with a diagnostic splint utilized at the time of the radiographs and at surgery, enables the clinician to assess the exact dimension of the needed implants and to precisely place implants in the correct location with the proper angulation.

At present, cone-beam computed tomography (CBCT) is the ideal method for obtaining diagnostic data. It provides precise measurement of bone dimensions in three planes and has significantly less radiation exposure than conventional CT scans.

Interactive simulation software programs are useful for complex cases because they allow the clinician to simulate a variety of implant sizes and placement techniques so that the most appropriate placement can be selected. A further application of three-dimensional digitized data is that is allows the clinician to have a surgical guide and a final prosthesis developed using computer-aided design/computer-aided manufacturing (CAD/CAM) technology. These approaches require a sophisticated surgical technique and may need further refinement to ensure the proper fit of the prosthesis.

Panoramic radiographs may be valuable as screening views but are not accurate enough for the precise measurements required for implant surgery. Periapical radiographs are a useful adjunct to CBCTs because they help more clearly visualize retained roots and other intrabony pathologic lesions.

CONCLUSION

Many radiographic projections are available for the evaluation of implant placement, each with advantages and disadvantages. The clinician must follow sequential steps in patient evaluation, and radiography is an essential diagnostic tool for implant design and successful treatment of the implant patient. Selection of appropriate radiographic modalities will provide the maximum diagnostic information, help avoid unwanted complications, and maximize treatment outcomes while delivering "as low as reasonably achievable" (ALARA) radiation dose to the patient.[15]

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 71

Standard Implant Surgical Procedures

Perry R. Klokkevold, Thomas J. Han, and Kwang Bum Park

CHAPTER OUTLINE

GENERAL PRINCIPLES OF IMPLANT SURGERY
Patient Preparation
Implant Site Preparation
One-Stage Versus Two-Stage Implant Placement Surgery
TWO-STAGE "SUBMERGED" IMPLANT PLACEMENT
Flap Design, Incisions, and Elevation (online only)
Implant Site Preparation
Flap Closure and Suturing (online only)
Postoperative Care
Second-Stage Exposure Surgery
ONE-STAGE "NONSUBMERGED" IMPLANT PLACEMENT (online only)
Flap Design, Incisions, and Elevation (online only)
Implant Site Preparation (online only)
Flap Closure and Suturing (online only)
Postoperative Care (online only)
CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

The surgical procedures for the placement of nearly all endosseous dental implants currently used are based on the original work of Professor Per-Ingvar Brånemark and colleagues in Sweden in the 1960s and 1970s.[4,5] Their landmark research evaluated the biologic, physiologic, and mechanical aspects of the titanium screw-shaped implant, subsequently known commercially as the Nobelpharma "Brånemark" implant system and currently manufactured by Nobel Biocare. The original Brånemark implant was a parallel-walled, cylindrically shaped, threaded implant with an external hex connection and a machined surface. In the last few decades since Brånemark's original work, many different designs of root form implants have been developed, modified, and studied. At present, there are more than 50 implant manufacturers in the United States and many more worldwide that make implants with various shapes, dimensions, and surface characteristics. Readers are referred to online materials and other resources for detailed descriptions of the various implant geometries and surface characteristics. Regardless of which implant system is used, the same fundamental principles of atraumatic, precise implant site preparation apply. Briefly, this includes a gentle surgical technique and progressive, incremental preparation of the bone for a precise fit of the implant at the time of placement.

This chapter presents general surgical considerations and outlines the standard surgical procedures for the placement of endosseous dental implants utilizing either a one stage or two stage protocol. The principles described here are intentionally generic and designed to serve as guidelines and to be applicable to most of the common implant systems. However, it is important to recognize that each implant system is designed with specific armamentarium and recommendations for use (e.g., drilling speeds) and it is advisable to follow the detailed, step-by-step description found in the manufacturer's manual when using any particular implant system.

GENERAL PRINCIPLES OF IMPLANT SURGERY

Patient Preparation

Most implant surgical procedures can be done in the office using local anesthesia. For some patients, depending on individual preferences, health status, and complexity of the case, conscious sedation (oral or intravenous) may be indicated (see Chapter 55). The risks and benefits of implant surgery specific to the patient's situation and needs should be thoroughly explained at an appointment before the day of surgery. Once the patient understands the proposed treatment and the patient's questions are answered, a written, informed consent should be obtained for the procedure.

Implant Site Preparation

Some basic principles must be followed to achieve osseointegration with a high degree of predictability[3,4,7] (Box 71-1). The surgical site should be kept aseptic, and the patient should be appropriately prepared and draped for an intraoral surgical procedure. Rinsing or swabbing the mouth with chlorhexidine gluconate for 1 to 2 minutes immediately before the procedure will aid in reducing the bacterial load present around the surgical site. Every effort should be made to maintain a sterile surgical field at all times and to avoid

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BOX 71-1 Basic Principles of Implant Therapy to Achieve Osseointegration

1. Implants must be sterile and made of a biocompatible material (e.g., titanium).
2. Implant site preparation should be performed under sterile conditions.
3. Implant site preparation should be completed with an atraumatic surgical technique that avoids overheating of the bone during preparation of the recipient site.
4. Implants should be placed with good initial stability.
5. Implants should be allowed to heal without loading or micromovement (i.e., undisturbed healing period to allow for osseointegration) for 2 to 4 or 4 to 6 months, depending on the bone density, bone maturation, and implant stability.

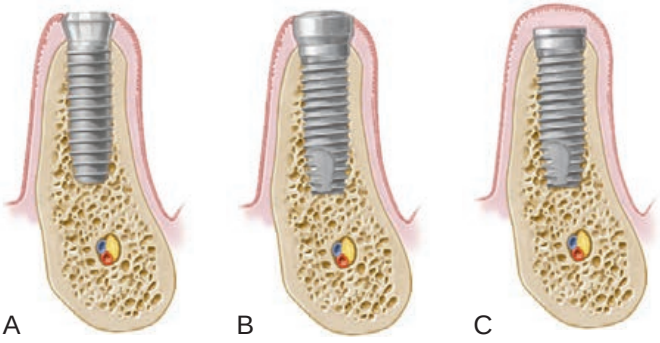

Figure 71-1 One-stage implant versus two-stage implant surgeries. **A,** One-stage surgery with the implant designed so that the coronal portion of the implant extends through the gingiva. **B,** One-stage surgery with implant designed to be used for two-stage surgery. A healing abutment is connected to the implant during the first-stage surgery. **C,** In the two-stage surgery, top of the implant is completely submerged under gingiva.

contamination of the implant surface. Implant sites should be prepared using gentle, atraumatic surgical techniques with a continuous effort to avoid overheating the bone. Finally, it is essential for implants to have good primary stability and be allowed to heal without adverse forces, trauma, or infection for a period of time to allow for osseointegration.

When these clinical guidelines are followed, successful osseointegration occurs predictably for submerged[4] and nonsubmerged[11] dental implants. Well-controlled studies of patients with good plaque control and appropriate occlusal forces have demonstrated that root form, endosseous dental implants show little change in bone height around the implant over years of function.[1] After an initial remodeling in the first year that results in 1.0 to 1.5 mm of bone reduction (described as "normal remodeling around an externally hexed implant"),[1] the bone level around healthy functioning implants remains stable for many years, allowing implants to be a predictable means for tooth replacement. The average annual crestal bone loss after the first year in function is expected to be 0.1 mm or less.

Regardless of the type of surgical approach, the implant must be placed in healthy bone to achieve osseointegration, and an atraumatic technique must be followed to avoid damage to bone or vital structures. The anatomic features of bone quality (dense compact versus loose trabecular) at the recipient site influence the interface between bone and implant.[9] Compact bone offers a much greater surface area for mineralized tissue-to-implant contact than cancellous bone. However, areas that consist entirely of dense, cortical bone (or entirely of loose trabecular bone) may be less desirable. Dense cortical bone sites may be problematic because of a decreased blood supply, fewer cells, and less bone remodeling, despite having a greater bone-to-implant contact, whereas areas consisting mainly of loose trabecular bone may be unable to withstand the forces of occlusion leading to loss of support or integration. Clinical studies have shown that areas of the jaw exhibiting thin layers of cortical bone and large cancellous spaces, such as the posterior maxilla, have significantly lower success rates than areas of denser bone structures.[9] The best results are achieved when the bone-to-implant contact is intimate at the time of implant placement and the blood supply and bone turnover is adequate.

Traumatic or aggressive surgical preparation of bone at the recipient site can adversely affect healing and potential for osseointegration. Drilling of the bone without proper cooling generates increased temperatures that can injure the bone and increase the risk of implant failure.[12] Over-preparation of the site can result in a loose implant fit, poor implant stability, and lack of osseointegration.

One-Stage Versus Two-Stage Implant Placement Surgery

Currently, most threaded endosseous implants can be placed using either a one-stage (nonsubmerged) or a two-stage (submerged) protocol. In the one-stage approach, the implant or the abutment emerges through the mucoperiosteum/gingival tissue at the time of implant placement, whereas in the two-stage approach, the top of the implant and cover screw are completely covered with the flap closure (Figure 71-1). Implants are allowed to heal, without loading or micromovement, for a period of time to allow for osseointegration. In two-stage implant surgery, the implant must be surgically exposed following an undisturbed healing period. Some implants (e.g., Straumann) are specifically designed with the coronal portion of the implant positioned above the crest of bone and extending through the gingival tissues at the time of placement in a one-stage protocol (Figure 71-1, *A*). Other implant systems, designed to be placed at the level of bone, require a healing abutment to be attached to the implant at the time of placement to be used in a one-stage approach[8] (Figure 71-1, *B*).

A one-stage surgical approach simplifies the procedure because a second-stage exposure surgery is not necessary. The two-stage, submerged approach is advantageous for situations that require simultaneous bone augmentation procedures at the time of implant placement because membranes can be covered by primary flap closure, which will minimize postoperative exposure. This approach also prevents movement of the implant by the patient, who may inadvertently bite on the healing abutment during the healing period (one-stage protocol). Mucogingival tissues can be augmented if desired at the second-stage surgery in a two-stage protocol. However, without the second-stage surgical opportunity, mucogingival augmentation must be managed at the initial surgery or an additional, separate surgery. Fundamental differences in flap management for these two surgical techniques are described separately.

TWO-STAGE "SUBMERGED" IMPLANT PLACEMENT

In the two-stage implant surgical approach, the first-stage or implant placement surgery ends by suturing the soft tissues together

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over the implant cover screw so that it remains submerged and isolated from the oral cavity. In areas with dense cortical bone and good initial implant support, the implants are left to heal undisturbed for a period of 2 to 4 months, whereas in areas of loose trabecular bone, grafted sites, and sites with lesser implant stability, implants may be allowed to heal for periods of 4 to 6 months or more. Longer healing periods are indicated for implants placed in less dense bone or when there is less initial implant stability (i.e., slight looseness caused by limited bone-to-implant contact), regardless of jaw or specific anatomic location. Over time, osteoblasts migrate to the surface and form bone adjacent to the implant (osseointegration).[6] Shorter healing periods are indicated for implants placed in good quality (dense) bone and for implants with an altered surface microtopography (e.g., acid etched, blasted, or etched and blasted). Readers are referred to online material and other resources for more information about implant surface microtopography.

In the second-stage (exposure) surgery, the implant is uncovered and a healing abutment is connected to allow emergence of the implant/abutment through the soft tissues, thus facilitating access to the implant from the oral cavity. The restorative dentist then proceeds with the prosthodontic aspects of the implant therapy (impressions and fabrication of prosthesis) after soft tissue healing.

The following paragraphs describe the steps for osteotomy preparation and the first-stage implant placement surgery of the two-stage protocol. Figures 71-2 and 71-3 illustrate the procedures via diagrams, and Figure 71-4 depicts the procedures with clinical photographs.

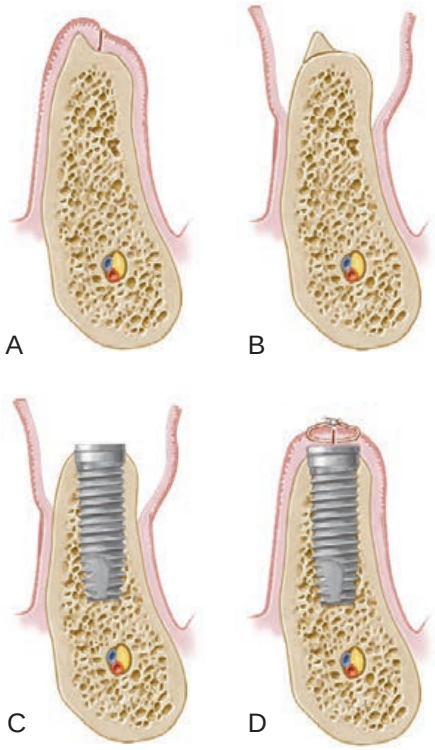

Figure 71-2 Tissue management for a two-stage implant placement. **A,** Crestal incision made along the crest of the ridge, bisecting the existing zone of keratinized mucosa. **B,** Full-thickness flap is raised buccally and lingually to the level of the mucogingival junction. A narrow, sharp ridge can be surgically reduced/contoured to provide a reasonably flat bed for the implant. **C,** Implant is placed in the prepared osteotomy site. **D,** Tissue approximation to achieve primary flap closure without tension.

For information on flap design, incisions, and elevation, please visit the companion Expert CONSULT website at www.expertconsult.com.

Implant Site Preparation

Once the flaps are reflected and the bone is prepared (i.e., all granulation tissue removed and knife-edge ridges flattened), the implant osteotomy site can be prepared. A series of drills are used to prepare the osteotomy site precisely and incrementally for an implant (Figure 71-5). A surgical guide or stent is inserted, checked for proper positioning, and used throughout the procedure to direct the proper implant placement (see Figure 71-4, *E*).

Round Bur. A small round bur (or spiral drill) is used to mark the implant site(s). The surgical guide is removed, and the initial marks are checked for their appropriate buccal-lingual and mesial-distal location, as well as the positions relative to each other and adjacent teeth (see Figure 69-9). Slight modifications may be necessary to adjust spatial relationships and to avoid minor ridge defects. Any changes should be compared to the prosthetically-driven surgical guide positions. Each marked site is then prepared to a depth of 1 to 2 mm with a round drill, breaking through the cortical bone and creating a starting point for the 2-mm twist drill (see Figure 71-3, *A*).

The 2-mm Twist Drill. A small twist drill, usually 2 mm in diameter and marked to indicate various lengths (i.e., corresponding to the implant sizes), is used next to establish the depth and align the long axis of the implant recipient site (see Figure 71-3, *B*). This drill may be externally or internally irrigated. In either case, the twist drill is used at a speed of approximately 800 to 1500 rpm, with copious irrigation to prevent overheating of the bone. Additionally, drills should be intermittently and repeatedly "pumped" or pulled out of the osteotomy site while drilling to expose them to the water coolant and to facilitate clearing bone debris from the cutting surfaces. In other words, in an effort to reduce heat generation and the resistance of drills while in bone, clinicians should pump the drill (up and down) intermittently and avoid preparing the bone with a unidirectional "push" of the drill in the apical direction only.

When multiple implants are being placed next to one another, a guide pin should be placed in the prepared sites to check alignment, parallelism, and proper prosthetic spacing throughout the preparation process (see Figure 71-3, *C*). The relationship to neighboring vital structures (e.g., nerve and tooth roots) can be determined by taking a periapical radiograph with a guide pin(s) or radiographic marker(s) in the osteotomy site(s) (see Figure 70-13). Implants should be positioned with approximately 3 mm between one another to ensure sufficient space for interimplant bone and soft tissue health and to facilitate oral hygiene procedures. Therefore the initial marks should be separated by at least 7 mm (center to center) for 4 mm standard-diameter implants. Incrementally more space is needed for wide-diameter implants (see Figure 69-9).

The 2-mm twist drill is used to establish the final depth of the osteotomy site corresponding to the length of each planned implant. The clinician should also evaluate the bone quality (density) with this drill while preparing the osteotomy to assess the need for modification of subsequent drills used (Box 71-2). If the vertical height of the bone was reduced during the initial ridge preparation, this must be taken into account when preparing the site for a

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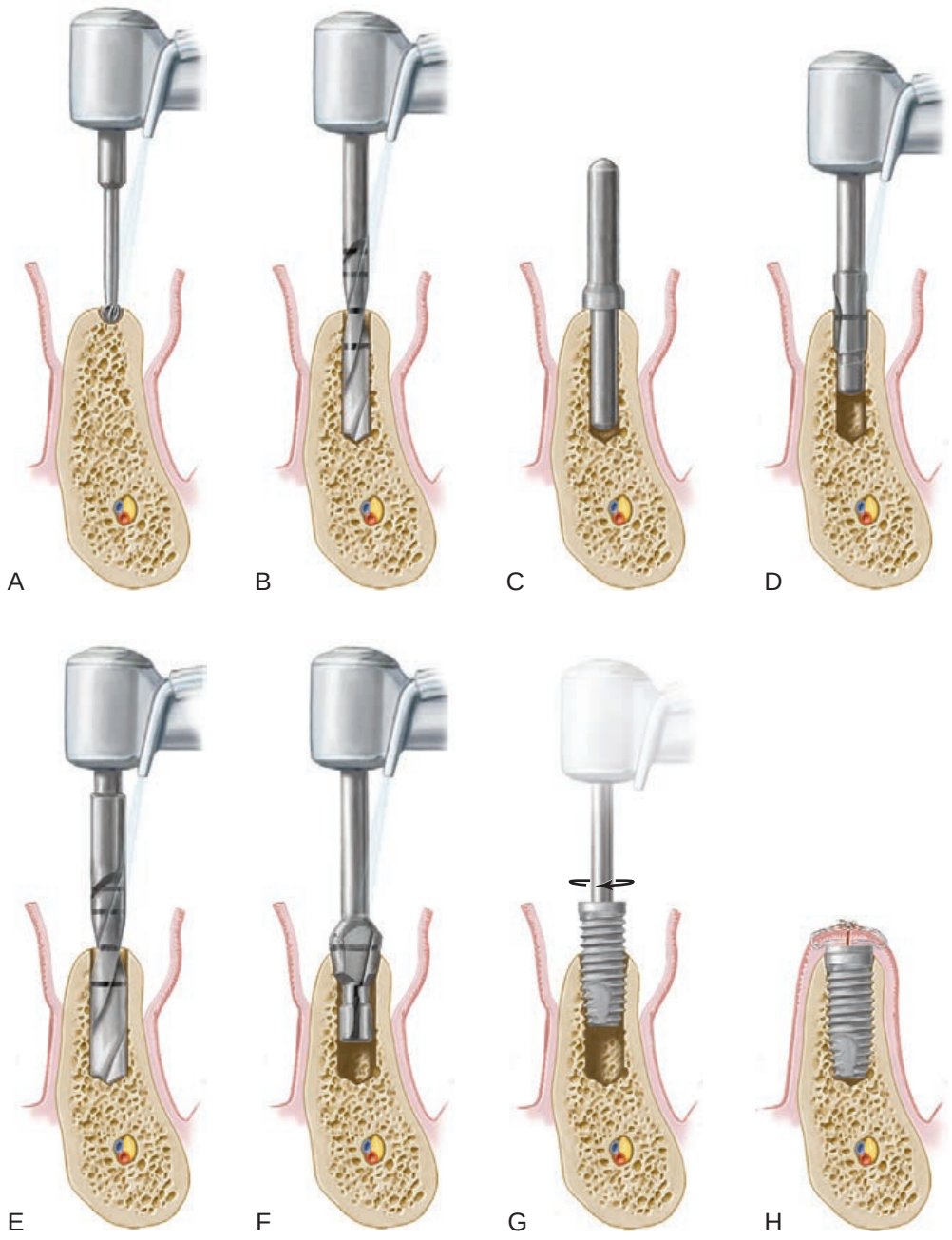

Figure 71-3 Implant site preparation (osteotomy) for a 4.0-mm diameter, 10-mm length screw-type, threaded (external hex) implant in a subcrestal position. **A,** Initial marking or preparation of the implant site with a round bur. **B,** Use of a 2-mm twist drill to establish depth and align the implant. **C,** Guide pin is placed in the osteotomy site to confirm position and angulation. **D,** Pilot drill is used to increase the diameter of the coronal aspect of the osteotomy site. **E,** Final drill used is the 3-mm twist drill to finish preparation of the osteotomy site. **F,** Countersink drill is used to widen the entrance of the recipient site and allow for the subcrestal placement of the implant collar and cover screw. NOTE: An optional tap (not shown) can be used following this step to create screw threads in areas of dense bone. **G,** Implant is inserted into the prepared osteotomy site with a handpiece or handheld driver. NOTE: In systems that use an implant mount, it would be removed prior to placement of the cover screw. **H,** Cover screw is placed and soft tissues are closed and sutured.

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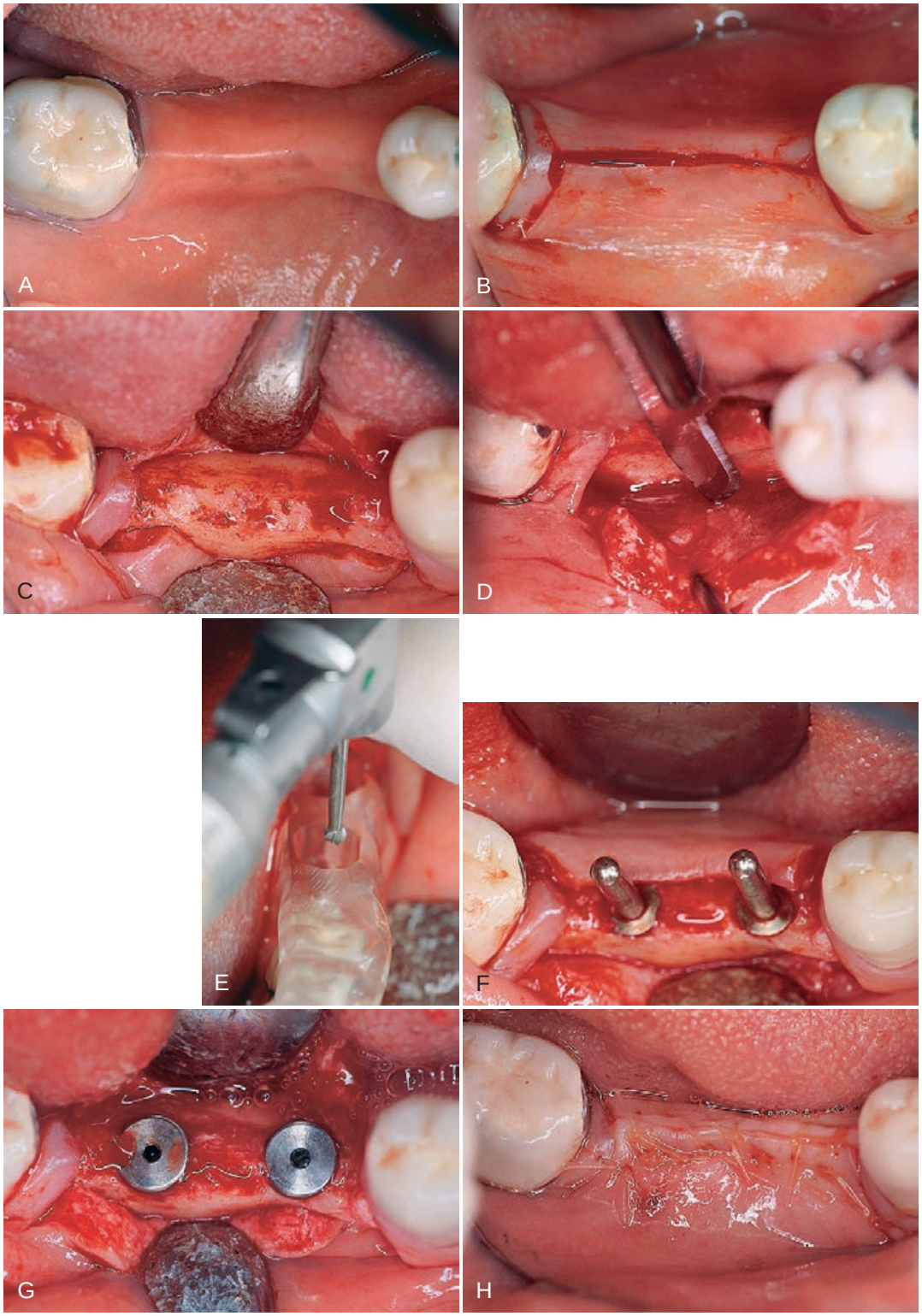

Figure 71-4 Clinical view of stage one implant placement surgery. **A,** Partial edentulous ridge; presurgical and prosthodontic treatment has been completed. **B,** Mesial sulcular and distal vertical incisions are connected by a crestal incision. Notice that bands of gingival collars remain adjacent to the distal molar tooth. **C,** Minimal flap reflection is used to expose the alveolar bone. Sometimes a ridge modification is necessary to provide a flap recipient bed. **D,** Buccal flap is partially dissected at the apical portion to provide a flap extension. This is a critical step to ensure a tension-free closure of the flap after implant placement. **E,** It is important to use the surgical stent to determine the mesial-distal and buccal-lingual dimensions and proper angulation of the implant placement. **F,** Frequent use of the guide pins ensures parallelism of the implant placement. **G,** After placement of two Nobelpharma implants, the cover screws are placed. The cover screws should be flush with the rest of the ridge to minimize the chance of exposure. This is especially important if the patient will wear a partial denture during the healing phase. **H,** Suturing completed. Both regular interrupted and inverted mattress sutures are used intermittently to ensure tension-free, tight closure of the flaps.

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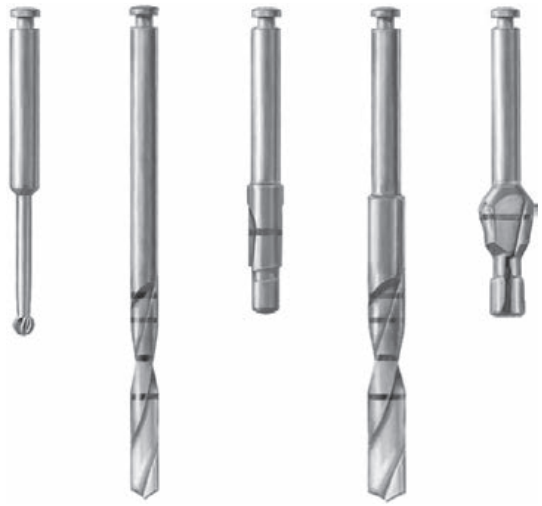

Figure 71-5 Sequence of drills used for standard-diameter (4.0-mm) implant site osteotomy preparation: round, 2-mm twist, pilot, 3-mm twist, and countersink. Bone tap (not shown here) is an optional drill that is sometimes used in dense bone before implant placement.

predetermined implant length. For example, if it appears that the implant will be too close to a vital structure, such as the inferior alveolar nerve canal, the depth of the implant osteotomy site and length of the implant may need to be reduced.

The next step is to use a series of drills to incrementally increase the width of the osteotomy site to accommodate the planned implant diameter. The styles, shapes, and final diameter of the drills will differ slightly among different implant systems, but their general purpose is to prepare a recipient site with a precise diameter (and depth) for the selected implant without unduly traumatizing the surrounding bone. It is important to use copious irrigation and a "pumping" action for all drilling.

Pilot Drill. Following the 2-mm twist drill, a pilot drill with a noncutting 2–mm-diameter "guide" at the apical end and a cutting 3–mm-diameter (wider) midsection is used to enlarge the osteotomy site at the coronal end, thus facilitating the insertion of the subsequent drill in the sequence (see Figure 71-3, *D*).

The 3-mm Twist Drill. The final drill in the osteotomy site preparation for a standard-diameter (4.0 mm) implant is the 3-mm twist drill. It is last drill used to widen the site along the entire depth of the osteotomy from the previous diameter (2 mm) to final diameter (3 mm). This final drill in the sequence finishes preparing the osteotomy site and consequently is the step that dictates whether the implant will be stable or not (Figure 71-3, *E*). It is critically important that the final diameter drilling be accomplished with a steady hand, without wobbling or changing direction so that the site is not overprepared. Finally, depending on bone density, the diameter of this final drill may be slightly increased or decreased to enhance implant support (see Box 71-2).

Countersink Drill (Optional). When it is desirable to place the cover screw at or slightly below the crestal bone, countersink drilling is used to shape or flare the crestal aspect of the osteotomy site allowing the coronal flare of the implant head and cover screw to fit within the osteotomy site (Figure 71-3, *F*). As with all drills in the sequence, copious irrigation and gentle surgical techniques are used.

BOX 71-2 Clinical Advice to Enhance Precision of Final Implant Site Preparation

Clinical Situation 1

If the final drill hits the bottom of the recipient site before reaching the desired depth, the added hand pressure necessary to achieve the proper depth often causes wobbling and funneling of the recipient site. This is especially true with "cannon" drills (used for cylindrical implants). To minimize this effect, a smaller-diameter drill should be used to prepare the site slightly deeper (e.g., 0.5 mm less). This narrower drill allows the desired depth to be reached without affecting the side walls and facilitates a more precise osteotomy preparation with the final drill.

Clinical Situation 2

If the final drill is inserted at an inaccurate angle, the result is funneling of the coronal portion of the implant site. To minimize this potential problem, when drilling multiple implant sites, the operator should always keep a direction indicator in an adjacent site. For single-implant sites, the adjacent teeth and surgical guide should serve as direction indicators. When dealing with dense bone, a precise recipient site can be achieved more predictably if there is minimal diameter change from drill to drill. For example, switching from 3.0 to 5.0 mm is much less precise than proceeding from 3.0 to 3.3 to 4.2 to 5.0 mm.

Clinical Situation 3

If the bone is "soft" (e.g., loose trabecular bone), it may be advantageous to underprepare the osteotomy site. A slightly underprepared site can be accomplished by using the final drill to a shallower depth than the previous drill (e.g., half the depth of the osteotomy site). This avoids removing too much bone and increases the implant stability or tightness at the time of placement. Another method to achieve an underprepared site is to use a drill with a slightly smaller diameter as the final drill in the preparation (e.g., a 2.75-mm drill as the final drill rather than a 3.0 mm or a 3.25 mm as the final drill for a 4.0-mm implant).

Bone Tap (Optional). As the final step in preparing the osteotomy site in dense cortical bone, a tapping procedure may be necessary (not shown). With self-tapping implants being almost universal, there is less need for a tapping procedure in most sites. However, in dense cortical bone or when placing longer implants into moderately dense bone, it is prudent to tap the bone (create threads in the osteotomy site) before implant placement to facilitate implant insertion and to reduce the risk of implant binding (Figure 71-3, *G*).

Clinical comment: When faced with a very soft, poor-quality bone (e.g., loose trabecular bone in the posterior maxilla), tapping is not necessary or recommended (see Box 71-2). It is better to allow the threaded implant to "cut" its own path into the osteotomy site.

Bone tapping and implant insertion are both done at very slow speeds (e.g., 20 to 40 rpm). All other drills in the sequence are used at higher speeds (800 to 1500 rpm).

It is important to create a recipient site that is very accurate in size and angulation. In partially edentulous cases, limited jaw opening or proximity to adjacent teeth may prevent appropriate positioning of the drills in posterior edentulous areas. In fact, implant therapy may be contraindicated in some patients because of a lack of interocclusal clearance, lack of interdental space, or a

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lack of access for the instrumentation. Therefore a combination of longer drills and shorter drills, with or without extensions, may be necessary. Anticipating these needs before surgery facilitates the procedure and improves the results.

Note on wide-diameter implant site preparation: It is sometimes desirable to replace molars with wide-diameter implants because the larger diameter better approximates the size and the emergence profile of molar-sized teeth and may improve support for occlusal forces. When wide-diameter drills are used for implant site preparation, it is advisable to reduce the drilling speed, according to the manufacturer's guidelines, to prevent overheating the bone. Copious external irrigation is critical. In the case of wide diameter implants, a specific pilot drill is often indicated as a transition between each of the subsequent wider drills.

Implant Placement. Implants are inserted with a handpiece rotating at slow speeds (e.g., 25 rpm) or by hand with a wrench. Insertion of the implant must follow the same path or line as the osteotomy site. When multiple implants are being placed, it is helpful to use guide pins in the other sites to have a visual guide for the path of insertion.

For information on flap closure and suturing, please visit the companion Expert CONSULT website at www.expertconsult.com.

Postoperative Care

Simple implant surgery in a healthy patient usually does not require antibiotic therapy. However, patients can be premedicated with antibiotics (e.g., amoxicillin, 500 mg three times a day [tid]) starting 1 hour before the surgery and continuing for 1 week postoperatively if the surgery is extensive, if it requires bone augmentation, or if the patient is medically compromised. Postoperative swelling is likely after flap surgery. This is particularly true when the periosteum has been incised (released). As a preventive measure, patients should apply an ice pack to the area intermittently for 20 minutes (on and off) over the first 24 to 48 hours. Chlorhexidine gluconate oral rinses can be prescribed to facilitate plaque control, especially in the days after surgery when oral hygiene is typically poorer. Adequate pain medication should be prescribed (e.g., ibuprofen, 600 to 800 mg tid).

Patients should be instructed to maintain a relatively soft diet after surgery. Then, as soft tissue healing progresses, they can gradually return to a normal diet. Patients should also refrain from tobacco and alcohol use at least 1 week before and several weeks after surgery. Provisional restorations, whether fixed or removable, should be checked and adjusted so that impingement on the surgical area is avoided.

Second-Stage Exposure Surgery

For implants placed using a two-stage "submerged" protocol, a second-stage exposure surgery is necessary after the prescribed healing period. Box 71-3 lists the objectives for second-stage implant exposure surgery. Thin soft tissue with an adequate amount of keratinized attached gingiva, along with good oral hygiene, ensures healthier periimplant soft tissues and better clinical results. The need for periimplant keratinized tissue is somewhat controversial, depending on the type of implant prosthesis and location of the implant. However, one long-term study indicated that, at least in the posterior mandible and in partially edentulous patients, the presence of keratinized tissue is strongly correlated with soft and hard tissue health.[2]

BOX 71-3 Objectives of Second-Stage Implant Surgery

1. To expose the submerged implant without damaging the surrounding bone.
2. To control the thickness of the soft tissue surrounding the implant.
3. To preserve or create attached keratinized tissue around the implant.
4. To facilitate oral hygiene.
5. To ensure proper abutment seating.
6. To preserve soft tissue esthetics.

Simple Circular "Punch" or Crestal Incision. In areas with sufficient zones of keratinized tissue, the gingiva covering the head of the implant can be exposed with a circular or "punch" incision (Figure 71-6). Alternatively, a crestal incision through the middle of the keratinized tissue and full-thickness flap reflection can be used to expose implants. This latter approach may be necessary when bone has grown over the implant and needs to be removed.

Partial-Thickness Repositioned Flap. If a minimal zone of keratinized tissue exists at the implant site, a partial-thickness flap technique can be used to fulfill the objective of the second-stage surgery (exposing the implant) while increasing the width of keratinized tissue. The initial incision is made within the zone of keratinized tissue so that it becomes the outer edge of the reflected split-thickness flap. Vertical releasing incisions are used on both the mesial and the distal end of the flap (Figure 71-7, *A* and *B*). A partial-thickness flap is then raised in such a manner that a nonmobile, firm periosteum remains attached to the underlying bone. The flap, containing a narrow band of keratinized tissue, is then repositioned to the facial side of the emerging head of the implant and sutured to the periosteum with a fine needle and resorbable suture such as a 5.0 gut suture (Figure 71-8). If the initial amount of keratinized tissue is less than 2 mm, the flap may be started at the labial edge of the keratinized tissue allowing that zone to remain on the lingual aspect of the implant. A partial-thickness flap is apically displaced and sutured to the periosteum without exposing the alveolar bone (Figure 71-7, *C*). A free gingival graft may be harvested from the palate and sutured to the periosteum on the labial surface of the implants to increase the zone of keratinized tissue (not shown).

After the flap is repositioned and secured with periosteal sutures, the excess tissue coronal to the cover screw is excised, usually with a surgical blade (Figure 71-8, *B*). However, if removal of this tissue would jeopardize the amount of remaining keratinized tissue around the lingual aspect of the implant(s), a similar partial-thickness flap can be elevated and repositioned on the lingual side as well. Extra care must be taken when creating a split-thickness flap on the lingual surface of mandibular sites because the tissue is often very thin. Alternatively, a full-thickness lingual flap will be safer and will serve a similar purpose of preserving keratinized tissue on the lingual surface of the implant(s).

When the excess tissue over the cover screw is removed or displaced, the outline of the cover screw is visible. A sharp blade is used to eliminate all tissues coronal to the cover screw (Figure 71-7, *D*). The cover screw is then removed, the head of the implant is thoroughly cleaned of any soft or hard tissue overgrowth, and the healing abutments or standard abutments are placed on the implant (Figure 71-7, *E* and *F*). The fit of the healing abutments to the

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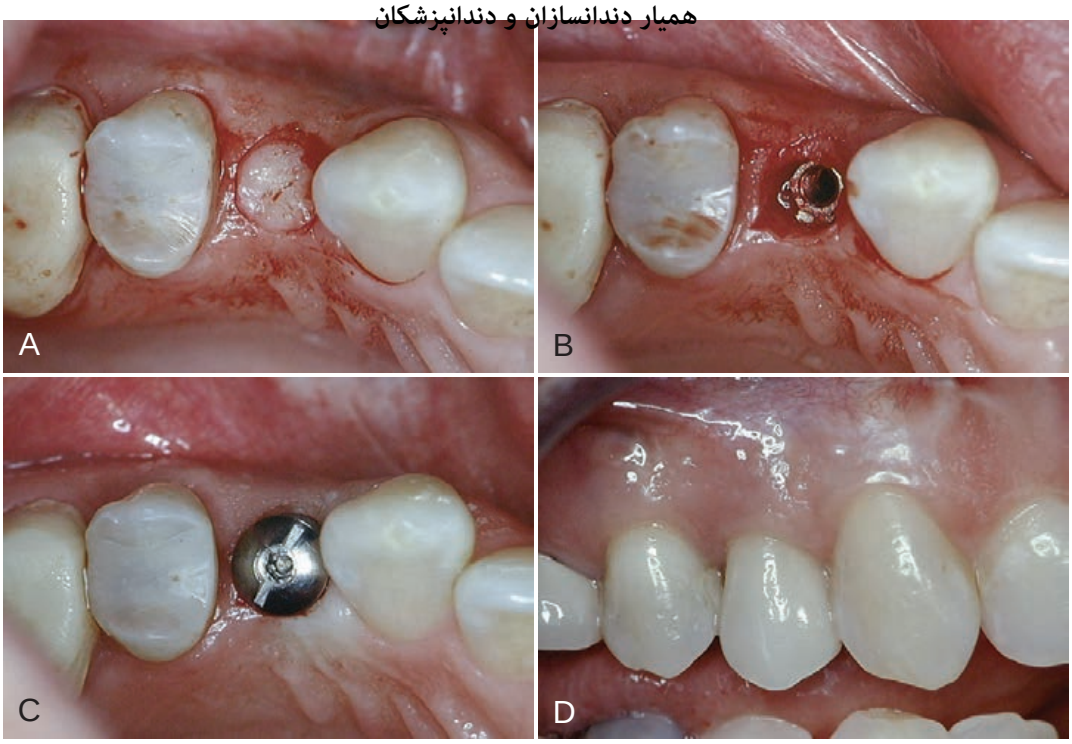

Figure 71-6 Clinical view of stage two, implant exposure surgery in a case with adequate keratinized tissue. **A,** Simple circular "punch" incision used to expose implant when sufficient keratinized tissue is present around the implant(s). **B,** Implant exposed. **C,** Healing abutment attached. **D,** Final restoration in place, achieving an esthetic result with a good zone of keratinized tissue.

Figure 71-7 Clinical view of stage two implant exposure surgery in a case with inadequate keratinized tissue. **A,** Two endosseous implants were placed 4 months previously and are ready to be exposed. Note the narrow band of keratinized tissue. **B,** Two vertical incisions are connected by crestal incision. If facial keratinized tissue is insufficient, it is necessary to locate the crestal incision more lingually so that there is at least 2 to 3 mm of keratinized band. **C,** Buccal partial-thickness flap is sutured to the periosteum apical to the emerging implants. **D,** Gingival tissue coronal to the cover screws is excised using the gingivectomy technique. **E,** Cover screws are removed, and heads of the implants are cleared. **F,** Abutments are placed. Visual inspection ensures intimate contact between the abutments and the implants. **G,** Healing at 2 to 3 weeks after second-stage surgery. **H,** Four months after the final restoration. Note the healthy band of keratinized attached gingiva around the implants.

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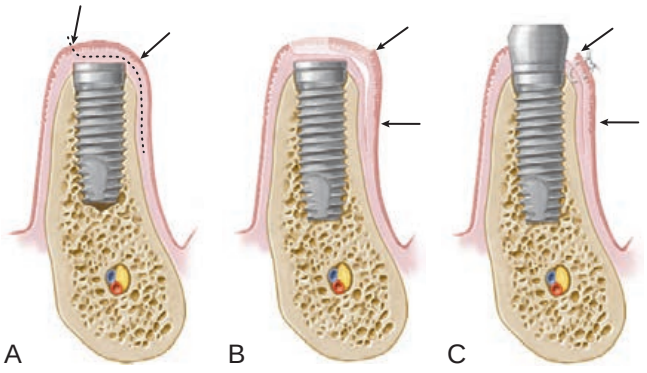

Figure 71-8 Illustration depicting the use of a split-thickness flap that is repositioned to the labial surface in order to preserve and increase the amount of keratinized tissue. **A,** Partial-thickness flap is created from the lingual aspect of the crest toward the labial surface in order to preserve the keratinized tissue on the crest (over the implant). NOTE: This tissue might be excised in a simple implant exposure. **B,** The split-thickness flap is repositioned to the labial surface. **C,** The flap is sutured to the periosteum at a more apical position preserving the amount of keratinized tissue *(arrows).* Finally, the remaining connective tissue over the cover screw **(B)** is excised with a sharp blade to expose the implant. Care should be taken to avoid removing keratinized tissue from the lingual aspect of the implant.

implants can often be visually evaluated. However, if it is not possible to visualize clearly the intimate connection between the implant and the abutment, an intraoral periapical x-ray film should be taken to confirm complete seating. Bone may need to be removed around the top of the implant to get the abutment to seat properly. Readers are referred to online material for a discussion of bone profiling.

Postoperative Care. Once the implant is exposed and soft tissues are sutured, it is important to remind the patient of the need for good oral hygiene around the implant and adjacent teeth. A chlorhexidine rinse can be used to enhance oral hygiene for the initial few weeks after implant exposure while the tissues are healing. Care should be taken during oral hygiene procedures to avoid dislodging any repositioned or grafted soft tissues. Furthermore, any direct pressure or movement directed toward the soft tissue from a provisional prosthesis can delay healing and should be avoided. Tissues should be monitored regularly and the provisional prosthesis should be adjusted as needed. Impressions for the final prosthesis fabrication can begin about 2 to 6 weeks after implant exposure surgery, depending on healing and maturation of soft tissues. Figure 71-7, *G* and *H*, shows the postoperative results in a clinical case after 2 to 3 weeks and 4 months, respectively.

For information on one-stage, nonsubmerged implant placement, please visit the companion Expert CONSULT website at www.expertconsult.com.

CONCLUSION

It is essential to understand and follow basic guidelines to predictably achieve osseointegration. Fundamental protocols must be followed for implant placement (stage one) and implant exposure surgery (stage two). These fundamentals apply to all implant systems.

References and suggested reading can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

Clinicians need to pay close attention to detail and follow clearly delineated protocols with skillful and gentle surgical techniques in all cases, even those that are relatively simple. In this way, the high success rate of implant therapy reported in a multitude of clinical trials with a wide variety of implant types can be transferred to individual dental practices.

The choice of a one- or two-stage approach should be available for all patients because each technique has its specific application. One-stage procedures offer simplicity and may be quicker for patients. They also allow better control of soft tissue contours that maximize the esthetic outcome. Two-stage procedures are valuable in patients with poor quality bone and in those cases requiring bone augmentation.

Implant stability at the time of placement is essential and depends on biologically-based, skilled surgical procedures. Implants have the least amount of bone support in the third and fourth week after placement as the new bone slowly integrates with the titanium surface. At this time, attention to functional loading must include eliminating or significantly minimizing forces on the new implant. Thus one-stage procedures with immediate placement of a temporary prosthesis should be restricted to implants with a high level of initial stability at the time of placement. New methodologies to measure implant stability, including measurements of resonance frequency response, will become more widely applicable as reliable data on their productive value become available.

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CHAPTER 72

Localized Bone Augmentation and Implant Site Development

Perry R. Klokkevold

CHAPTER OUTLINE

GUIDED BONE REGENERATION (online only)
- Barrier Membranes
- Bone Graft Materials
- Autogenous Bone

LOCALIZED RIDGE AUGMENTATION
- Flap Management
- Horizontal Bone Augmentation
- Simultaneous Implant Placement
- Complications

ALVEOLAR RIDGE PRESERVATION/MANAGEMENT OF EXTRACTIONS
- Delayed Implant Placement
- Staged Implant Placement
- Delayed versus Staged Technique
- Immediate Implant Placement

CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures may be out of numeric order in this printed chapter.

One of the most critical aspects of creating an esthetic implant restoration is surgical placement of the implant in a "prosthetically-driven" position to restore the natural tooth position and to emulate the natural emergence of a tooth from the soft tissues. Implants placed without regard for prosthetic position often result in dental restorations that are functionally and esthetically compromised, and patients are left with a less-than-optimal reconstruction. Placement of dental implant(s) in an esthetically and functionally optimal position requires reconstruction of deficient alveolar ridges (or preservation of ridges when teeth are to be extracted).

Periodontal bone loss, gingival recession, tooth loss, and long-term use of removable appliances typically result in alveolar defects that prevent the placement of implants in an optimal prosthetic position. It also leads to soft tissue deficiencies that are unacceptable. Fortunately, continuous innovations in surgical techniques and advances in the biologic understanding of bone-regenerative techniques have led to advanced implant procedures and an increased predictability in the reconstruction of alveolar ridge defects.[27,38]

Standard implant placement surgery, as described in Chapter 71, is based on adequate bone volume and quality in the desired implant location. The time-tested standard protocol allows for adequate remodeling and maturation of bone, with healing periods of 3 to 6 months. Recent implant procedures often challenge these original conventions by placing implants in areas with inadequate bone volume, simultaneously augmenting bone, and restoring or loading implants after shorter healing periods. This chapter presents an overview of surgical bone augmentation procedures used to correct or to prevent alveolar ridge deficiencies for the optimal placement of dental implants.

For information on guided bone regeneration, please visit the companion Expert CONSULT website at www.expertconsult.com

LOCALIZED RIDGE AUGMENTATION

Patients often present for implant planning after tooth loss and alveolar ridge resorption. In these cases, the clinician is obligated to perform augmentation procedures to reconstruct lost bone and place implants in a prosthetically-driven position.

Surgical reconstructive procedures for the preparation and placement of dental implants have become more numerous and

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complex. Depending on the size and morphology of the defect, various augmentation procedures can be used. These procedures have been categorized according to the deficient dimension: horizontal or vertical. Methods used to augment horizontal, as well as vertical, bone deficiencies include particulate bone grafts and monocortical block grafts. Barrier membranes can be used with bone grafts to reconstruct all types of alveolar bone defects. See Chapter 73 for a review of procedures used to achieve vertical augmentation. All the proven principles of GBR and flap management must be followed to achieve good results. These include generating a blood supply; maintaining a stable, protected space for bone growth; and achieving tension-free flap wound closure.

Flap Management

Soft tissue management is a critical aspect of bone augmentation procedures. Incisions, reflection, and manipulation should be designed to optimize blood supply and wound closure. The design and management of mucoperiosteal flaps must consider the increased dimensions of the ridge after augmentation as well as esthetics and approximation of the wound margins. The surgical procedure must be executed with the utmost of care in order to preserve the vascularity of the flap and to minimize tissue injury.[1]

Several flap techniques maintain a "submerged" position of bone grafts and barrier membranes during the entire healing process, including a remote or displaced incision.[9,25] The advantage of a remote incision is that the wound opening is positioned away from the graft. A conventional crestal incision can be used, even in large supracrestal defects, as long as a periosteal releasing incision and coronal advancement of the flap achieve a tension-free closure.[30] Most reports suggest removing sutures approximately 10 to 14 days after surgery. It is also suggested that no prosthesis be inserted for 2 to 3 weeks after surgery to avoid pressure over the wound during the early healing phase.

General concepts for flap management associated with ridge augmentation include the following:

1. It is desirable to make incisions remote relative to the placement of barrier membranes (e.g., vertical releasing incisions at least one tooth away from the site to be grafted). In the anterior maxilla, keeping vertical incisions remote is also an esthetic advantage.
2. Full mucoperiosteal flap elevation at least 5 mm beyond the edge of the bone defect is desirable.
3. The use of vertical incisions, although often required for surgical access, should be minimized.
4. Use of a periosteal releasing incision to give the flap elasticity and permit tension-free suturing is essential. This permits complete closure without stress on the wound margins.
5. Removable appliance should not be inserted over the wound for 2 weeks or more to avoid postoperative trauma to the surgical site.
6. Wound closure should incorporate a combination of mattress sutures to approximate connective tissues and interrupted sutures to adapt wound edges.

Horizontal Bone Augmentation

A deficiency in the horizontal dimension of bone may be minimal, such as a dehiscence or fenestration of an implant surface, or it may be more significant, such that the implant would have more than one axial surface exposed while having some bone along the entire vertical length. Dehiscence defects can usually be managed during implant placement because most of the implant is covered and stabilized by native bone. If the horizontal deficiency is large and the implant placement would result in significant exposure (i.e., implant body is significantly outside the alveolar bone), it may be better to reconstruct the bone first, in a staged approach, with a subsequent surgery for implant placement.

Although reconstruction of deficient ridges with bone grafts alone (i.e., without barrier membrane) has proved to be effective, variable resorption of the grafted bone has been reported. Preliminary results in a 1- to 3-year study using autografts harvested from the maxillary tuberosity showed an increased ridge width, but resorption of 50% of the graft volume was also noted.[55] Buser et al[9] investigated the lateral ridge augmentation procedure using an autograft from the retromolar or symphysis area covered by a membrane in 40 consecutively treated patients and noted no clinical signs of resorption of the block graft. The researchers emphasized a remote incision technique, perforation of the cortex, stable placement of corticocancellous autografts, precise adaptation and stabilization (with miniscrews) of the ePTFE membranes, and a tension-free primary soft tissue closure. After 7 to 13 months, the sites were reopened for membrane removal and implant placement. Of the 40 patients, 38 exhibited excellent ridge augmentation, whereas 2 sites showed some soft tissue encapsulation of the grafted bone.

Nevins and Mellonig[42] and Doblin et al[14] reported an increased amount of new bone using freeze-dried bone allografts (FDBAs) with membranes, even in the presence of membrane exposure. The biopsies showed viable bone cells and visible osteocytes in lacunae, and a 9-month specimen showed no remaining allograft material.

On the other hand, there are some contradictory results using DFDBA and membrane combinations.[2,7,8] In a human study, seven paired extraction sockets were grafted with either DFDBA or autologous bone. Sites were reentered and biopsied after 3 to 13 months to evaluate bone formation. Histologic specimens revealed dead particles of DFDBA with no evidence of bone formation on the surface and no evidence of osteoclastic resorption. Conversely, the autogenous sites revealed vascular channels with woven and lamellar bone. Some nonvital, cortical bone chips were observed with osteoclastic resorption.

Figure 72-1 (online) shows an example of the lack of bone formation around DFDBA particles used in a ridge augmentation procedure after more than 20 months of healing under a nonresorbable ePTFE barrier membrane.

To view Figure 72-1, please visit the companion Expert CONSULT website at www.expertconsult.com.

Particulate Bone Graft. Advantages of particulate bone grafts (or bone chips) are that the smaller pieces of bone demonstrate more rapid ingrowth of blood vessels (revascularization), larger osteoconduction surface, more exposure of osteoinductive growth factors, and easier biologic remodeling compared with a bone block for reconstruction of large defects. However, particulate grafts often lack a rigid, supportive structure and are much more easily displaced than block grafts.

Autologous particulated bone grafts can be harvested from any edentulous jaw site, either in smaller "particle" or in a larger "block" of bone. If the bone has been harvested in a block, a bone mill is necessary to "particulate" the bone and prepare it for transplantation into the bone defect (Figure 72-2, online).

To view Figure 72-2, please visit the companion Expert CONSULT website at www.expertconsult.com.

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Particulate grafts are indicated (1) in defects with multiple osseous walls that will contain the graft or (2) in dehiscence or fenestration defects when implants are placed during the bone augmentation procedure. If a bone defect does not have sufficient osseous walls to contain the graft, the barrier membrane (placed in contact with the native bone) should be secured along the periphery with tacks, screws, or sutures. This bone graft and secured barrier membrane combination becomes an environment that is stable and supports new bone formation. Figure 72-3 depicts the use of a barrier membrane in combination with a particulate bone graft to treat a mandibular horizontal defect.

Monocortical Block Graft. Horizontal alveolar deficiencies that might be challenging to reconstruct with particulate grafts can easily be reconstructed with a monocortical block bone graft. The technique uses a cortical block of bone harvested from a remote site and used to increase the width of bone. The block graft taken from an intraoral (e.g., mandibular symphysis or ramus) or extraoral (e.g., iliac crest or tibia) site is fixated to the prepared recipient site with screws. The overlying soft tissues can be separated from the bone graft with a barrier membrane or simply covered with the mucoperiosteal flap. Fixation hardware (screws and plates) should be removed after an adequate period of healing (approximately 6 months). The disadvantage of this technique is the biologic limitation of revascularizing large bone blocks. It therefore is crucial to have sufficient osteogenic cells in the residual surface of the surrounding bone and to limit this technique to horizontal augmentation and only minimal vertical defects.

Figure 72-4 shows the use of a monocortical block graft to reconstruct a horizontal deficiency in the posterior right mandible. The patient presented with a loss of the buccal cortical plate of bone after a traumatic extraction of endodontically treated tooth #29. The surgical extraction also resulted in a nonrestorable cut into the mesial root of tooth #30. Recommended treatment included extraction of tooth #30, monocortical block graft reconstruction of the buccal defect at site #29, and bone augmentation of the extraction socket #30. The procedure for this case follows.

Procedure. After local anesthesia, an incision was made in keratinized tissue along the crest and around the molar tooth (#30) with a vertical releasing incision mesial to the first bicuspid (#28). A full-thickness flap was elevated to expose the alveolar bone (Figure 72-4, *D*). All soft tissues were thoroughly removed from the recipient site before bone grafting. After simple forceps delivery of tooth #30, the defect to be grafted was measured to determine the size of block graft to harvest from the mandibular symphysis. Several bleeding points were created using a small round bur.

The autogenous monocortical block graft was harvested from the mandibular symphysis. It was cut to an appropriate size and mortised to intimately fit the recipient defect site. Once properly positioned, the graft was fixated with two fixation screws (Leibinger, Kalamazoo, MI) that passed through the graft and into the existing native alveolar bone. A periosteal releasing incision was used to sever the periosteum from anterior to posterior and facilitate coronal advancement of the mucogingival flap.

After 6 months of healing, a full-thickness mucoperiosteal flap was elevated to expose the alveolar bone sites #29 and 30. Mild resorption of the monocortical block graft is evident. Notice that the position of the head of the fixation screws (especially the posterior screw) is more protruded than the grafted bone as a consequence of bone remodeling and resorption (see Figure 72-4, *H* and *I*).

The fixation screws are removed, and the sites are prepared in the usual manner for the placement of two screw-type, wide-diameter implants (Implant Innovations, Palm Beach Gardens, FL). Care is taken to avoid preparing the grafted site too wide or too far labially because the grafted bone may be vulnerable to fracture or additional resorption (Figure 72-4, *J*).

Simultaneous Implant Placement

Large alveolar bone defects need to be augmented before implant placement and require a healing period of 6 months or longer. In selected cases, it is possible to perform a bone augmentation procedure simultaneously with the implant placement. It is essential to achieve good implant stability in the existing native bone so that endosseous integration can occur.

A very predictable osseous defect to manage with simultaneous implant placement is the implant dehiscence or fenestration defect. *Fenestration* defects are exposures of the implant's axial surface that do not include the coronal aspect of the implant (Figure 72-5). *Dehiscence* defects expose a part of the axial surface, including the coronal aspect of the implant, while maintaining sufficient bone volume around all remaining implant surfaces (Figure 72-6). In a dehiscence defect the implant remains within the confines of the existing bone.

Fenestration and dehiscence defects have been managed with barrier membranes or simply with flap closure. Bone grafts have also been used. The only controlled comparison studies between membrane treatment and periosteal flap coverage of exposed implant surfaces in humans demonstrated that the membrane treatment was far superior with regard to bone fill.[11] Another controlled study in humans showed better results in the membrane groups; four of six sites (66%) treated with a membrane resulted in 95% to 100% elimination of the dehiscence and total coverage of the threads. In the control sites, only 2 of 6 sites (33%) showed moderate-to-complete bone fill.[43] All other clinical studies are in the form of case reports.[38] Figure 72-7, *M,* demonstrates coverage of an implant dehiscence using a barrier membrane. Admittedly, without a biopsy, it cannot be determined whether the tissue covering the implant is bone or firm connective tissue.[22,26]

A 1-year multicenter study evaluating 55 Brånemark implants (i.e., machined-surface, external hex) with bone dehiscence in 45 patients, treated by ePTFE membrane alone, demonstrated an average bone fill of 82%.[12] The average initial defect height was 4.7 mm. The 1-year follow-up of these implants demonstrated a favorable response to loading. Of the 55 implants, a total of 6 failed, corresponding to a cumulative survival rate of 84.7% in the maxilla and 95.0% in the mandible, which is similar to previously published results for this implant design.

A clinical report on the use of TR membranes demonstrated the biologic potential to fill a large protected space in four patients.[24] Bone dehiscence at implant sites ranged from 5 to 12 mm (mean, 8.2 mm). They were covered with a TR membrane alone (no graft). Reentry after 7 to 8 months of submerged healing found complete bone coverage over all implants. Radiographic evaluation demonstrated the implants functioning with normal crestal bone support after 1 year.

No clinical comparisons are available in the literature evaluating the placement of bone grafts with or without barrier membranes on implant dehiscence defects. Most evidence supports the use of graft materials in conjunction with membrane treatment, especially the use of FDBA in conjunction with GBR. In a study with 40 patients, 110 implants were placed in conjunction with barrier membranes and FDBA; a success rate of 96.8% was achieved with complete bone fill (defined as >90% fill of dehiscence).[45] This study

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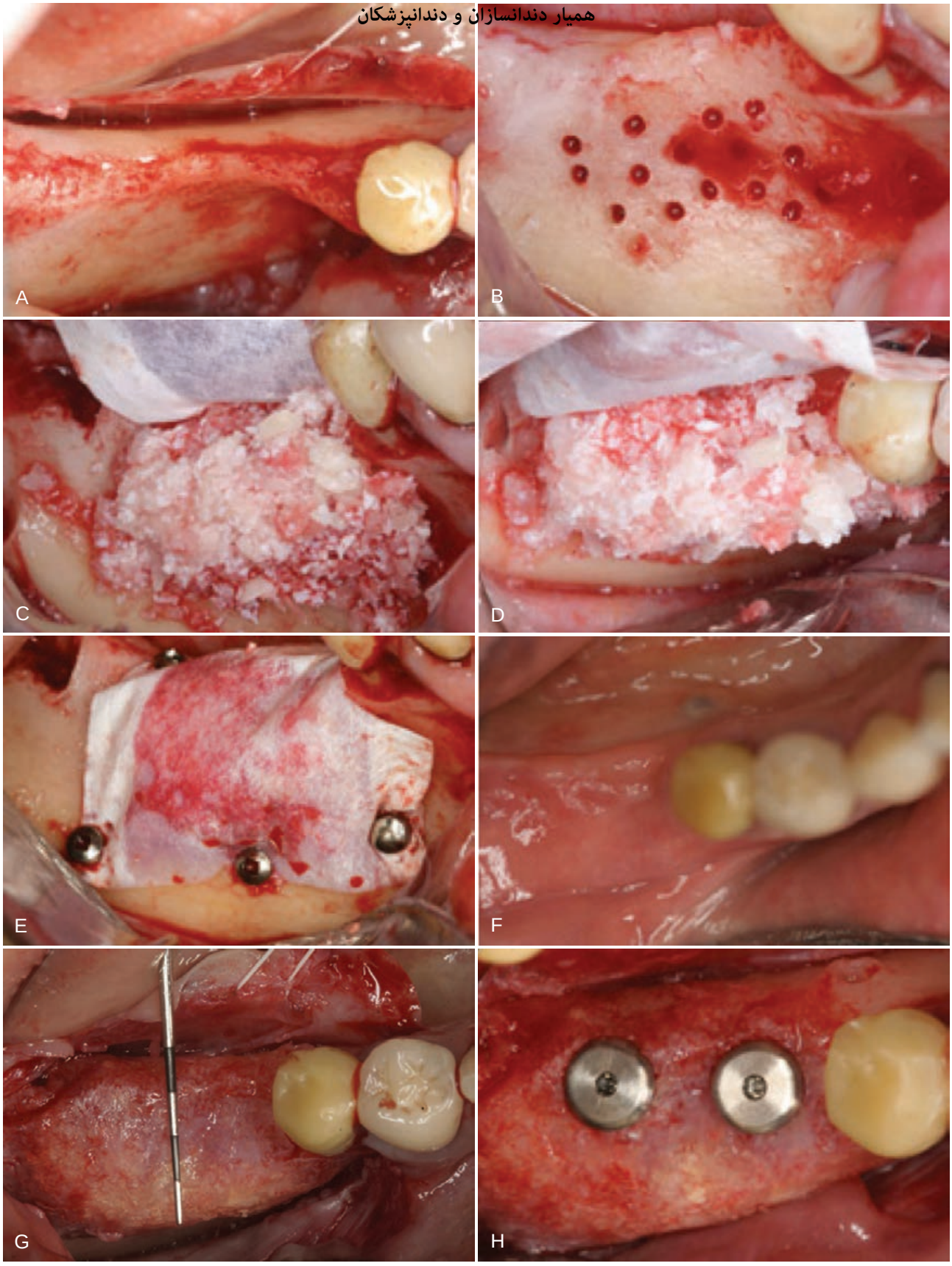

Figure 72-3 Horizontal bone augmentation with expanded polytetrafluoroethylene (ePTFE) barrier membrane. Bone graft consists of a mixture of autogenous particles harvested from the alveolar ridge and Bio-OssOsteohealth Company, A Division of Luipold Pharmaceuticals, Inc., Shirley, NY. **A,** Partially edentulous posterior mandible with narrow buccolingual dimensions. **B,** Cortical perforations made in buccal bone with small round bur to enhance blood supply to grafted area. **C,** Placement of ePTFE barrier membrane and mixture of autogenous and Bio-Oss particulate bone graft. **D,** View of C from occlusal perspective to visualize horizontal augmentation. **E,** Barrier membrane secured to native bone with fixation screws. **F,** Clinical photograph of healed site prior to membrane removal. Soft tissue closure maintained throughout healing without membrane exposure. **G,** Clinical photograph of healed ridge after membrane removal. Dimensions of alveolar ridge are significantly wider as demonstrated with periodontal probe. **H,** Two standard-diameter implants placed in "prosthetically-driven" ideal position. Notice there are no implant exposures. *(Images courtesy Dr. Istvan A. Urban, Budapest, Hungary.)*

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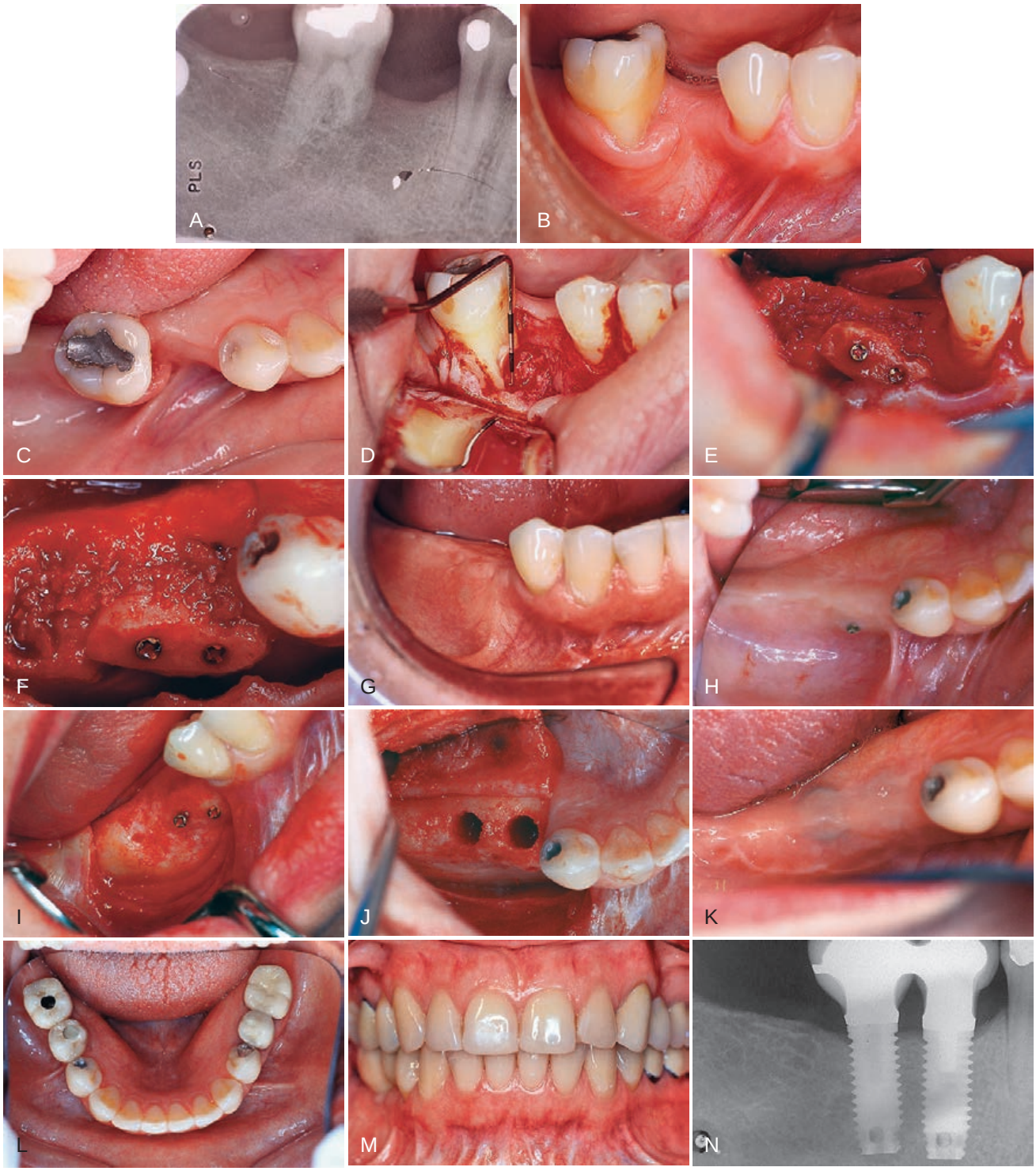

Figure 72-4 Use of a monocortical block graft to reconstruct a horizontal deficiency in the posterior right mandible. **A,** Periapical radiograph shows missing tooth #29 and damaged (i.e., cut) mesial root #30. **B** and **C,** Labial and occlusal views, respectively, of site reveal deficient alveolar ridge on buccal side of #29. **D,** Full-thickness flap reflection reveals the extent of missing bone in the buccal aspect of site #29, as well as the periodontal defect and damaged mesial root #30. **E** and **F,** Autogenous monocortical bone block graft secured to native alveolar bone with fixation screws. **G,** Good tissue healing after block graft, with evidence of a widened alveolar ridge. **H,** After 6 months of healing, posterior fixation screw is observed protruding through the mucosa. **I,** Full-thickness flap reveals that bone resorption has resulted in exposure of part of the fixation screw. **J,** Osteotomy prepared for wide-diameter implants, taking care to avoid making the labial bone "graft" too thin. **K,** Complete closure and good healing of wound after implant placement. **L** and **M,** Clinical photographs of completed restorations. **N,** Final radiograph shows good restoration contours on wide-diameter implants.

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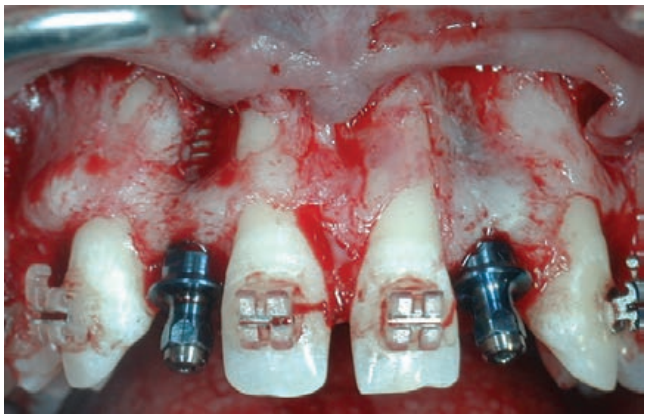

Figure 72-5 Fenestration defect observed in very thin maxillary anterior sites. Implants placed in the maxillary lateral incisor positions demonstrating fenestration defects due to the concavities in the apical area. Notice that the natural teeth (centrals and cuspids) also demonstrate fenestrations. Bone will grow across this type of defect in a very predictable manner because the implants are stable; the defect is small and surrounded by bone.

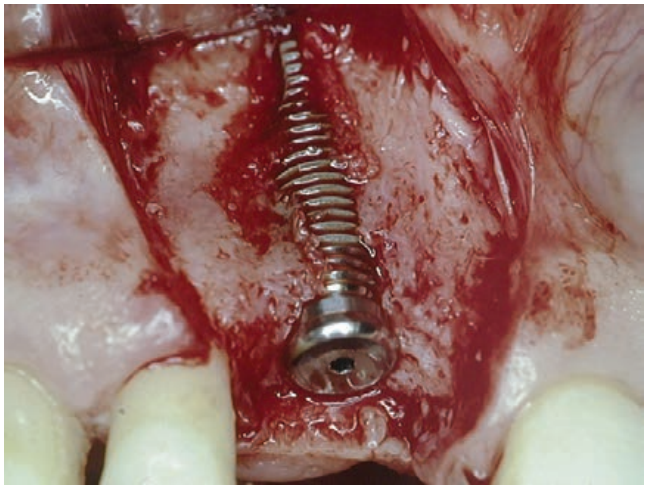

Figure 72-6 Dehiscence defect observed during placement of an implant in the maxillary central incisor position. Note that the implant is completely surrounded by existing native bone except for the facial surface, which is exposed. Bone will grow across this type of defect in a very predictable manner because the implant is stable and the exposed surface is relatively flat with bone on all surfaces.

reported a membrane exposure rate of 29% but noted little adverse effect on the bone regeneration.

With respect to ridge preservation, Becker et al[6] recently reported the effect of barrier membranes and autogenous bone grafts on the preservation of ridge width around implants. They evaluated the ridge width around 76 implants in 61 patients from a case series database. Three groups were compared, including 34 implants treated with GBR (barrier membranes), 27 implants treated with autogenous bone grafts, and 15 implants placed without the need for ridge preservation/augmentation procedures (control group). The results revealed that implant sites treated with barrier membranes and autogenous bone grafts lost an average of 0.1 mm and 0.8 mm, respectively, more than the no augmentation group.

Another study evaluated the possibility of regenerating bone around implants placed in extraction sockets.[30] Bone augmentation was achieved with human DFDBA particles mixed with tetracycline packed around the exposed implant surfaces. Implants and graft material were covered with e-PTFE barrier membranes with complete flap closure for 4 to 6 months. The results demonstrated complete bone regeneration in all cases except when barrier membranes were prematurely exposed and removed. One-year posttreatment histologic evaluation of regenerated bone revealed remnants of the DFDBA graft particles in direct contact with vital bone tissue (woven and lamellar bone). Osteoblasts were observed and appeared to be actively engaged in bone formation adjacent to graft particles. Figure 72-7, *L* and *M*, demonstrates a simultaneous GBR and implant placement in a dehiscence defect.

Complications

Bone augmentation procedures used to increase the bone volume in deficient alveolar ridges have been successful and have enabled the placement of implants into prosthetically-driven positions.[51] Unfortunately, these augmentation procedures carry an increased risk of morbidity and can require secondary surgeries to correct problems resulting from the procedure.[60] The subsequent corrective surgeries required to correct problems add surgical time and complexity to the implant therapy.

Surgical complications are reported for a variety of bone reconstructive techniques.[15] A review assessed the number and types of complications associated with bone reconstructive procedures for endosseous implants.[56] The review of literature (1976–1994) included 2315 implants in 733 autogenous block, particulate, and various other bone graft materials. Complications reported included bleeding, postoperative infection, bone fracture, nerve dysfunction, perforation of the mucosa, loss of a portion of the bone graft, pain, decubital ulcers, sinusitis, and wound dehiscence. Wound dehiscence seemed to have the most deleterious effect on implant survival. This finding emphasizes the importance of flap management.

Typical findings include less bone fill with early exposure and membrane removal versus retaining the membrane without exposure for 6 to 8 months.[25,53] Buccolingual ridge deficiencies were treated in a prospective study involving 19 patients using ePTFE membranes and miniscrews as fixation and tenting devices.[32] The group of defects, which healed uneventfully, yielded on reentry a 90% to 100% bone regeneration compared with the maximal volume of the space defined by the membrane placement. In the exposed-membrane group, the percentage of regenerated bone ranged from 0% to 62%. When a late membrane removal was performed (3 to 5 months postsurgically), the regeneration varied between 42% and 62%. The authors concluded that the length of membrane healing and size of the defect played a significant role in the amount of new bone formation.[32]

Other authors have reported successful bone fill in situations in which the membranes had to be removed because of an early exposure.[42,52] A significantly greater fill of the osseous defects at the grafted sites was noted. The authors concluded that the regeneration of bone around the implants appeared most dependent on the anatomy of the bony defect at the time of implant placement.

Although the effect or amount of regenerated bone with regard to membrane exposure is somewhat contradictory, the goal should be to keep the membranes covered during the healing period so that the risk of infection and soft tissue and esthetic problems can be minimized or eliminated. Again, the importance of flap management for ridge augmentation procedures should be stressed. See Chapter 77 for more information and details regarding surgical complications and failures.

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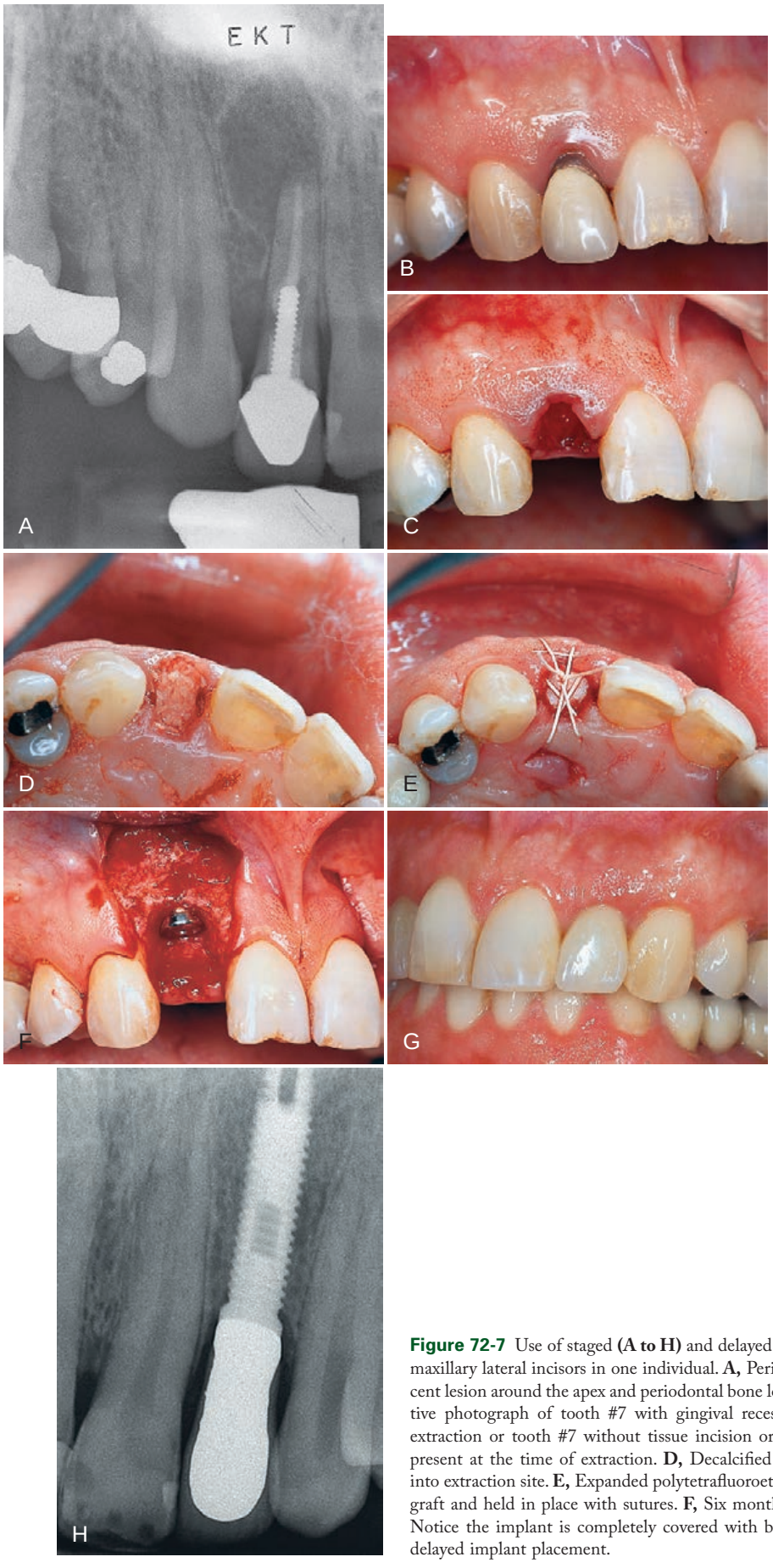

Figure 72-7 Use of staged **(A to H)** and delayed **(I to O)** implant placement after extraction of two maxillary lateral incisors in one individual. **A,** Periapical radiograph of tooth #7 with a large radiolucent lesion around the apex and periodontal bone loss along the distal interproximal area. **B,** Preoperative photograph of tooth #7 with gingival recession and marginal inflammation. **C,** Atraumatic extraction or tooth #7 without tissue incision or tissue elevation. Palpation reveals no facial bone present at the time of extraction. **D,** Decalcified freeze-dried bone allograft (DFDBA) condensed into extraction site. **E,** Expanded polytetrafluoroethylene (ePTFE) barrier membrane positioned over graft and held in place with sutures. **F,** Six months after the extraction/graft, the implant is placed. Notice the implant is completely covered with bone. **G,** Final restoration. **H,** Final radiograph of delayed implant placement.

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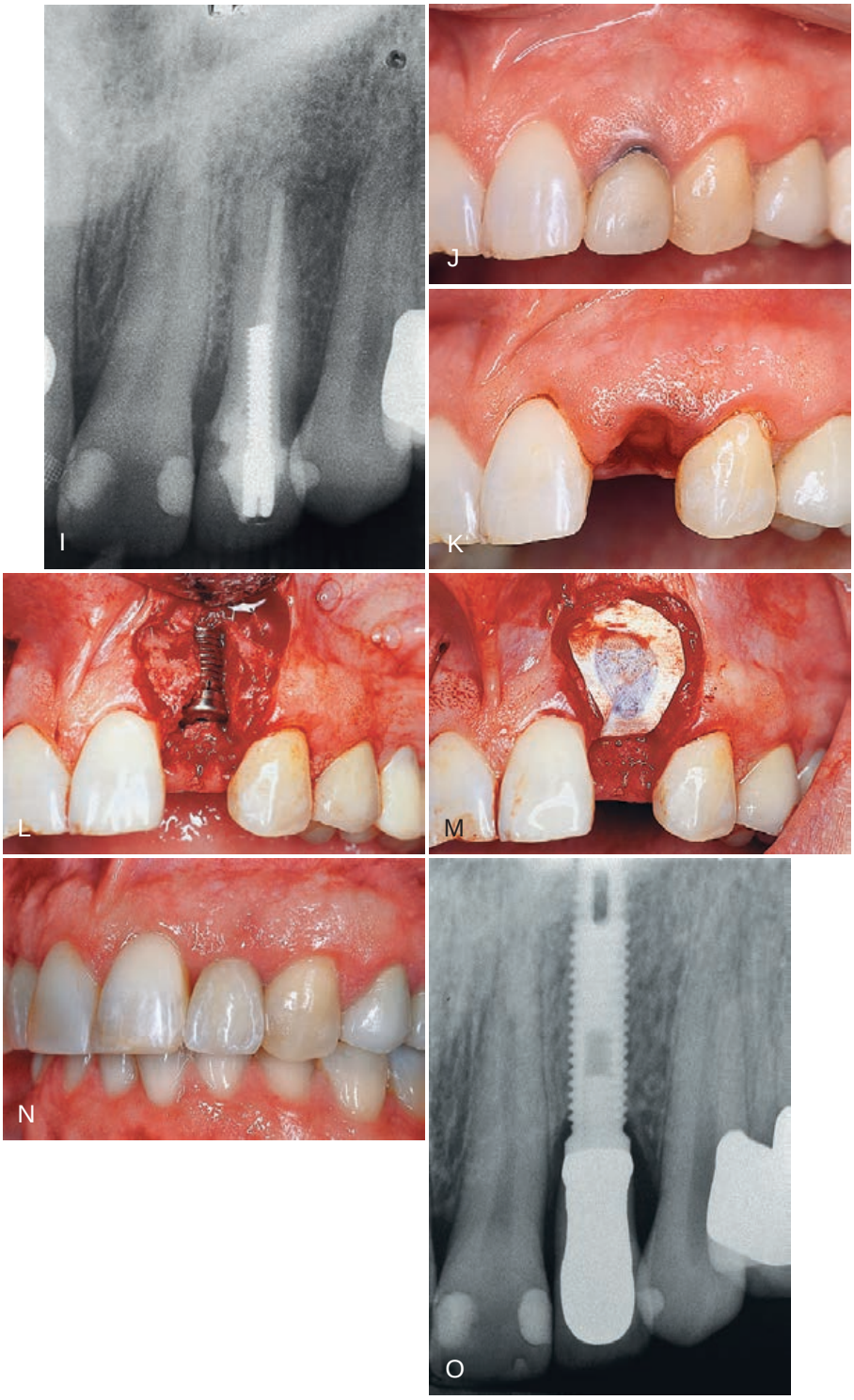

Figure 72-7, cont'd **I,** Periapical radiograph of tooth #10. **J,** Preoperative photograph of tooth #10 with exposed gingival margin. **K,** Atraumatic extraction of tooth #10 without tissue incision or tissue elevation. Palpation reveals no facial bone present, and dehiscence is expected. **L,** Two months after extraction, the implant is placed with dehiscence defect. **M,** Guided bone regeneration (GBR) accomplished with ePTFE barrier membrane positioned over the dehiscence. **N,** Final restoration. **O,** Final radiograph. *(Figures A, B, C, F, G, J, M, N, and O from Klokkevold PR et al:* Pract Periodont Aesthet Dent *11:603, 1999.)*

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ALVEOLAR RIDGE PRESERVATION/ MANAGEMENT OF EXTRACTIONS

Because tooth extraction (or tooth loss) often results in alveolar ridge resorption or collapse, preservation of bone volume at the time of extraction is a desirable goal. Most bone loss after extraction occurs in the first 6 to 24 months.[10] Therefore, when clinicians are afforded the opportunity to intervene at the time of extraction, preservation of alveolar bone should be initiated. A conservative approach to the management of extraction sites can eliminate or significantly reduce the necessity of advanced bone augmentation procedures.

When extracting a tooth and preparing for implant placement, alveolar bone resorption should be prevented. Experimental animal studies have shown that the use of a barrier membrane enhances the predictability of bone fill in the extraction site and therefore maintains original bone volume when compared with mucoperiosteal flap coverage alone.[7] Clinical studies have also demonstrated the benefits of a regenerative approach to tooth extraction.[34,35,41] These authors found that a nonresorbable barrier membrane resulted in minimal resorption of alveolar ridge size and shape.

Although earlier studies have proposed the concept of treating extraction sites without flap closure (i.e., an exposed membrane used to cover the graft), more recent studies concluded that complete wound closure over the physical barrier might be associated with greater bone fill.[5,53] The decision about whether to advance a flap to achieve wound closure must be weighed against the soft tissue changes that will be created and may need to be corrected (i.e., mucogingival junction discrepancies and esthetic problems).

The timing of implant placement relative to the time of extraction has been widely debated. Depending on the quantity, quality, and support of existing bone, as well as the preferences of the clinician and patient, the placement of implants after tooth extraction can be immediate, delayed, or staged. By definition, *immediate* implant placement occurs at the time of extraction. Delayed implant placement is performed approximately 2 months after extraction to allow for soft tissue healing. *Staged* implant placement allows for substantial bone healing within the extraction site, which typically requires 4 to 6 months or longer.

Tooth extraction is managed with an atraumatic surgical technique that uses a narrow, flat instrument (e.g., Periotome, Hu-Friedy, Chicago) directed apically into the sulcus to sever the periodontal ligament and slightly expand the adjacent periodontal tissues. The tooth is elevated and removed with forceps using a gentle, rotational movement. New techniques for atraumatic extraction are described in Chapter 75. Buccolingual forces are avoided to prevent damaging the integrity of the labial bone. No incisions are made, and care is taken to avoid soft tissue reflection. In this manner, soft tissues maintain their structural anatomy, and the periosteum (blood supply to the bone) remains intact. If the tooth has multiple roots, curved roots, or other anatomic features that make removal difficult, it may be necessary to cut the tooth using a high-speed rotary drill or other cutting device and remove it in smaller pieces. It is important to cut only tooth structure and avoid cutting (overheating) bone when using high-speed rotary drills. The bone within the extraction site is completely debrided of soft tissue with surgical curettes. After debridement, the extraction site is thoroughly irrigated with sterile saline. Finally, the clinician can evaluate the bone level and socket anatomy to determine whether to bone-graft the site and when to place the implant (immediate, delayed, or staged placement).

Delayed Implant Placement

Delayed implant placement shares some advantages of immediate implant placement, including extraction site preservation, and offers additional advantages. Unlike immediate implant placement, which is deficient of soft tissue for coverage, the delayed–implant placement technique allows time for soft tissue healing to close the wound.[23] The delayed-placement technique still reduces the length of treatment by several months because it is not necessary to wait for complete bone healing. Furthermore, because bone formation is active within the first few months after tooth extraction, the delayed technique may facilitate more osteogenesis adjacent to the implant.

The primary advantage of delayed implant placement is that by allowing for soft tissue healing and closure of the extraction site, mucogingival flap advancement is not necessary. This alleviates the need for additional surgeries to correct mucogingival discrepancies. Delayed implant placement also allows time for resolution of infections that may have been present within the extraction site.

As with immediate implant placement, similar limitations of bone support and implant stability exist for delayed placement. The normal osseous healing that occurs within the first 2 months does not significantly affect the anatomy of the alveolar bone. Therefore limitations in bone support after 2 months of healing are similar to those that exist at the time of extraction.

Staged Implant Placement

Staged implant placement allows adequate time for osseous healing. This may be complete osseous healing of an extraction site without a bone graft (if circumferential bone support is good) or with a bone graft. Staged implant placement, by definition, allows for complete hard and soft tissue healing and permits the placement of implants into healed bone sites with adequate coverage by hard and soft tissues.[51] This eliminates the necessity of mucogingival flap advancement, allows for the resolution of preexisting infections, and prevents soft tissue invasion. Furthermore, by using an extended healing period, the grafted bone also has the opportunity to become vascularized. Bone grafts performed simultaneously with implant placement do not share this advantage.

The primary disadvantage of staged implant placement is the length of time required for bone healing.

Delayed Versus Staged Technique

Delayed and staged techniques for implant placement are demonstrated here in one individual using two extraction sites with similar bone morphologies in the anterior maxilla (see Figure 72-7). Both techniques facilitate the esthetic placement of implants into prosthetically-driven positions. Delayed and staged approaches maintain alveolar bone volume, reduce the need for advanced bone augmentation, and eliminate the need for subsequent mucogingival surgery. The timing and management of delayed versus staged implant placement is described in the next section.

To decide which implant placement method to use, the quantity and location of bone surrounding the tooth should be assessed. Once the patient has been anesthetized, a periodontal probe can be used to "sound" for the level of bone support through the soft tissue. Using this method, the bone levels surrounding the tooth can be mapped. Bone support that surrounds the extraction site can also be evaluated and confirmed after tooth removal by palpation, probing, and direct (internal) visualization.

If the tooth to be extracted has sufficient bone support on all surfaces, the extraction site can be expected to fill with bone without additional augmentation procedures, except when the

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labial bone is very thin. A simple extraction followed by a healing period of 4 to 6 months could be sufficient for complete osseous healing. Subsequently, an implant could be placed in the usual manner without the need for bone augmentation. Conversely, if little or no bone exists on the labial surface, it should be anticipated that the site would require bone augmentation to facilitate placement of the implant. In this case, bone grafting at the time of extraction can be used to maintain the alveolar ridge dimensions occupied by the tooth.

Immediate Implant Placement

The primary advantage of immediate implant placement is the reduction of the healing time, which translates to an earlier restorative time (i.e., shorter time to completion for the patient).[33,39,50,59] Because the implant is placed at the time of extraction, the bone-to-implant healing begins immediately with extraction site healing. Another advantage is that the normal bone healing, which generally occurs within the extraction site, takes effect around the implant. This bone-forming activity may enhance the bone-to-implant contact compared with an implant placed in a less osteogenically active site.

Possible disadvantages of immediate implant placement include the need for subsequent mucogingival surgeries to correct tissues moved by repositioned flaps and the need for bone grafting to fill extraction site defects around the implant. If inadequate bone exists to stabilize the implant, immediate implant placement is not recommended.

When a two-stage implant is placed at the time of tooth extraction, the mucogingival flap is advanced, with releasing incisions, to cover the implant completely (exception: one-stage implants). It may also be necessary to graft bone into the extraction site in areas that do not contact the implant to avoid soft tissue invasion around the implant.[50] A 1-year study of 49 immediate extraction site implants treated by a membrane alone demonstrated a 93.6% bone fill. After 1 year (postloading), the implant success rate was 93.9%.[5]

Although some have advocated submerging implants placed in extraction sockets with flap advancement,[23] others have demonstrated success with a nonsubmerged approach. Implants may be placed in extraction sockets along with bone augmentation without flap advancement using a one-stage implant placement approach. Clinical studies evaluating the outcome of bone augmentation around implants placed in extraction sockets reveal good bone fill.[31] The placement of 21 transmucosal implants in immediate extraction sites treated with a barrier membrane were tested for the implant success rate and the percentage of bone fill. Of 21 transmucosal implants, 20 yielded complete bone fill and coverage of the entire plasma-coated implant surface.

A clinical report on the use of resorbable collagen membranes around extraction site implants demonstrated a variable degree of bone fill in nine patients.[44] More clinical review of the use of resorbable membranes for GBR is required because evidence is insufficient to evaluate the predictability.

In a study of 30 patients, the use of autografts alone in 54 extraction sites was highly effective for simultaneously placed implants completely within the envelope of bone.[3] The study showed that extraction sites, including those with a buccal dehiscence, could be treated with autografts alone. Because ungrafted sites were not evaluated, the absolute need to graft small defects adjacent to implants was not ascertained by this study. In another study, implants placed in extraction sockets were tested for their potential to regenerate bone with allograft alone, a membrane alone, and a combination treatment.[19] Reentry confirmed 100% thread coverage in all but one implant in the "no-wall" group treated with DFDBA alone. A clinical study of five patients evaluated different treatment modalities for extraction site implants together with bone graft combinations.[54] This small study supported the concept that "non–space-making defects" are best treated with a combination of barrier membrane and an autograft or allograft as compared to treatment with a nonreinforced membrane alone (without a graft).

Immediate placement of an implant into the extraction socket in a one-stage approach along with immediate provisionalization is perhaps the best way to manage the hard and soft tissues following extraction (Figure 72-8, *A* to *J*). The immediate placement of a provisional restoration is the best way to support the soft tissues (papilla and marginal gingiva) following tooth extraction.

SCIENCE TRANSFER

Implants may be placed immediately into extraction sites when there is sufficient bone apically and horizontally to stabilize and cover the implant surfaces. This gives the simplest method to replace an extracted tooth and to maintain soft tissue contours. In cases where there is insufficient bone for this procedure, a variety of bone graft materials can be placed in the socket, covered with a resorbable collagen membrane and sutures placed to hold the soft tissues without the need for complete flap closure. Implants can then be placed in the augmented site in 4 to 6 months.

Bone dehiscences or fenestrations present during implant placement are the easiest defects to resolve. These are successfully treated with a variety of bone graft materials used in conjunction with resorbable barrier membranes.

Localized ridge augmentation in a horizontal direction is generally carried out with autogenous veneer bone grafts harvested from the mandibular ramus or with bone graft materials covered with a titanium reinforced Teflon membrane with water tight flap closure being essential. These procedures may result in some postoperative morbidities including pain, swelling, and bruising.

Vertical augmentation of deficient alveolar ridges using bone grafts and membranes is more difficult and less predictable than techniques to improve the width of the alveolus. For this reason, bone distraction techniques are becoming more often applied to these difficult cases. Bioactive molecules such as BMP II are currently being evaluated in clinical trials and will add additional bone forming capacity to current surgeries.

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Figure 72-8 Immediate implant placement after extraction of a maxillary first premolar. **A,** Preoperative clinical photograph of tooth #12, which fractured and was deemed nonrestorable. **B,** Periapical radiograph of tooth #12. It is root canal–treated and has a full-coverage porcelain-fused-to-metal (PFM) crown. **C,** Atraumatic extraction of tooth #12 without incisions or tissue elevation. **D,** Extracted tooth. **E,** Palpation and examination with probe reveals intact bony walls around the extraction socket. The facial bone wall is observed to be about 2 mm below the facial gingival margin. **F,** The palatal aspect of the extraction socket is prepared, and a tapered implant is placed to emerge through the central fossa. Autogenous bone chips (harvested from the maxillary tuberosity) are condensed into the labial aspect of the extraction socket to support the facial bone and soft tissue contours. **G,** An immediate provisional restoration is fabricated (indirectly in the laboratory) and delivered at the time of implant placement. The palatal cusp is not replaced in this provisional to avoid function and the occlusion is checked for light centric contacts only. **H,** Facial view of provisional restoration at the time of delivery. Notice how the interdental papilla and facial gingival margin is well supported by the contours of the provisional restoration. **I,** Final restoration at time of delivery (approximately 4 months after implant placement). **J,** Final radiograph of definitive restoration.

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CONCLUSION

Localized bone augmentation procedures allow clinicians to reconstruct horizontal alveolar ridge deficiencies and replace missing teeth with dental implants in a prosthetically-driven position with natural appearance and function. In many cases, implants can be placed simultaneously with the augmentation procedure. In cases of advanced bone resorption, ridge augmentation before implant placement may be a better choice. Bone augmentation procedures can also be used to preserve alveolar dimensions following tooth extraction. If adequate bone exists to stabilize an implant, these procedures may be combined. The predictable outcome of these procedures depends on several biologic principles that must be followed. Diagnosis, treatment planning, careful execution of the surgical treatment, postoperative follow-up, and appropriate implant loading are all important factors in achieving success.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 73

Advanced Implant Surgical Procedures

Perry R. Klokkevold, Istvan A. Urban, and David L. Cochran

CHAPTER OUTLINE

MAXILLARY SINUS ELEVATION AND BONE AUGMENTATION
Indications and Contraindications
Surgical Procedures for Sinus Elevation
Risks and Complications
SUPRACRESTAL/VERTICAL BONE AUGMENTATION
Guided Bone Regeneration and Augmentation
Distraction Osteogenesis
GROWTH FACTORS IN BONE AUGMENTATION
Bone Morphogenetic Proteins
Autologous Platelet Concentration
CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures may be out of numeric order in this printed chapter.

The high predictability of endosseous dental implants has led to routine use and an expectation for success. However, the ultimate success for any patient or any particular implant relies on several factors, the most important of which is the *availability of bone*. The loss of teeth, whether caused by disease or trauma, can result in severe deficiency of the alveolar bone. Horizontal bone deficiencies are managed quite predictably with localized bone augmentation procedures (see Chapter 72). However, vertical bone deficiencies are much more challenging. The edentulous posterior maxilla is challenging as the result of a general lack of bone volume and the omnipresent poor bone quality of the area; that is, posterior maxillary bone often consists of a thin cortical shell filled with sparse trabecular bone. Edentulous sites, in any anatomic location, that have suffered significant vertical alveolar bone loss are especially challenging to reconstruct.

This chapter reviews advanced surgical procedures used to treat the most challenging type of bone loss, which is *a deficiency in vertical bone height*. Maxillary sinus elevation and bone augmentation, vertical bone augmentation, and distraction osteogenesis are reviewed. The role of growth factors in bone augmentation procedures is also discussed.

MAXILLARY SINUS ELEVATION AND BONE AUGMENTATION

Rehabilitation of the edentulous posterior maxilla with endosseous dental implants often represents a clinical challenge because of insufficient bone volume resulting from *pneumatization* of the maxillary sinus along with resorption or loss of alveolar crestal bone. Before the utilization of bone augmentation procedures, patients with missing teeth and deficient bone in the posterior maxilla could only be rehabilitated with removable prostheses, short implants, or cantilevered restorations (i.e., supported by adjacent teeth or implants). Historically, the failure rate for implants in the posterior maxilla has been significantly higher than failure rates for implants in all other anatomic locations.[16] Therefore procedures such as the maxillary sinus elevation and bone augmentation are needed to increase the amount of vertical bone height in the posterior maxilla for the placement of implants.

In 1980, Boyne and James[3] first described a procedure to graft the maxillary sinus floor with autogenous marrow and bone for placing an implant (blade type). Access to the maxillary sinus was gained through a "Caldwell-Luc" procedure (i.e., an opening into the maxillary sinus created at the anterior-superior aspect). Since then, several other techniques have been described, including variations on the lateral window osteotomy and a variety of techniques to lift the sinus floor from a crestal approach.

Various bone graft materials have been used to augment the maxillary sinus. The 1996 Consensus Conference on Maxillary Sinus Bone Grafting reviewed available data and concluded that allografts, alloplasts, and xenografts, alone or in combination with autogenous bone, can be effective as bone substitute graft materials for sinus bone augmentation.[17] More importantly, it was concluded that the sinus graft procedure with implant placement is a highly

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predictable and effective therapeutic modality for the rehabilitation of the posterior maxilla. Sinus floor elevation with bone augmentation of the maxillary sinus is now a well-accepted procedure used to increase bone volume in the posterior maxilla. Numerous reports have validated the safety and efficacy of this procedure.[6,9,11] Implant success rates are equal to or better than that of implants placed in non-grafted maxillary bone (i.e., areas of the posterior maxilla with adequate height of existing native bone). Thus bone augmentation of the maxillary sinus is a viable option for the vertically deficient posterior maxilla in which the interocclusal dimension is normal or only moderately increased.

Indications and Contraindications

As with any therapeutic procedure, treatment success depends on appropriate patient selection, careful evaluation of the anatomy, identification and management of any pathology, sound surgical procedures, and appropriate postsurgical management. The primary indication for maxillary sinus elevation and bone augmentation, specific for the placement of endosseous dental implants, is an alveolar bone height in the posterior maxilla that is deficient (e.g., less than 7 mm of existing vertical bone height). Other factors that must be considered include the health of the patient, the condition of the remaining dentition, and the likelihood of a beneficial outcome. A thorough evaluation of the patient and the judgment of the clinician ultimately determines whether the procedure is indicated for any particular individual.

Contraindications to maxillary sinus elevation and bone augmentation are similar to contraindications for other surgical procedures, with the added consideration of the maxillary sinus (Box 73-1). Patients must be in good general health and free of diseases that affect the maxilla or maxillary sinus. Local factors that are considered contraindications to maxillary sinus elevation and bone augmentation include the presence of tumors, maxillary sinus infection, severe chronic sinusitis, scar or deformity of the sinus cavity from previous surgery, dental infection, severe allergic rhinitis, and chronic use of topical steroids. Systemic contraindications to treatment include radiation therapy, uncontrolled metabolic disease (e.g., diabetes), excessive tobacco use, drug or alcohol abuse, and psychologic or mental impairment.

Surgical Procedures for Sinus Elevation

The goal of sinus elevation and bone augmentation is to lift the schneiderian membrane from the floor of the sinus, raising it up into the sinus cavity to create a new, more superiorly located sinus floor with a space between it and the deficient alveolar ridge. The newly created space can then be filled with bone (or a suitable bone substitute material) to increase the total vertical height of bone in the posterior maxilla for the placement of endosseous implants.

The maxillary sinus bone augmentation procedure was first described in the 1960s by Boyne (unpublished oral presentations to United States [US] Navy Dental postgraduates, 1965–1968) and originally used as a preprosthetic surgical procedure for patients with large tuberosities and pneumatized sinuses.[2] To reduce the size of the tuberosity without creating an oral-antral defect, bone was grafted into the sinus cavity to increase the volume of bone within the maxillary tuberosity. After a period of healing, the tuberosity was able to be reduced surgically from the alveolar ridge crest. As stated earlier, Boyne and James[3] were the first to describe the use of the maxillary sinus bone-grafting procedure for placement of an implant (blade type) to retain a prosthesis. Greater use and further development of this procedure evolved along with the success of endosseous dental implants and the desire to replace missing posterior maxillary teeth with implant-retained restorations.

The techniques used for sinus elevation and bone augmentation are primarily differentiated by the anatomic location of the osteotomy used to gain access to the maxillary sinus. Specifically, four different anatomic locations have been described: (1) the superior lateral wall, or "Caldwell-Luc," opening, which is located just anterior to the zygomatic arch; (2) the middle lateral wall opening, which is located midway between the alveolar ridge and the zygomatic arch; (3) the inferior lateral wall opening, which is located at the level of the alveolar ridge; and (4) the crestal osteotomy approach, which is an opening through the alveolar bone crest superiorly toward the floor of the sinus. At present, the most common procedures used for sinus elevation and bone augmentation are the lateral wall antrostomy (middle or inferior approach) and the crestal approach osteotomy.

Presurgical Evaluation of Maxillary Sinus. Presurgical evaluation of the maxillary sinus is primarily accomplished using radiographic examination techniques (Figure 73-1). Several observations about the anatomy can be made with a periapical or panoramic projection, but the internal anatomy is more accurately assessed with a three-dimensional scan, such as computed tomography (CT) or cone-beam CT (CBCT) scan. The maxillary sinus should be evaluated for any pathology, masses, or the presence of septa. If three-dimensional scans are available, the lateral wall should also be evaluated for the presence of medium or large intraosseous vascular channels (see Figure 53-15). Medium- to large-sized vessels occasionally traverse the lateral wall of the maxillary sinus, and identifying them is helpful in avoiding a bleeding problem during surgery (see Chapter 53).

Simultaneous Implant Placement. Simultaneous implant placement is possible with sinus elevation and bone augmentation procedures as long as the implant can be stabilized in the desired location with the existing native bone (Figure 73-2, online).

To view Figure 73-2, please visit the companion Expert CONSULT website at www.expertconsult.com.

It has been suggested that a minimum of 5 mm of existing native bone in the alveolar crest is required for simultaneous implant placement. However, some clinicians claim that it is

BOX 73-1 Contraindications for Maxillary Sinus Elevation and Bone Augmentation

Local Factors
Tumors or pathologic growth in the sinus
Maxillary sinus infection
Severe chronic sinusitis
Surgical scar/deformity of sinus cavity
Dental infection involving or in proximity to sinus
Severe allergic rhinitis/sinusitis
Chronic topical steroid use

Systemic Factors
Radiation therapy involving the maxillary sinus
Metabolic disease (e.g., uncontrolled diabetes mellitus)
Excessive tobacco use
Drug/alcohol abuse
Psychologic/mental impairment

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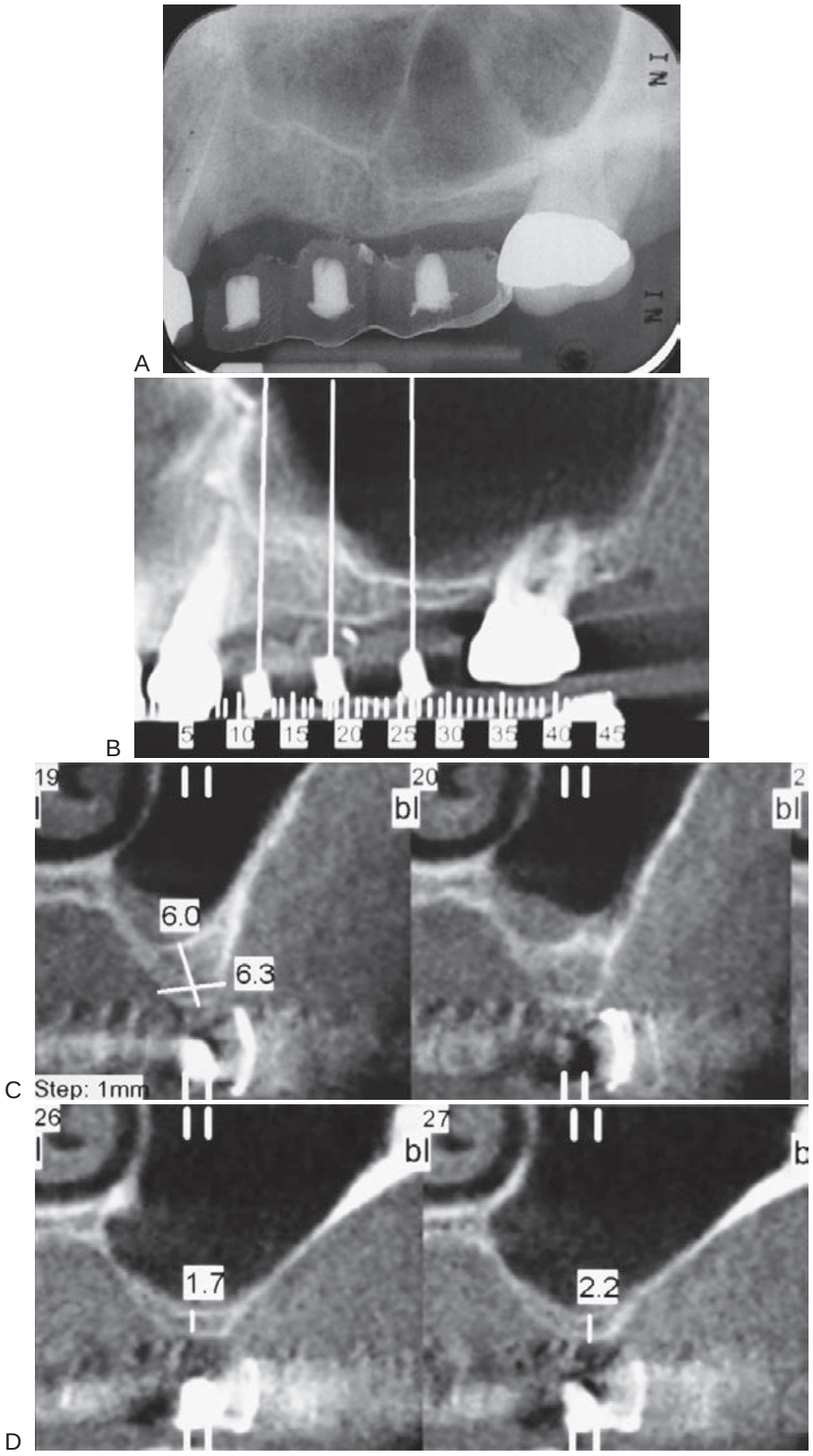

Figure 73-1 Presurgical evaluation of the maxillary sinus. **A,** Periapical radiograph. **B,** Panoramic projection from cone-beam scan. Note the presence of maxillary septa in the premolar region. **C,** Cross-sectional image in premolar region showing about 6 mm of bone height and presence of maxillary septa. **D,** Cross-sectional image in molar region showing about 2 mm of bone height.

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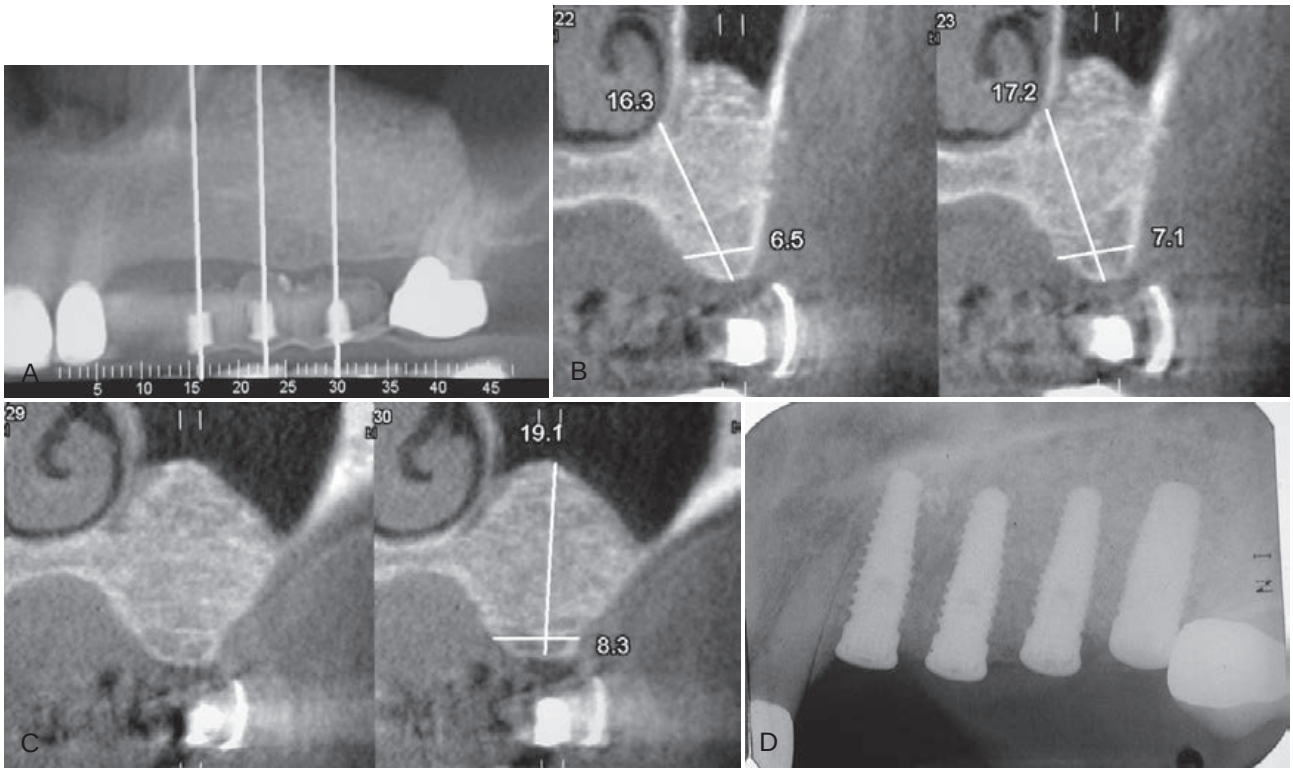

Figure 73-3 Staged implant placement after sinus elevation and bone augmentation procedure. Same patient as in Figure 73-1. See preoperative radiograph and preoperative cross-sectional images. **A,** Postsurgical panoramic view of bone-augmented maxillary sinus. The maxillary left cuspid has been extracted because of a vertical fracture. **B,** Postsurgical cross-sectional image in premolar region demonstrating more than 17-mm vertical bone height. **C,** Postsurgical cross-sectional image in molar region demonstrating 19.1-mm vertical bone height. **D,** Postsurgical radiograph of implants placed in the previously grafted maxillary sinus (and cuspid site).

possible to place implants simultaneously with as little as 1 mm of remaining bone.[22,42] The most important factor in determining whether implant(s) can be placed at the time of sinus bone augmentation is the ability to achieve implant stability in the existing bone rather than any measure of bone height. Factors that influence implant stability include bone height, bone quality, precision of osteotomy preparation, and the surgeon's skill and experience. If inadequate native bone exists to place implants at the time of the bone augmentation procedure, they can be placed at a subsequent surgery after an appropriate healing period (Figure 73-3).

Bone Graft Materials. *Autogenous bone* is often considered the "gold standard" for bone augmentation because of its osteoconductive, osteoinductive, and osteogenic properties.[3] However, harvesting autogenous bone from intraoral or extraoral locations creates a second surgical site with additional morbidity. Numerous studies have demonstrated clinical success using many variations of bone graft materials and combinations.[17]

Several recent clinical studies and reports have attempted to evaluate the maxillary sinus augmentation procedures using a variety of bone-grafting materials, including autogenous bone from the iliac crest or oral cavity and bone substitutes such as freeze-dried demineralized bone, resorbable and nonresorbable hydroxyapatite, and xenografts. However, only a few studies have critically evaluated the long-term clinical outcome of this procedure, and most have used a small study population. Short- to long-term clinical studies of dental implants placed into grafted sinuses demonstrate an equivalent or higher survival rate as compared to implants placed in native maxillary bone (i.e., without the need of sinus augmentation).[17] The results of these studies support the clinical predictability of maxillary sinus augmentation procedures for the rehabilitation of the edentulous posterior maxilla with implant-supported prostheses.

The use of bone-substitute graft materials can reduce the morbidity introduced by a second surgical site while maintaining equally good implant success rates.[17] These materials form an osteoconductive scaffold for bone growth but do not have any osteoinductive properties. A possible exception is demineralized freeze-dried bone allograft (DFDBA). This material has demonstrated osteoinductive potential[40] but has not proved to be particularly advantageous in the maxillary sinus bone augmentation.[17,40] In fact, the bone volume gained with the use of DFDBA is less than that achieved with mineralized graft materials as a result of moderate postoperative shrinkage of DFDBA-grafted bone.

Crestal Osteotomy Technique. In cases with moderate bone height (e.g., 7 to 9 mm) that require limited sinus bone augmentation, a crestal approach to elevation may be desirable. The osteotome technique is a procedure that uses osteotomes (Figure 73-4, online) to compress bone (internally from the alveolar crest upward) against the floor of the sinus, ultimately leading to a controlled "inward fracture" of the sinus floor bone along with the schneiderian membrane, which should remain intact above the inward-fractured bone.

To view Figure 73-4, please visit the companion Expert CONSULT website at www.expertconsult.com.

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The osteotome sinus floor elevation (OSFE) technique was described by Summers.[33,34] It is considered to be a "conservative" approach to sinus elevation, but it is also a "blind" technique because it does not allow the operator to visualize the schneiderian membrane during the osteotomy. For this reason, it is a technique-sensitive procedure as well (i.e., the operator must "feel" the bone fracture and the membrane elevation). Verification of success can only be observed with radiographs and visualization of the bone graft material in the sinus cavity.

Procedure. An osteotomy site is prepared with a series of drills (e.g., initial drills used for implant site preparation) to a depth that is approximately 1 to 2 mm from the floor of the maxillary sinus. Osteotomes are used to increase compressive forces gradually against the floor of the sinus by adding incremental quantities of graft material until the floor of the sinus fractures inward (Figure 73-5). After the controlled inward fracture of the maxillary sinus floor, bone graft materials continue to be slowly introduced, through the osteotomy site and into the maxillary sinus, which continues to elevate the membrane and thus allows a vertical expansion of the bone height in a localized area of the maxillary sinus. Once the sinus membrane is elevated with bone graft material to the desired height, the implant osteotomy can be completed, with a final drill used to finish preparation of the site, and an implant can be inserted.

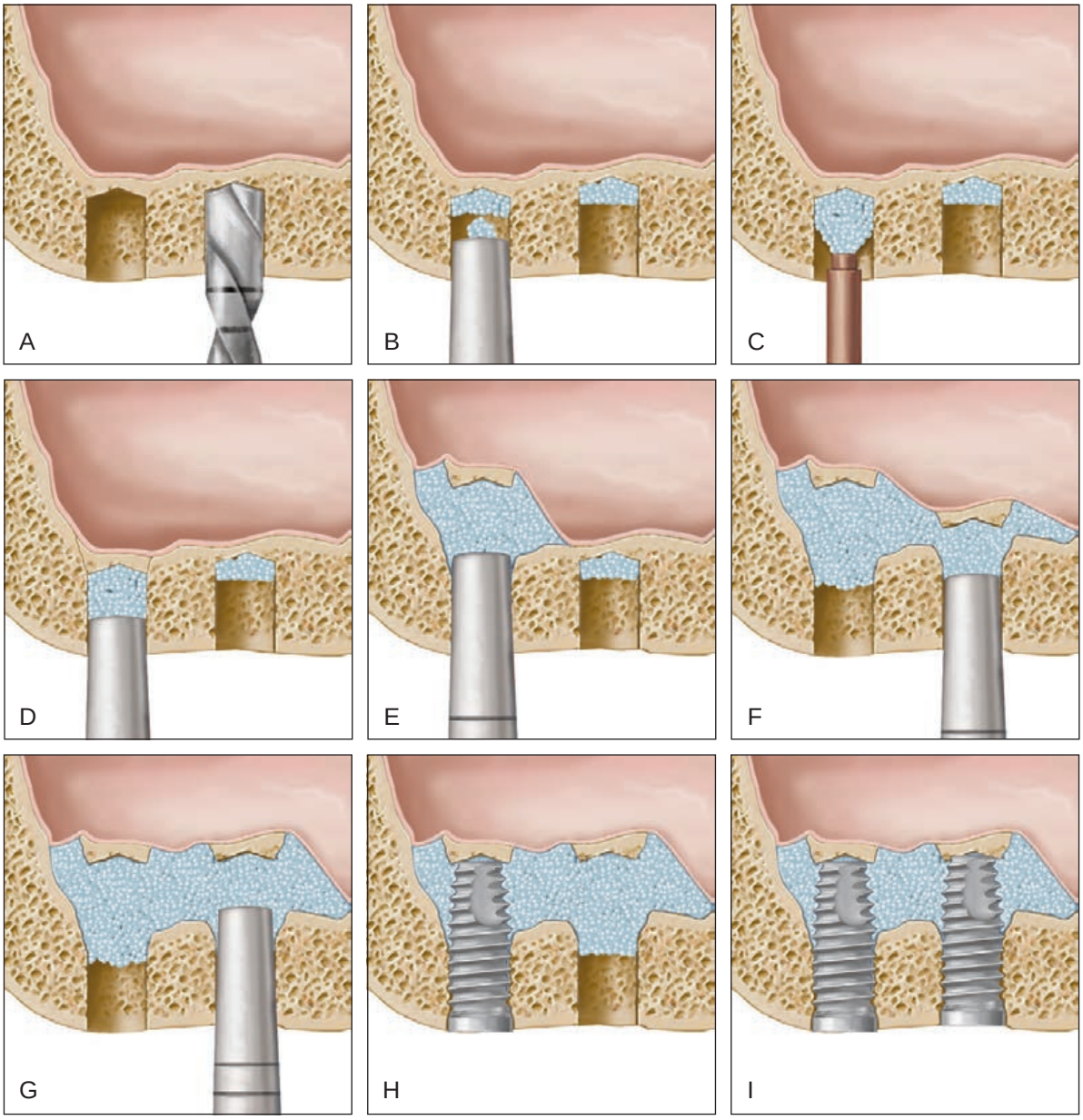

Figure 73-5 Illustration of the osteotome sinus floor elevation (OSFE) technique. **A,** Osteotomy prepared with drills to a depth that is near the maxillary sinus floor. **B,** Graft material introduced into osteotomy and condensed with osteotome. **C,** Additional bone graft material is added to the osteotomy. **D,** Bone graft continues to be condensed by osteotomes. **E,** This process is continued until floor of sinus is "fractured" up internally and the floor is lifted with the bone graft material. **F,** Continuation of process shown in **E** for second site. **G,** Bone graft material continues to be added gradually to both sites with osteotome condensation to elevate the schneiderian membrane away from the bone (maxillary sinus walls) until sufficient height and volume are created for the placement of implants. **H,** The coronal aspect of the osteotomy is carefully prepared for the placement of implants (instrumentation not shown) and the implant is placed. **I,** Final view of two implants placed in the grafted maxillary sinus using the OSFE technique.

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BOX 73-2 Clinician Comments on Use of Osteotome Technique

Clinical Perspective 1

The osteotome procedure involves repeated tapping of osteotomes with a mallet to create the necessary pressure to fracture the floor of the maxillary sinus. This tapping can be bothersome to some individuals, especially those patients who are not sedated for the procedure. The tapping procedure tends to be more bothersome for patients with dense cortical bone and for those with loose trabecular bone. In fact, a specific postoperative complication, called *benign paroxysmal positional vertigo* (BPPV), has been associated with the osteotome sinus elevation technique. During the osteotomy preparation and sinus floor elevation, the trauma induced by percussion of the osteotome with the surgical hammer, along with hyperextension of the neck during the operation, can displace otoliths in the inner ear and induce BPPV (see Chapter 77).

Clinical Perspective 2

The osteotome technique requires that the osteotome be properly aligned in the direction of the long axis of the planned implant. Thus patients must be able to open wide enough to allow a direct insertion of the osteotome into the osteotomy site. Offset osteotomes are available that can facilitate the correct angulation (see Figure 73-4, *B,* online).

Multiple individual sites can be elevated and prepared simultaneously through separate osteotomy sites.

Published reports of this technique have demonstrated increased bone height from 2 to 7 mm (average, 3.8 mm).[37] Thus the crestal approach is a useful technique for increasing the vertical height of bone up to approximately 4 mm. If more vertical bone height is needed, the lateral wall osteotomy approach may be more advantageous. In addition to the usual precautions and contraindications for sinus elevation and bone augmentation procedures, the osteotome technique may be contraindicated for sinuses that have an acutely sloped floor or septa in the location of the planned osteotomy. An acutely sloped sinus floor will tend to deflect the osteotome in an undesirable direction rather than allowing the osteotome to penetrate into the sinus space, and the presence of septa makes it virtually impossible to fracture the sinus floor inward. Box 73-2 provides additional precautions and clinician comments on using the osteotome technique. Several new instruments and techniques have emerged that improve the ability to create a crestal approach osteotomy while avoiding trauma or injury to the schneiderian membrane. These instruments/techniques vary from drill systems to piezoelectric bone surgery. See Chapter 75 for a description of piezoelectric bone surgery.

Lateral Window Technique. The lateral window technique is probably the most effective and efficient way to access the maxillary sinus and elevate the sinus floor. In this procedure, an opening into the maxillary sinus is created to elevate the schneiderian membrane and to place bone graft in the space immediately above the existing alveolar bone. The osteotomy can be prepared with a high-speed round diamond or a piezoelectric bone surgery device (see Chapter 75). Some clinicians prefer to cut the lateral window (outline cut through to the membrane) and use the impacted bony wall as the superior wall of the space created for bone grafting (Figure 73-6, *A* to *C*), and others prefer to eliminate the bony window entirely by reducing it to a paper-thin layer, which is easily removed (Figure 73-6, *D* to *F*). With the former technique, it is important to create a window that is small enough, relative to the mediolateral width of the maxillary sinus, to allow the "window" to be pushed completely into the sinus cavity. If the window cannot be inserted completely, it must be carefully separated from the membrane and removed. The bone removed from the lateral window osteotomy can be harvested and incorporated into the bone graft.

Elevation of the schneiderian membrane is accomplished with hand instruments that are inserted along the internal aspect of the bony walls of the sinus (Figure 73-7, online).

To view Figure 73-7, please visit the companion Expert CONSULT website at www.expertconsult.com.

Great care is taken to avoid perforation of the membrane. Small instruments, such as the De Marco curette, are introduced along the inferior, anterior, posterior, and superior aspects of the prepared antrostomy window, gradually inserting further along the bone until the membrane begins to separate and lift away from the bone. Subsequently, larger instruments, such as the Gracey 13/14 curette, are gently introduced along the bone to continue lifting the membrane to the desired levels (height, width, and depth). Instruments must always be kept in contact with the bone surface while elevating the schneiderian membrane to avoid perforation. Regardless of whether implants are being placed simultaneously or not, it is useful to estimate the dimensions of the augmentation needed by inserting a surgical guide with holes or markers indicating the ideal location for planned implants. Once elevated, the space can be grafted with bone (autogenous, bone substitute, or a combination). If implants are being simultaneously placed, the implant osteotomy sites should be prepared, and implants placed after the medial, anterior, and posterior aspects are filled with bone, thereby supporting the schneiderian membrane up and away from the drills and implants. After implant placement, the remaining lateral aspect is packed with bone graft. Finally, the antrostomy and bone graft is covered with a barrier membrane (e.g., resorbable membrane) and the flap is sutured.

Risks and Complications

The maxillary sinus elevation and bone augmentation procedure is technique sensitive, requiring meticulous surgical skills. Risks and complications of the procedure include tearing or perforation of the schneiderian membrane, intraoperative/postoperative bleeding, postoperative infection, and loss of bone graft or implants (see Chapter 77).

The reported incidence of perforation or tearing of the schneiderian membrane varies greatly (up to 60%) and depends largely on the anatomy of the sinus and the skill and experience of the operator.[19,23,28] The presence of septa in the maxillary sinus increases the likelihood of membrane perforation. If the perforation is small, it can often be managed with a resorbable barrier membrane placed over the opening, followed by careful packing of the bone augmentation material. If a perforation or tear is extensive (see Figure 77-18, *A*), it may be necessary to abort the procedure, close the wound, and attempt it again at a later date.

Infections have been reported in a small but significant number of cases (up to 10%) after maxillary sinus elevation and bone augmentation procedures.[28] Prevention of infection is crucial for bone augmentation procedures. Surgery should always be performed using sterile technique. Patients should use a presurgical antimicrobial mouth rinse (e.g., chlorhexidine), and postoperative antibiotics should be prescribed.

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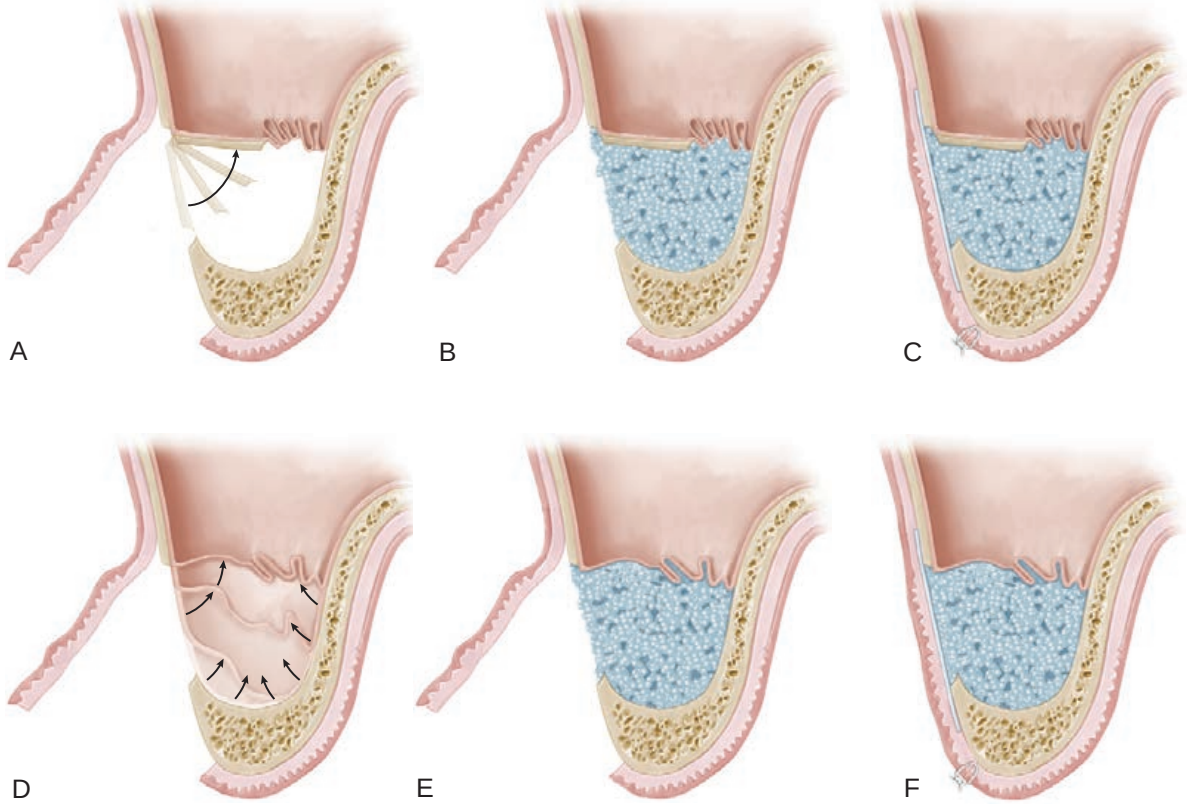

Figure 73-6 Illustrations showing two techniques for the lateral window procedure to access the maxillary sinus for bone augmentation. The first technique **(A** to **C)** preserves the bone of the lateral window elevating it up into the sinus cavity to create a new sinus floor. The second technique **(D** to **F)** completely removes the lateral window as part of the preparation. **A,** Lateral window is cut at the periphery of the access window, leaving the lateral bony wall in the center of the window intact. The bony window is then pushed inward to become the new, elevated sinus floor/superior wall of the grafted maxillary sinus space. **B,** Bone graft material is packed into the newly created space. **C,** A barrier membrane is placed over the lateral window and bone graft material. The full-thickness flap is sutured over the barrier membrane. **D,** Lateral window is removed entirely during the preparation of the osteotomy. The schneiderian membrane is elevated inward and upward to become the superior containment of the grafted maxillary sinus space without a superior bony wall. **E,** Bone graft material is packed into the newly created space. **F,** A barrier membrane is placed over the lateral window and bone graft material. The full-thickness flap is sutured over the barrier membrane.

Opening a window through the lateral wall is accomplished by completely cutting through the bone of the lateral wall up to the schneiderian membrane. The membrane is highly vascularized and may bleed significantly. However, a more serious bleeding problem can arise if an intraosseous artery is severed in the process. Bone wax and topical hemostatic agents must be available to manage such an urgent surgical complication. If a medium-to-large vascular channel is identified (with CT or CBCT scan) presurgically, it can usually be avoided.

SUPRACRESTAL/VERTICAL BONE AUGMENTATION

Supracrestal or vertical bone augmentation presents one of the greatest challenges of bone regeneration in implant dentistry. This is primarily a result of the difficulty of the surgical procedure and its potential complications. Since vertical augmentation can be a very challenging procedure with a relatively high rate of complications, it is necessary to justify this particular treatment for each patient. The rate of complications associated with vertical bone augmentation procedures, including membrane exposure and/or postoperative infection, is reported to range from 12.5% to 17%.[31,32,36] Alternative treatment options, albeit with limited outcomes, need to be considered. For example, regenerating a vertically deficient defect may not be necessary if (1) implants can be placed in adjacent sites, (2) short implants can be used, or (3) pink ceramic can be used to create an illusion of "normal" soft tissue anatomy.

Historic attempts to vertically increase alveolar bone height using modalities such as onlay bone grafting have failed. More recent treatment modalities developed for vertical bone growth include distraction osteogenesis and vertical guided bone regeneration (GBR). The techniques and evidence of success are presented.

Guided Bone Regeneration and Augmentation

The surgical technique of GBR for supracrestal regeneration was described in the 1990s.[36] Both animal and human studies demonstrated successful vertical bone augmentation with histologic evidence.[18,32] The available evidence describing and supporting the use of supracrestal GBR is limited. Some studies have evaluated the effect of space creation by a membrane alone, whereas others have used an autogenous bone graft to maintain space under the membrane. Animal (dog) studies demonstrated the ability to gain about 2.7 mm (0.4 to 4.0 mm) of vertical bone height around simultaneously placed implants.[18] In one clinical study, 5 patients were treated with a supracrestal exposure of 3 to 7 mm (average of 4.67 mm) and showed a clinical bone gain of 0.5 to 4 mm (average of 2.97 mm) after 9 months.[30] These studies suggest that supracrestal bone formation up to 3 mm is predictable using the GBR technique with a membrane–blood clot combination.

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Simion et al[32] and Jovanovic et al[18] used a titanium-reinforced (TR) membrane for vertical bone regeneration around dental implants without the placement of supportive bone substrates. Instead, the space was filled with a blood clot facilitated by perforating the cortical bone surface or by injecting the space with venous blood. Supracrestal bone regeneration achieved was 3.3 mm[32] and 1.82 mm in these studies, respectively.[18,32] Recent studies showed that supracrestal bone formation is more predictable using a bone graft filler material placed under the TR membrane. Therefore, at present, advanced surgical GBR augmentation for vertical bone gain should be achieved with a nonresorbable TR membrane that is supported by a bone graft (Figure 73-8).

The long-term results of vertical GBR after 1 to 5 years of prosthetic loading were examined in a retrospective multicenter study evaluating 123 implants.[30] Three treatment modalities (nonresorbable regenerative membranes in combination with blood clot

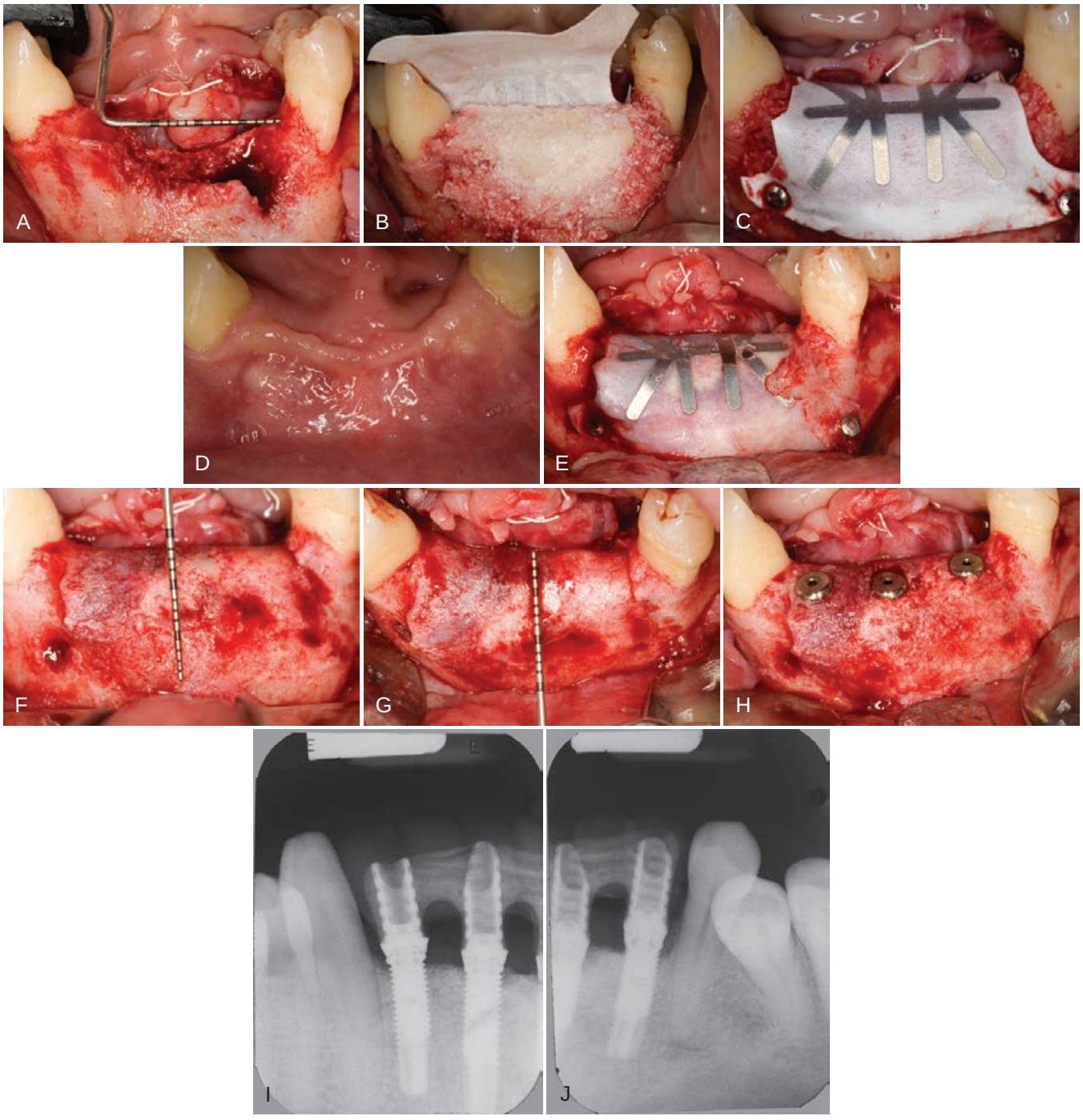

Figure 73-8 Representative case of a patient treated with supracrestal/vertical alveolar ridge bone augmentation (1:1 mixture of autogenous particulated bone and anorganic bovine bone mineral). **A,** Vertical defect in the anterior mandible. **B,** Buccal view of autogenous particulated bone mixed with anorganic bovine bone mineral. **C,** Guided tissue regeneration membrane/titanium-reinforced (GTRM-TR) membrane is fixed in place with titanium pins. **D** and **E,** Following 9 months of uneventful healing, the expanded polytetrafluoroethylene (ePTFE) membrane has maintained its position. **F** and **G,** At membrane removal the area demonstrates complete vertical bone gain. Note the excellent incorporation of both graft materials. **H,** Three regular platform implants are placed into ideal positions. **I** and **J,** Radiographs of implants restored in function. Note the appearance of good quality bone around all implants.

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only, DFDBA, or autogenous bone chips) were studied, and the results from this investigation revealed that vertical bone regeneration greater than 4 mm could only be achieved with the use of autogenous bone chips. These authors reported an overall success rate of 97.5%, leading them to conclude that vertically augmented bone using GBR techniques supports implant placement in a fashion similar to native, nonregenerated bone.

Urban et al[39] used TR membranes with autogenous particulated bone for vertical bone augmentation before implant placement. The study included 35 patients with 36 vertical bone defects. Eighty-two implants were placed in a staged approach and a resorbable collagen membrane (Bio-Gide Resorbable Bilayer Membrane, Osteohealth, Shirley, NY) was placed over the newly formed crestal bone at the implant placement surgery to protect the graft from early resorption after removal of the expanded polytetrafluoroethylene (ePTFE) membrane. Implants were followed from 1 to 6 years after prosthetic loading.

Treatment groups included single and multiple tooth sites, as well as vertical defects in the posterior maxilla. Vertical bone augmentation of the posterior maxillary ridge was done simultaneously with sinus bone augmentation (Figure 73-9). At membrane removal, the mean vertical bone augmentation was 5.5 mm (±2.29 mm). The mean combined crestal remodeling was 1.01 mm (±0.57 mm) at 12 months, which remained stable through the

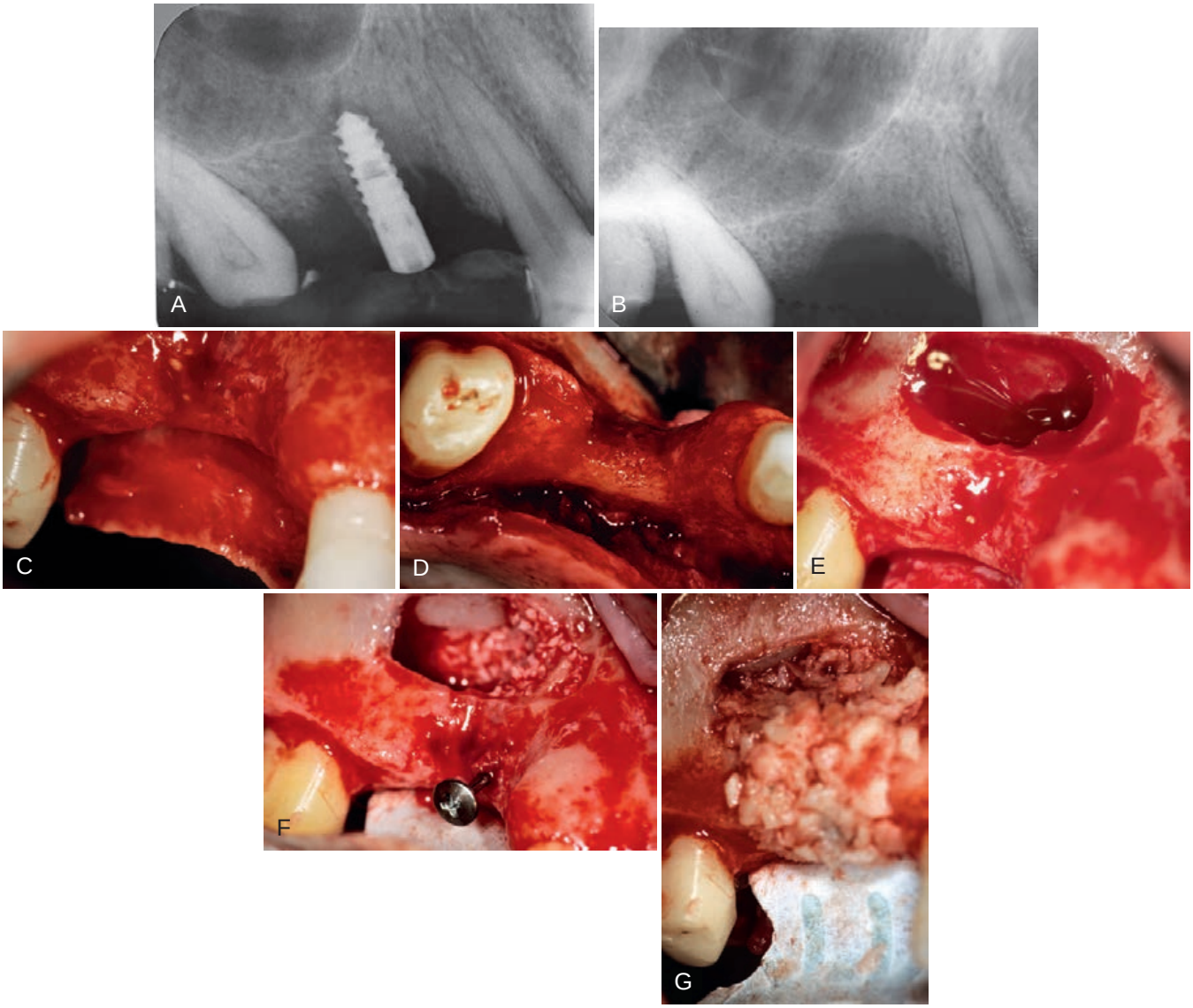

Figure 73-9 Simultaneous vertical ridge and sinus bone augmentation. Representative case requiring posterior maxillary vertical and sinus augmentation. **A,** Failing implant and severe periimplant bone defect on the right maxillary premolar area. **B,** Radiograph 3 months after implant removal demonstrates vertical deficiency and sinus proximity. **C** and **D,** Remote full thickness flap is elevated exposing the facial bone of the posterior maxillary region. Buccal and occlusal views of esthetically and functionally demanding vertical defect. **E,** The lateral window of the sinus is outlined and fractured inward. The schneiderian membrane is carefully elevated until the correct vertical height is reached. **F** and **G,** Anorganic bovine-derived bone mineral (ABBM) and autogenous bone was applied and packed to the subantral space of the elevated sinus. Guided tissue regeneration membrane/titanium-reinforced (GTRM-TR) membrane was fixated on the palatal bone and a titanium tenting screw is placed protruding to the desired vertical position. Autogenous particulated bone was applied to the vertical defect.

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Figure 73-9, cont'd **H** and **I,** Buccal and occlusal views of GTRM-TR membrane secured over the graft with titanium pins. **J,** After 9 months of uneventful healing, the GTRM-TR membrane has maintained its position. **K** and **L,** Buccal and occlusal views of the regenerated alveolar bone crest. **M,** The final reconstruction demonstrates a functional and esthetic outcome. **N,** Stable crestal bone around the implants after 6 years of loading.

6-year follow-up period. There were no statistically significant differences between the treatment groups in mean marginal bone remodeling. The overall implant survival rate was 100% with a cumulative success rate of 94.7%.

The efficacy of a 1:1 mixture of anorganic bovine-derived bone mineral (ABBM)* (Bio-Oss, Geistlich Pharma AG, Wolhusen, Switzerland) and autogenous particulated bone graft using ePTFE membranes was evaluated histologically and histomorphometrically in 8 patients (10 ridge defects).[29] After a healing period of 6 to 9 months, a mean vertical gain of 3.15 mm (SD ±1.12 mm) was achieved. The histologic analysis demonstrated new bone formation and ongoing remodeling of autogenous particulated bone and the ABBM particles. However, long-term results are not available to confirm the predictability of the procedure or implant success. More long-term studies are needed.

Distraction Osteogenesis

Distraction osteogenesis is a surgical technique that has been developed to increase vertical bone height in the deficient jaw site and contrasts with the more conventional method of bone grafting with or without membranes. Under the proper circumstances, most cells in bone can differentiate into osteogenic (or chondrogenic) cells needed for repair. Ilizarov popularized the concept of distraction osteogenesis in the 1980s by developing a protocol for "cutting" long bones and "stretching" them during the healing process.[13–15]

Based on experimental and clinical studies over 35 years, distraction osteogenesis can provide a surgeon with the ability to treat small bones in the hands and feet using external fixation devices. Recently, new intraoral devices for vertical bone growth of the alveolar process have been developed and successfully applied in preparation for the placement of dental implants. Other important advantages of distraction osteogenesis are that it may avoid the need for a second surgical site to harvest bone, and the newly created bone has native bone at the crest, which is thought to withstand forces better than regenerated bone.

One of the most significant disadvantages of the distraction osteogenesis procedure for intraoral application is the unidirectional limitation of current devices. The application of distraction osteogenesis for vertical bone growth has shown good predictability. However, limitations have been encountered in achieving horizontal bone growth with this method. Secondary bone grafting is frequently needed following vertical distraction osteogenesis, especially for the extremely resorbed (narrow) ridges. Consequently, distraction osteogenesis cannot be expected to solve vertical ridge defects without the use of additional augmentation procedures.

*Anorganic bovine-derived bone mineral (ABBM) has also been referred to in the literature as *deproteinized bovine bone mineral, deproteinized anorganic bovine bone,* and *anorganic bovine bone.*

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GROWTH FACTORS IN BONE AUGMENTATION

Current research is exploring the use of various growth factors to induce bone formation.[5] These bone-inductive mediators are powerful modifiers of the healing process. Incorporation of osteoinductive mediators into bone graft material or other carriers can be applied to sites in conjunction with conventional bone augmentation procedures to enhance the outcome.

Most of the clinical techniques available to try and stimulate new bone growth at such sites utilize some type of bone replacement graft, including autograft, allograft, xenograft, or alloplast. These bone graft materials have both advantages and limitations. Consequently, attempts have been made to improve bone grafting by either using growth factors alone or growth factors combined with a bone replacement graft.[1,26,27] The rationale is that bone formation can be further enhanced when a growth factor is used to stimulate osteoblast precursor or osteoblast cells and hence to push the bone formation/bone resorption balance in favor of bone formation.[25]

Bone Morphogenetic Proteins

Perhaps the most studied of all the osteoinductive growth factors are the bone morphogenetic proteins (BMPs), which belong to the transforming growth factor beta (TGF-β) superfamily. Of this family, *recombinant human bone morphogenetic protein-2* (rhBMP-2) has shown significant signs of bone-enhancing potential.

One commercial bone growth factor product has been approved by the Food and Drug Administration (FDA) for use in the oral cavity to stimulate bone formation. As was noted in the use of growth factors for periodontal regeneration, the growth factor protein must be combined with a carrier to have activity and be used clinically. The commercially available bone growth product contains rhBMP-2 combined with a bovine type I collagen sponge. This product has been cleared for oral cavity use in both sinus lift augmentation therapy and for ridge augmentation in maxillary anterior extraction sites with bone loss.[4,5,10,12,38]

The BMPs are a group of related proteins that are found in the body and are important for skeletal development.[7,8,43] Each of the proteins has relatively specific functions, and BMP-2 has been shown to have some of the strongest bone-producing activity. BMP-7 (also called *osteogenic protein-1* [OP-1]) and BMP-3 (also known as *osteogenin*) have also been shown to stimulate bone formation. The BMPs were originally isolated from bovine bone by Marshall Urist.[40] As a growth factor, the BMPs induce the differentiation of mesenchymal stem cells to become bone-producing osteoblast cells. Thus, unlike proliferation factors that increase the number of cells, the BMPs are differentiation factors that stimulate cells to produce bone tissue. Many carriers have been tested with the BMPs, but the binding and release kinetics using bovine type I collagen have proved to be the most useful clinically. BMP binds tightly to the collagen within minutes and has been shown to be released over time for 2 to 3 weeks at the defect site. The extended release kinetics likely allow the undifferentiated mesenchymal cells migrating into the wound site over time to be exposed to the growth factor. These BMPs have been extensively studied and have been shown to stimulate bone formation in many animal models and in humans under a variety of conditions and indications.[41] In fact, the same product approved for use in the oral cavity had previously been approved by the FDA for use in single-level spine fusion procedures and in open tibial fractures in humans.

The BMPs have specific receptors on the surfaces of cells.[24,35,44] Binding of the BMP causes dimerization of the receptor proteins, which in turn stimulates a cascade of events intracellularly involving a set of proteins called *SMADs*. The end result of the intracellular cascade is the activation of genetic activity and bone cell differentiation.

The preclinical results of using rhBMP-2 in animals with critical-sized defects have reported rapid new bone formation. The inductive capacity of rhBMP-2 was demonstrated by impregnating a polymer carrier and placing the substrate in critical-sized rat mandibular defects with or without a barrier membrane.[20] The study evaluated 12- and 24-day healing times and showed a bony union of the defects for rhBMP-treated sites. The control sites were not closed at the same intervals.

The clinical results of using rhBMP-2 and collagen in patients are remarkable considering that only one bone differentiation agent is being delivered to the bone site and it is only delivered at the time of the surgical procedure. Relatively high doses of the protein are often used in some sites however, possibly as the result of the need to release effective doses at later times. Future research directions will likely focus on more targeted delivery of the bone growth factor at specified times during the wound healing process.

The use of growth factors for bone augmentation has the potential to completely eliminate the need of bone harvesting and any bone filler materials, and preliminary results are encouraging. However, the clinician must recognize that there is limited clinical information available on these new modalities, and no information on resorption of the regenerated bone, implant survival, or bone remodeling around implants. Further documentation from long-term, randomized, controlled clinical studies are necessary.

Autologous Platelet Concentration

In recent years, a new approach to enhance the vitality of bone grafts has been introduced by using platelet concentrate or platelet-rich plasma (PRP).[21] PRP is an autologous source of platelet-derived growth factors and transforming growth factors that is obtained by sequestering and concentrating platelets by centrifugation. The patient's own blood is withdrawn and separated into platelet-rich and platelet-poor components. The PRP is the important content, containing a high-level mixture of platelets and a concentration of growth factors. This PRP mixture is added to the autologous bone graft and has been shown to increase the quality of and reduce the time needed for bone regeneration. Clinical and experimental studies are scarce but suggest that this technique holds promise for large bone defects and bone defects with low osteogenic potential. Extrapolation to the possible effect of PRP on other bone-filler materials cannot be made at this time.

CONCLUSION

Bone augmentation and advanced implant surgery procedures allow clinicians to reconstruct vertical alveolar bone deficiencies and replace missing teeth with endosseous dental implants. As stated in Chapter 72, the predictable outcome of these procedures depends on several biologic principles that must be followed. Diagnosis, treatment planning, careful execution of the surgical treatment, postoperative follow-up, and appropriate implant loading are all important factors in achieving success. Sinus elevation and bone augmentation has become a widely used and predictable procedure to augment the deficient posterior maxilla (i.e., pneumatized maxillary sinus). Reconstruction of vertically deficient ridges remains a significant challenge despite the reported progress and success of new techniques.

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SCIENCE TRANSFER

A variety of bone grafting techniques and materials are successful in creating sufficient bone volume in maxillary sinuses where insufficient bone was available to place implants. In cases in which there is 5 mm or more of bone height, the space for grafts can be completed by elevating the sinus membrane through the osteotomy created for implant placement. This can be accomplished with osteotomes or threaded elevators. In addition, there are special surgical instruments designed to release and lift the membrane through the apical end of the osteotomy so that grafts can be placed. In more advanced cases, a lateral window can be created to gain access for elevating the sinus membrane and for placing graft material.

Implants can be placed simultaneously with sinus grafting if sufficient bone is available to give initial stabilization of the implants. This generally requires at least 5 mm of bone height. In those cases in which stability of implants cannot be obtained, it is necessary to wait a minimum of 5 months for bone growth into the sinus graft before placing implants.

In deficient ridges, bone height can be increased by using special surgical procedures with bone grafts covered by titanium reinforced nonresorbable membranes. This can give an average of 3 to 4 mm gain but requires a meticulous management of flaps so that they are released and can be sutured with a watertight closure.

Distraction osteogenesis is used in cases where significant improvements in height of bone is needed. This is also a complex surgical procedure that requires highly skilled surgeons. As much as 7 or 8 mm of additional bone height can be obtained and once the initial distraction has occurred, there is a wait of at least 5 months before implant placement. These cases frequently require subsequent bone grafts to obtain sufficient width of the implant site.

Platelet-rich plasma can be used to supplement bone graft materials, however most cases are successfully treated using bone grafts alone. Recently, bone morphogenetic protein has become available for clinical use with a collagen carrier. This material has been successful in a number of applications including maxillary sinus grafts and regenerative surgery at the time of tooth extraction.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 74

Esthetic Management of Difficult Cases (Minimally Invasive Approach)

Thomas J Han, Kwang Bum Park, and Perry R. Klokkevold

CHAPTER OUTLINE

SURGICAL STRATEGY FOR PREDICTABLE ESTHETICS
- Determine the Level of Surgical Esthetic Goal (online only)
- Visualize the Final Outcome (online only)
- Preserve Existing Tissues Important for Esthetics (online only)
- Always Overbuild Bone and Soft Tissue in Augmentation Surgeries (online only)

IMMEDIATE IMPLANT PLACEMENT FOR PREDICTABILITY AND ESTHETICS

SURGICAL MANAGEMENT OF DIFFICULT CASES (MINIMALLY INVASIVE APPROACH)
- Components of Esthetic Examination
- Case Presentation 1
- Case Presentation 2 (online only)
- Case Presentation 3 (online only)

CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.

Some figures may be out of numeric order in this printed chapter.

In recent years, implant dentistry has been increasingly influenced by esthetic considerations. In addition to successful osseointegration, harmonious soft and hard tissues must surround the implant restorations so that they look natural and healthy. A major challenge in implant dentistry is that, in many instances, dental implants need to be placed in an esthetic zone in which there is extensive alveolar bone deficiency from tooth loss, dentoalveolar infection, or other pathology (Figure 74-1, *A* to *C*). Gingival morphology follows the shape of the underlying bone, and it is difficult to build esthetically acceptable gingiva in areas with deficient supporting bone. Furthermore, using conventional surgical approaches to accomplish the goal in a patient-friendly manner with minimal trauma and clinical predictability is an extremely demanding task.

Treatment planning in a complex implant case today is often confusing because there are many different surgical and restorative approaches to solve the same problem. Many times these procedures seem to conflict. When considering the surgical placement of implants, clinicians must consider the approach (conventional two-stage approach versus a one-stage approach) and the timing of implant placement (immediate versus delayed or staged placement). For bone augmentation procedures, there is the more conventional approach of augmenting the ridge first and placing the implant(s) after healing, or the simultaneous implant placement with bone augmentation approach in which bone grafting is done at same time as implant placement. All of these approaches can provide a successful outcome if patient selection is appropriate and techniques are performed properly.[9,22,24,32] However, depending on the situation, some of these techniques are more advantageous than others in achieving esthetic results with better predictably and less patient discomfort.

It is easy for a clinician to "dogmatically" choose one approach, usually choosing the one that he or she feels most comfortable with, and to treat all patients with the same approach. However, with heightened expectations of esthetics pushing the art and science of dentistry, it is necessary more than ever for clinicians to fully understand all the available options of treatment and decide appropriately which, where, when, and how to utilize these options for each patient.

An important recent clinical development in dental implant surgery is the concept of "minimally invasive" approaches to treatment. Specifically, there is a new drive toward minimally invasive implant placement. With advances in technologies, materials, and biologic sciences in dentistry, this approach to implant surgery is

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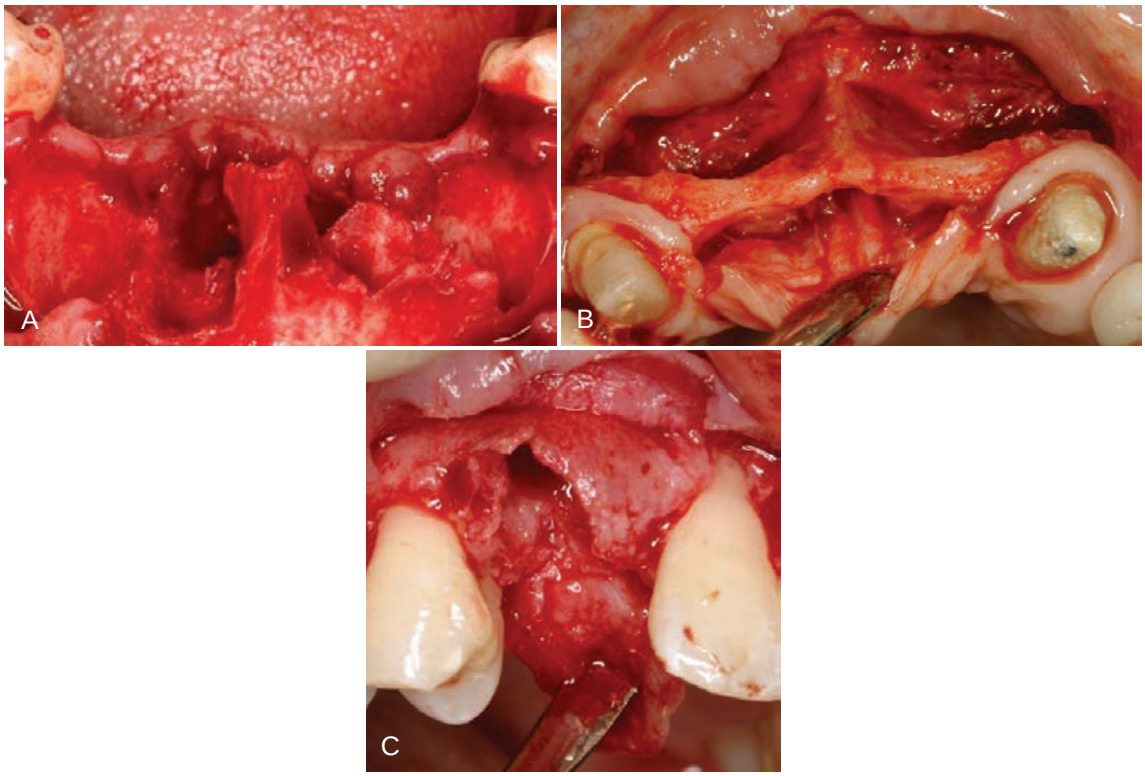

Figure 74-1 Implant sites in the anterior, esthetic zone with extensive bone loss. **A,** Anterior mandibular site showing extensive bone loss following extraction of hopelessly infected mandibular incisors. **B,** Extremely narrow (labial-palatal) ridge width in the anterior maxilla that was missing centrals and laterals for many years. **C,** Moderate bone loss associated with the extraction socket of a maxillary cuspid.

becoming more popular among clinicians and will most likely dominate the way dental implants will be placed in the near future.

Minimally invasive surgery in implant dentistry implies a surgical approach that minimizes the extent and number of surgeries while providing esthetics, predictability, and longevity with minimal surgical morbidity and discomfort to patients. The surgical approach most often required to achieve these goals for anterior implant therapy involves immediate implant placement, one-stage surgical approach, with or without flap, and simultaneous bone grafting. Furthermore, these surgical techniques need to be guided by a consistent surgical strategy that provides predictable esthetic results in implant dentistry. A minimally invasive approach to implant therapy in posterior sextants in which esthetics is not a major concern can be predictably achieved with the use of short wide implants.[2,15,16,27]

This chapter introduces surgical strategies that enhance the esthetic predictability in implant dentistry. The minimally invasive surgical approach to manage difficult esthetic and anatomically deficient cases is discussed with examples. The one-stage, immediate implant placement technique, which is the foundation of a patient-friendly approach to esthetic implant dentistry, is presented in detail. The thought processes involved in case selection, their scientific rationale, and proper techniques of soft and hard tissue management are described with cases.

SURGICAL STRATEGY FOR PREDICTABLE ESTHETICS

Observation of and adherence to the following surgical strategies enhance predictability and outcomes for esthetic dental implant surgeries with minimal discomfort to patients:

1. Determine the level of surgical esthetic goal to be achieved.
2. Visualize the final outcome.
3. Preserve existing tissues important for esthetics.
4. Always overbuild bone and soft tissue in augmentation surgeries.

For more information on these surgical strategies and examples of esthetic surgical goals, please visit the companion Expert CONSULT website at www.expertconsult.com.

IMMEDIATE IMPLANT PLACEMENT FOR PREDICTABILITY AND ESTHETICS

Immediate implant placement in a one-stage approach, in which a healing abutment or provisional restoration is attached to the implant and remains exposed, provides more predictable preservation of the interproximal periimplant gingival tissue with less patient discomfort and treatment time.[21,22] This approach to implant placement is the foundation of a minimally invasive approach to esthetic implant dentistry. However, as with any surgical technique, it takes learning and practice to perform it properly. The criteria and techniques for proper immediate implant placement have previously been established and reported with successful long-term outcomes.[18,32]

One of the more difficult aspects of immediate implant placement is positioning the implant with sufficient primary stability in an extraction socket, often without elevating a flap. The alveolar architecture in relation to the angle of the implant to be inserted; the presence or absence of a bone concavity apical to the extracted tooth; the amount of existing bone apical and palatal to the

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extraction socket, which can provide primary stability for the immediate implant; and the quality of the bone and soft tissues of the ridge should all be thoroughly evaluated clinically and radiographically before surgery.[18,32] Many clinicians perform successful immediate implant placement without the aid of a three-dimensional scans (e.g., computed tomography [CT] or cone-beam CT [CBCT]). However, if the tooth involved is long and large or if there is an alveolar concavity or other anatomic variations, the use of a CT/CBCT scan is advised.

A major disadvantage of placing implants immediately in the changing alveolar bone of an extraction socket is that it may result in progressive recession of the gingival labial margin over the implant restoration.[4,17] Therefore, when placing an immediate implant with one-stage surgical approach in an esthetic zone, a prudent strategy would be to improve the quality and quantity of labial gingival tissue, which seems to be vital for the stability of the labial gingival margin involving immediate implants.[19,31] One of the most effective ways to keep the implanted socket from collapsing and improve the labial gingival biotype is to simultaneously fill the labial socket void with particulate bone and augment the labial gingival tissue with soft tissue.[3,23,26,30,32]

An effective bone and gingival tissue augmentation technique used with flapless immediate implant placement in a one-stage approach is the bone and crescent-shaped free gingival grafting technique.[18] In this technique, the space between the inner surface of the labial bony wall and the labial surface of the implant is filled with slow-resorbing mineralized, freeze-dried particulate bone allograft (FDBA) or particulate xenograft to help preserve the horizontal dimension of the ridge (Figure 74-5, *A*). Then, a crescent-shaped soft tissue graft is harvested from the ipsilateral palate (Figure 74-5, *B*) and transplanted into the labial recipient site coronal to the particulate bone graft (Figure 74-5, *C*). To provide/maintain the blood supply to the donor tissue, it is important that the outer surface of the crescent graft fits with an intimate contact with the bleeding lamina propria of the labial gingiva. Proper suturing ensures good proximity and prevents the graft from being displaced coronally out of the recipient site (Figure 74-5, *D*).

The advantage of this approach to gingival augmentation is simplicity and minimal surgical morbidity. In addition to providing a sealed protection for the bone graft, it prevents resorption of the sensitive labial crestal bone. Preparation of the recipient site involves no surgical manipulation other than de-epithelialization as described. Gingival walls are completely intact with a full blood supply. The donor site wound is small (approximately 3 mm depth × 3 mm height at the widest point) with intact epithelium around the wound, which epithelializes within a week and causes minimal discomfort for the patient. Since each donor tissue graft is small, multiple grafts can be harvested from a single palate, so that multiple immediate implants can be augmented at same time. Furthermore, this gingival augmentation technique frequently improves an unfavorable initial gingival margin since the grafted gingival margin is always coronal to the existing gingival margin. This minimizes a need for other time-consuming techniques, such as orthodontic extrusion, or a delayed approach to implant placement when the initial gingival margin is not esthetic or ideal, as recommended by many authors.[5,33]

The risk-to-benefit ratio of the crescent grafting technique is favorable enough that if the graft does not survive, or if more than expected horizontal resorption of the ridge occurs, traditional techniques (e.g., subepithelial connective tissue graft) can be performed to augment the results. This is usually possible without refabrication of the implant restoration because the vertical height of the interdental papilla is sufficiently preserved with immediate implant placement in a one-stage approach.

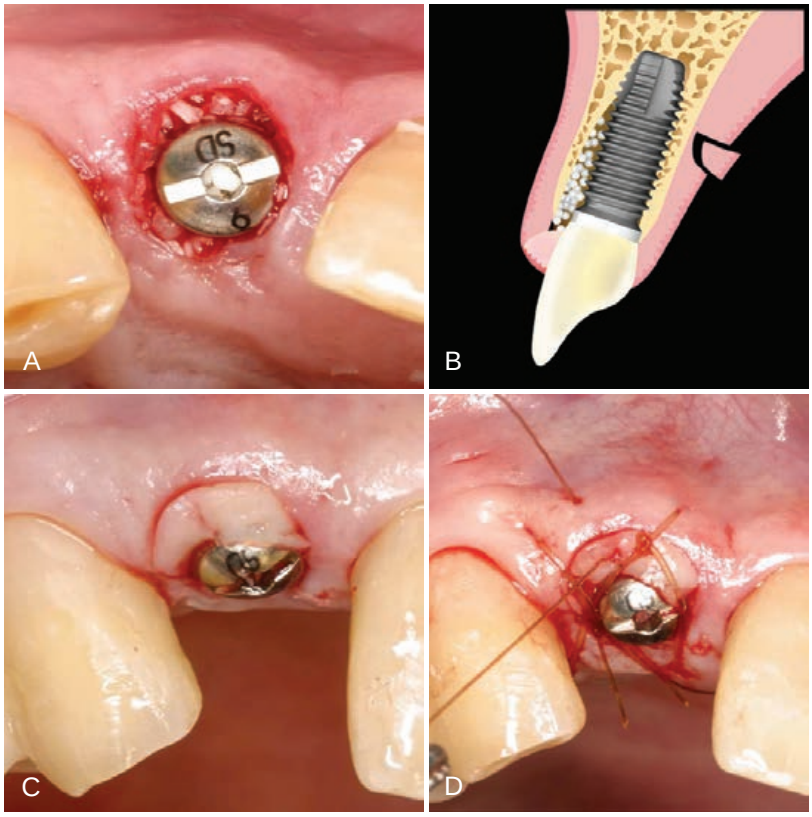

Figure 74-5 **A,** Bone graft material is lightly packed into the extraction socket gap between the labial wall and the implant surface. **B,** Diagram of crescent-shaped, free gingival graft tissue being harvested from the palate. **C,** Clinical photograph of crescent-shaped, free gingival graft tissue positioned over the bone graft to fit snuggly in the gap between the gingival wall and the implant surface. **D,** Crescent-shaped, free gingival graft is sutured in place using 5-0 gut sutures. The sutures pass through the gingival graft toward the labial gingival margin and are tied. Then, without cutting the ends, the suture is passed over the graft and tied to the palatal gingival tissue. Three sutures are needed to keep the graft in position.

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SURGICAL MANAGEMENT OF DIFFICULT CASES (MINIMALLY INVASIVE APPROACH)

The cause of the failure to satisfy the esthetic needs of a patient frequently starts with an inadequate examination of the soft and hard tissues surrounding the surgical site and the natural dentition. This can result in an incorrect diagnosis, which leads to incorrect treatment plan. The wrong treatment plan combined with selection of inappropriate surgical approaches or techniques can result in a disastrous esthetic outcome and unnecessary patient suffering. The first two case presentations describe the examination and thought processes involved in determining the diagnosis and treatment planning of complex anterior cases with extensive alveolar bone loss. The proper application of the surgical strategies and the minimally invasive techniques, previously described, are illustrated. The final case presentation illustrates the minimally invasive approach to posterior implant therapy with the use of short wide implants, in which esthetics is not the major concern.[2,15,16,27]

Components of Esthetic Examination

The patient's chief complaint, esthetic zone, tooth positions, gingival form, osseous crest position, biotype, tooth shape, horizontal and vertical ridge deficiency, and occlusal status all play an important role in deriving an accurate esthetic treatment plan for the patient. Therefore developing the necessary skill and knowledge to examine and recognize problems of these components is an essential first step to clinical success.

Case Presentation 1

The first case presented is a simultaneous bone and soft tissue augmentation with multiple maxillary anterior immediate implants placed in a one-stage, flapless approach.

Patient Dental History and Chief Complaint. The patient is a 70-year-old female with severe mobility and discomfort of the maxillary four incisors. Except for a generalized feeling of weakness, she is healthy and does not have any medical contraindications for dental treatment. She presented with a desire to replace her maxillary incisors with dental implants, but she is very concerned about the physical discomfort she may experience from the implant surgery. She does not want to wear a removable prosthesis at all, not even for a short time. She is content with her present dental esthetics.

Examination and Diagnosis. The maxillary incisors exhibit moderate-to-severe periodontitis with 4 to 7 mm periodontal probing depths. They exhibit severe (2+) mobility with fremitus. The periodontal and restorative prognosis of these teeth is poor. She has an excessive overbite with evidence of moderate mandibular incisor wear, indicating possible parafunctional habits. Her incisors are slightly elongated, but the dentogingival symmetry is acceptable (Figure 74-6, *A*). The shape of the incisors is slightly triangular with sufficient interdental papilla volume and height. Interproximal gingival tissue is not swollen or edematous. Her gingival biotype appears to be on the thin side with slight marginal inflammation, and the position of the labial gingival margins is already high (i.e., maximal apical position). Any further recession will be unaesthetic.

Radiographic evaluation demonstrates moderate-to-severe vertical and horizontal periodontal bone loss. The osseous crest position, in relation to the gingival margins, appears too apical to provide adequate gingival support (Figure 74-6, *B*). The restorative and periodontal status of the canines is healthy.

Treatment Objectives. Considering her age, reasonable esthetic expectation, concern for surgical morbidity, and a lack of willingness to wear a removable provisional prosthesis, the treatment objective for this patient would be to utilize a surgical approach that minimizes the extent and number of surgical procedures while providing predictability, longevity, and acceptable esthetics. Recommending orthodontic extrusion or multiple surgeries to achieve ideal esthetics on this patient would be considered an excessive treatment plan and would not provide any additional value or benefits for her.

Treatment Options

For more information on the following treatment options, please visit the companion Expert CONSULT website at www.expertconsult.com.

1. Extract four maxillary incisors and replace them with a conventional (6-unit) fixed prosthesis supported by the canines.
2. Extract four maxillary incisors and wait for healing of the sockets and ridge while temporarily replacing the missing teeth with a removable partial denture. Plan to place two or four implants after 3 to 6 months of healing using either a one- or two-stage approach.
3. Extract four maxillary incisors, immediately place two implants in the lateral positions and replace the missing teeth with a 4-unit provisional fixed partial denture supported by two implants.
4. Extract four maxillary incisors, immediately place four implants in the sockets and replace the missing teeth with a 4-unit provisional fixed partial denture supported by four implants.

Surgical Strategy for Predictable Esthetics. Because of the patient's age and reasonable expectation, the level of the esthetic goal determined for this patient is not ideal but is acceptable. This level of esthetics can be predictably achieved in a minimally invasive, patient-friendly manner. By mentally visualizing the surgical and prosthetic goal, an acceptable esthetic outcome can be achieved for this patient if the existing heights of the interproximal gingival tissues can be maintained. Additionally, if the existing biotype and the level of the labial gingival margin can be overbuilt with hard and soft tissue augmentation, the final esthetic outcome is enhanced. If this can be accomplished with simultaneous implant placement, it minimizes the number of surgeries required. In this case, especially since the patient appears to have parafunctional habits, it is desirable to place more implants and use them to support the immediate provisionalization.

Considering all of these treatment options, the treatment that is most compatible with this patient's surgical strategy for predictability, acceptable esthetics, and long-term results is treatment option 4, extraction of the incisors followed by immediate placement of four implants with an immediate provisional restoration supported by the implants.

Treatment Plan and Rationale. Four maxillary incisors are to be extracted, and four implants placed immediately in a one-stage approach with simultaneous bone and soft tissue grafting. The bone and crescent-shaped free-gingival grafting technique is used to overbuild labial gingival biotype.[18] A tapered implant with platform-switching design is to be utilized. The tapered design has shown to promote primary stability in sockets, and the

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Figure 74-6 **A,** Clinical photograph of periodontally compromised maxillary anterior teeth with long clinical crowns. The presenting esthetic status is not ideal. However, the dentogingival symmetry is fair since the gingival margin is approximately the same for all incisors (cuspids, laterals, and centrals). **B,** Periapical radiographs of the maxillary anterior teeth reveal moderate to severe horizontal periodontal bone loss with vertical intrabony defects. **C,** Simple, atraumatic extraction of the maxillary incisors reveals sockets with unsupported interdental tissues. Note that there are no incisions or flap reflection, which has preserved the blood supply and integrity of the interdental soft tissues. **D,** Guide pins placed in prepared implant sites reveal good position within the extraction sockets achieving good primary stability from the palatal aspect of the socket. **E,** Implants with temporary abutments and crescent shaped, free gingival grafts (not yet sutured). **F,** Implant provisional abutments are prepared and provisional restorations are fabricated using conventional methods.

platform-switching design better maintains the interimplant bone.[8,25] An immediate provisional restoration without centric contacts is attached to the implants.

Treatment Sequence. In complex treatment involving dental implants, the treatment plan must be sequenced and coordinated between the restorative dentist and the surgeon before starting the treatment. This is especially true when immediate provisionalization is planned. This will enhance success and help make the treatment more patient friendly.

Surgical Procedure. Once the patient is anesthetized, the incisors are extracted atraumatically, ensuring that gingival tissues, especially the interdental papilla are not damaged (Figure 74-6, *C*). The sockets are prepared to receive implants by removing the sulcular epithelium, and by completely and thoroughly removing all granulation tissue. Immediately placed implants must have complete primary stability at the time of placement. Proper vertical position, buccolingual position, and mesiodistal position, as well as buccolingual angulation, are all critical factors for a successful outcome. Implants in anterior sockets are prepared and placed toward the palate (Figure 74-6, *D*) to ensure sufficient labial bone thickness for labial margin stability.[9] Once the provisional healing abutments are accurately seated (may require bone profiler), the labial void of the sockets are grafted with particulate bone and a crescent-shaped free gingival tissue graft is harvested and placed over the graft (Figure 74-6, *E*) as described previously and in the literature.[18] Using this technique, all four teeth can be augmented in one surgery with minimal discomfort to the patient. Once the soft tissue crescent grafts are secured with sutures, the provisional abutments are prepared for provisional crowns. The provisional crowns (splinted) are fabricated using conventional methods (Figure 74-6, *F*). Contours of the provisional crowns may need to be modified as the healing and remodeling occurs. Impression taking for the final restoration is relatively easy because the provisional abutments placed at the time of surgery and modified during healing nicely shape the tissues for an optimal prosthetic emergence profile.

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Figure 74-6, cont'd **G,** One-year postloading results show nearly complete preservation of the interproximal papilla height and improved labial gingival biotype. Labial gingival margins are substantially coronal in position as compared to the initial levels. **H,** The final restorations have a more normal length offering the patient a much more youthful smile. Preexisting moderate to severe attrition is noted on the incisal edges of the mandibular anterior teeth. **I,** Periapical radiographs at 1 year of the implants with final restorations reveal good preservation of interproximal bone height with minimal saucerization. The platform-switched implant restorations likely contribute to the preservation of bone.

Results. The 1-year result shows nearly complete preservation of the interproximal papilla height, an improved labial biotype with labial gingival margins that are substantially coronal to the original gingival margin level (Figure 74-6, *G*). The final result is crowns with a more normal incisor length and a more youthful smile (Figure 74-6, *H*). There is some gingival irregularity noted, which appears to be a result of the soft tissue grafting. These areas could easily be smoothed with gingivoplasty, but the patient refused. Radiographs reveal preservation of interproximal bone with minimal saucerization around the implants with a platform-switch design (Figure 74-6, *I*).

After the surgical strategies described and utilizing minimally invasive surgical techniques, this 70-year-old patient received immediate implant-supported replacement of severely compromised maxillary incisors with an acceptable (or better) esthetic result and minimal treatment discomfort. With one surgery, including the extractions, she was provided with a fixed implant restoration on four implants. In the same surgery, the bone and soft tissue was augmented while preserving the interdental papilla. She experienced very little postoperative pain and was pleased with her new, more youthful-looking smile. The treatment time was only 6 months from extraction to final restoration. She was never without teeth and never wore a removable prosthesis. The improvement of the labial gingival margin was achieved without orthodontic extrusion or multiple surgeries.

To view an additional case on simultaneous extensive bone augmentation with multiple mandibular anterior immediate implants, please visit the companion Expert CONSULT website at www.expertconsult.com.

To view an additional case on the use of short wide implants, please visit the companion Expert CONSULT website at www.expertconsult.com.

CONCLUSION

The clinical science in dentistry has evolved to where placement of dental implants and restoring them requires sufficient knowledge in several disciplines of dentistry. In addition to mastering surgical objectives and techniques, periodontists must be able to evaluate and accurately diagnose (and treat or refer) a wide range of related "restorative" issues including but not limited to esthetics, occlusion, temporomandibular joint (TMJ) function, vertical dimension, and dental-skeletal relationships. These "other" aspects of diagnosis are essential for the development of an appropriate treatment plan, which is necessary for a successful outcome in esthetic implant dentistry.

In addition, with heightened expectations of esthetics pushing the art and science of dentistry, it is necessary more than ever for clinicians to fully understand all the available treatment options and to be able to appropriately determine which option to choose, when to use certain techniques, and how to apply them for each patient scenario.

The minimally invasive approach and techniques presented in this chapter are not the only ways to manage difficult esthetic cases, and some may consider them controversial. Certainly, these techniques require learning and practice to make them effective in each clinician's hands. However, they present a sound approach to what is possible in hard and soft tissue management for esthetic implant surgery and provide effective strategies and techniques for solving many difficult esthetic cases in a patient-friendly manner.

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SCIENCE TRANSFER

Implants may be placed immediately after tooth extraction if there is sufficient bone to give initial implant stability utilizing apical bone and bone in the walls of the socket. In some upper incisor cases, it is necessary to prepare the osteotomy slightly palatally to the original socket so that appropriate crown access holes are positioned in the cingulum. Most cases are improved by the use of soft tissue enhancing techniques with connective tissue grafts or crescent-shaped gingival grafts. These allow the surgeon to plump out the labial gingiva so that esthetic gingival contours are obtained since there is very often a loss of labial thickness of tissue after implant surgery.

Support of gingival tissues to maintain soft tissue contours is a valuable attribute of the minimally invasive technique. In cases in which implants are very stable at placement, an immediate placement of an esthetic but nonfunctional treatment crown can be considered. This will allow for tissue management by changing the crown contours so that over 3 to 4 months, ideal gingival contours are obtained. It has been suggested that the use of resonance frequency measurements of implant stability can be the basis for deciding whether to immediately place a crown; readings of 50 or greater are an indication that sufficient bone support is present for immediate crown placement. A one-stage surgical approach can be used, and if implants are not suitable for immediate crown placement, the use of healing abutments gives some support of soft tissue during the osseointegration period.

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 75A

Technologic Advances in Implant Surgery: Dental Implant Microsurgery—Immediate Placement

Dennis Shanelec and Leonard S. Tibbetts

CHAPTER OUTLINE

IMPLANT MICROSURGERY
- Microsurgical Tooth Extraction
- Implant Drilling in the Extraction Site
- Bone Grafting
- Buccal Gingival Grafting
- Immediate Provisional Fabrication
- Immediate Implant Occlusion
- Custom Impression Transfer Coping
- Final Implant Restoration

CONCLUSION

The success of dental implants in extraction sites combined with immediate provisionals for newly placed dental implants has brought a convergence in restorative and surgical practice for treatment planning of dental implants. This convergence reaches its summit in the approach to failing teeth in the maxillary esthetic zone. For anatomic reasons, maxillary anterior teeth are at high risk for traumatic injury[13,14] (Figure 75A-1). Traumatized teeth frequently receive endodontic treatment that may be followed by horizontal or vertical root fracture.[1,2,15] Dentistry's historic answer to tooth loss has been the fixed bridge. Tooth preparation necessary for a fixed bridge often results in significant reduction of tooth structure.[22] Inherent esthetic limitations of fixed bridges include loss of gingival papillae and resorption of the buccal alveolar plate. For these reasons, dental implants are a preferred choice for tooth replacement in the maxillary esthetic zone.

IMPLANT MICROSURGERY

Microsurgery is associated with enhanced soft tissue procedures and fine suturing. This is part of the scope of implant microsurgery but additional benefits include dental implant drilling precision. The ability to discern minute dimensional differences permits implant osteotomy preparations centered exactly between reference points such as adjacent teeth, adjacent implants, or buccal and lingual ridge anatomy. More profoundly, the microscope allows immediate detection of subtle changes in drill position so appropriate feedback corrections can be applied to the handpiece. Enhanced angular perception is also important. The drill's angular position can be oriented relative to small landmarks such as the implant platform surface level or the angle of adjacent implant healing caps. This permits optimal parallel positioning and depth of adjacent implants. The implant drill angle can also be accurately oriented to root surface angulation utilizing just 3 to 4 mm of root anatomy exposed between the cementoenamel junction and the osseous crest. These reference points are simply not visible without a microscope. The detection of subtle angulation reference points and changes in drill position permit feedback correction, which is important for osteotomy preparation in extraction sockets. The microscope-enhanced accuracy of osteotomy microsurgery permits socket implant placement in an ideal position followed by an esthetic implant-supported provisional (Figure 75A-2). As a flapless procedure, this is accomplished with minimal patient morbidity.[22]

Microsurgical Tooth Extraction

Tooth extraction has been traumatic for centuries. Conventional tooth extraction may require mucogingival flaps and bone removal, resulting in compromised esthetics. Employing a microscope with minimally invasive principles reduces trauma and results in predictable esthetic outcomes.[6] Instrument selection influences the trauma of tooth removal. Periotome luxation or leveraged mechanical extraction employing tapped root anchorage systems can carefully separate a tooth from its surrounding ligament and lift it vertically from the socket. This limits injury to papillae and preserves natural gingival anatomy (Figure 75A-3). Subtle nuances in luxation direction can be microscopically detected for root removal in a proper anatomic path of extraction. Increased visibility under the microscope permits most extractions without mucogingival flaps. Greater

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Figure 75A-1 X-ray of typical fractured central incisor.

Figure 75A-2 Flapless dental implant microsurgery.

visibility also permits atraumatic sectioning of ankylosed roots to leave alveolar bone and soft tissue uninjured. Apical granulomatous lesions can be completely debrided with full visibility. Such minimally invasive microsurgical techniques translate into reduced patient morbidity with enhanced healing.

Implant Drilling in the Extraction Site

Microsurgical implant drilling in the extraction socket is unique. Under the microscope, a socket appears as large as a room with its apex and walls clearly visible. A different set of skills is required for socket drilling. For placement of maxillary anterior implants, the most favorable bone lies to the palatal (Figure 75A-4). Drilling therefore must be done at an angle to the palatal socket wall. Twist drills innately track toward the direction of less dense bone and into the open socket. Drilling sockets under a microscope utilizes visual feedback to constantly redirect the drill to the correct position and angulation. This avoids the common mistake of an implant being placed too far toward the buccal. With the magnification and lighting a microscope provides, implants can be placed in the palatal socket wall with good initial stability and ideal esthetic position. Microscope-enhanced dimensional and angular perceptions allow adjustments to correct drilling speed. Too little or too much drill pressure or excessive drill speed causes frictional heat. This adversely affects implant osteointegration. Detecting

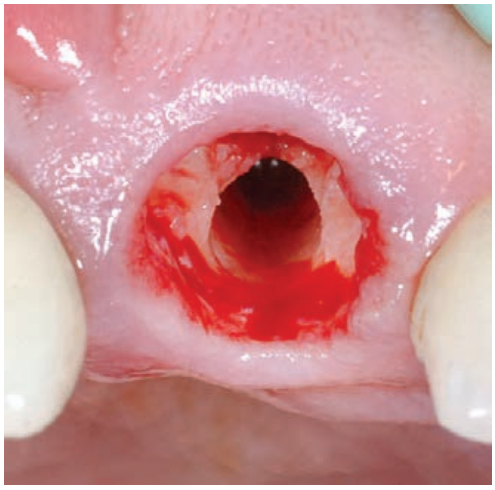

Figure 75A-4 Palatal wall socket osteotomy.

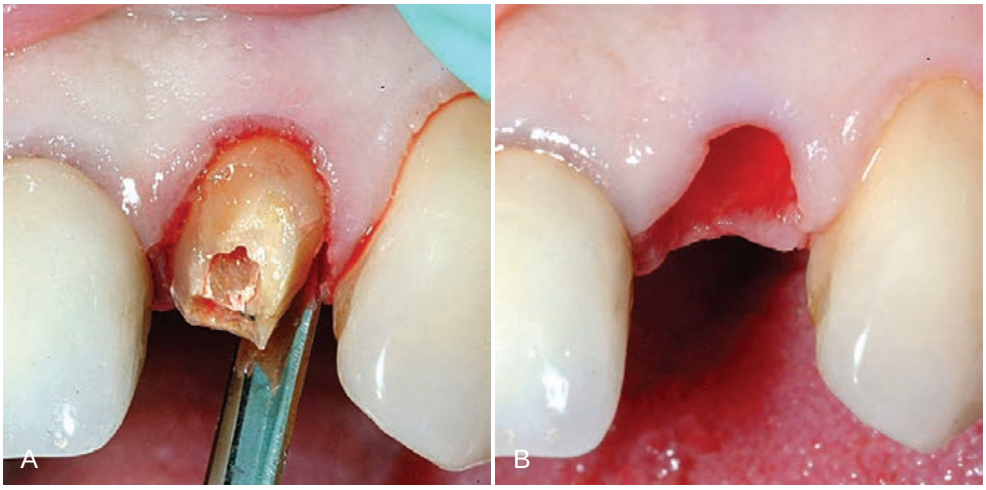

Figure 75A-3 **A,** Periotome extraction of lateral incisor. **B,** Noninvasive extraction of lateral incisor.

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micromovement of the advancing drill assures that proper pressure and rotational speed are applied to varying bone densities encountered in the socket. The angular velocity at the cutting edge of a 4-mm drill is several times faster than the velocity at the cutting edge of a 2-mm drill. For this reason, pressure and rotation of larger diameter drills must be decreased to compensate for their faster cutting speed. Enhanced visual feedback for speed and pressure correction is accomplished through directly viewing the advancing drill under the microscope.

Bone Grafting

The osteotomy in an extraction socket is prepared in the palatal wall. This placement results in a gap between the implant and the buccal socket wall. To avoid displacing a particulate graft, the provisional is finished before a socket bone graft is placed. The socket is filled with xenograft to within a millimeter of its crest. Xenograft is selected to reduce remodeling resorption of the buccal bone. Filtered bone from the osteotomy preparation is rinsed with 3% tetracycline solution then condensed on the top of the xenograft. Finally, it is covered with a layer of microfibrillar collagen to contain the graft.

Buccal Gingival Grafting

Recession on the buccal gingival margin around anterior implants placed in extraction sockets has been well documented.[11,17] Multiple factors, such as the periodontal biotype, presence or absence of the buccal cortical plate, surgical trauma, implant position, and the emergence profile of both the provisional and final restorations, are associated with such recession.[9,12,16,18-20] A subepithelial connective tissue graft is therefore harvested from the palate and transferred into a split-thickness envelope incision on the buccal of the implant. A connective tissue graft is done to maintain or augment gingival height and thickness that may have been lost as the result of injury (Figure 75A-5). Even gingival tissue at a normal height can be expected to recede as much as 1.5 mm unless gingival grafting is performed. Placing a subepithelial connective tissue graft concurrent with implant placement insures stability of the postoperative gingival level.[7,21]

Figure 75A-5 Facial connective tissue graft.

Immediate Provisional Fabrication

To preserve natural esthetics and provide support for gingival anatomy, an implant provisional must emerge from the surrounding gingival tissue exactly like the extracted tooth.[4,23] The surgical microscope gives dentists the visibility necessary to fabricate ideal anatomy for implant provisional crowns (Figure 75A-6). Subgingival provisional contours created under the microscope provide tissue support and well-finished margins. The provisional crown on the implant serves a number of functions, as follows:

1. It provides optimal esthetics and function.
2. It minimizes tissue collapse by supporting the gingival tissue.
3. It obturates the surgical extraction socket to contain particulate and soft tissue grafts.

Creating an implant provisional crown begins before the tooth is removed.[8] A clear silicone impression captures the dentogingival junction and its proximal tooth contours (Figure 75A-7). Tooth

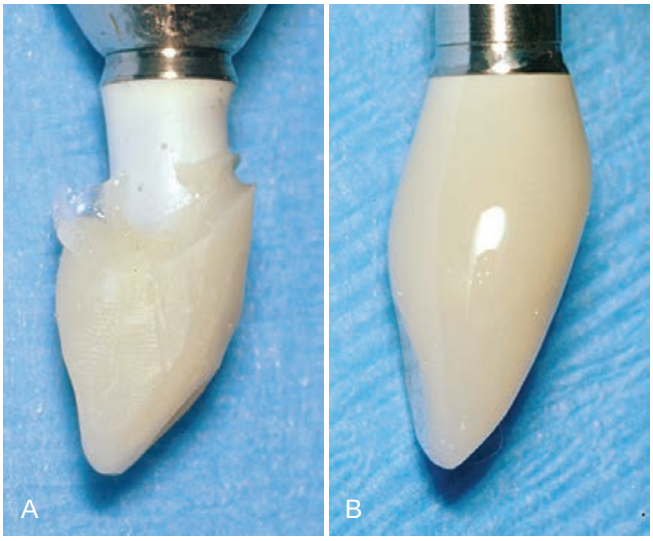

Figure 75A-6 **A,** Screw-retained provisional before finishing. **B,** Screw-retained provisional after finishing.

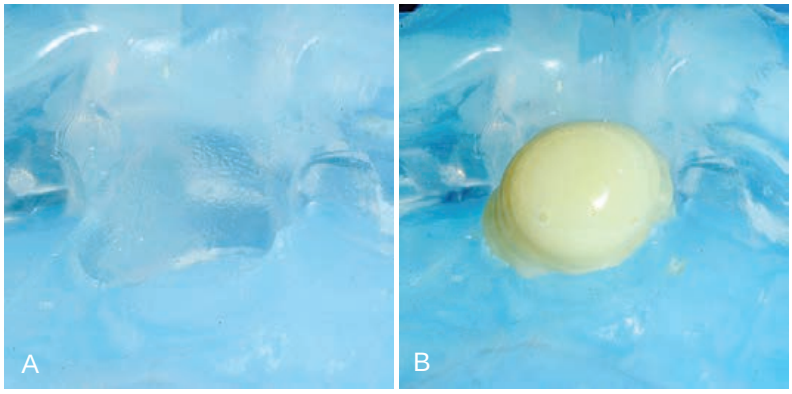

Figure 75A-7 **A,** Clear silicone impression of failing tooth crown. **B,** Clear silicone impression filled with composite.

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color matching is done and a light-cured flowable composite resin duplicate of the tooth is created using the impression (Figure 75A-8). The duplicate crown is trimmed to the exact location of the dentogingival junction then hollowed to create a shell crown. It is fitted to a screw-retained titanium provisional abutment that is opaque for a color match (Figure 75A-9). Great attention is paid to the incisal edge position of the shell crown before luting it to the abutment with flowable composite resin. Screw access for removal of the provisional is accomplished by drilling through the incisal one-third of the provisional crown.

Figure 75A-8 Shell composite crown created from impression.

The luted abutment and crown are removed and placed on a laboratory handle to facilitate shaping and polishing (see Figures 75A-12 and 75A-13). Each provisional crown has a uniquely shaped subgingival emergence profile that duplicates the original tooth. Voids and rough edges are eliminated, and the provisional is carefully shaped under the microscope to provide gingival support (Figure 75A-10). Shaping the provisional is accomplished with a 12-fluted finishing bur, glass nail file, and a prophylaxis cup with pumice. Attention to detail is critical. As a final step, the provisional crown is glazed and thoroughly cured. Light curing the composite assures that no free monomer is present to irritate soft tissue or bone. The machined titanium provisional abutment assures good marginal fit and reduces the possibility for the loosening.

Immediate Implant Occlusion

Early loading bone trauma is minimized in multiple immediate implant provisional cases by splinting. Early loading bone trauma in single implant cases is reduced by lessening occlusal forces. Symmetric and light mesial and distal proximal contacts are established and the provisional is taken out of centric and lateral occlusal contact using 1 mm green occlusal indicator wax. This technique allows patients to leave the dental office with a nonloaded esthetic provisional tooth securely anchored to the implant (Figure 75A-11).

Custom Impression Transfer Coping

A custom impression transfer coping is necessary to preserve and communicate gingival supporting contours to the ceramicist. The

Figure 75A-9 **A,** Opaque titanium temporary abutment. **B,** Composite crown luted to opaque titanium temporary abutment.

Figure 75A-10 Provisional supporting gingival tissue.

Figure 75A-11 Provisional 1 week after implant microsurgery.

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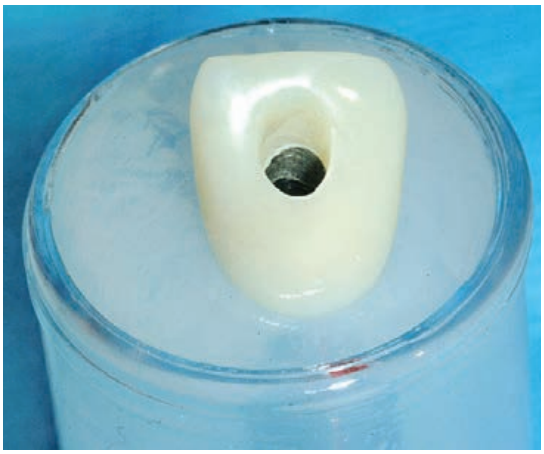

Figure 75A-12 Clear silicon impression of provisional.

Figure 75A-13 Custom impression transfer coping reproducing emergence profile.

chairside-created provisional anatomy must be precisely reproduced for the dental laboratory via the custom impression transfer coping.[5,10] To make the custom transfer coping, an impression of the gingival third of the provisional crown is made with an implant analog attached (Figure 75A-12). This registers the implant platform orientation and preserves the provisional anatomy. The crown is removed, and a standard impression coping is attached to the implant analog. Acrylic powder is filled into the gap between the impression coping and the clear silicone impression. Monomer liquid is then infused into the powder to create a hard acrylic copy of provisional anatomy.[3,5] Using a 25-gauge needle to apply monomer to the powder from the base up minimizes inclusion of air bubbles in the custom transfer coping. For orientation, a mark is initially applied to the labial of the provisional impression. It is transferred to the custom transfer coping to provide orientation for the restorative dentist during final impressions (Figure 75A-13). This technique allows precise communication from the surgeon to the restorative dentist to the ceramicist of the anatomy required for a final restoration that esthetically supports gingival tissue.

Final Implant Restoration

Final impressions are taken, utilizing the custom impression transfer coping. Computer-assisted scanning and machining creates a

Figure 75A-14 Preoperative microsurgery.

Figure 75A-15 Immediate postoperative microsurgery.

Figure 75A-16 Provisional 8 weeks postoperative.

zirconia abutment and zirconia coping for an all-ceramic crown (Figures 75A-14 to 75A-16). Zirconia has the benefit of tissue biocompatibility and light translucency. The sequence described assures a final implant abutment and crown that exactly matches both the provisional emergence profile and the original tooth shape. Working as a team, surgeon, restorative dentist, and laboratory

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SCIENCE TRANSFER

The use of a microscope accompanied by special microsurgical instruments provides an enhancement of implant surgery particularly in the placement of immediate implants in the anterior esthetic zone. Exquisite surgical skills are needed, and specialized training in microsurgical procedures is required, as well as extensive clinical experience, and only a few gifted clinicians have mastered this demanding approach to therapy. The results of such precise procedures are optimal healing and optimal maintenance of excellent esthetics.

Figure 75A-17 Pre-operative microsurgery.

Figure 75A-18 Immediate postoperative microsurgery.

Figure 75A-19 Final restoration.

technician can create a final restoration in harmony with preserved gingival architecture (Figures 75A-17 to 75A-19).

CONCLUSION

Some of the advantages of microsurgery for extraction and implant placement have been described with an emphasis on tissue management and the ability to enhance visualization of details, which translates into better results. This microsurgery protocol advances dentistry from an era of traumatic tooth extraction to one of seamless immediate tooth replacement utilizing implant microsurgery.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 75B

Technologic Advances in Implant Surgery: Piezoelectric Bone Surgery

Tomaso Vercellotti and Perry R. Klokkevold

CHAPTER OUTLINE

CLINICAL CHARACTERISTICS OF ULTRASONIC CUTTING
- Microprecision
- Selective Cutting
- Maximum Visibility
- Excellent Healing

CLINICAL APPLICATIONS
- Periodontal Surgery (online only)
- Crown Lengthening (online only)
- Tooth Extraction Techniques (online only)
- Implant Site Preparation
- Sinus Lift (online only)
- Ridge Expansion
- Bone Harvesting

CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures may be out of numeric order in this printed chapter.

Ultrasound has been used for many years in periodontics to remove tartar, debride root surfaces, and to degranulate periodontal defects. In the last decade a novel family of ultrasonic powered devices has been developed that is revolutionizing maxillofacial bone surgery.

A new surgical technique, known as *piezosurgery,* was invented by Vercellotti and developed by Mectron Medical Technology. The piezosurgery device (Figure 75B-1) consists of a piezoelectric ultrasonic transducer powered by an ultrasonic generator, capable of driving a range of specially designed cutting inserts (Figures 75B-2 to 75B-10).[17,22] Box 75B-1 describes the main cutting properties of piezosurgery by Mectron, and Box 75B-2 describes piezosurgery inserts. Piezoelectric bone surgery techniques have been developed for clinical applications in dentistry and are becoming state of the art for a variety of procedures.[18-20,22,28,29] Recently, Piezosurgery Medical expanded development of clinical applications to other fields of medicine. The extraordinary cutting properties of piezoelectric bone surgery have been introduced and applied in maxillofacial surgery, cranial and spinal neurosurgery, and hand-foot surgery.[3-13]

The most compelling characteristics of piezoelectric bone surgery are low surgical trauma, exceptional control during surgery, and a fast healing response of tissues. Clinical studies have demonstrated that the specificity of operation and the techniques employed with piezoelectric bone surgery make it possible to advantageously exploit differences in hard and soft tissue anatomy.[3,23,25,26] This not only increases treatment effectiveness but it also improves postoperative recovery and healing. Experimental studies on animals have shown faster tissue healing when compared to traditional cutting instruments.[3]

Ideally, surgical trauma should be minimized to obtain the optimal healing, which depends on gentle management of soft and hard tissues. Surgery, by definition, alters normal physiology by interrupting the vascular supply of tissues. The degree of surgical invasiveness is extremely important for the quality of tissue healing and may effect whether wounds heal by repair or regeneration. Indeed, when surgical trauma is kept to a minimum it generates enough stimulation to favor healing mechanisms that lead to regeneration. On the other hand, surgical techniques that are more traumatic often lead to greater inflammatory responses with slow healing that may lead to repair and scarring rather than regeneration. For this reason, it is desirable to choose the least traumatic surgical instruments and techniques for any surgical procedure. Piezoelectric bone surgery was conceived and developed precisely to overcome the limits of traditional bone cutting instruments and to achieve the most effective treatment with the least morbidity.

From a mechanical standpoint, the effect of burs or twist drills on bone is characterized by lamellar fracturing in areas adjacent to the cut surface and the deposition of large bone fragments and debris in the endosteal spaces. This finding is thought to be, at least in part, responsible for the inflammatory process that takes place in the immediate postsurgical wound healing and for the delay of osteogenesis observed in these wounds. On the other hand, the

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Figure 75B-1 Piezosurgery device by Mectron Medical Technology.

Figure 75B-2 Piezosurgery osteotomy 7 insert: OT7 (0.5 mm of thickness).

Figure 75B-3 Piezosurgery osteotomy 7 special insert: OT7S (0.35 mm of thickness).

Figure 75B-4 Piezosurgery osteoplasty 1 insert: OP1.

BOX 75B-1 Description and Cutting Properties of Piezosurgery by Mectron

Piezosurgery is an electronic device that generates ultrasonic microvibrations at variable frequency. Its technology is unique in the world (US Patent 6,695,047B2) for the characteristic vibrations generated in parallel and variable frequency modulation for precise cutting of bone with different degrees of density.

The unit has a display that allows the operator to select either the BONE or ROOT operating modes. The BONE cutting mode is used to cut bone with selections that are specific to bone type or density. The ROOT mode is used to shape, debride, and smooth root surfaces (both external: periodontal and internal: endodontic).

Bone Operating Mode

Cortical Bone

The basic low ultrasonic frequency (30 kHz) is overmodulated by sound waves for cutting and removing small cortical bone fragments.

Spongy Bone

The basic low ultrasonic frequency is overmodulated by sound waves that are slower than those for cortical bone, which is better for cutting and removing cancellous bone fragments.

Root Operating Mode

Periodontal Surgery

The low ultrasonic frequency without overmodulation is set at an ideal power level for scaling, debridement, and root planing.

Endodontic Surgery

The ultrasonic frequency is set at an ideal power level for retrocanal and intracanal debridement after root canal treatment.

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Figure 75B-5 Piezosurgery osteoplasty 3 insert: OP3.

Figure 75B-6 Piezosurgery implant site preparation 1 insert: IM1.

Figure 75B-7 Piezosurgery implant site preparation 2 insert: IM2.

Figure 75B-8 Piezosurgery implant site preparation 3 insert: IM3.

BOX 75B-2 Description of Piezosurgery Inserts

The mechanical action of bone cutting takes place thanks to the linear microvibrations of inserts with a variable range from 20 to 80 μm, depending on efficiency. Piezosurgery inserts are classified based on their functional and clinical characteristics.

Functional Classification

Sharp: These inserts have sharp ends for osteotomy and osteoplasty. They are made of nitride titanium steel and they are gold in color.

Smoothing: These nitride titanium inserts are diamond coated and gold in color. Their different granulometry produces a smoothening action that is generally used to complete the cut near soft tissue.

Blunt: These steel color inserts are characterized by rounded ends and are generally used to refine the cut in contact with soft tissue.

The inserts described in the functional classification as sharp, smoothing, and blunt have clinical classification codes that relate to their specific use.

Clinical Classification

OT: The identification code for inserts used to perform osteotomy is *OT* followed by a number.

OP: The identification code for inserts used to perform osteoplasty is *OP* followed by a number.

EX: The identification code for inserts used to perform extraction is *EX* followed by a number.

IM: The identification code for inserts used to perform implant site preparation is *IM* followed by a number.

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Figure 75B-9 Piezosurgery periodontal surgery 2 insert: PS2.

Figure 75B-10 Piezosurgery elevation (of schneiderian membrane) 1 insert: EL1.

micromechanical cutting action of piezoelectric bone surgery results in micronization of the cut bone and does not cause lamellar fracturing in adjacent bone, which may favor exposure and release of bone morphogenetic proteins and be responsible for the early onset of osteogenesis at these sites (see below). Furthermore, there may be a diminished inflammatory response since there is little or no need to remove damaged bone and surgical debris as compared to conventional rotary drilled sites.

CLINICAL CHARACTERISTICS OF ULTRASONIC CUTTING

The primary clinical characteristics of the piezosurgery cutting action include microprecision, selective cutting, maximum visibility, and excellent healing.

Microprecision

Piezosurgery cuts mineralized tissues with microprecision. The cut is made by mechanical microvibrations at a linear range of about 80 µm and a frequency of 30,000 times a second. A sound wave is overmodulated on this base frequency, which generates a hammering action with very little heat because the mechanical energy necessary to produce the microvibrations is very low. This action, along with the water spray, facilitates removal of bone debris.

Piezoelectric osteotomies are easy to create, but it is important to recognize that the technique and instrument handling are different than the technique using a traditional handpiece with rotary instruments. The piezosurgery insert is applied to the bone with a relatively light stroke similar to the smooth precision used to draw a picture. Heavy pressure or force is not required. Indeed, the pressure applied by the surgeon to the piezosurgery handpiece is much lower than the pressure typically applied to a rotary or oscillating type handpiece, which uses mechanical macrovibrations for cutting. This characteristic provides maximum control during surgery and makes this technique unique especially in areas with delicate anatomy.

Selective Cutting

Piezosurgery ultrasonic microvibrations are low frequency and selective for cutting mineralized tissue only. These microvibrations are physically unable to cut soft tissue. Clearly, the most significant demonstrated benefit of piezosurgery selective cutting is the ability to preserve the integrity of soft tissues, such as the alveolar nerve, the infraorbital nerve, the maxillary sinus membrane, and the dura mater, while effectively cutting the mineralized tissue (bone) in close proximity to these tissues.

Maximum Visibility

Piezosurgery creates a surgical field that is blood free during cutting because of its cavitation effect. Cavitation is a physical phenomenon that, from a clinical standpoint, happens with the nebulization of the saline solution. The slight hydropneumatic pressure applied by piezosurgery temporarily stops bleeding from both hard and soft tissues. It is important for the liquid pump to be flowing properly and for the cutting action to be intermittent to maintain optimal surface microcirculation, especially for long surgical procedures. Tissue perfusion resumes shortly after cutting (and cavitation action) is stopped.

Excellent Healing

Clinical studies comparing use of piezoelectric bone surgery with traditional rotary instruments for third molar extractions[14] and periodontal surgery[25] have reported better recovery and fewer postoperative symptoms in those treated with piezosurgery. Postoperative healing after piezoelectric bone surgery is characterized by minimal swelling and little bleeding and postoperative morbidity is lower compared to traditional techniques.[24] Gingival tissue is typically light in color when compared to the appearance of autogenous gel of platelet-rich plasma.

CLINICAL APPLICATIONS

There are many important clinical applications of piezoelectric bone surgery in dentistry. In fact, nearly all techniques previously performed with burs, twist drills, chisels, or oscillating saws have the potential to be performed with piezosurgery. For 10 consecutive years the author (TV) has used piezosurgery on a daily basis for oral, periodontal, and implant surgical procedures, enabling him to develop techniques and protocols for each. Readers are referred to

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online materials and other publications for detailed descriptions and step-by-step instructions on these piezosurgery protocols. A brief overview of piezosurgery clinical applications for tooth extraction, implant site preparation, sinus lift, ridge expansion, and bone harvesting are described here.

For more information on piezosurgery clinical applications for periodontal surgery, crown lengthening, and tooth extraction techniques, please visit the companion Expert CONSULT website at www.expertconsult.com.

Implant Site Preparation

Special piezosurgery inserts developed for bone perforation have enabled the development of a new technique for ultrasonic implant site preparation (UISP). Extensive experience gained over 5 years using the technique to prepare more than 1500 implant sites has culminated in the development of a protocol with significant clinical advantages.

The first advantage of UISP is related to the cutting characteristics of piezosurgery, which facilitate differential preparation of the cortical and cancellous bone. A surgical bone classification system was developed that simplifies the diagnosis and surgical decision-making process by exploiting differences in bone anatomy (Figure 75B-27). The differential implant site preparation (DISP) technique can be used within the initial osteotomy site to correct the implant axis by selectively directing the cutting action in the desired direction.[22] DISP can also be used in combination with twist drills to facilitate preservation of alveolar crestal bone while achieving maximum primary stability.[32]

The second advantage of UISP is the fast clinical healing of both soft and hard tissue. Animal research has demonstrated that new bone formation (neo-osteogenesis) is active at implant sites prepared with piezoelectric bone surgery earlier than at sites prepared with traditional techniques.[3] A histomorphometric study on mini-pigs showed more bone formation and a greater density of periimplant osteoblasts at implant sites prepared with piezosurgery as compared to sites prepared with twist drills.[3]

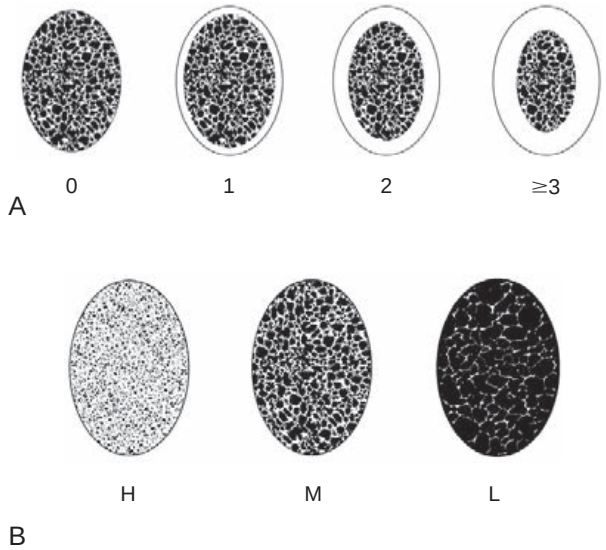

Figure 75B-27 Surgical bone classification by Tomaso and Giuseppe Vercellotti. **A,** Cortical crestal thickness in millimeters. **B,** Radiographic cancellous bone classification.

The improved healing response observed at piezoelectric bone surgery–treated sites (compared to sites prepared with traditional drills) may be explained by differences in the level of bone morphogenetic proteins (BMPs), growth factors, and cytokines. In an animal study comparing the biomolecular profile of sites prepared with piezoelectric bone surgery or rotary drills, BMP-4 increased earlier at implant sites prepared with piezoelectric bone surgery.[3] As compared to drilled sites, the levels of BMP-4 at piezoelectric bone surgery–treated sites were 18.5 times higher at 7 days, 15 times higher at 14 days, and 2 times lower at 56 days. The peak levels of BMP-4 were observed at 14 days for piezoelectric bone surgery–treated sites. The level of BMP-4 at drilled sites did not reach the same level of BMP-4 achieved at piezoelectric bone surgery–treated sites until day 56. There was also a greater increase in the level of transforming growth factor beta2 (TGF-β2) cytokine at implant sites prepared with piezoelectric bone surgery techniques. As compared to drilled sites, the levels of TGF-β2 at piezoelectric bone surgery treated sites were 3.5 times higher at 7 days, 19 times higher at 14 days, and diminished below baseline at 56 days. The level of TGF-β2 at drilled sites never exceeded baseline levels.

Proinflammatory cytokine tumor necrosis factor alpha (TNF-α), proinflammatory and bone-resorbing cytokine interleukin-1 beta (IL-1β), and antiinflammatory cytokine IL-10 were also measured. In general, the expression of these proinflammatory cytokines was higher in the early experimental phases of drilled sites only, which also showed more inflammatory cells. The piezoelectric bone surgery–treated sites showed a higher expression of the proinflammatory and bone-resorbing cytokine IL-1β at 56 days. This latter finding might be indicative of bone remodeling at day 56 in piezoelectric bone surgery treated sites (IL-1β is involved in osteoclast differentiation).

It is assumed that the micronization of the bone reduces cut trauma while saline solution cavitation produces debridement of the osteotomy surfaces with exposure and preservation of a high number of bone morphogenetic proteins and growth factors. These proteins are capable of increasing the mitosis of stromal stem cells and osteoblasts.

The surgical technique entails an osteotomy preparation using a series of piezosurgery insert. The preparation is initiated with the narrow, pointed IM1 insert (Figure 75B-28). Parallel pins are used throughout the procedure to check alignment (Figure 75B-29). The 2-mm diameter IM2 insert is used to further prepare the osteotomy (Figure 75B-30). The coronal aspect of the preparation is widened

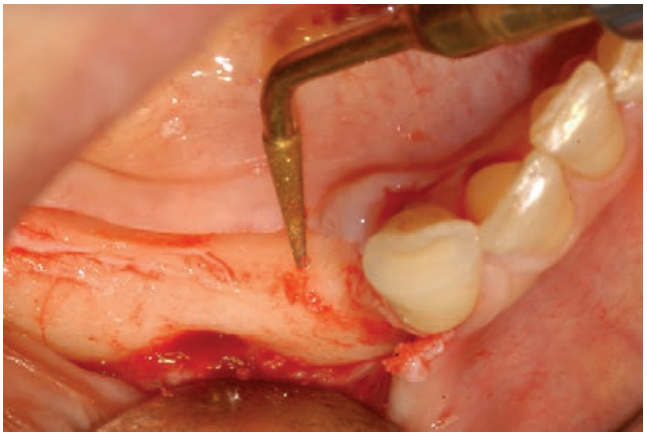

Figure 75B-28 Piezosurgery insert (IM1) used to start the implant site preparation.

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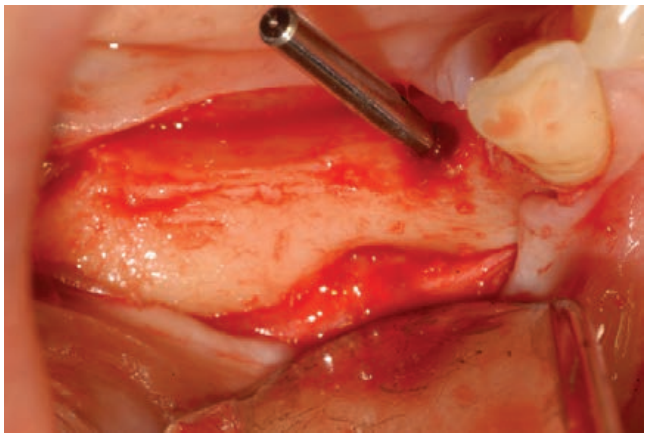

Figure 75B-29 Parallel pin in place to check the direction of osteotomy.

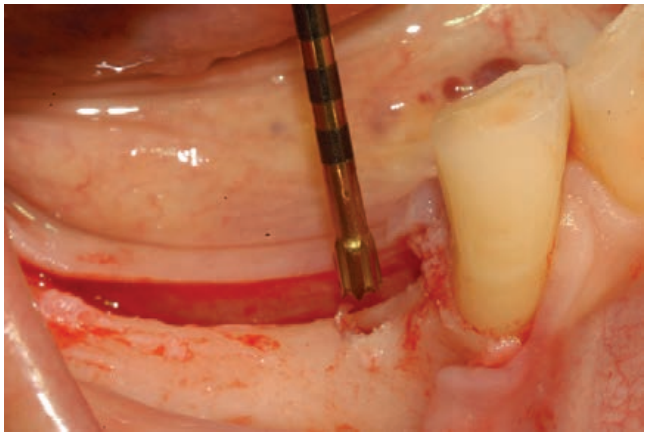

Figure 75B-30 Piezosurgery 2-mm diameter insert (IM2) for pilot osteotomy.

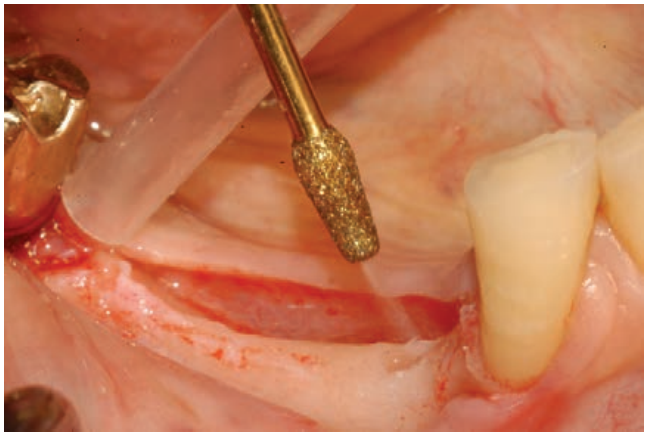

Figure 75B-31 Piezosurgery insert for cortical preparation (pilot 2/3).

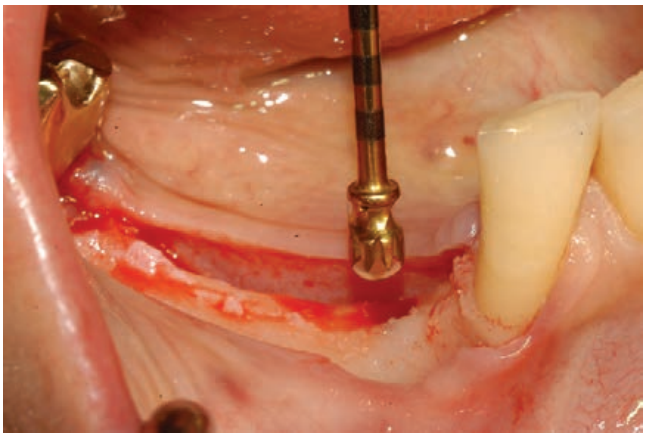

Figure 75B-32 Cancellous bone preparation with piezosurgery insert (IM3).

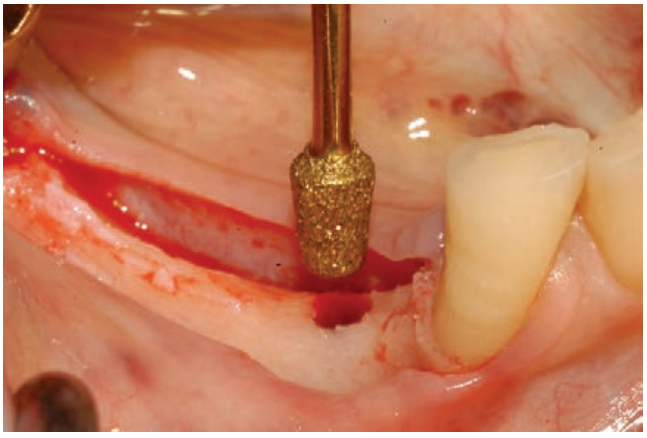

Figure 75B-33 Piezosurgery diamond-coated insert (IM4) for final cortical bone preparation.

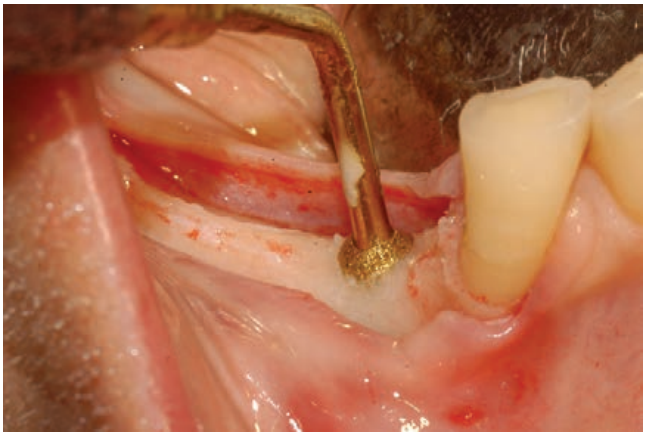

Figure 75B-34 Piezosurgery pilot insert 3/4 in action.

with the pilot 2/3 insert (Figure 75B-31), and the osteotomy is finished with the IM3 insert (Figure 75B-32). Final cortical preparation is accomplished with the IM4 insert (Figure 75B-33). After pilot 3/4 insert (Figure 75B-34), the implant osteotomy is completed (Figure 75B-35). If desired, before implant placement, osteoplasty of the crestal bone is possible with the OP3 insert (Figure 75B-36). Figure 75B-37 shows the occlusal view of a well-placed implant in a narrow ridge.

For more information on sinus lift, please visit the companion Expert CONSULT website at www.expertconsult.com.

Ridge Expansion

Horizontal alveolar ridge expansion is an extremely useful technique for increasing bone width and simultaneously placing

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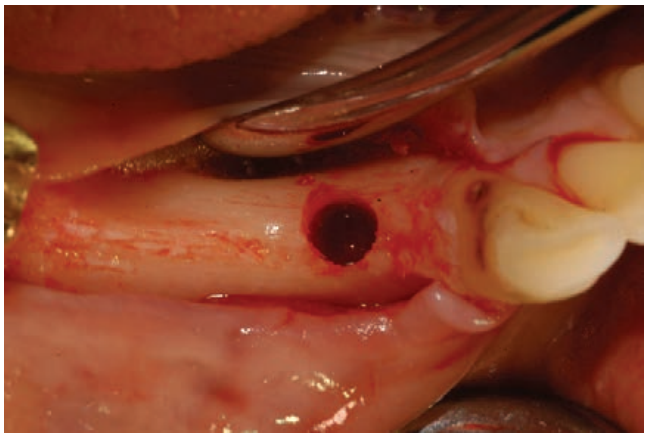

Figure 75B-35 Occlusal view of completed implant site preparation.

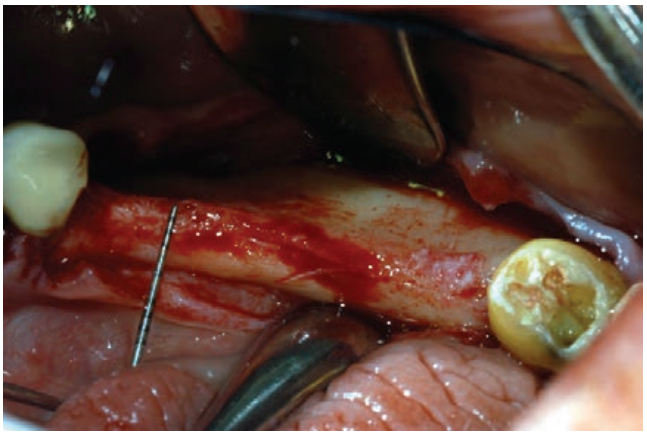

Figure 75B-46 Evaluation of narrow (3 mm wide) posterior mandibular ridge with a periodontal probe.

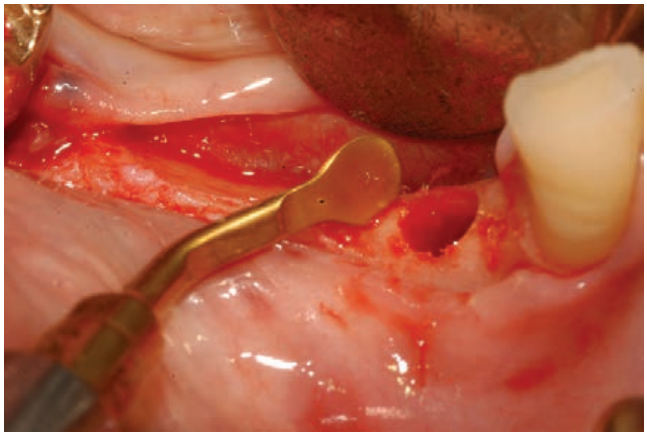

Figure 75B-36 Osteoplasty of crestal bone in the periimplant area using piezosurgery insert (OP3).

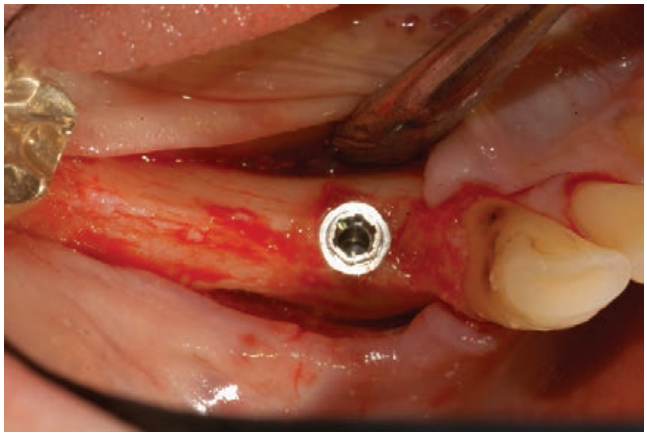

Figure 75B-37 Occlusal view of well-placed implant despite the narrow ridge.

implants in narrow ridges. The great advantage is that both augmentation and implant placement are accomplished in one surgical procedure. A possible disadvantage is that crestal bone loss may be greater in cases that begin with very narrow ridges.

Clinical and radiographic evaluation of the size, shape, and morphologic characteristics of the alveolar bone in the ridge is necessary to determine whether to use a ridge expansion or a traditional bone augmentation technique (Figure 75B-46). In brief, it can be said that this technique exploits bone elasticity so it is necessary to know the bone anatomy of each implant site. In particular, the morphology of the crestal bone and the thickness of the buccal cortical and lingual cortical plates in relation to the amount of cancellous bone present in the ridge greatly influences the ease and ability to prepare an osteotomy and to expand the ridge. The technique requires preservation of periosteum integrity while elevating mucogingival flaps enough for access to create the appropriate osteotomy. Piezosurgery is an indispensable tool used to create a horizontal osteotomy through the alveolar bone crest caused by its precise (narrow) cutting action. In some cases (e.g., areas of dense bone with little elasticity), it may also be necessary to make one or two vertical cuts in the alveolar bone to allow ridge expansion. The horizontal osteotomy is expanded in subsequent steps using piezoelectric inserts for implant site preparation together with screw-type or fan-type expanders for increasing the diameter or section, respectively.

Expanding a narrow ridge by the entire width (diameter) of the planned implant(s) may be challenging because of the density of cortical bone and limited elasticity. The need to expand or displace the buccal cortical bone can be reduced (i.e., less than the implant diameter) with rigorous application of DISP to the lingual cortical bone (i.e., preferential preparation of the lingual side), which is generally thicker than the buccal cortical. This is difficult to do with traditional twist drills because they cannot limit cutting to one side and tend to get displaced by areas of dense bone into areas of loose or missing bone (i.e., twist drills are deflected away from dense cortical bone). In fact, attempting to prepare the site (between separated cortical plates) with traditional drills can fracture the weaker, inelastic buccal cortical bone, making simultaneous implant placement impossible.

Piezoelectric bone surgery has radically simplified the ridge expansion technique in the horizontal osteotomy phase, which can be performed with micrometric precision, and in the site preparation phase, which exploits the different degree of cortical resistance compared to cancellous bone (Figures 75B-47 to 75B-51).[17,31]

Bone Harvesting

The bone harvesting technique using piezosurgery is effective for harvesting bone fragments and for removing blocks of monocortical-cancellous bone.

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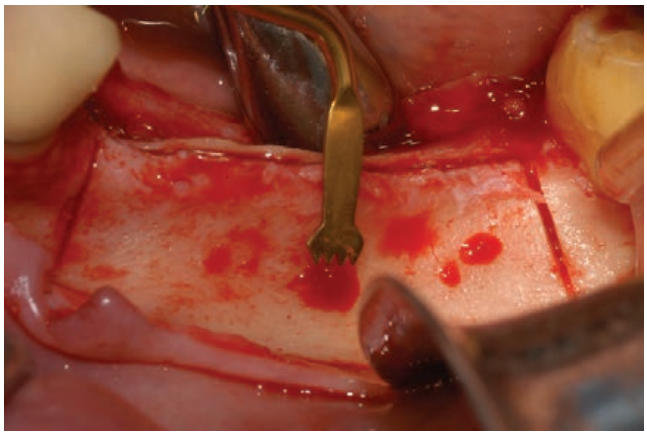

Figure 75B-47 Horizontal and vertical osteotomies prepared with narrow (0.35 mm) piezosurgery insert (OT7S).

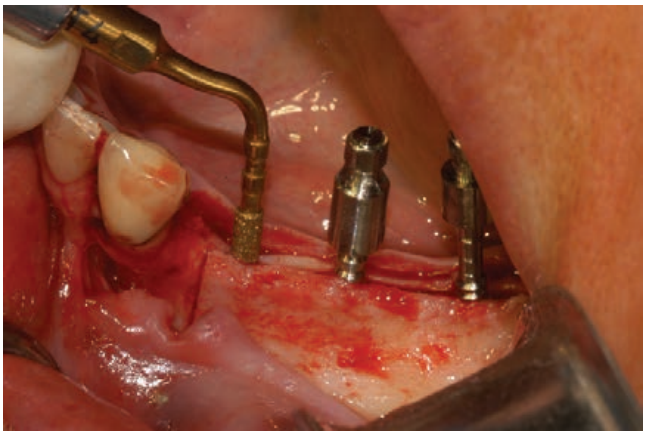

Figure 75B-50 Differential implant site preparation (DISP) using piezosurgery insert (OT4).

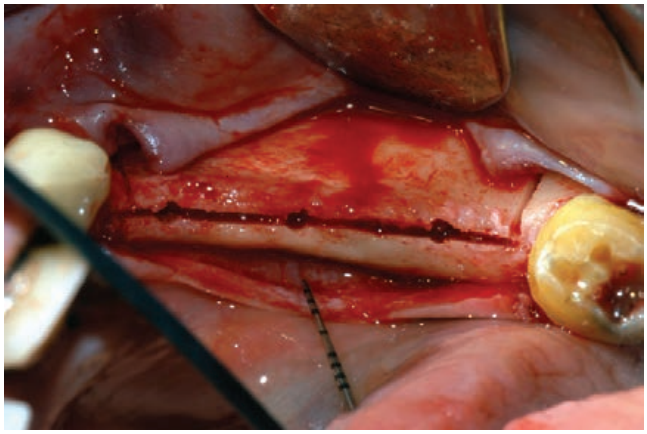

Figure 75B-48 Initial implant site preparation started with piezosurgery insert (IM1).

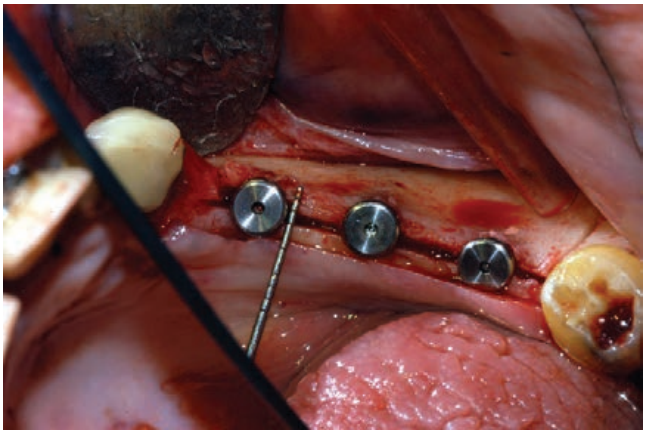

Figure 75B-51 Mandibular ridge expanded, implant sites prepared, and three implants simultaneously placed. Periodontal probe demonstrates final ridge width.

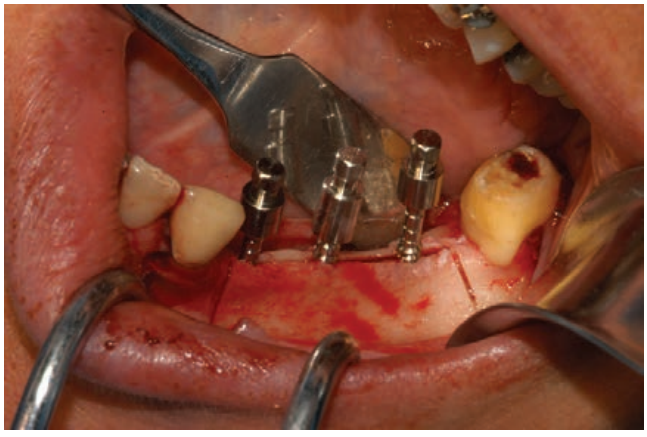

Figure 75B-49 Screw expanders used to separate bone segments and begin expanding alveolar ridge.

Particulate Bone Chip Harvesting Technique. Particulate bone chips are harvested using special osteoplasty inserts to scrape the bone surface. The flow of the irrigation fluid should be reduced to prevent particles from being dispersed after harvesting. The end of the aspirator tip should be kept away from the collecting particles or placed in the opposite direction of the insert movement.

There are two piezosurgery inserts used for performing this bone chip harvesting technique. Osteoplasty insert no. 1 (OP1) is the most effective because it is large and quickly harvests bone fragments. However, the size and surgical movement requires donor sites with ample surface area. It is generally used in bone grafting techniques for implant surgery. Osteoplasty insert no. 3 (OP3) is smaller and tends to harvest smaller bone fragments in any anatomic situation. It can be used in any site to collect bone for any bone grafting technique but is best used in limited areas to harvest smaller quantities of bone for periodontal osseous defects.

Clinical Note It is advantageous to use OP1/OP3 inserts to collect bone (for grafting defects) while performing other procedures (e.g., in maxillary sinus lift surgery while removing bone from the lateral window osteotomy or in periodontal surgery while recontouring bone with osteoplasty/ostectomy).

Block Harvesting Technique. Choosing an appropriate donor site and selecting the best instruments and technique for harvesting block bone are important decisions in the success of bone augmentation with monocortical bone block grafts.

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SCIENCE TRANSFER

The use of a piezoelectric cutting instrument with an amplitude of 80 μm and a frequency of 30,000 cycles per second provides a precise, minimally traumatic alternative to the use of rotary cutting instruments. It can be used for periodontal surgery, implant surgery, and tooth extractions but has particular advantages when used in lateral wall sinus lift surgeries. The cutting tip used for this procedure only cuts bone and does not cut soft tissue so it reduces the risk of surgically induced tearing of the sinus lining membrane. Although it takes a little longer than conventional rotary bone cutting, piezoelectric bone surgery is also valuable for harvesting bone blocks for bone augmentation procedures.

Harvesting block bone with piezosurgery is extremely simple, precise and fast.

The most common donor sites for harvesting monocortical-cancellous block bone are the mandibular ramus and symphysis (chin). The surgical operation with piezosurgery offers considerable precision and operating sensitivity during surgery so that the operator is able to easily recognize differences in cortical and cancellous bone allowing him/her to preserve the integrity of important structures.

Harvesting a monocortical-cancellous bone block from the mandibular ramus is generally preferable to the symphysis region because of the reduced level of morbidity. Evaluation of presurgical three-dimensional (3D) scan images facilitates measurements and determines how much bone can be harvested with respect to the position of the inferior alveolar nerve canal, the position of molar roots, and the width of the mandible in the ramus area. Osteotomies performed with piezosurgery allow an extremely precise, clean, and smooth vertical cut through bone with excellent visibility.[2] The result is that a cortical block can be removed with a portion of spongy bone and its surface is extremely regular compared to blocks harvested from the chin.

This technique is very predictable because it facilitates harvesting the maximum amount of bone with respect to the donor site. By comparison, bone blocks harvested using traditional rotary cutting instruments reduce the width of the cortical bone by at least 1 mm circumferentially around the entire graft. Furthermore, traditional cutting instruments are unable to effectively cut the internal cancellous bone. The result is that the block is detached by using scalpels or chisels to break it away, which makes the width irregular. Subsequently, it becomes necessary to further reduce the block width.

CONCLUSION

Piezosurgery is a new surgical technique for bone surgery with many clinical applications in dentistry. The extraordinary cutting properties and applications of piezoelectric bone surgery were described. The most compelling characteristics of piezoelectric bone surgery are low surgical trauma, exceptional precision, and fast healing response. As a result, piezosurgery has the ability to increase treatment effectiveness while improving postoperative recovery and healing.

Although a great deal of scientific research focuses on new products for tissue engineering and bone regeneration, the importance of minimal surgical trauma for optimal bone healing and regeneration should not be overlooked. A new appreciation for the effectiveness of piezosurgery has the potential to redefine the concept of minimally invasive surgery in osteotomy and osteoplasty procedures.

References and Suggested Readings can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 75C

Technologic Advances in Implant Surgery: Computer-Assisted Implant Surgery

Daniel H. Etienne, Raymond R. Derycke, and Perry R. Klokkevold

CHAPTER OUTLINE

COMPUTER-ASSISTED IMPLANT SURGERY
- Uses and Requirements
- Sequence of Steps
- Preoperative Data Acquisition
- Identification and Registration
- Navigation and Positional Tracking
- External Viewer, Augmented Reality, and Three-Dimensional Projection Screens
- Advantages and Disadvantages

CONCLUSION

Implant surgical procedures have more or less remained the same since the introduction of osseointegrated dental implants. Briefly, implant placement surgery involves the use of a full-thickness flap elevation and a sequential series of drills with increasing diameters under profuse irrigation to create a precise osteotomy site in the bone for the placement of a dental implant (see Chapter 71). The site is prepared, and the implant positioned to avoid important anatomic structures, such as the inferior alveolar nerve, sinus cavities, and teeth, and to optimally support and emulate the planned prosthetic tooth replacement(s). Proper implant position is important for optimal function and esthetics.

Clinicians determine implant positions based on presurgical diagnostic imaging, study models, and the use of a diagnostic wax-up of the planned tooth replacement(s). The actual or final position of the implant(s) results from the surgeon's interpretation of diagnostic information and his or her ability to translate that information to the patient at surgery. Most often, the surgical "guide" between the diagnostic information and the patient is an acrylic stent, fabricated by a laboratory technician, that may or may not have precise guide channels for implant positioning. Therefore inaccuracies in the stent fabrication and movement of the stent during surgery, as well as variations in the use of the stent during surgery, can lead to imprecise implant positioning.

Recent advances in implant surgical technology include the use of the following:

- Computer-imaging software is used preoperatively to "simulate" the implant position(s) into a virtual patient, that is, a three-dimensional (3D) computer image of the patient's jaw created from the computed tomography (CT) scan data.[19]
  - Computer-generated surgical guides with drill holes based on the presurgical "virtual" implant positioning are used to more accurately place implants in the "planned" locations.[8,10,14]
- Computer-assisted implant surgery (CAIS) uses simultaneous tracking and "guidance" of the implant instrumentation to accurately follow the planned treatment during surgery.[4]

CAIS is the most sophisticated and perhaps the most promising of these technologies because it has the greatest potential to reduce surgical time, minimize surgical invasiveness, and result in a more precise translation of implant planning to the actual surgical procedure.[18] Understandably, CAIS is also the technique that requires the greatest amount of preparation and coordination of the patient, image data, and surgical instrumentation. The following provides a conceptual overview of the techniques and terminology used in CAIS.

COMPUTER-ASSISTED IMPLANT SURGERY

Uses and Requirements

Computerized navigation surgery evolved from early applications in neurosurgical procedures and continues to evolve today with applications in many surgical specialties.[13,15] Clearly, the advantage of using a computer to assist surgery is the precision that it offers. There is also a real-time safety control obtained with multiple imaging sources facilitating a minimally invasive approach to surgical procedures. As in medicine, 3D imaging is used in dentistry to facilitate presurgical planning and to guide the surgical placement of dental implants. This allows precise positioning while avoiding injury to nearby important anatomic structures. Several different approaches to computers have been used in dental implant surgery, from simple imaging software to visualize the implant positions in a 3D virtual patient to more complex, simultaneous image monitoring and instrument navigation used to perform surgery.[4]

The use of CAIS requires precise and continuous coordination of the patient, the image data, and the surgical instrumentation. Therefore CAIS requires an accurate alignment (identification and registration) of the patient with the patient's image data (3D CT scan data) and a system of tracking the precise movements of the surgical instrumentation (e.g., handpiece, drills) in relation to the actual patient. A variety of systems have been developed to acquire and register image data and to coordinate and track movements. These methods are generically described in the following sections.

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Sequence of Steps

The clinical sequence of steps required for CAIS is as follows:

1. *Data acquisition*. The patient is scanned for image data acquisition (e.g., CT scan) with fiducial (artificial) radiographic markers (e.g., stent with markers or intentionally placed pins or screws into jaw) or anatomic (natural) markers such as teeth or bony landmarks. If fiducial markers are placed in a stent, the patient must have it in place when scanned.
2. *Identification*. The anatomic or fiducial markers will be identified with a probe tracked by the system. If markers were incorporated into a radiographic stent, the stent will again be placed in the mouth and the markers identified by hand with a probe tracked by the stereovision system.
3. *Registration*. After identification of the predetermined markers, the software will indicate the best localization or "match" on the arch between the image data and the patient. If registration is not validated, matching can be improved with additional points. An invalidated registration may be caused by an improper initialization or CT scan data.
4. *Navigation*. Ultimately, the operator will be able to visualize surgical instrument navigation (movement). The drilling instruments will be guided to a target point of impact with a 3D spatial orientation.
5. *Accuracy*. Sustained-accuracy procedures are critical during surgery and should prove reliability in regard to the system's overall accuracy. This sustained-accuracy procedure will be done through contact of the drill on the handpiece with selected teeth, by visualization of markers, which can be viewed by the stereovision system, or repositioning of the radiomarker stent.
6. *Feedback*. Variations from the ideal position can be restricted by the software through inactivation of the drill (stop-and-go action) or by an audible or visual cue.

Preoperative Data Acquisition

CT scans and the more-recent cone-beam CT (CBCT) scans are widely used for 3D patient imaging (see Chapter 70). Factors that must be considered when deciding to use a CT scan include radiation exposure, limitations in accuracy, and the possibility of diffracted images as a result of metallic restorations. The evolution of scanner technology (spiral CT scan, CBCT scan) has made it possible to reduce the radiation dose to the level of a conventional panoramic radiograph while maintaining adequate diagnostic quality for preoperative implant planning.[6]

Similar to implant planning using conventional diagnostic methods, radiographically identifiable markers are important for CAIS. However, unlike conventional planning, in which orientation and simulation of implant positions are related to the planned prosthetic crown positions, CAIS markers must relate the image data to the actual patient anatomy. In other words, the position of the surgical instrumentation (and ultimately the implant) must be related to the scan image data of the patient's jaw morphology. Thus it is critically important to scan the patient with markers that are identified in the scan and correlated to the patient at surgery. Markers can be anatomic markers, such as teeth or specific bony landmarks, or artificial markers (fiducial), such as small tacks or screws that are secured in the bone.

Identification and Registration

After scan acquisition, the 3D image data of the patient's jaw(s) are interpreted by software as anatomic geometric elements. As previously stated, a sufficient number of markers (anatomic or fiducial) must be identified in the patient's mouth to correlate the image data with the patient anatomy. Several devices have been used to capture the actual patient anatomy for registration with the scan data, including a touch pointer and an ultrasound probe. The touch pointer allows the operator to touch specific anatomic points (or fiducial markers) while a tracking device "sees" the instrument and records each point of reference. This device is fairly accurate, but if the clinician is not careful, there is a tendency to define points that are not in contact to a real surface, thus creating a false mapping. The ultrasound probe has a lower accuracy than the touch pointer but has the advantage of being able to capture continuous data of bone morphology through the mucosa or gingiva. Echo quality is subject to variations with the type of penetrated tissue.

Matching between the geometry of the image data with the patient anatomy is called registration. Five registration methods are used to compare anatomic points of the preoperative image data and the intraoperative patient anatomy: (1) point-based, (2) line-based or curve-based, (3) surface-based, (4) volume-based, and (5) projective methods. Point-based and line-based methods are described here to illustrate the requirements for an adequate registration.

In the point-based method, a few particular points are identified in the preoperative image data and the patient anatomy. These can be natural (anatomic points) or artificial (fiducial) markers. In either case, points must be well defined and stable so that they can be precisely measured. The operator can manually click, with a tracking device, on the point in the patient corresponding to the image point. After several points are matched, the computer calculates a transformation equation that minimizes the mean distance between matched points to complete the registration. Three nonaligned points are necessary to obtain a nonambiguous result. The registration accuracy can be predicted, depending on the distribution of points (e.g., an equilateral tripod will give more accurate results than three co-linear points).[7,12]

The most intuitive algorithm may be the Hough transformation, in which the most simple geometric invariant, the distances between points, are computed, compared, and used to find a triangle in the preoperative set of points and a triangle in the intraoperative set of points that shares similar edge lengths. The triangle points are then registered using the best-fit algorithm. Once a first estimate of the transformation has been computed, the clinician can apply the transformation to the intraoperative points.

Line-based or curve-based methods are derived from point-based methods but require a few lines or curves to be measured and identified. Artificial lines can be extracted from the CT scan image data and measured intraoperatively by the operator with the tracking device. All lines and surfaces measured on image planning (after segmentation of anatomic structure of the jawbone in the CT scan), and the points taken on the patient anatomy by the tracking device are known as a set of points. These sets may be dense or sparse; a segmented bone surface may have hundreds or thousands of points. However, it is not practical to measure hundreds of points.

When the segmented surface is dense, one can assume that almost all points measured with the tracking device will be identified as points of the segmented surface. Thus, algorithms developed for the point-based registration, based on identification of matching between the preoperative and the intraoperative data, can be easily adapted. However, the number of possible errors in the identification process increases dramatically in line-based or curve-based methods.

Navigation and Positional Tracking

Numerous commercial products exist for navigation or positional tracking, but few meet the *computer-aided surgery* (CAS)

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requirements in terms of accuracy (about 1 mm in one cubic meter), reliability, and clinical usability. The "real-time" navigational technology is based on the global positioning system (GPS) technology.[16] Some of the technologies used in medical CAS to track movement include mechanical, magnetic, and optical tracking systems.

Mechanical tracking systems use a six-axis coding robot with a passive arm. The system is very reliable and highly accurate but has limitations when more than one instrument or patient marker needs to be located. Thus mechanical tracking is less desirable for CAIS, which requires the use of several different instruments and multiple markers.

Magnetic tracking systems use a magnetic source and a field receiver. The system loses accuracy in the presence of magnetic field interference. Relative inaccuracies result from changes in the magnetic field, which may be caused by any metallic mass that may be present, such as a drill motor (with or without activation).[2,3] Thus the obligatory presence of drill motors in the operatory during implant surgery makes magnetic trackers impractical for CAIS.

Optical tracking systems are recognized for their dependability and accuracy. Positioning is made by intersecting the vision plane between two or three cameras to locate markers with stereovision. A passive system absorbs and processes ambient light, whereas an active system interprets reflected light.

Active markers with infrared light–emitting diodes (IRED) have been widely used with superb accuracy but are sensitive to reflections and interference with the line of sight between the IRED markers and the cameras.[9,11] Although variations in optical localizers are adequate for medical applications,[1,5] they need to be improved for use in dental implant surgery. This is particularly problematic with the typical seating arrangement of surgeon and assistant (i.e., the direct line of sight to the cameras may be interrupted by the operators). A stereovision with natural-light cameras is a less expensive alternative compared with IRED. However, natural-light systems are more sensitive to surrounding light, background, and the shape of markers. In comparison, infrared cameras are less sensitive to these light variations.

With optical tracking devices, the surrounding light in the operatory is important, and a headset light is preferred with a camera sensitive to natural light. Patient motion will be tracked efficiently if the marker is stable during surgery. In case of loose teeth or unstable markers, cortical bone screws should be used.

External Viewer, Augmented Reality, and 3D Projection Screens

Once registration between the data and the actual patient has been established, the instrumentation can be coordinated with the system and observed by the surgeon/operator (Figures 75C-1 and 75C-2). Instrument movement relative to the image data (and through registration to the patient) may be viewed on an external monitor, visually projected in the surgeon's field of vision (resulting in a superimposed visual image seen over the surgical field) using a head-mounted projection system or projected on a 3D projection screen (Figure 75C-3).[20] In this manner, the image on the external monitor (see Figure 75C-2), the surgical field, or the 3D projection screen (Figures 75C-4 and 75C-5) will guide the surgeon to perform the planned procedure.

Side viewers display target data in two dimensions and require the surgeon to look away from the surgical field. However, see-through viewers display target data transparently in the surgeon's field of view and allow the operator to observe the surgical field continuously. An augmented-reality viewer allows the surgeon to see target data in three dimensions, superimposed over the surgical site through projected images in both eyes.[21] The augmented-reality method allows the operator to adapt to the system more naturally and therefore more rapidly, but there do not appear to be advantages over the use of a two-dimensional (2D) side-viewer devices in terms of accuracy.[17] Both systems allow simultaneous viewing of virtual information of the implant (axis and target) and a real vision of the surgical site. Augmented-reality devices are very sensitive to calibration before surgery and require care and monitoring intraoperatively to prevent misalignment during the surgery. The relative stability of a headset is critical to maintaining accuracy.

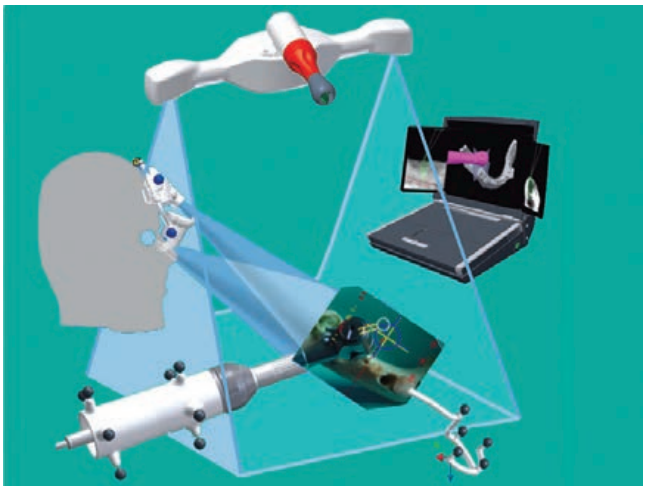

Figure 75C-1 Global setup for surgical navigation. The basic setup for a navigation system consists of stereovision cameras with several tools. The contra-angle probe, the ultrasound probe and the patient jig need to have markers, which are tracked by the cameras. The occlusal stent, with markers in the standard process and without markers in ultrasound registration, used during computed tomography (CT) scan acquisition will be recognized in its three dimensions for prosthetic planning.

Alternatively, 3D projection screens provide a "real 3D vision" viewed on a specialized monitor (see Figure 75C-3) without the need for viewers (i.e., operator glasses).

Two types of 3D projection screens are available, as follows:

1. The multiplane device has a screen that provides three simulated real-time planes. The object is projected in the screen with a back-field simulated projection. A plane within the screen surface and the forward plane are at a maximum focus of 10 to 20 cm. This device provides a 3D view that is extremely dependent on the shape.
2. A new generation device uses nanolenses on each pixel of screen resolution. It provides a natural light splitting effect similar to the effect of a natural eye separating the three basic colors. Consequently, it is perceived as a natural view. A lateral displacement of the operator's head provides 8 to 12 simulated views, which together create a natural "volume" effect. No adaptation time is required and viewing is intuitive.

This 3D projection screen technology cannot provide a true holographic 3D view because of the eye's plane of focus on a flat screen, but it does provide a 3D "real volumetric view." In addition to this improvement in 3D vision, a 3D sequence of images can be modified for specific applications such as navigation. For example, a 3D sequence of 8 views can be modified to facilitate the observation of a projected view of the object at 90° with a minimal head side shift of only 5 degrees. These real-time, 3D front and side views

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Figure 75C-2 Images of computer screen and surgeon's view of computer-aided implant surgery (CAIS) navigation simulated with dry mandible. During navigation, the surgeon concentrates on visual cues seen on the eye viewer.

Figure 75C-3 Dental implant real-time navigation system with infrared stereovision cameras and a three-dimensional (3D) monitor display showing the reconstruction of a computed tomography (CT) scanned mandible.

provide intuitive 3D data views in spite of the 2D parameters used to visualize them.

Advantages and Disadvantages

Benefits and advantages of computer-assisted implant surgery include the following:

1. CAIS results in improved accuracy and real-time safety with information obtained from multiple data sources for redundancy and precision.
2. Surgeon validation and expertise are maintained.
3. Security features can stop the rotary instrument in proximity to important structures.
4. Simulation can be visualized before surgery. A real 3D simulation with a 3D projected screen can be visualized before surgery and all along the surgery.
5. Implant position can be planned before surgery.
6. Real-time information is provided to the surgeon.
7. Inexperienced surgeons will improve their skill with training.
8. Experienced surgeons can treat more challenging cases with greater comfort and confidence.
9. Surgical time is reduced using a surgical guide.
10. Noninvasive surgery is possible; some cases may be performed with minimal or no flap reflection.

Limitations and disadvantages of CAIS are as follows:

1. Initial cost of the system.
2. Increased installation time for surgery.
3. Training time is mandatory.
4. Accuracy depends on the various components of the system.
5. Inaccurate data (e.g., CT scan, displaced markers) can lead to difficulties in registration.
6. Three anatomic or fiducial markers must be visible.

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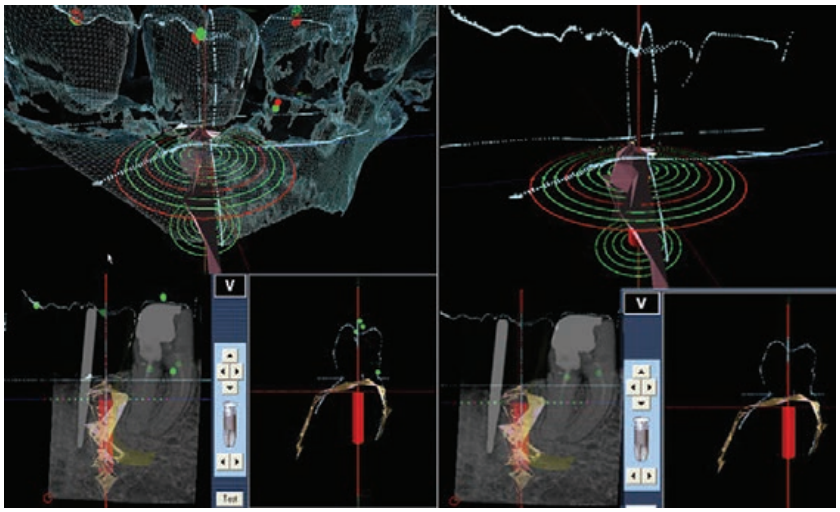

Figure 75C-4 Conventional x-ray and ultrasound acquisition for alveolar bone topography (in light brown, *lower left*) combined with an optical scan image showing crown and alveolar bone morphology (dark green). The implant position is shown in red. This two-dimensional view is obtained without a computed tomography (CT) scan.

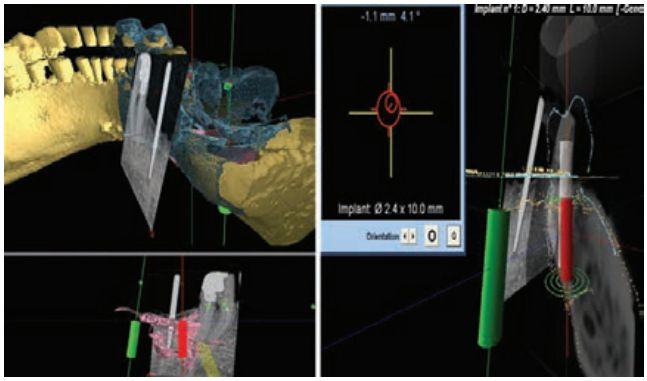

Figure 75C-5 Screen views available during surgery. *Upper left,* Simulated implants in green and red. The computed tomography (CT) scan reconstruction of the mandible and teeth is observed in yellow. Surface mapping of bone and crown morphology (dark green/blue) obtained from ultrasound or optical scanner. The computer compares the data from the three-dimensional (3D) topography to the two-dimensional (2D) x-ray. *Lower left,* The 2D x-ray is matched to a 3D topography of the patient "matrix" with the simulated implant positions in green and red. *Upper right,* The target and position of the drill/implant are observed. *Lower right:* Real-time navigation with a combination of superimposed images from a 2D x-ray, optical scanner, CT scan, and 3D ultrasound.

CONCLUSION

The use of computer-assisted implant surgery with simultaneous tracking and "guidance" of the implant instrumentation facilitates the precise surgical placement of implants with "real-time" visual assessment of anatomic structures. The system is complex, and several steps must be coordinated for CAIS to be accurate and useful. Newer generation systems are becoming more intuitive with 3D projected vision with improved software interface for the operator.

CAIS systems are expensive and therefore not readily available. Surgeons require training and experience to perform CAIS well. As a result, the more complex or difficult cases may be the most likely types of patients to benefit from the use of CAIS. The newer systems are continuing to improve, which may make utilization of CAIS more accessible to surgeons and patients.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

SCIENCE TRANSFER

Computer-assisted surgery (CAS) involves the use of digitalized three-dimensional (3D) data obtained by the use of cone-beam computer-generated radiographs, markers placed in a surgical stent, and a computer-directed guidance system used during implant placement. This new technology is being developed to enhance accuracy of implant placement and to reduce angulation and alignment errors. At present, these systems require extensive training by the clinician, and the intraoperative surgical guidance methodology is being improved so that this approach can have wider utilization.

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CHAPTER 76

Biomechanics, Treatment Planning, and Prosthetic Considerations

Nick Elian, Perry R. Klokkevold, David G. Kim, Ting-Ling Chang, and John Beumer

CHAPTER OUTLINE

BIOMECHANICAL CONSIDERATIONS
- Load-Bearing Capacity
- Angulation and Arrangement

TREATMENT PLANNING WITH DENTAL IMPLANTS
- Edentulous Maxilla (online only)
- Edentulous Mandible (online only)
- Partially Edentulous Patients

STRATEGIES TO AVOID IMPLANT OVERLOAD
- Occlusal Design
- Cantilevers
- Connecting Implants to Teeth

CONCLUSION

Refer to the Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures may be out of numeric order in this printed chapter.

Clinical research has demonstrated excellent long-term success rates for implant-supported restorations in fully and partially edentulous patients. The replacement of missing teeth with an implant-retained prosthesis restores function and esthetics, improves confidence and self-esteem, and enhances quality of life. However, it is important to recognize that implant success is achieved by following sound principles. Good treatment planning is essential for predictability. Perhaps, the most critical factor to be considered in the planning is the control and distribution of stress for preservation of the "biologic" connection between the implants and bone. The quantity and quality of bone available to support implants play an important role in stress distribution,[2] maintenance of bone apposition, and support under functional loading.

This chapter discusses important biomechanical considerations and treatment-planning guidelines for the use of dental implants in fully edentulous, partially edentulous, and single-tooth cases. Occlusal design, cantilevers, and connecting implants to teeth are addressed as well.

BIOMECHANICAL CONSIDERATIONS

Osseointegrated dental implants provide a predictable means of replacing missing teeth and restoring dental function. It is now clear that a thorough understanding of implant biomechanics is essential if implant-retained restorations are to be employed predictably. The load-bearing capacity of implants supporting the restoration must be greater than the anticipated loads during function. If applied loads exceed load-bearing capacity, it may lead to mechanical and/or biologic failure. In the case of *mechanical failure,* screws that secure the restoration may bend, loosen, or fracture. The most devastating type of mechanical failure is fracture of the implant. In the case of *biologic failure,* a resorption-remodeling response of the bone around the implant(s) is provoked, leading to progressive bone loss.[10,18] In some cases, bone loss around the implant progresses until the implant is no longer supported and osseointegration is lost.

The function and support of implant-retained restorations is quite different from tooth-retained restorations. Teeth are suspended within the supporting alveolar bone by a periodontal ligament, which allows slight physiologic "movement" of teeth in function. If forces are excessive, teeth have the capacity to adjust or move in response to the applied forces. In the absence of inflammatory periodontal disease, teeth will adapt to these forces without appreciable bone loss. Orthodontic movement of teeth through alveolar bone is the ultimate example of the capacity of teeth to adapt to excessive applied forces. As orthodontic forces move a tooth, bone resorbs in response to pressure and forms in response

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BOX 76-1 Implant Biomechanics

Load-Bearing Capacity

Implant
- Number
- Length
- Angulation

Quality of bone-implant interference

Anticipated Load (Affected By)

Occlusal factors
Cusp angles
Width of occlusal table
Guidance type
- Anterior guidance
- Group function

Cantilever Forces

Connection to natural dentition
Size of occlusal table
- Buccolingual dimension
- Mesiodistal dimension

Cantilevered extensions

Parafunctional Habits

Chronic bruxism
Clenching habit
Grinding habit

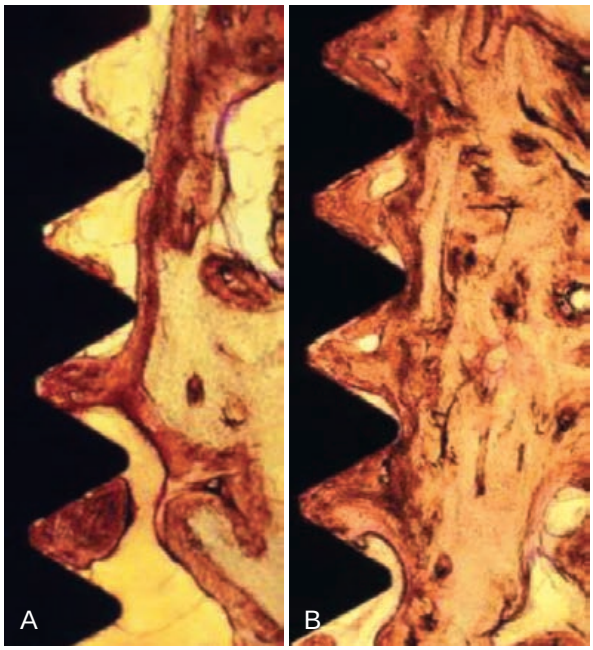

Figure 76-1 Bone quality influences the load-bearing capacity. In these histologic images obtained with light microscopy, note the difference in bone appositional index achieved in **A,** sparse "poor-quality" bone, often found in the posterior maxilla, versus **B,** dense bone, typical of the anterior mandible.

to tension applied by the "attached" periodontal ligament fibers. *Osseointegrated* dental implants, by definition, are in direct contact with the alveolar bone without intervening soft tissues; there is no periodontal ligament and should movement occur, it would indicate a loss of osseointegration (e.g., fibrous encapsulation).[9] Excessive forces are destructive. Thus it is crucial to have a good understanding of the biomechanical properties and the limitations of dental implants to ensure proper treatment planning that will sustain the anticipated occlusal forces.

Load-Bearing Capacity

The load-bearing capacity of implants is influenced by several factors, including the number and size of implants, the arrangement and angulation of implant positions, and the quality of the bone-to-implant interface (Box 76-1). The *bone appositional index* (percentage of bone-to-implant contact) may be the most important factor to consider when evaluating the load-bearing capacity of any given implant(s). Less bone density and lower bone-to-implant contact provide less support and resistance to occlusal loading. As an example, consider the bone support of implants in the posterior maxilla; the bone quality is particularly poor as compared with the anterior mandible. The trabecular bone is less dense and the cortical bone layer is thin in the posterior maxilla. As a result, the bone appositional index in the posterior maxilla is significantly less than what can be achieved in the anterior mandible, where the trabecular bone is typically dense with a thick cortical bone layer. The bone appositional index for implants in the posterior maxilla typically ranges from 30% to 60%, whereas the bone appositional index for implants placed in the anterior mandible typically ranges from 65% to 90% (Figure 76-1).

Technologic advances in surface modification (as compared to the original machined Brånemark surface) may enhance bone apposition and support for implants placed in poor quality/quantity bone, thereby gaining anchorage and improving the biomechanics.[4,24,25,42] These "rougher" implant surfaces appear to have a significant effect on bone anchorage.[24,25,34] Surfaces with an altered microtopography achieve higher bone appositional indices than the machined surfaces and appear to facilitate the biologic processes of bone formation, resulting in greater deposition of bone onto the surface of the implant. In addition, bone deposited on the surface of acid-etched implants appears to be harder and denser and may be more resistant to resorptive remodeling.[40]

A lack of bone height limits the amount of available bone for placement of long implants and reduces the potential for bone-to-implant contact. In the posterior mandible the inferior alveolar nerve and vessels travel through the body of the mandible until exiting the mental foramen in the premolar region (see Chapter 53). The height of bone available for placement of implants in the posterior mandible is determined by the location of the neurovascular bundle and the resorption of the alveolar ridge. Lateral nerve repositioning is possible but has a moderately high morbidity rate and is not advised. In the posterior maxilla, pneumatization of the maxillary sinus combined with loss of alveolar bone results in a decreased total height of available bone in the posterior maxilla, which limits the length of implants that can be used. Sinus floor elevation and bone augmentation procedures have enabled clinicians to increase the height of bone available in the posterior maxilla, allowing for the placement of longer implants with improved success. Vertical ridge augmentation is challenging and less predictable for all sites.

Historically, the use of longer (>7 mm) implants has been advocated as the result of reported higher rates of failure for shorter (≤7 mm) implants.[17,20,29,39] Logic suggests that biomechanical stress around "short" implants leads to greater bone loss and a higher rate of implant failure. On the other hand, Pierrisnard[37] showed that the maximum implant stress increased somewhat with implant

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BOX 76-2 Finite Element Analysis

Finite element analysis (FEA) is a computerized investigative method that uses a mathematic model to assess stress in various objects and their surroundings when subjected to forces. It is useful in generating a hypothesis and testing basic biomechanical mechanisms but cannot be relied on for definitive answers. Only hard clinical evidence is undisputed and any assumption or predictions that are made by FEA needs to be validated clinically.

length and bicortical anchorage, encouraging use of short implants in selected cases. He also states that the low anchorage stiffness of short implant might reduce the mechanical stress to the implant because of the flexibility of the bone. Using the finite element analysis (FEA), Pierrisnard and colleagues[36] showed that greater implant length did not positively affect the way stresses were transferred to the implant but found that increasing implant diameter reduced the intensity of stress along the length of the implant. Iplikcioglu and Akca[19] using the same method observed that wider implants rather than longer implants registered lower stress value to the whole system, suggesting that the use of short, wide implants could increase the load-bearing capacity of implants and implant prosthesis. Baggi and colleagues also used FEA to show that increases in implant width reduced stress more than increases in length[5] (Box 76-2).

Angulation and Arrangement

The angulation and arrangement of implants used to retain and support a prosthesis influence the load-bearing capacity of the system (i.e., implants, components, prosthesis, and supporting bone).

In the 1980s, conventional dogma stated that once an implant became "osseointegrated," it did not matter whether occlusal loads were applied axially. However, as more clinical follow-up and animal research data became available, the adverse effect of nonaxial loads on implants became increasingly more apparent. The angulation of implants in relation to the plane of occlusion and the direction of the occlusal load is an important factor in optimizing the transfer of occlusal forces to implants and surrounding bone. Axial loads are tolerated well. Minor discrepancies in angulation are probably not clinically significant, but if loads are applied at an angle of 20 degrees or more to the long axis of the implant, load magnification can result, provoking a resorptive remodeling response of the adjacent bone.

Nonaxial loads lead to implant overload through load magnification at the bone-to-implant interface, which in turn precipitates a resorptive remodeling response of the bone around the neck of the implants. This concept is supported by numerous finite element analysis (FEA) studies, which clearly demonstrate that nonaxial forces significantly increase the stress concentration to the cortical bone around the neck of the implant (Figure 76-2).[3,11,33,44]

When excessive loads persist, bone loss continues and may progress to implant failure. Brunski et al[10] proposed that excessive occlusal loads lead to microdamage (fractures, cracks, delamination) of the bone adjacent to the implant, which provokes a resorptive remodeling response. The bone remodeling at the implant surface results in less bone density adjacent to the implant, especially around the coronal aspect of the implant. This bone has a poor capacity for repair that is the result in part to the absence of a periodontal ligament fiber attachment (i.e., no bone-forming tension exists). Thus a vicious cycle ensues in which continued excessive loading leads to more microdamage and progressive bone loss until the implant fails (Box 76-3).

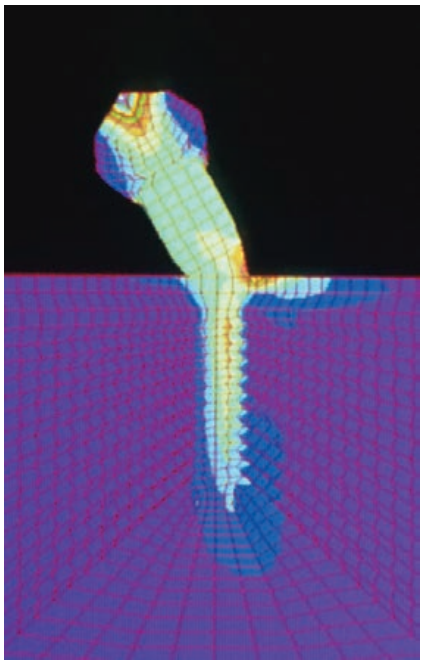

Figure 76-2 Nonaxial loads result in load magnification. Using finite element analysis, Cho and co-workers demonstrated that nonaxial loads concentrated stresses around the neck of the implant. *(Courtesy Dr. In Ho Cho.)*

BOX 76-3 Implant Overload and Bone Resorption: Proposed Mechanisms of Implant Failure

- Excessive occlusal loads
- Load resulting in microdamage: fractures, cracks, and delaminations
- Resorption-remodeling response of bone
- Loss of bone at the bone-to-implant interface as a result of remodeling
- Vicious cycle of continued loading, additional microdamage, and bone loss progressing to implant failure

When implants are arranged in a linear fashion, the biomechanics with respect to anticipated bone response are quite unfavorable compared with a configuration in which the implants are arranged in a nonlinear (curvilinear or staggered) fashion. Arranging implants in a nonlinear configuration creates a more stable base that is more resistant to the torquing forces created by off-center contacts and lateral loads (Figure 76-3).

Short-span, linear implant configurations in the posterior segments are particularly prone to bone loss when loads are not applied axially. Bone loss in posterior areas can be more damaging because implants in these areas are primarily supported by the cortical bone around the most coronal aspect of the implant. In the posterior maxilla, the cortical bone can be very thin, and unless the implant is positioned to engage the superior cortical bone (floor of maxillary sinus), the apical aspect of the implant is usually poorly supported by loose trabecular bone. The load-bearing capacity is poor, and nonaxial loads can be very destructive.

Therefore every attempt should be made to position posterior implants with the long axis directed toward the opposing stamp

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cusp and aligned perpendicular to the occlusal plane. Then, occlusal forces can be directed down the long axis of the implant, which is tolerated far better than nonaxial forces. The restoration is also simpler and more cost effective to fabricate, since angled or custom abutments are not required.

Before the widespread use of bone augmentation procedures in the posterior maxilla (i.e., sinus augmentation), many implants were positioned with excessive buccal angulations or were restored with buccal cantilevers. Similarly, distal cantilevered pontics were used to replaced missing maxillary posterior teeth. Implant-supported restorations with a cantilevered pontic transfer nonaxial occlusal forces to the adjacent implant and surrounding bone resulting in bone loss. Occlusal forces directed to the pontic created torquing forces around the implant adjacent to the cantilever. As a result, load magnification caused bone loss around the neck of the implant closest to the cantilever (Figure 76-4). Excessive distal angulations, which also created nonaxial loading and bone loss were also recognized problems. These implants often exhibit signs of overload (i.e., progressive and irreversible bone loss around neck of implant) when nonaxial occlusal loads are applied to the prosthesis (Figure 76-5).

TREATMENT PLANNING WITH DENTAL IMPLANTS

The success of implant-retained restorations is measured not only by successful osseointegration but also by the lack of problems associated them. Implant complications and failures have taught clinicians that the "engineering" of implant support for prosthetic appliances is an essential part of treatment planning and the key to success for implant-retained restorations (see Chapter 77 for a description of implant complications and failures). Treatment planning with dental implants in the edentulous or partially edentulous patient must carefully consider biomechanical issues to minimize failures and optimize success.

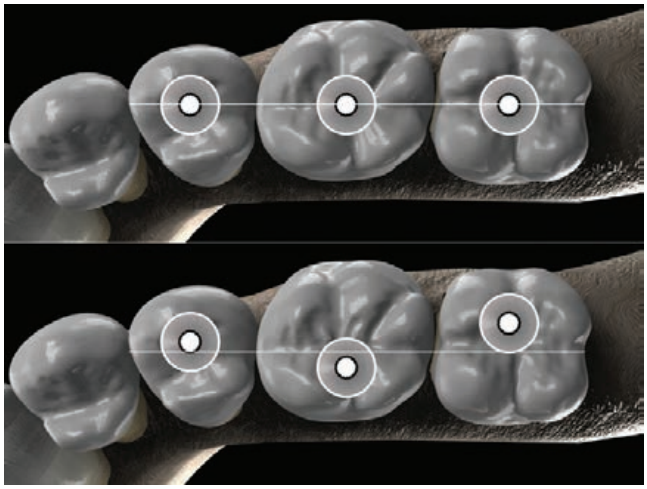

Figure 76-3 Placement of implants in a linear arrangement for a short-span, unilateral posterior restoration allows lateral forces to induce adverse bending of the implants leading eventually to bone loss and implant failure. If the implants are placed in a nonlinear "staggered" or "tripod" arrangement, lateral forces will be greatly diminished, and with a good occlusal scheme, will be counteracted by more favorable axial forces.

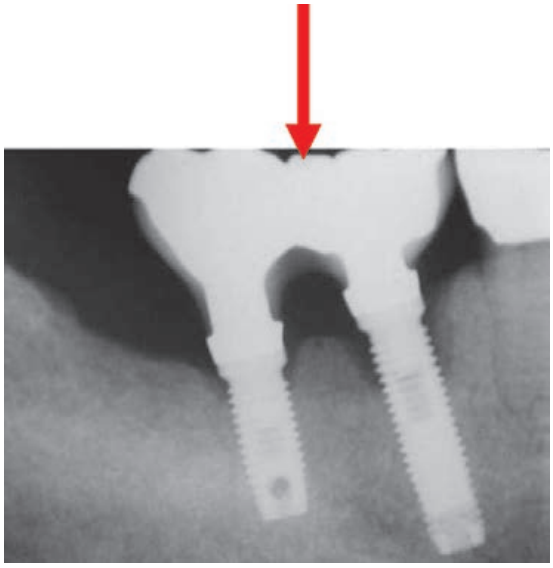

Figure 76-5 Because of the curve of Spee and the distal angulation of these implants (posterior mandible), the occlusal loads *(arrow)* are nonaxial. The nonaxial loading results in implant overload and bone loss around the neck of both implants.

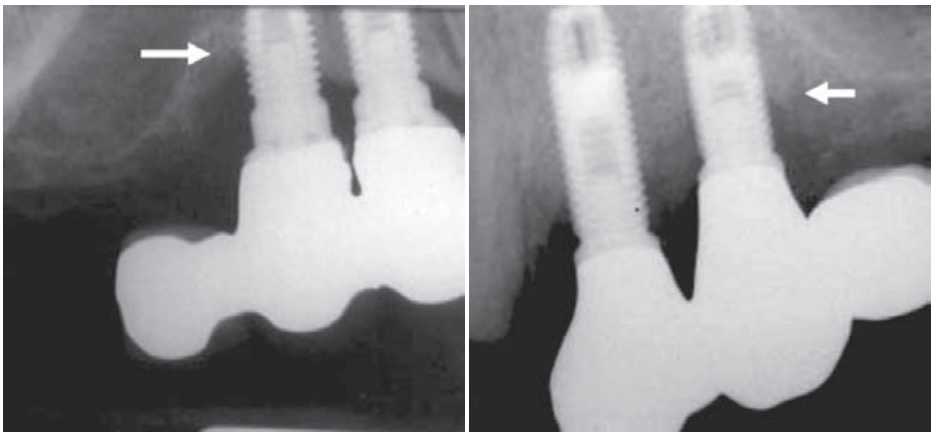

Figure 76-4 Cantilevers in posterior quadrants of partially edentulous patients should be avoided. These radiographs demonstrate two cases of bone loss *(arrows)* around the neck of short implants (closest to the cantilever) in which load magnification occurs.

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For more information about treatment planning implants in the edentulous maxilla and mandible, please visit the companion Expert CONSULT website at www.expertconsult.com.

Partially Edentulous Patients

Prosthetic options for patients who are partially edentulous (missing one to several teeth) include a conventional removable partial denture, a fixed partial denture (tooth-borne), or an implant-supported prosthesis.

Multiple Tooth Sites. Unilateral, implant-supported fixed partial dentures are predictable when they are supported by an adequate number of implants with good quality bone surrounding them. The lowest success rates for implants have consistently been reported for short-span (segmental) restorations in the posterior maxilla.[21] Anatomic limitations can prevent placement of implants of adequate length in the posterior sextants. In the posterior maxilla, the maxillary sinus limits the lengths used, and in the mandible, the inferior alveolar nerve limits the lengths of the implants used. The bone-implant interface for osseointegrated implants in the posterior maxilla is compromised because the bone quality is poor. Anchorage for an implant placed in this region may be improved by engaging the cortical bone of the floor of the sinus, but there is generally minimal bone-implant interface along the course of the implant. Because many patients possess insufficient bone in the posterior maxilla to receive an implant of suitable length, bone augmentation of the alveolar ridge or maxillary sinus has been suggested.[35,41] Vertical augmentation of the alveolar ridge has not been predictable, but the "sinus lift and graft procedure" appears predictable and has achieved wide acceptance[22,43] (see Chapter 73).

The number of implants used will influence the load-bearing capacity. In the 1980s and early 1990s, many posterior quadrants of the maxilla were restored with one or two implants, and in some patients, two implants were used to support restorations with three or four dental units. In many cases, soon after loading, a distinct pattern of bone loss was observed that led to loss of the implants (see Figure 77-16). Additional implants significantly improve the biomechanics of these implant-supported fixed partial dentures.

Currently, it is imperative to "engineer" the treatment of unilateral, posterior segments with an implant used to support each missing tooth that will be restored. If space and available bone permits, it is desirable to use a minimum of three implants to replace three missing posterior teeth in the maxilla. The same rule (one implant for every missing tooth being restored) applies to replacing teeth with implants in the posterior mandible as well. However, greater bone density (i.e., thickness of cortical bone) in the posterior mandible sometimes permits the use of fewer implants. A three-unit bridge supported by two implants in the posterior mandible is widely accepted (Figure 76-14). However, the decision to restore three missing teeth supported by two implants should always be made with consideration for the quantity and quality of bone available to support the implants (i.e., effective load-bearing capacity of the implants).

Single Tooth Sites. Implants have been used successfully to replace missing single teeth despite the fact that the loading limit of a single implant remains unknown. Single tooth implant restorations have replaced the traditional three-unit bridge and gained acceptance and patient satisfaction.

Standard-diameter implants have been used to restore single-tooth defects in the posterior quadrants of the maxilla and mandible with mixed results. Solitary implants restoring maxillary molars have had unpredictable results, whereas single implants used to restore mandibular molars have had a better prognosis. In the 1980s, clinicians attempted to restore mandibular first molar sites with conventional implants 3.75 or 4.0 mm in diameter. Unfortunately, the results were quite disappointing. Occlusal overload led to bone loss and in some cases, fractured implants, especially for the 3.75 mm diameter implant.[12]

The most common problem observed, particularly when external hex-headed implants were used, was loosening of the screw

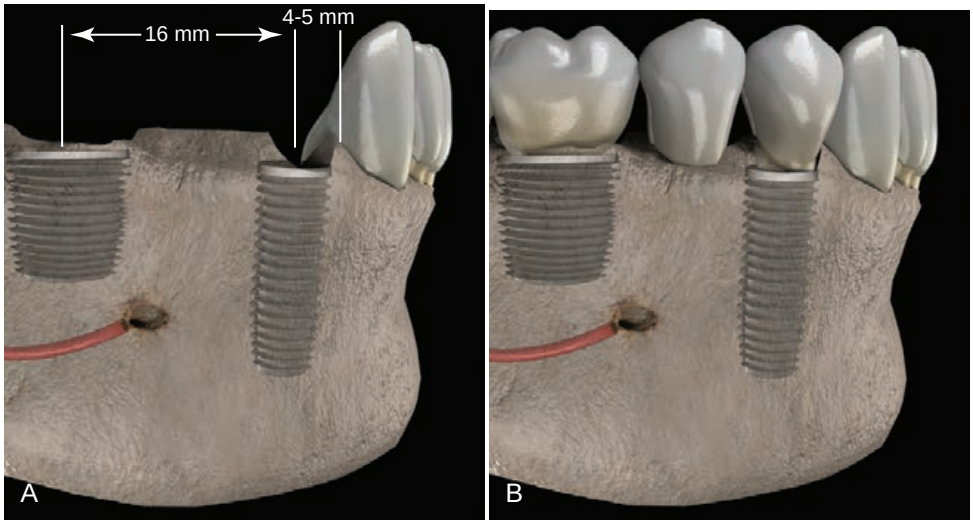

Figure 76-14 **A,** Placement of two implants in the mandible to replace three missing teeth. Note that the distance from the center of a standard implant (4-mm diameter) to the adjacent root is 4 to 5 mm, which accounts for the minimum of 1.5 distance between implant and root and the radius of the implant body. This distance is double if two implants are to be placed next to each other, making it 3 mm on each side if 3 implants were to be placed in a row. Following these recommendations, the necessary mesiodistal distance from the center of a wide body implant of 6 mm to the center of a standard implant of 4 mm that would replace 3 missing teeth is 15 to 16 mm. **B,** Restoration of the implants with an implant-supported three-unit fixed partial denture. Note that the spacing of the implants from center to center (15 to 16 mm) allowed a normal esthetically acceptable-size pontic.

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retaining the restoration. This problem results because the diameter of the head of the implant is much smaller than the size of the occlusal surface. The buccolingual width of the crown can be minimized by narrowing the dimension of the restoration, but there is less control over the mesiodistal dimension because contacts need to be established and the space is most often filled. Consequently, a mesiodistal cantilever, albeit small, is created. Tipping of the restoration during function eventually leads to stretching and loosening of the screw that secures the crown to the implant fixture. If hex-headed implants are used, the use of wide-diameter implants greatly diminishes the screw-loosening complication (Figure 76-15). The larger platform reduces the potential for tipping forces to stretch or break the screw. Internal connection implants are far less susceptible to screw loosening.

If the first molar is lost and suitable abutments are present, a conventional three-unit, fixed partial denture is still a viable option, especially if the adjacent teeth already have large restorations. However, if adjacent teeth have small or no restorations, it is more conservative and preferable to replace the missing single tooth with an implant. This restoration is cost effective and predictable. In distal extension areas when restoring one molar tooth, two conventionally sized implants may be placed (with appropriate spacing) to improve biomechanical support (see Figure 69-9).

Single implants can be used with a high rate of success in the anterior and premolar areas. The bone-implant interface achieved in this area is good, and the size of the occlusal surface is generally small. Although similar considerations exist regarding screw loosening for single-implant restorations in the anterior and premolar regions, these concerns are less significant because of a more anterior position (lesser forces). Typically, there is better bone quantity and quality compared with more posterior sites.

STRATEGIES TO AVOID IMPLANT OVERLOAD

Multiunit implant restorations should be splinted to maximize implant support (sharing the loads), and emergence profiles should be developed with open embrasure spaces to facilitate oral hygiene (Figure 76-16).

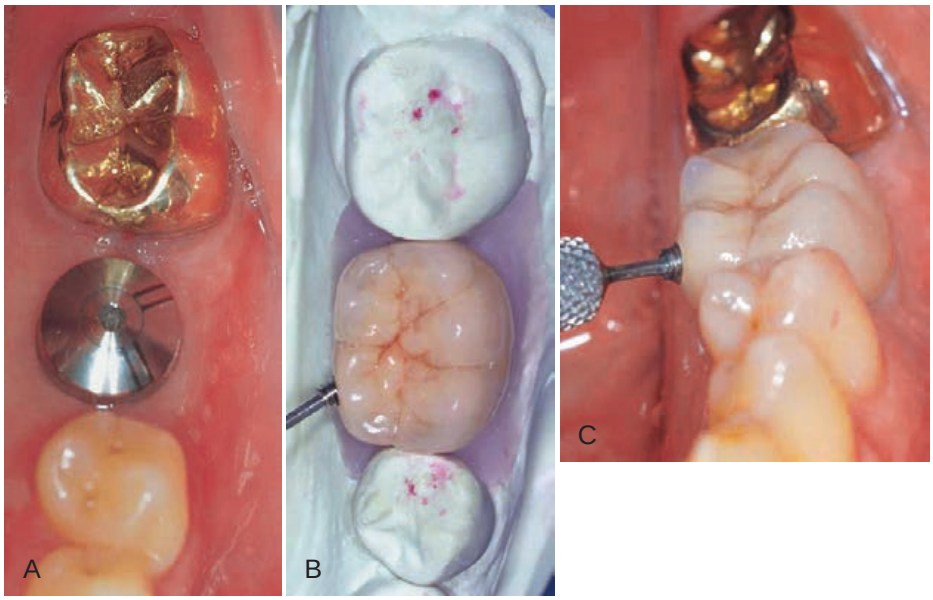

Figure 76-15 Single-tooth restoration in the posterior mandible supported by a wide-diameter implant. **A,** Clinical photograph of healing abutment on wide-diameter implant. **B,** Photograph of laboratory model with single molar. **C,** Clinical photograph of molar crown supported by wide-diameter implant. The use of wide-diameter (external hex) implants eliminates the problem of screw loosening for single-tooth, posterior, implant-supported crowns. NOTE: This crown is secured to the abutment via a lingual set screw (small hex driver seen attaching set screw).

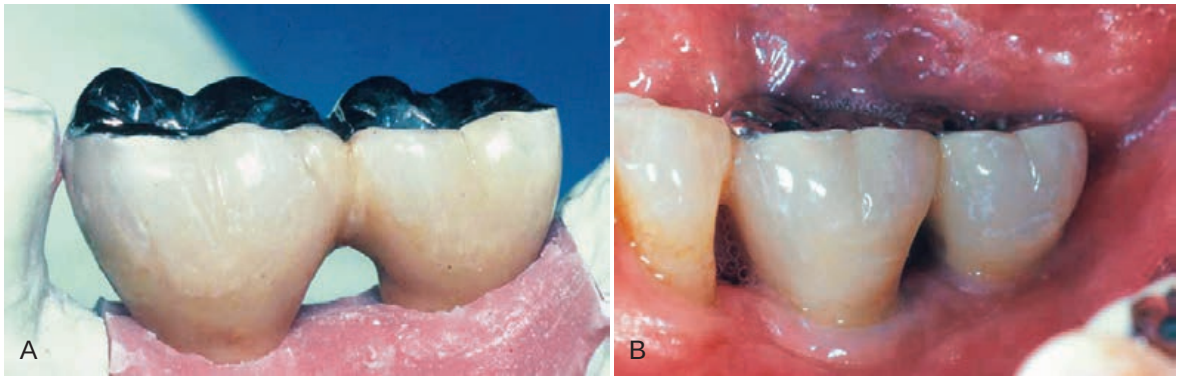

Figure 76-16 Guidelines for restoring posterior quadrants. Embrasures should be large enough for access with a proxy brush. The occlusal table must be narrow, cusp angles flattened, and proper emergence profiles developed. Anterior guidance should be restored with the natural dentition whenever possible. **A,** Laboratory model and, **B,** clinical photograph of two-unit, fixed partial denture supported by two implants in the posterior mandible demonstrate these qualities.

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It is important to "engineer" implant-retained restorations well. In fact, some advocate "over engineering" to avoid failures. Implants should be adequate number, size and position to sustain the anticipated occlusal loads. Implants with smaller diameters have a lesser capacity to support bending forces than do implants with larger diameters. Implants with a minimum of 4 mm diameter should be used in areas expected to sustain greater occlusal loads (i.e., the posterior regions). Wider-diameter implants can be used when adequate bone and space exists (Figure 76-17). Thus, when the residual ridge and site permits, wide diameter implants should be used for molar replacement. Standard diameter (4 mm) implants may be best for premolar replacement since it approximates the premolar size and will provide a better emergence profile. In the case of multiple implant restorations, when ridge width permits, offsetting the implant location is encouraged.

Occlusal Design

Occlusal design for implant-supported prostheses is an essential and integral determinant of overall treatment planning.[28,38] The risk of implant overload can be minimized by limiting the width of the occlusal table of the implant-supported fixed partial denture, flattening the cusp angles, avoiding the use of cantilevered restorations, and restoring the anterior guidance provided by the anterior dentition. Box 76-4 lists strategies to avoid implant overload.

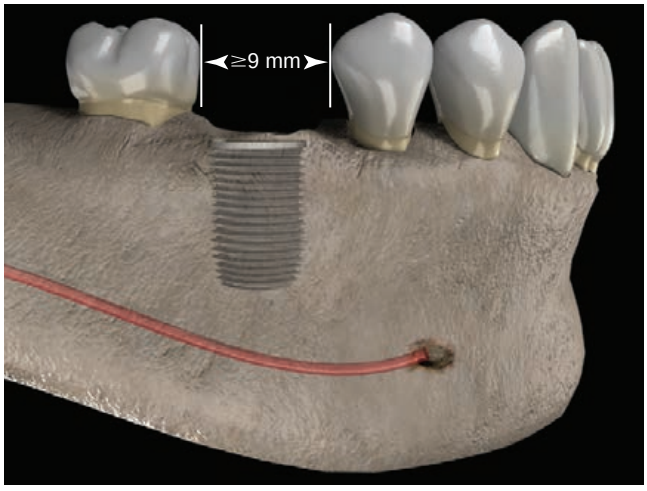

Figure 76-17 Placement of a wide platform implant 6 mm in diameter to replace a first mandibular molar. A minimum distance of 1.5 mm between the walls of the implant and the roots of the adjacent teeth on each side is to be considered, making 9 mm the minimum mesiodistal distance necessary.

BOX 76-4 Strategies to Avoid Implant Overload

- Place implants perpendicular to the occlusal plane
- Place implants in tooth positions
- Use an implant for each unit being replaced
- Avoid the use of cantilevers in linear configurations
- Avoid connecting implants to teeth
- If connecting implants to teeth, use a rigid attachment
- Control occlusal factors such as cusp angles and width of occlusal table
- Restore anterior guidance if possible

The intensity and the vector of the force depend on several variables including the amount of bone contact (osseointegration), the quality and the quantity of the supporting bone and the implant surface characteristics. Regardless of the direction or the amount of this force, it always leads to micromotion at the bone-implant interface, which is translated into a transfer of stress to the adjacent bone. Splinting implant crowns together and placing longer and wider implants, as well as enhanced implant surface features, increase the bone-to-implant contact, or *bone appositional index*, and reduce stress forces, thereby increasing the load-bearing capacity of the implant(s).

Cantilevers

The use of cantilevered implant-supported restorations (i.e., unilateral, linear configurations) in posterior quadrants of the mandible or maxilla is strongly discouraged and should be avoided. Cantilever extensions result in load magnification that can cause overloading of the implant next to the cantilever extension, which in turn may lead to progressive bone loss and implant failure (see Figure 76-4).

Connecting Implants to Teeth

In general, it is advisable to keep implant-supported restorations separate from natural teeth because implants and teeth function differently and connecting them can lead to complications such as screw loosening and intrusion of natural dentition. Teeth have the capacity to move under functional occlusal loads while implants do not. Connecting implants to teeth may create a cantilever effect on the implant.[8,27,30,31] Furthermore, designing the restoration so that it is independent of the natural dentition simplifies the biomechanics.

In contrast to this widely accepted view, a recent critical review of the literature suggests that these concerns may have been overstated.[13] There are situations that may warrant connection of implants to teeth. Several authors have shown (animal and human research) that success is possible when specific guidelines are followed.* Specifically, if implants are to be connected to the natural dentition, it should be done in a rigid manner, either with screw-retained attachments or with copings secured by permanent cement. Tooth preparation should allow good retention, teeth should be periodontally healthy and stable, and the occlusal scheme should be good. Gulbransen[15] showed that if implants are connected to the natural dentition with a rigid system of attachment, the implant failure and complication rates were dramatically reduced. It appears that the intrusion problem (and other complications) is increased when implants and natural teeth are connected with "nonrigid" and "semirigid" attachments. In addition, the well-documented phenomenon of intrusion of the natural tooth abutment associated with the use of semiprecision attachments is prevented.[14]

CONCLUSION

The importance of biomechanics and the limitations of implant systems were initially underestimated. It was believed that multiunit fixed bridges could be supported by as few as two implants, as it was done with the natural dentition. Over the years, clinical experience and research underscored the importance of biomechanics in the success and predictability of implant-retained prostheses. The rigid nature of implant-retained restorations and the

*References 1, 6, 7, 14, 16, and 32.

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SCIENCE TRANSFER

Excessive loading of implants can cause failure by loss of bone integration, fracture and loosening of retaining screws, and even fracture of the implant body. Many implant patients should wear protective occlusal splints to minimize nonfunctional forces. Implants should be as long as possible, and wider implants distribute loads better than narrow implants. Nonaxial forces should be avoided, particularly in posterior implants in which angulations greater than 20 degrees are often destructive.

The occlusal pattern on implant-supported crowns in partially edentulous cases should assure that the immobile implant is not prematurely loaded because natural teeth will be compressible during function. Centric contacts should be lighter on implants than on the adjacent teeth. Working and balancing contacts on implant crowns should be avoided and occlusal surfaces should be narrower than natural teeth to protect against forces transferred by the food bolus.

Sufficient implants should be in place. Most maxillary posterior cases should have one implant per missing tooth. In mandibular posterior cases with long implants in dense bone, two implants can be used to replace three teeth. Cantilevers should be reserved for use in anterior cases where they can assist in providing better soft tissue contours than two adjacent implants.

Fully edentulous maxillas are usually treated with four to six implants together with a removable prosthesis. This will provide lip support for good esthetics and prevent aberrations of speech and stop uncontrolled saliva leakage around crowns. Fully edentulous mandibular cases can be treated with two implants supporting a bar and clip-retained overdenture or four to five implants with a fixed bone-anchored bridge with posterior cantilevers not to exceed 20 mm.

lack of forgiveness in these systems demands a revised approach to treatment planning that is now applied. The biomechanics must be factored into the planning at the beginning of any implant treatment to achieve long-term, predictable success.

When clinicians follow a strategy that avoids implant overload and places an adequate number of implants in optimal positions, abutment selection is simplified, proper emergence profiles can be developed, space is available in interproximal areas for hygienic access and tissue health, the clinician has better control over the occlusal anatomy (narrowed occlusal table and flat cusp angles), and occlusal loads are better controlled.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 77

Implant-Related Complications and Failures

Stuart J. Froum, Perry R. Klokkevold, Sang Choon Cho, and Scott H. Froum

CHAPTER OUTLINE

DEFINITION OF IMPLANT SURVIVAL AND IMPLANT SUCCESS
TYPES AND PREVALENCE OF IMPLANT COMPLICATIONS
DIFFERENT TYPES OF DENTAL IMPLANTS
SURGICAL COMPLICATIONS
- Hemorrhage and Hematoma
- Neurosensory Disturbances
- Implant Malposition

BIOLOGIC COMPLICATIONS
- Inflammation and Proliferation
- Dehiscence and Recession
- Periimplantitis and Bone Loss
- Implant Loss or Failure

COMPLICATIONS RELATED TO AUGMENTATION PROCEDURES
- Autogenous Bone Harvesting/Grafting
- Guided Bone Regeneration
- Sinus Bone Augmentation (online only)

COMPLICATIONS RELATED TO PLACEMENT AND LOADING PROTOCOLS
- Immediate Implant Placement (online only)
- Immediate Loading After Implant Placement (online only)
- Implant Placement Using a Flapless Approach (online only)

PROSTHETIC OR MECHANICAL COMPLICATIONS
- Screw Loosening and Fracture
- Implant Fracture
- Fracture of Restorative Materials

ESTHETIC AND PHONETIC COMPLICATIONS
- Esthetic Complications
- Phonetic Problems

CONCLUSION

Refer to Expert CONSULT companion website at www.expertconsult.com for additional content.
Some figures may be out of numeric order in this printed chapter.

Patients have experienced much success with endosseous dental implants.[2,85] Yet, despite the long-term predictability and success of implants, complications do occur in a percentage of cases.[3,25] Some complications are relatively minor and easy to correct, but others are more significant, resulting in loss of implants, failure of prostheses, and occasionally in severe loss of tissues in the area of implant failure. Complications may be surgical, biologic, mechanical, or esthetic.

Biologic complications are those that involve the periimplant supporting hard and soft tissues. Changes in the periimplant soft tissues may be minor, such as inflammation and proliferation, or more significant, involving progressive bone loss. The ultimate biologic complication is implant loss or failure, which may also result in soft and hard tissue defects. Biologic loss of implants may be caused by a lack of osseointegration in the early stages before restoration or by a loss of osseointegration as a result of bone loss after the restoration is installed and functioning.

Surgical complications are those problems or adverse outcomes that result from surgery, including procedures used for implant placement, implant exposure, and augmentation procedures. Complications arising from implant malposition are those resulting from an implant being placed in a nonideal position. Malpositioned implants , which usually are the result of poor presurgical treatment planning and/or surgical technique, can lead to an array of implant problems ranging in severity from minor to major. These complications include compromised esthetic and/or prosthetic results, soft tissue and bone dehiscences, impingement on anatomic structures, and possibly implant failure. Unfortunately, the extent of the problems that arise from implant malposition are often not recognized until the time of implant restoration.

Prosthetic or mechanical complications and failures typically occur in the form of material failure such as abutment and prosthetic screw loosening or fractures. The patient can recover from many of these mechanical problems if they are minor and

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recognized early. However, some complications, such as implant fractures, are not salvageable.

Esthetic complications arise when patient expectations are not met. Patient satisfaction with the esthetic outcome of the implant prosthesis will vary from patient to patient, depending on several factors. The risk for esthetic complications is increased for patients with high esthetic expectations and less-than-optimal patient-related factors (e.g., high smile line, thin gingival tissues, or inadequate bone quantity and quality).

This chapter reviews several of the more common implant-related complications. A summary of findings from the literature is presented to offer some insight into the prevalence of various types of implant-related complications. Implant failure, surgical complications related to site development, and different implant placement protocols are discussed as well.

DEFINITION OF IMPLANT SURVIVAL AND IMPLANT SUCCESS

Implant success (or failure) is reported in many ways. Case reports, case series, retrospective studies, controlled studies, and prospective studies all report levels of success and failure as related to the population being evaluated. Each type of report or study has recognized limitations. However, recognizing the tremendous variation in the way individual investigators measure and interpret success may be more important. At times, outcomes are measured simply by the presence or absence of the implant(s) at the time of the last examination, which is only a measure of implant survival and should not be confused with implant success. In contrast to this simplified assessment, some investigators use detailed criteria to measure implant success and failure, with variations of successful outcomes separated and defined by additional criteria.

Implant survival is simply defined as any implant that remains in place at the time of evaluation, regardless of any untoward signs, symptoms, or history of problems. Clearly, there is a difference between implants that are present and functioning under an implant-retained restoration and implants that are present but not connected to any restoration and not providing support or function. These implants are sometimes referred to as "sleepers" and should not be considered successful merely because they are present and remain osseointegrated. Rather, sleeper implants should be included in the discussion as "surviving" but counted as "failures" because they failed to fulfill the originally intended treatment.

Implant success, on the other hand, is defined not only by the presence of the implant but also by criteria evaluating the condition and function of the implant at the time of examination. Criteria for implant success and failure have been defined over the years, but not all investigators use them. In the classic definition, Albrektsson et al[6] defined implant success as an implant with no pain, no mobility, no radiolucent periimplant areas, and no more than 0.2 mm of bone loss annually following the first year of loading. Roos-Jansaker et al[115] added to this definition by defining a successful implant as one that lost no more than 1 mm of bone during the first year in function. Sometimes these criteria are used as proposed, whereas at other times, they are used by investigators with modifications and new criteria. Consequently, it is inherently difficult to make comparisons between studies and often impossible to make absolute conclusions about any aspect of implant success or failure based on one or a few studies.

If one considers "success" using a strict definition as the outcome without any adverse effects or problems, then "implant success" should be defined as any implant-retained restoration in which (1) the original treatment plan is performed as intended without complications, (2) all implants that were placed remain stable and functioning without problems, (3) periimplant hard and soft tissues are healthy, and (4) both the patient and the treating clinician(s) are pleased with the results. When these strict criteria are used, implant success (i.e., absence of complications) is projected to be only about 61% after 5 years for implant-supported fixed partial dentures (FPDs) and 50% after 10 years for combined tooth/implant FPDs.[83]

An additional criteria of implant success that is often unreported but that must be considered is the "esthetic" success or patient satisfaction with the outcome. There have been a number proposed methods to evaluate esthetic results. A restorative index was proposed by Jensen et al[72] to appraise the esthetics of the final restoration. The index uses a scale from 1 to 10 with 1 being an extremely poor result and 10 being a superlative esthetic result. The index, based on both subjective and objective criteria, evaluates the size and shape of the implant restoration in comparison to the equivalent contralateral tooth, blending into the arch, as well as the presence of the papillae, gingival form, color, and other factors considered essential in determining an esthetic result. The pink esthetic score (PES) is an index proposed by Furhauser et al[55] that considers seven soft tissue parameters, including an evaluation of the color, contour, and texture of the surrounding soft tissues (papilla and facial mucosa). Each parameter is given a score of 0, 1, or 2, which allows the best score of 14 to determine the highest level of esthetics. Other indices were proposed for single-tooth implant restorations in the esthetic zone.[19,89] The most recent, proposed by Belser et al,[19] combines a modified PES index with a white esthetic score (WES) focusing on the visible part of the implant restoration scoring five parameters from general tooth form to hue, value, surface texture, and translucency. The maximum possible WES score is 10. These indices are aimed at quantifying the esthetic result, which could then present an objective method of judging "implant esthetic success."

TYPES AND PREVALENCE OF IMPLANT COMPLICATIONS

The prevalence of implant-related complications has been reported in several reviews. In a systematic review of reports on the survival and complication rates of implant-supported FPDs, Pjetursson et al[83] found that the most common technical complication was fracture of veneers (13.2% after 5 years), followed by loss of the screw access hole restoration (8.2% after 5 years), abutment/occlusal screw loosening (5.8% after 5 years), and abutment/occlusal screw fracture (1.5% after 5 years; 2.5% after 10 years). Fracture of implants occurred infrequently (0.4% after 5 years; 1.8% after 10 years). A retrospective evaluation of 4937 implants by Eckert et al[44] found that implant fractures occur more frequently in partially edentulous restorations (1.5%) than in restorations of completely edentulous arches (0.2%), and all observed implant fractures occurred with commercially pure 3.75-mm diameter threaded implants.

In a literature review that included all types of implant-retained prostheses, Goodacre et al[59] found that the most common technical complications were loosening of the overdenture retentive mechanism (33%), resin veneer fracture with FPDs (22%), overdentures needing to be relined (19%), and overdenture clip/attachment fracture (16%). Their review, with the inclusion of edentulous patients having overdentures, seemed to indicate a significantly higher percentage of complications than Pjetursson's systematic review[107] of patients with implant-supported FPDs. Goodacre et al[58] found it impossible to calculate an overall prosthesis complication rate

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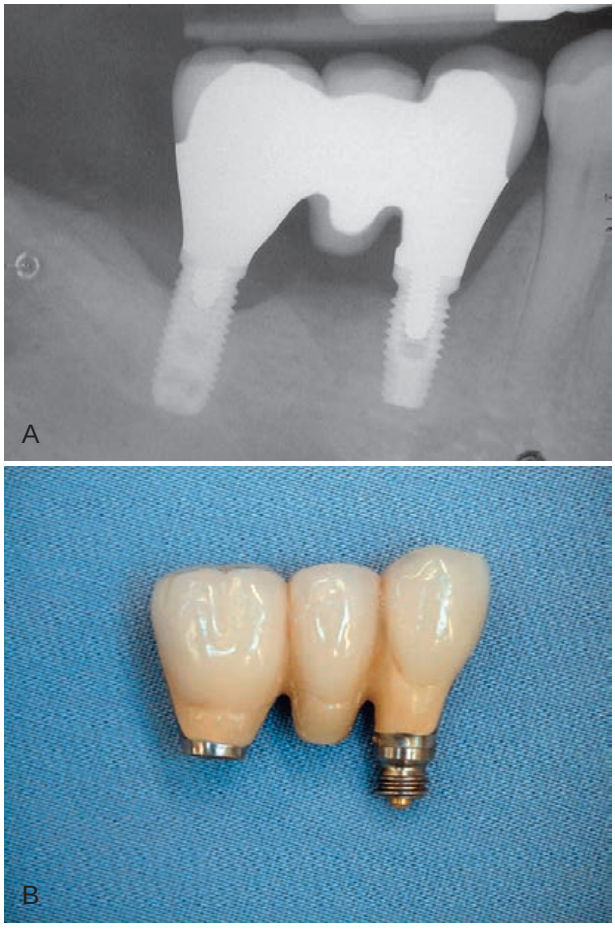

Figure 77-1 **A,** Radiograph of a three-unit, posterior, fixed partial denture (FPD) supported by two standard-diameter, screw-shaped threaded implants. Notice long crown height, relatively short implant length, and bone loss around posterior implant. **B,** Photograph of the ultimate implant-supported restoration failure. The anterior implant fractured between the second and third threads, which resulted in loss of the restoration.

because most studies included in their review did not report on several of the complication categories.

The most common complication reported for single crowns was abutment or prosthesis screw loosening. Abutment screw loosening varied dramatically from one study to another, ranging from 2% to 45%.[59] The highest rate of abutment screw loosening was associated with single crowns, followed by overdentures. The rate of prosthesis screw loosening was similar, ranging from 1% to 38% in various studies. A higher frequency was reported for single crowns in the posterior areas (premolar and molar) than in the anterior region.

Implant fracture is an uncommon but significant complication. Goodacre et al[58]reported a 1.5% incidence in their literature review. The incidence of implant fracture was higher in FPDs supported by only two implants. Consistent with this finding, Rangert et al[111] reported that most fractured implants occurred in single- and double-implant–supported restorations. They also indicated that most of these fractures were in posterior partially edentulous segments, in which the generated occlusal forces can be greater, as opposed to anterior segments (Figure 77-1).

In a systematic review of prospective longitudinal studies (minimum of 5 years) reporting both biologic and technical complications associated with implant therapy (all restoration types included), Berglundh et al[20] found that the incidence of technical complications was consistent with Pjetursson's findings, with implant fracture occurring in less than 1% (0.08% to 0.74%) of cases. Interestingly, consistent with the findings in Goodacre's review, technical complications were higher for implants used in overdenture therapy than implants supporting fixed prostheses.

In Pjetursson's systematic review[83] of survival and complication rates for implant-supported FPDs, biologic complications, such as periimplantitis and soft tissue lesions, occurred in 8.6% of implant-supported FPDs after 5 years. However, in a more recent literature review of the prevalence of periimplant diseases, Zitzmann and Berglundh[144] reported that although cross-sectional studies are rare, data from the only 2 studies available showed that periimplant mucositis occurred in 80% of the subjects and 50% of implant sites. Periimplantitis on the other hand was identified in 28% and ≥56% of subjects and in 12% and 43% of implant sites in the two studies that followed subjects with implants in function at least 5 years. A critical review of the literature by Esposito et al[51] included 73 publications reporting early and late failures of Brånemark implants; biologically-related implant failures were relatively low at 7.7%. The treatments involved all anatomic areas and all types of prosthetic design. The authors concluded that the predictability of implant treatment was especially good for partially edentulous patients compared with totally edentulous patients, with failures in the latter population being twice as high. Also, the incidence of implant failure was three times higher for the edentulous maxilla than for the edentulous mandible, whereas failure rates for the partially edentulous maxilla were similar to those for the partially edentulous mandible.

Risk factors, such as smoking, diabetes, and periodontal disease, may contribute to implant failure and complications. Several studies with numerous implants and years of follow-up have concluded that smoking is a definite risk factor for implant survival.[11,41,42,46,97] A systematic review of the effect of risk factors on implant outcomes concluded that smoking has an adverse affect on implant survival and success, with the effects being more pronounced in areas of loose trabecular bone (i.e., posterior maxilla).[81] The review suggested that type 2 diabetes may have an adverse effect on implant survival rates but did not have enough studies to permit a definitive conclusion.[81] Finally, the same review also concluded that while patients with a history of treated periodontitis did not show any decrease in implant survival, they did experience more implant complications and lower success rates.[81]

DIFFERENT TYPES OF DENTAL IMPLANTS

When analyzing the data concerning prevalence of implant complications (i.e., failure, fracture, periimplantitis, etc) in the published systematic reviews, the reader should be cognizant of the fact that many of the studies reviewed for these complications reported on implants with previous designs (i.e., machined surfaces and external connections). Currently, nearly all implant systems feature surfaces with an altered microtopography (rough) and many have internal connections. As a result, the prevalence and type of complications associated with these newer implant designs may be different.

Many modifications have been developed to try to improve the long-term success rates of implants. More than 1300 types of dental implants are now available, in different materials, shapes, sizes, lengths, and with different surface characteristics or coatings. There is limited evidence to show that implants with relatively smooth surfaces (i.e., machined) are less prone to bone loss from chronic infection (periimplantitis) than implants with rougher surfaces. On the other hand, implants with a turned (smoother) surface might be at greater risk to fail early than implants with roughened surfaces since the bone-to-implant contact is

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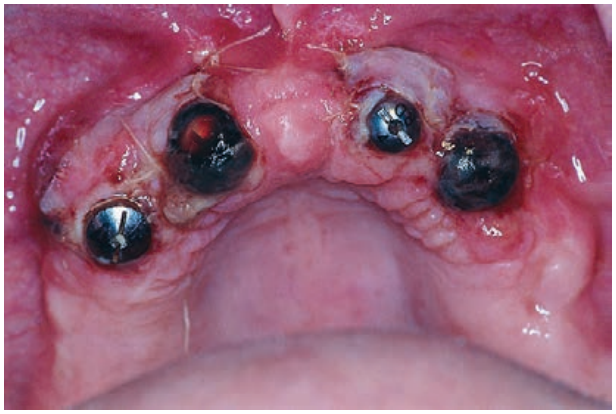

Figure 77-2 Clinical photograph of postoperative bleeding around healing abutments after second-stage implant exposure surgery.

Figure 77-3 Clinical photograph of postoperative (extraoral) bruising indicative of subdermal bleeding into connective tissues spaces. This is a normal expectation that resolves within 7 to 14 days.

comparatively less. To date, there is no evidence showing that any particular type of dental implant has superior long-term success.[48]

Different implant designs and surfaces must be studied in prospective human trials over long periods of time. At present, these data are not available, and the clinician should be aware that the data we currently have, even on rates of implant failure, may not reflect the outcome for implants being used today. Moreover, new implant surfaces and designs are continually being introduced with even less data available on potential complications.*

SURGICAL COMPLICATIONS

As with any surgical procedure, there are risks involved with implant surgery. Proper precautions must be taken to prevent the risk of injury resulting from surgical procedures, including but not limited to (1) a thorough review of the patient's past medical history, (2) a comprehensive clinical and radiographic examination, and (3) good surgical techniques. Surgical complications include perilous bleeding, damage to adjacent teeth, injury to nerves, and iatrogenic jaw fracture. Postoperative complications include bleeding, hematoma, and infection. They may be minor, transient, and easily managed or more serious and require postoperative treatment.

Hemorrhage and Hematoma

Bleeding during surgery is expected and usually easily controlled. However, if a sizable vessel is incised or otherwise injured during surgery, the hemorrhage can be difficult to control. Smaller vessels will naturally constrict or retract to slow the hemorrhage. If bleeding continues, it may be necessary to apply pressure or to suture the hemorrhaging vessel. This can be especially difficult if there is a vascular injury to an artery that is inaccessible such as in the floor of the mouth or posterior maxilla. Serious bleeding from an inaccessible vessel can be life threatening, not by exsanguination but rather as a result of airway obstruction. This is most problematic when the point of bleeding is inaccessible and internal (within the connective tissues and soft tissue spaces).

Postoperative bleeding is an equally important problem to manage (Figure 77-2). Patients should be given postoperative instructions on normal expectations for bleeding and how to prevent and manage minor bleeding. Historically, standard practice has recommended that they should be advised, with their physician's approval, to discontinue or reduce medications that increase bleeding tendency 7 to 10 days before surgery. However, recent evidence suggests that this may not be necessary and may increase the risk of hematologic or cardiovascular problems[73,74,97,116] (see Chapter 37). Dental health care providers should consult with medical health care providers regarding the best management for each individual patient. Furthermore, dental health care providers and patients should always include the treating medical practitioner in the management decisions if postoperative bleeding is excessive or persistent. Submucosal or subdermal hemorrhage into the connective tissues and soft tissue spaces can result in hematoma formation. Postoperative bruising is a typical example of minor submucosal or subdermal bleeding into the connective tissues (Figure 77-3). Bruising and small hematomas typically resolve without special treatment or consequence. However, larger hematomas or those that occur in medically compromised individuals are susceptible to infection as a result of the noncirculating blood that sits in the space. Thus it is prudent to prescribe antibiotics for patients who develop a noticeably large hematoma.

Although the incidence of a life-threatening hemorrhage from implant surgery is extremely low, the seriousness of the problem warrants the attention of everyone who participates in this type of surgery. Potentially fatal complications have been reported for implant surgical procedures in the mandible (especially the anterior region).* Massive internal bleeding in the highly vascular region of the floor of the mouth can result from instrumentation or implants that perforate the lingual cortical plate and sever or injure the arteries running along the lingual surface. Depending on the severity and location of the injury, bleeding may be apparent immediately or only after some delay. In either case, the progressively increasing hematoma dissects and expands to displace the tongue and soft tissues of the floor of the mouth, ultimately leading to upper airway obstruction. Emergency treatment includes airway management (primary importance) and surgical intervention to isolate and stop the bleeding. Clinicians must be aware of this risk and must be prepared to act quickly. It is important to recognize that bleeding, although considered a complication at the time of surgery, may be a serious complication in the hours and days after surgery.[38]

Neurosensory Disturbances

One of the more problematic surgical complications is an injury to nerves. Neurosensory alterations caused by damage to a nerve may

*References 7, 9, 33, 43, 103, 104, 128, 132, and 145.

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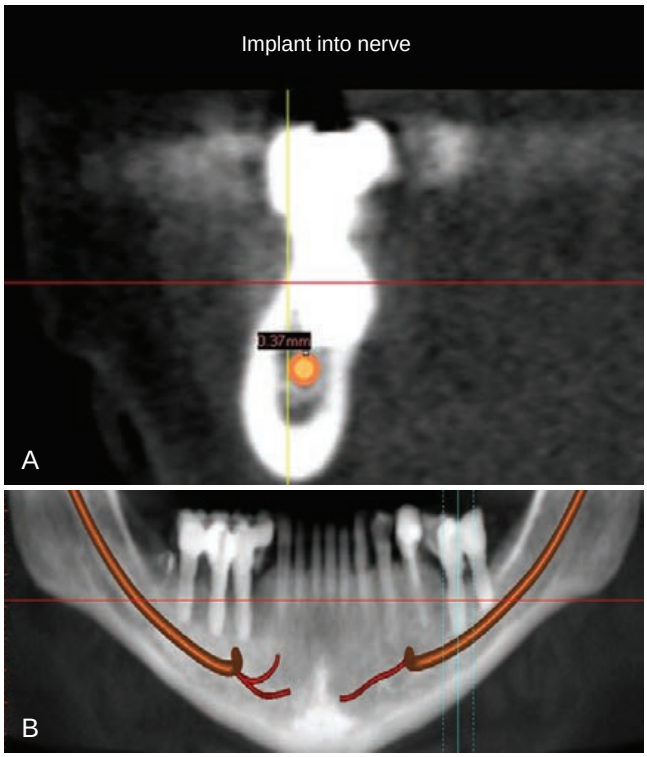

Figure 77-4 **A,** Cross-sectional image from computed tomography (CT) scan showing implant impinging on inferior alveolar nerve canal. **B,** Panoramic image of CT scan showing implant in lower left first molar area impinging on inferior alveolar nerve canal. Nerve is marked by tracing with software.

be temporary or permanent. Neuropathy can be caused by a drilling injury (cut, tear, or puncture of the nerve) or by implant compression or damage to the nerve (Figure 77-4; also see Figure 70-16). In either case, the injury produces neuroma formation, and two patterns of clinical neuropathy may follow. *Hypoesthesia* is a neuropathy defined by impaired sensory function that is sometimes associated with phantom pain. *Hyperesthesia* is a neuropathy defined by the presence of pain phenomena with minimal or no sensory impairment.[61] Some neuropathies will resolve, whereas other will persist. The type of neuropathy is not indicative of the potential for recovery.

It is likely that neurosensory disturbances occur more frequently after implant surgery than currently reported in the literature for several reasons. First, many of these changes are transient in nature, and most patients recover completely or at least recover to a level that is below a threshold of annoyance or daily perception. Second, wide variation exists in the postoperative evaluation of patients by clinicians. Some clinicians do not examine or inquire about postsurgical neurosensory disturbances at all, thus allowing this complication to go unnoticed. Likewise, some patients may think that the altered sensation is part of the expected "side effect" of surgery and may never acknowledge or comment on its presence, especially if the disturbance is minor. Therefore it is likely that minor neuropathies exist but go unrecognized and unreported.

Neurosensory disturbances reported in the literature are most prevalent and significant when they are more serious and occur more frequently, such as those associated with lateral transposition of the mandibular nerve.[71,78] This relatively uncommon procedure is used to reposition the nerve and allow longer implants to be placed in the atrophic posterior mandible. Lateral nerve transposition procedures are associated with almost 100% incidence of neurosensory dysfunction immediately after surgery. More than 50% of these neurosensory changes are permanent (ranging from 30% to 80%).[78]

Figure 77-5 Clinical photograph of gingival recession around a maxillary anterior implant (left central incisor) resulting in exposure of the crown margin, the implant collar, and several threads of the implant.

Implant Malposition

Many of the aforementioned complications that arise during implant surgery can be attributed to the dental implant being placed in an undesired or unintended position. Malpositioning of dental implants is usually the result of poor treatment planning before the implant surgery, lack of surgical skill by the implant surgeon, and/or poor communication between implant surgeon and restorative dentist. Optimal implant esthetics and the avoidance of positional complications can be achieved by placing the implant in a prosthetically driven manner.[90,102] In other words, the implant should be placed with reference to the three dimensions dictated by the position of the final restoration and not by the availability of bone. The angulation is another important determinant (fourth dimension) of implant position that will affect outcome esthetics. The ideal implant position entails an accurate preparation, insertion, and placement of the implant into the alveolus in a proper three-dimensional geometry according to apicocoronal, mesiodistal, and buccolingual parameters, as well as implant angulation relative to the final prosthetic restoration and gingival margins.[82,117] (see Chapter 71).

Apicocoronally, the implant should be placed so the dental implant platform is 2 to 3 mm apical to the gingival margin of the anticipated restoration.[24] The actual implant position varies slightly from one implant system to another, depending on abutment design and space requirements. If the implant platform is placed too coronal, there will not be sufficient room to develop a natural-looking emergence profile and the tooth may have a squarish, unesthetic contour. If the platform is placed at or above the level of the gingival margin, a metal collar or implant exposure can occur yielding an unesthetic result (Figure 77-5). Conversely, if the implant platform is placed too apically, a long transmucosal abutment will be necessary to restore the implant. This can lead to a deep pocket and difficult hygiene access for the patient and clinician.

Mesiodistal implants should be placed at a distance of 1.5 to 2 mm from a natural tooth and 2 to 3 mm from an adjacent implant to maintain an adequate biologic dimension.[62] Similar to natural teeth, violation of biologic width around an implant

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can lead to bone loss.[67] Implants that are placed too close to each other (Figure 77-6) or natural teeth can be difficult to restore. Impression copings and impression-taking techniques must be modified. Improperly spaced implants invariably lead to chronic inflammation and periimplantitis.[47,136] Conversely, implants placed with excessive distance from an adjacent tooth or implant may require prosthetic compensation in the form of mesial or distal cantilevers, which may predispose the implant to biologic (e.g., bone loss) and mechanical (e.g., screw loosening,[59] screw fracture,[133] or implant fracture[111]) complications, as well as difficulties with hygiene.[136]

Figure 77-6 Radiograph of two mandibular anterior implants placed too close together (no proximal space) resulting in implants that will be impossible to restore.

Ideally, implants should be placed, buccolingually so there is at least 2 mm of bone circumferentially around the implant.[127] Implant exposure through the lingual or buccal cortex can predispose an individual to abscess and/or suppuration.[32] Implants that are placed too far palatally/lingually require prosthetic compensation in the form of a buccal ridge lap, which may be difficult for the patient to clean and could lead to tissue inflammation.[18]

To obtain ideal esthetics, to avoid potential esthetic complications, and to correct bodily placement of the dental implant, the implant must be correctly angulated on insertion. In most anterior cases, it is desirable to have the implant long axis directed so it is emerging toward the cingulum. In the posterior region, the implant axis should be directed toward the central fossa or the stamp cusp of the opposing tooth. Implants that are placed with mild-to-moderate misangulations can often be corrected prosthetically with implant abutments. Minor misangulations (up to 15 or 20 degrees) can be corrected by with prefabricated stock-angled abutments; moderate misangulations (20 to 35 degrees) can usually be managed with customized UCLA-type abutments; extreme errors in implant angulations (more than 35 degrees) may deem an implant unrestorable and require it to be left submerged (i.e., sleeper) or to be removed (Figure 77-7).

The ultimate complication of malposed implant(s) is implant or instrument invasion into vital structures. The most common violation of neighboring anatomy is the placement of the dental implant

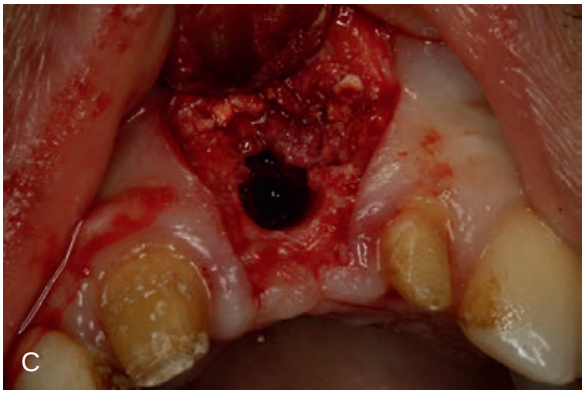

Figure 77-7 Clinical photograph of maxillary anterior implant (left central incisor) placed with an extreme facial angulation resulting in an implant that emerges through the gingiva at a level that is more apical than the adjacent natural tooth gingival margins. **A,** Surgical exposure of malpositioned implant. **B,** Surgically removed implant. **C,** Alveolar defect resulting from surgical removal of malpositioned implant.

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into the adjacent tooth root. Surgical procedures used to prepare osteotomy sites and place implants adjacent to teeth can injure the teeth either by directly cutting into the tooth structure or by damaging nearby supporting tissues and nerves. Instrumentation (e.g., drills) directed at or near the adjacent tooth may cause injury to the periodontal ligament, tooth structure, and nerve of the tooth. Depending on the extent of the injury, the tooth may require endodontic therapy or extraction. On insertion, dental implants will follow the trajectory of the osteotomy prepared by the surgical drills. Care must be taken when preparing the osteotomy to stay true to the planned path of insertion. Guidepin location radiographs taken during implant surgery can greatly reduce the potential for damaging adjacent teeth (see Figure 70-13). Radiographic analysis before implant surgery should include detection of curved, convergent, and/or dilacerated root structures of adjacent teeth that can limit implant placement.

Particular care must be taken when placing implants in the mandible so as to not encroach on the inferior alveolar canal or the mental foramen (see Chapter 53) for a description of this anatomy. Encroachment on the mandibular canal or mental foramen during osteotomy or implant placement via direct contact or mechanical compression of bone can result in injury to nerves and blood vessels. Paresthesia, hypoesthesia, hyperesthesia, dysesthesia, or anesthesia of the lower lip, skin, mucosa, and teeth may result, as well as arterial or venous bleeding.[60] The incidence of sensory disturbances after mandibular implant placement has been reported to range from 0% to 17.5%.[15]

In the maxilla, care must be taken to avoid dental implant perforation into the maxillary sinus. Displacement of the entire dental implant into the maxillary sinus cavity may require a Caldwell-Luc procedure for retrieval. See online section on sinus augmentation for further information about complications related to the maxillary sinus.

The risks of surgery are always present, but the complications can be minimized with an understanding of the etiologies and with proper diagnosis and treatment planning. Three-dimensional imaging (e.g., computed tomography [CT] and cone-beam CT [CBCT] scans) provides the surgeon with useful presurgical information for proper diagnosis and treatment planning (see Chapter 70). Careful surgical exposure for direct visualization and identification of the mental nerve may be indicated as well. Once identified, it is recommended to establish a "zone of safety" and to keep instrumentation and implants a safe margin away from the nerve (e.g., ≥2 mm).[60]

BIOLOGIC COMPLICATIONS

Biologic complications involve pathology of the surrounding periimplant hard and soft tissues. Frequently, soft tissue problems are an inflammatory response to bacterial accumulation. The cause of bacterial accumulation around implants is key to understanding the problem. For example, bacteria may accumulate at the junction of an ill-fitting implant-abutment or abutment-crown connection. Some of the highly textured, macroscopically rough implant surfaces (e.g., titanium plasma-sprayed [TPS] or hydroxyapatite [HA] coated) may also perpetuate the accumulation of bacteria on the implant surface.

Inflammation and Proliferation

Inflammation in the periimplant soft tissues has been found to be similar to the inflammatory response in gingival and other periodontal tissues. Not surprisingly, the clinical appearance is similar as well. Inflamed periimplant tissues demonstrate the same erythema, edema, and swelling seen around teeth. Occasionally, however, the reaction of periimplant soft tissues to bacterial accumulation is profound, almost unusual, with a dramatic inflammatory proliferation (Figure 77-8). This type of lesion is somewhat characteristic around implants and is indicative of either a loose-fitting implant to abutment connection or trapped excess cement that remains buried within the soft tissue space or "pocket." The precipitating local factor ultimately becomes infected with bacterial pathogens, leading to mucosal hypertrophy or proliferation and possible abscess formation (Figure 77-9). Correction of the precipitating factors (e.g., loose connection, retained cement) effectively resolves the lesion. Another type of lesion resulting from a loose abutment connection is the fistula (Figure 77-10). Again, correcting the etiologic factor quickly resolves the fistula.

Dehiscence and Recession

Dehiscence or recession of the periimplant soft tissues occurs when support for those tissues is lacking or has been lost. Recession is a common finding after implant restoration and should be anticipated especially when soft tissues are thin and not well supported (Figure 77-11). Improper implant positioning also predisposes periimplant tissues to recession. As noted earlier, placement or angulation of the implant too far to the buccal causes the buccal plate to resorb and has been shown to result in greater recession.[126] Another factor to consider is the thickness of the buccal plate of bone. Spray et al[127] recommended this thickness to be 2 mm or greater to support the buccal soft tissue. If this thickness is not present presurgical or simultaneous site development using guided bone regeneration is indicated. Recession is a problem that is particularly disconcerting in anterior esthetic areas. Patients with a high smile line or high esthetic demands consider such recession a failure (Figure 77-12).

The anatomy and soft tissue support around implants is different than that around teeth. Specifically, periodontal tissues have the distinct advantage of soft tissue support from circumferential and transeptal connective tissue fibers that insert into the cementum at a level that is more coronal than the supporting bone. In the absence of inflammation, these fibers support periodontal soft tissues far above the level of crestal bone. As a result, gingival margins and interdental papillae are supported and maintained around teeth even when the periodontal tissues are very thin. Periimplant soft tissues, on the other hand, are entirely dependent on the surrounding bone for support. Soft tissue thickness accounts

Figure 77-8 Inflammatory proliferation caused by a loose-fitting connection between the abutment and the implant.

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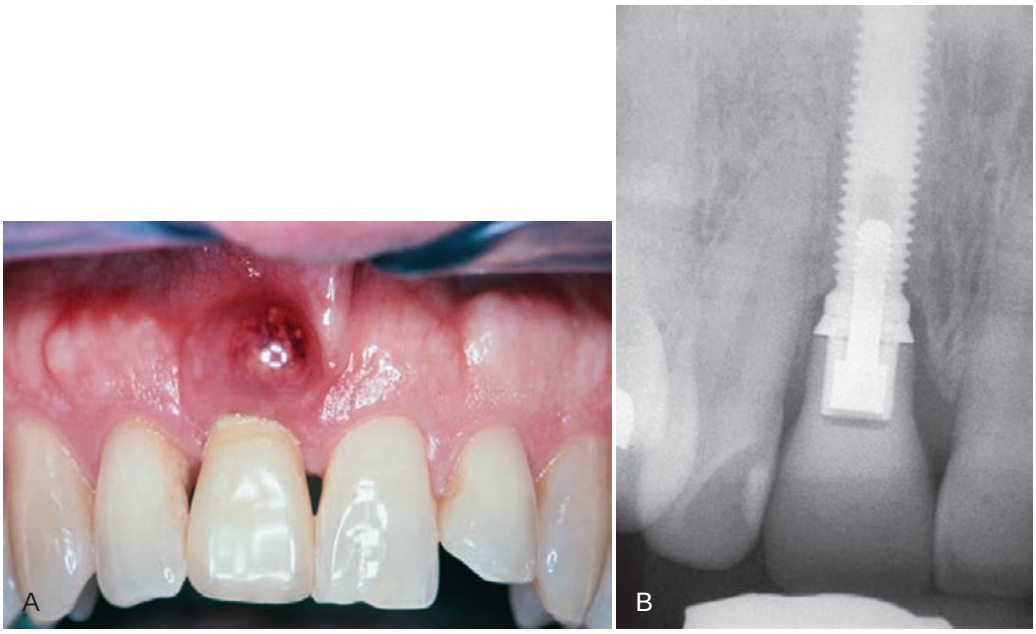

Figure 77-9 **A,** Clinical photograph of abscess caused by excess cement trapped within the soft tissues. **B,** Radiograph of implant with cemented crown (same patient as in A). Notice the subgingival depth of the crown-abutment (cement line) junction, which is below the level of the adjacent interproximal bone and therefore impossible to adequately access with explorer to remove excess cement.

Figure 77-10 Fistula caused by loose implant-abutment connection (maxillary left lateral incisor).

for some soft tissue height, but there are no supracrestal inserting connective tissue fibers to aid in the soft tissue support around an implant. Therefore soft tissue height around implants typically does not exceed about 3 to 4 mm, and bone loss around implants often leads to recession.

Periimplantitis and Bone Loss

Periimplantitis is defined as an inflammatory process affecting the tissues around an osseointegrated implant in function, resulting in loss of supporting bone.[94] To diagnose a compromised implant site, soft tissue measurements using manual or automated probes have been suggested. Although some reports state that probing is contraindicated, careful monitoring of probing depth over time seems useful in detecting changes of the periimplant tissue.[35,110,129,130] Standardized radiographic techniques, with or without computerized analysis, have been well documented and found to be useful in evaluating periimplant bone levels.[3,21,25,76,110] Together with periodic evaluation of tissue appearance, probing depth changes, and radiographic assessment as the best means of detecting changes in bone support. Clinicians should monitor the surrounding tissues for signs of periimplant disease by monitoring changes in probing depth and radiographic evidence of bone destruction, suppuration, calculus buildup, swelling, color changes, and bleeding.[95,99] Periimplantitis may be perpetuated by bacterial infection that has contaminated a rough (e.g., TPS, HA-coated) implant surface and by excessive biomechanical forces.[141,142] A classic trough-type defect is typically associated with periimplantitis (Figure 77-13). In cases with severely reduced bone support extending into the apical half of the implant (Figure 77-14) or in cases demonstrating mobility, implant removal should be considered.[6,98] The number and distribution of implants and the occlusal relationships influence the biomechanical forces applied to implants[109,112] (see Chapter 76). A recent review by Lindhe and Meyle from the Consensus Report of the Sixth European Workshop on Periodontology concluded that risk indicators for periimplantitis included (1) poor oral hygiene, (2) a history of periodontitis, (3) diabetes, (4) cigarette smoking, (5) alcohol consumption, and (6) implant surface.[87] Most of these risk factors (1 to 4) have been recognized and reported in the literature.[81] The report suggests that although data for the latter two risk factors (5 and 6) are limited, they appear to be relevant to periimplantitis.[87]

Implant Loss or Failure

Implant loss or failure is generally considered relative to the time of placement or restoration. Early implant failures occur before implant restoration. Late implant failures occur after the implant has been restored. When an implant fails before restoration, it probably did not achieve osseointegration, or the integration was weak or jeopardized by infection, movement, or impaired wound healing (Figure 77-15). Late implant failures occur after prosthesis installation for a variety of reasons, including infection and implant

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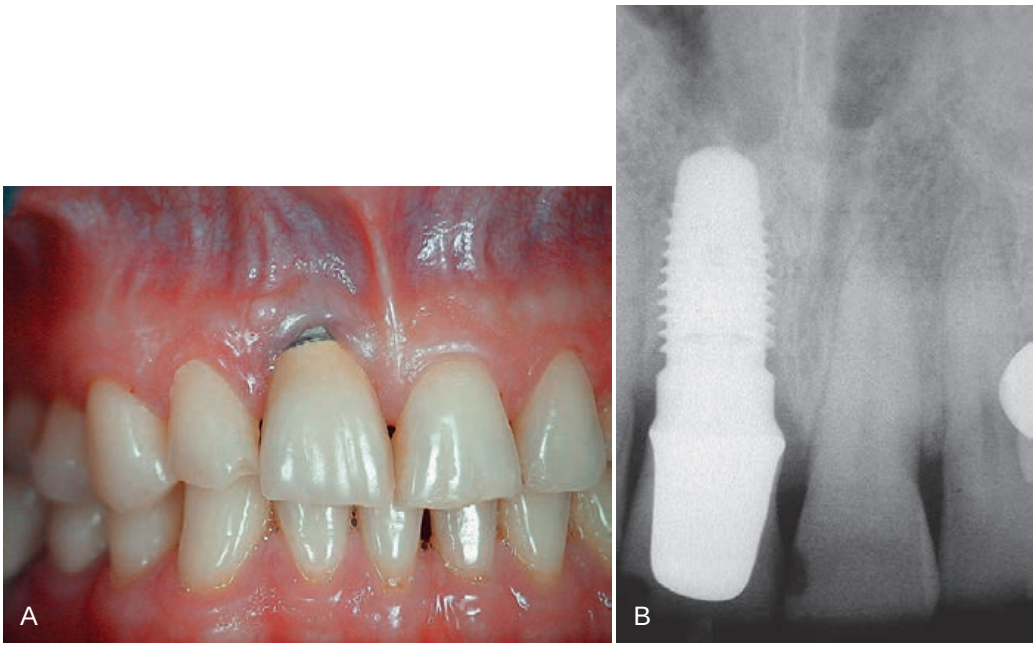

Figure 77-11 **A,** Clinical photograph of single-tooth implant crown (maxillary right central) with moderate recession that occurred 1 year after delivery of final restoration. Recession, in this case, most likely occurred because the labial bone around this wide-diameter implant was very thin or nonexistent. **B,** Radiograph of wide-diameter (6 mm) implant supporting maxillary central incisor crown (same patient as in **A**).

Figure 77-12 Poor esthetics resulting from gingival recession and exposure of crown margins, implant collars, and threads of several maxillary and mandibular implants supporting full-arch fixed partial dentures (FPDs). Notice the thin labial tissues and erythema (especially around mandibular implant sites).

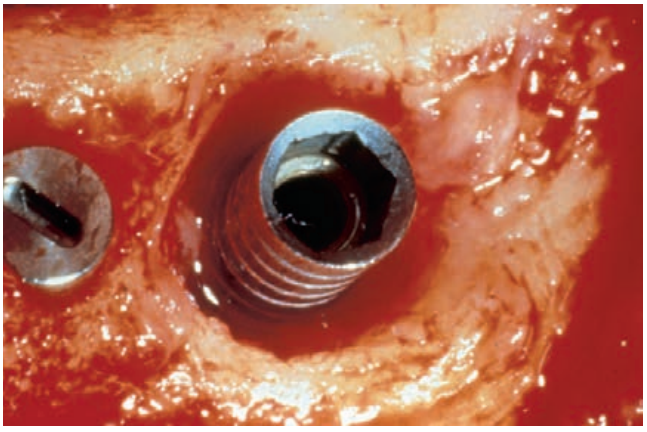

Figure 77-13 Moderately advanced bone loss around an implant with the typical circumferential trough type of boney defect. *(From Garg AK:* Implant dentistry: a practical approach, *ed 2, Mosby, St. Louis, 2010.)*

overload (Figure 77-16). In a review of the literature to evaluate biologic causes for implant failure, Esposito et al[50] found that infections, impaired healing, and overload were the most important contributing factors.

COMPLICATIONS RELATED TO AUGMENTATION PROCEDURES

A common problem encountered in implant dentistry is insufficient bone quantity to allow implant placement according to standard procedures. Deficiencies in alveolar bone result from developmental defects, periodontal disease, tooth loss, and trauma.[8,28,121] For most cases with alveolar ridge resorption, bone regenerative procedures are required to correct the defects before or simultaneous with implant placement. The success of bone augmentation procedures has been assessed by experienced clinicians in several workshops.[4,32,53,64,65] One systematic review of literature (the 2003 Workshop on Contemporary Science in Clinical Periodontics) found that survival rates of dental implants in augmented bone achieved a high level of predictability and showed that these rates were similar to implants placed in natural bone.[53] However, a more-recent systematic review of the literature (the 2008 Consensus Report of the Sixth European Workshop on Periodontology) concluded that bone augmentation procedures can

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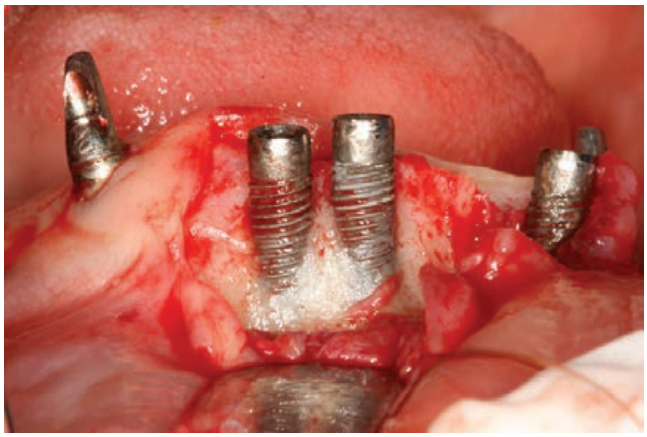

Figure 77-14 Severe horizontal and vertical bone loss around several mandibular implants.

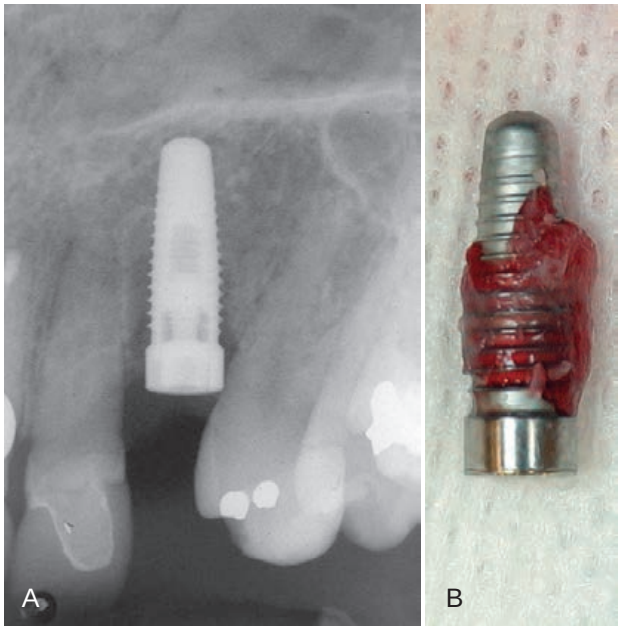

Figure 77-15 **A,** Radiograph of early failed implant caused by lack of osseointegration. In addition to the crestal bone loss, notice the radiolucency along the sides of the implant. **B,** Photograph of the failed (nonintegrated) implant (shown in **A**) that was easily removed along with surrounding connective tissue.

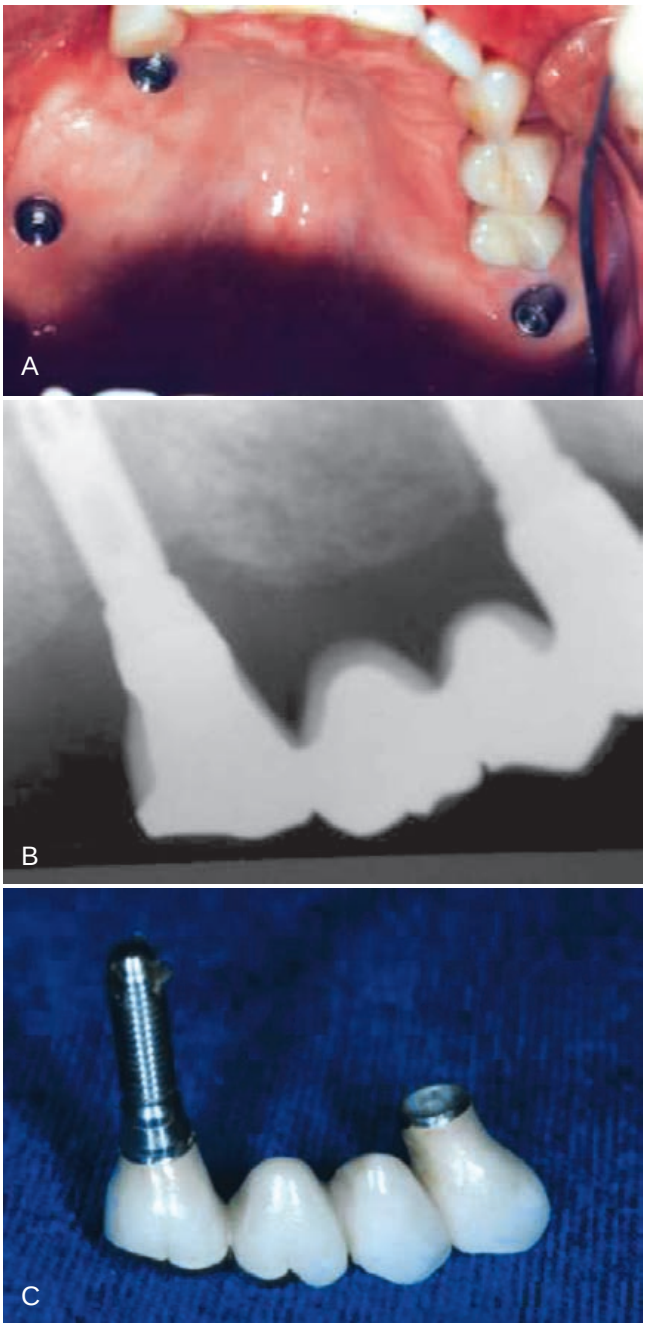

Figure 77-16 Four-unit fixed partial denture (FPD) in the posterior maxilla supported by only two implants. **A,** Clinical photograph of implant abutments in the posterior maxilla. **B,** Radiograph taken 30 months after restoration. Note the bone loss around the distal implant. **C,** Failed distal implant attached to failed prosthesis. The biologic failure of one (posterior) implant resulted in a long-span cantilever extension from the other (anterior) implant that ultimately led to its mechanical failure (i.e., abutment screw fracture). *(Courtesy Dr. John Beumer, UCLA Maxillofacial Prosthetics, Los Angeles, CA.)*

fail and that implants placed in these areas do not enjoy the high long-term survival rates of dental implants placed in pristine sites.[137] The consensus emphasized that research is needed to answer questions concerning (1) long-term performance of dental implants placed in augmented bone, (2) the clinical performance of dental implants placed in augmented or pristine sites, and (3) the clinical benefits of bone augmentation with respect to alternative treatments.

The procedures most often used to regenerate bone include autogenous bone harvesting/grafting, guided bone regeneration, and sinus bone augmentation (see Chapters 72 and 73 for detailed descriptions of these procedures). The following is a brief review of the most common complications related to these three augmentation procedures.

Autogenous Bone Harvesting/Grafting

Autogenous bone from a histologic and biologic point of view has been considered the gold standard for osseous reconstruction. Bone block grafts of autogenous bone from extraoral and intraoral sources have been documented as a predictable procedure for reconstruction of defective or atrophic alveolar ridges.[93]

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Complications can occur either at the donor or recipient sites. The most common extraoral donor sites include the ilium and tibia. Complications related to extraoral donor sites are beyond the scope of this chapter. The most common intraoral donor sites include the mandibular symphysis, mandibular ramus, and maxillary tuberosity. Although each donor area has specific associated complications, the most common complications of intraoral autogenous harvesting/grafting concern those from the mandibular symphysis.

The ramus donor site is associated with a much lower incidence of complications compared to the symphysis area.[63,92] Potential complications associated with surgical harvesting of bone from the ramus include damage to the inferior alveolar nerve and trismus after surgery. Damage to the buccal nerve, although rare, has been reported as well. Surgical harvesting of bone from the mandibular symphysis region are associated with a higher incidence of altered neurosensory disturbances to the mandibular anterior teeth and soft tissues of the chin area. The incidence of mental nerve paresthesia after symphysis grafts has been reported to be as high as 43%.[108] Many of these paresthesias are temporary.

In addition, fracture of the mandible has been reported after bone graft harvesting from the symphysis.[34] Many of these complications can be avoided by proper surgical technique, good planning, and an experienced operator.

Recipient site complications include wound dehiscence, flap necrosis, graft exposure, graft contamination, infection, and problems with bone graft incorporation and resorption. Proper flap reflection, intimate fixation of the graft, and flap coverage without tension can avoid many of the potential postsurgical complications. Bone graft material including barrier membranes should be immobilized (fixated if possible). Provisional restorations should be adjusted to prevent pressure over grafted areas. Block grafts, as well as other augmentation procedures, require experience in handling hard and soft tissue, as well as a clinician who is prepared to recognize and treat complications should they arise.

Guided Bone Regeneration

Guided bone regeneration (GBR) is a procedure that utilizes a barrier membrane to isolate an area for bone growth. The technique has been well documented for horizontal (and limited vertical) ridge augmentation[23,35,77] (see Chapter 72). The most common complication associated with GBR is premature exposure of the barrier membrane and necrosis of the overlying flap (Figure 77-17). Exposure rates of expandable polytetrafluoroethylene (ePTFE) membranes during various GBR procedures range from 41% when the technique was first introduced in the early 1990s for horizontal ridge augmentation[23] to 12.5% when surgical techniques improved and clinician's became more familiar with handling the material.[124] Once exposed to the oral environment, the membrane becomes colonized with bacteria within 3 to 4 weeks and the potential for bone regeneration under the membrane is limited to an area that is at least 2 to 3 mm from the contaminated surface.[17,125] Topical application of chlorhexidine to the exposed membrane has been advocated as a method of reducing the amount of bacteria, but it does not solve the problem and removal of the exposed membrane is necessary.

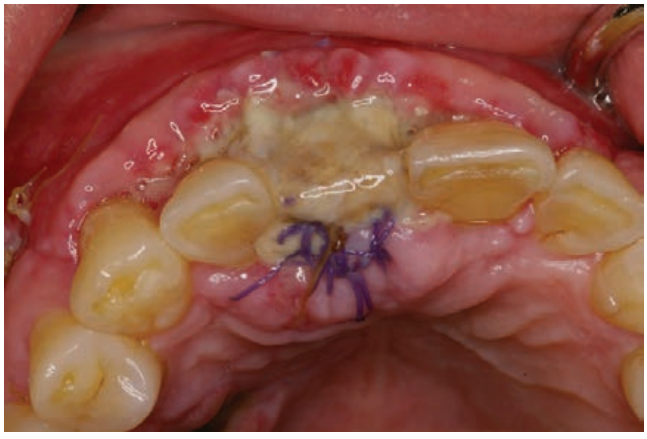

Figure 77-17 Clinical view of flap necrosis over area treated with guided bone regenerative procedure.

Other complications associated with GBR procedures include soft tissue or bone graft infection, failure to regenerate adequate bone volume and mucogingival problems, including loss of keratinized tissue and decrease in the vestibule.[23] Most of these complications are related to insufficient soft tissue healing after tooth extraction, inadequate flap design, movement of the membrane and/or graft caused by transmucosal loading and improper provisionalization, flap suturing under tension, poor surgical technique, contamination of the membrane or surgical site, compromise of the vascular supply and flap advancement for graft coverage that reduces the keratinized tissue, and vestibular depth. To prevent complications associated with GBR procedures, proper surgical technique should be employed.

For more information on complications related to augmentation procedures, including sinus bone augmentation, please visit the companion Expert CONSULT website at www.expertconsult.com.

COMPLICATIONS RELATED TO PLACEMENT AND LOADING PROTOCOLS

The traditional implant placement protocol required a healed edentulous ridge into which implants were placed and allowed to osseointegrate for a period of 3 to 6 months without occlusal loading (see Chapter 71). In contrast to these early standards, some current protocols advocate dramatically different approaches, including (1) implant placement immediately after tooth extraction, (2) implant loading immediately after placement, and (3) flapless implant placement surgery. Each of these approaches has distinct advantages but are also accompanied by specific risks of complications. The most common complications associated with these newer, developing protocols are discussed in this section.

For more information on complications related to implant placement and loading protocols, please visit the companion Expert CONSULT website at www.expertconsult.com.

PROSTHETIC OR MECHANICAL COMPLICATIONS

Prosthetic or mechanical complications occur when the strength of materials is no longer able to resist the forces that are being applied. As materials fatigue, they begin to stretch and bend; ultimately, depending on the applied forces, they will fracture. Material failures, in turn, lead to prosthetic complications such as loose, broken, and failed restorations.

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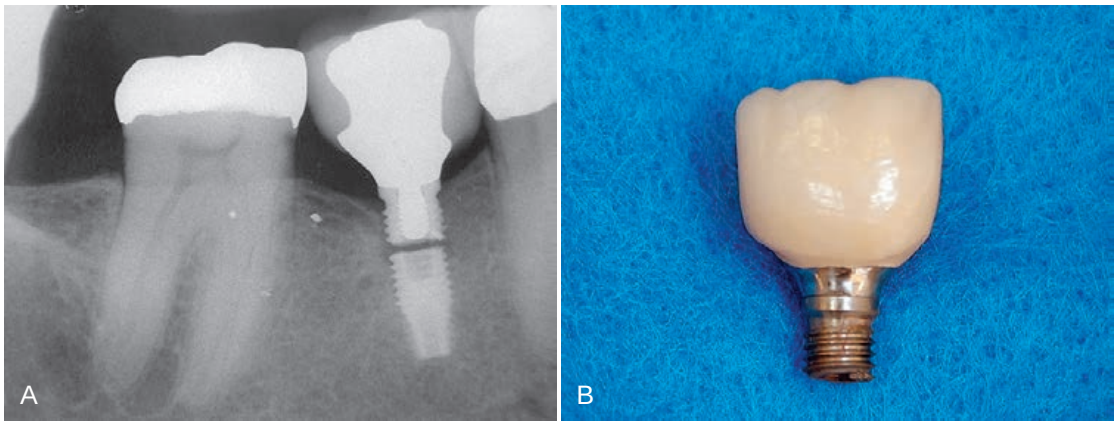

Figure 77-20 **A,** Radiograph of fractured implant (standard diameter) used to support a molar-sized single crown in the posterior mandible. **B,** Crown and coronal portion of implant (same as **A**) that fractured between the third and fourth threads.

Screw Loosening and Fracture

Screw loosening has been reported to occur quite frequently in screw-retained FPDs. Screw-retained single crowns attached to externally hexed implants (i.e., those with narrow- or standard-diameter restorative interface connection surfaces) are particularly prone to this type of mechanical complication. Studies have reported screw loosening in 6% to 49% of cases at the first annual check-up.[70,96] Screw loosening was a more prevalent problem with earlier designs. For example, abutment screws were previously made with titanium, which did not offer the clamping forces of current materials. Newer abutment designs and improved abutment screws allow for an increased clamping force to be achieved without excessive torque levels, which has helped to reduce the rate of screw loosening.

Abutment or prosthesis screw loosening is often corrected by retightening the screws. Over time, however, if screws continue to be stretched, they become fatigued and eventually fracture. This problem is evident in the patient with a loose single crown. However, in the patient with a prosthesis retained by multiple implants, the ability to detect a loose screw is greatly diminished and the problem may go unnoticed until additional screws stretch, fatigue, and fracture. In either case, the biomechanical support (and resistance) for the restoration must be evaluated and if possible, changed to prevent recurrence of the problem.

Implant Fracture

The ultimate mechanical failure is implant fracture because it results in loss of the implant and possibly the prosthesis (Figure 77-20). Furthermore, removal of a fractured implant creates a large osseous defect. Factors such as fatigue of implant materials (Figure 77-21) and weakness in prosthetic design or dimension are the usual causes of implant fractures.[3,13] Balshi[13] listed three categories of causes that may explain implant fractures: (1) design and material, (2) nonpassive fit of the prosthetic framework, and (3) physiologic or biomechanical overload. Patients with bruxism seem to be at higher risk for such events and therefore need to be screened, informed, and managed accordingly.[12,13] These patients should be fitted with occlusal guards in conjunction with placement of the final prostheses.

Fracture of Restorative Materials

Fracture or failure of materials used for implant-retained restorations can be a significant problem. This is particularly true for veneers (acrylic, composite, or ceramic) that are attached to superstructures (Figure 77-22).

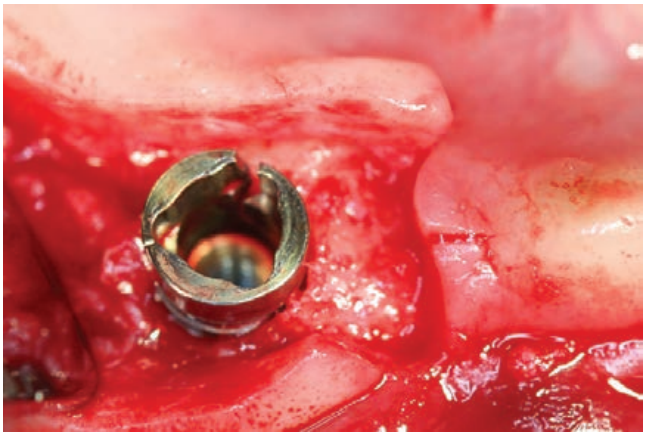

Figure 77-21 Implant fractured at internal connection collar resulting from fatigue. This collar fracture was caused by rotational forces applied to the implant at the time of placement into dense bone. The fracture was likely the result of a combination of material weakness and density of the prepared site.

ESTHETIC AND PHONETIC COMPLICATIONS

Esthetic Complications

The challenge of modern implant dentistry is achieving an esthetic, as well as a functional, implant restoration. Harmonious tooth shape, size, and ideal soft tissue contours are key factors for successful esthetic outcomes.[82]

Esthetic complications arise when patient expectations are not met. Patient satisfaction with the esthetic outcome of the implant prosthesis vary from patient to patient, depending on a number of factors. As mentioned earlier, the risk for esthetic complications is increased for patients with high esthetic expectations and less-than-optimal patient-related factors (e.g., high smile line, thin periodontal soft tissues, or inadequate bone quantity and quality). In addition to the actual appearance of the final restoration, individual perceptions and desires determine the acceptance of the

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Figure 77-22 Fractured porcelain from incisal edges of implant-supported fixed partial denture (FPD).

results. Esthetic complications result from poor implant position and deficiencies in the existing anatomy of the edentulous sites that were reconstructed with implants. An important prerequisite for achieving optimal gingival tissue contour is sufficient periimplant bone to support the soft tissues. Hard tissue defects can be treated by a variety of bone augmentation procedures.

Implant placement in the esthetic zone requires precise three-dimensional tissue reconstruction and ideal implant placement.[62,117] This reconstructive procedure enables the restorative dentist to develop a natural emergence profile of the implant crown. If the amount of available bone does not allow for ideal implant placement and if the implant is positioned too apical, buccal, or in the proximal space, an unesthetic prosthetic profile will be developed (Figure 77-23).

If crown contours and dimensions are not ideal or if gingival harmony around the implant restoration is unesthetic, the patient may consider the implants or restorations as complications because the outcome does not represent a natural appearance (Figure 77-24). Gingiva-colored materials utilized to replace lost gingival anatomy can offer an alternative to surgical augmentation in patients undergoing implant therapy (Figure 77-25). These restorations provide numerous advantages over conventional restorations, including improved lip support, masking of interproximal spaces, and restoration of gingival symmetry in selected cases.[66]

If the patient is truly dissatisfied with the esthetic result and there is a problem with the position of the implants that can be corrected (i.e., the patient's expectations are reasonable), the implants could be removed; the case could be reevaluated and possibly retreated. However, the clinician should consider prosthetic solutions before implant removal. Use of angulated abutments, superstructures, and gingiva-colored materials may result in an acceptable esthetic result, thus avoiding multiple surgeries necessary to rebuild hard and soft tissue if an integrated implant is removed.

Careful patient evaluation and treatment planning along with a solid understanding and appreciation for the predictability and limitations of implant procedures will minimize esthetic complications. Patients with a high smile line, high esthetic demands, thin periodontium, or lack of hard and soft tissue support in the anterior esthetic region are some of the most difficult cases to treat and should only be treated after extensive planning by experienced clinicians.

Phonetic Problems

Implant prostheses that are fabricated with unusual palatal contours (e.g., restricted or narrow palatal space) or that have

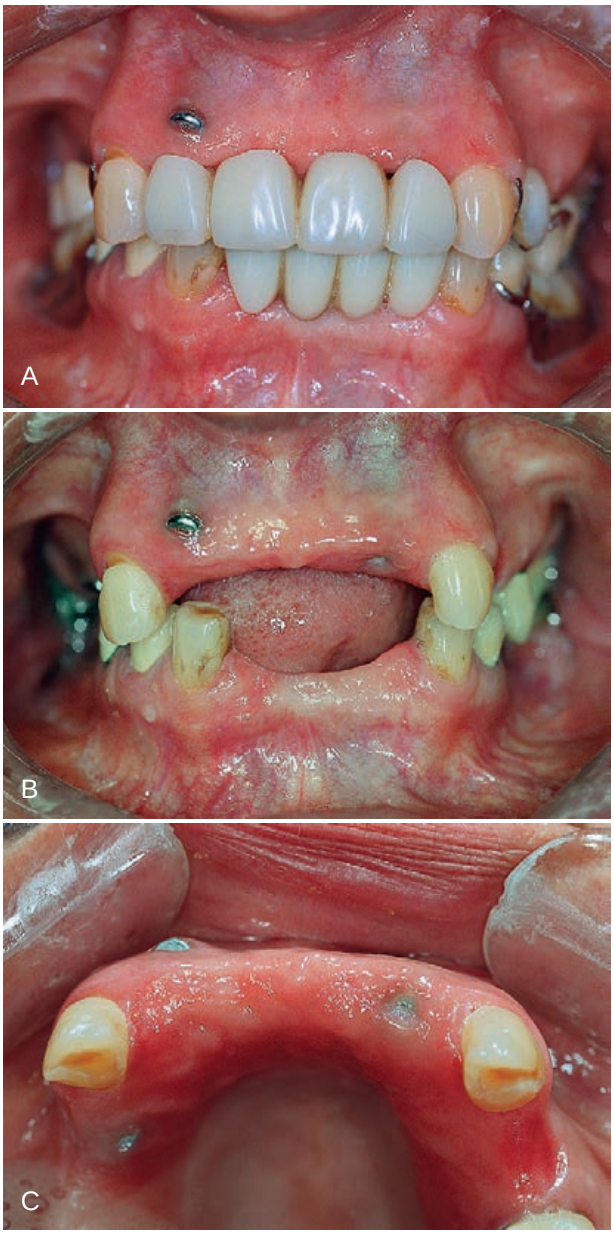

Figure 77-23 Poor implant position makes it impossible to restore with an esthetic, "natural appearing" restoration. **A,** Anterior view with removable partial dentures RPDs inserted. **B,** Anterior view without removable partial dentures. Notice the high level of implant cover screw/head exposure (maxillary right lateral incisor) that is significantly apical to the level of the adjacent natural tooth (cuspid) gingival margin. **C,** Occlusal view of same patient. Again, notice the labial projection of same implant (maxillary right lateral incision), as well as the palatal position of the implant in premolar area. Any attempt to restore the anterior implants would not be esthetically acceptable.

spaces under and around the superstructure can create phonetic problems for the patient. This is particularly problematic when full-arch, implant-supported, fixed restorations are fabricated for patients who have a severely atrophied maxilla. These patients are probably best served with an implant-assisted maxillary overdenture because the design facilitates replacement of missing alveolar structure and avoids creating spaces that allow air to escape during speech.

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SCIENCE TRANSFER

Implant treatment in general has a high success rate, although the clinician has the obligation to each patient to reduce the possibility of failure and complications in that particular case.

Many surgery-related problems can be avoided by proper presurgical work-ups. This includes the use of three-dimensional radiographs to clearly ascertain bone dimensions and anatomic structures. Appropriate three-dimensional radiologic data should preclude the risk of damage or injury to the inferior mandibular nerve and the mental nerve, and the use of intraoperative periapical radiographs is essential to prevent damage to adjacent tooth roots. Penetration of surgical burs or implants through the lingual plate of the mandible must be avoided because it can lead to serious hemorrhage into the sublingual tissues with risk of subsequent airway obstruction.

The most common prosthetic-related problems are fractures of veneers caused by heavy occlusal loading and loosening of prostheses from the implant by screw loosening or cement failure. Control of occlusal loading by narrowing occlusal tables and ensuring no balancing and minimal working and centric contacts should be part of all implant therapy. Screw-retained prostheses can be easily removed and repaired, whereas cemented restoratives often cannot be retrieved and can have excess cement causing periimplantitis. The use of torque-controlled screwdrivers and implants with internal fixation can reduce the risk of screw fracture and loosening.

Figure 77-24 High gingival margin on single-tooth implant crown in the maxillary lateral incisor position. Notice the discrepancy between gingival margin levels of the implant and the adjacent natural teeth.

Figure 77-25 Pink porcelain used on implant-supported fixed restoration to mask the high gingival margin and long implant crowns resulting from an uncorrected alveolar ridge defect.

CONCLUSION

Although implants offer a highly predictable treatment option for the replacement of single and multiple missing teeth, there are a variety of potential complications that may be surgical, biologic, mechanical, prosthetic, or esthetic. Careful diagnosis and treatment planning, along with the use of diagnostic imaging, surgical guides, meticulous techniques, and adherence to proven principles can prevent many of the problems discussed in this chapter. A thorough understanding of anatomy, biology, and wound healing can reduce the incidence of complications. However, there is no substitute for training, knowledge, and clinical experience for preventing, recognizing, and successfully managing complications. This chapter is a start. The clinician who places and/or restores implants must be aware of techniques and procedures for early intervention should a complication arise.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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PART 8

Supportive Treatment and Results of Treatment

Henry H. Takei

The successful management of periodontal disease requires a positive program directed at maintaining and improving the results of treatment, as well as preventing the development of new disease. The gradual microbial recolonization of tooth surfaces begins soon after plaque is eliminated; therefore the measures to stop recolonization also must be a constant effort on the part of the patient, following the professional instructions and guidance and including periodic professional care visits. This part of the text also presents the overwhelming evidence that has been accumulated about the effectiveness of periodontal therapy.

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CHAPTER 78

Supportive Periodontal Treatment

Robert L. Merin

CHAPTER OUTLINE

RATIONALE FOR SUPPORTIVE PERIODONTAL TREATMENT
MAINTENANCE PROGRAM
Examination and Evaluation
Checking of Plaque Control
Treatment
Recurrence of Periodontal Disease
CLASSIFICATION OF POSTTREATMENT PATIENTS
REFERRAL OF PATIENTS TO THE PERIODONTIST
TESTS FOR DISEASE ACTIVITY
MAINTENANCE FOR DENTAL IMPLANT PATIENTS

Preservation of the periodontal health of the treated patient requires as positive a program as that required for the elimination of periodontal disease. After Phase I therapy is completed, patients are placed on a schedule of periodic recall visits for maintenance care to prevent recurrence of the disease (Figures 78-1 and 78-2).

Transfer of the patient from active treatment status to a maintenance program is a definitive step in total patient care that requires time and effort on the part of the dentist and staff. Patients must understand the purpose of the maintenance program, and the dentist must emphasize that preservation of the teeth depends on maintenance therapy. Patients who are not maintained in a supervised recall program subsequent to active treatment show obvious signs of recurrent periodontitis (e.g., increased pocket depth, bone loss, or tooth loss).[7,10,16] The more often patients present for recommended supportive periodontal treatment (SPT), the less likely they are to lose teeth.[70] One study found that treated patients who do not return for regular recall are at 5.6 times greater risk for tooth loss than compliant patients.[16] Another study showed that patients with inadequate SPT after successful regenerative therapy have a fiftyfold increase in risk of probing attachment loss compared with those who have regular recall visits.[18]

Motivational techniques and reinforcement of the importance of the maintenance phase of treatment should be considered before performing definitive periodontal surgery.[10] Studies have shown that few patients display complete compliance with recommended maintenance schedules[1,33,37,39,40,69,71] (Figure 78-3). *It is meaningless simply to inform patients that they are to return for periodic recall visits without clearly explaining the significance of these visits and describing what is expected of patients between visits.*

The maintenance phase of periodontal treatment starts immediately after the completion of Phase I therapy (see Figures 78-1 and 78-2). While the patient is in the maintenance phase, the necessary surgical and restorative procedures are performed. This ensures that all areas of the mouth retain the degree of health attained after Phase I therapy.

RATIONALE FOR SUPPORTIVE PERIODONTAL TREATMENT

Studies have shown that even with appropriate periodontal therapy, some progression of disease is possible.[23,26,41,46,63,67] One likely explanation for the recurrence of periodontal disease is *incomplete subgingival plaque removal.*[66] If subgingival plaque is left behind during scaling, it regrows within the pocket. The regrowth of subgingival plaque is a slow process compared with that of supragingival plaque. During this period (perhaps months), the subgingival plaque may not induce inflammatory reactions that can be discerned at the gingival margin. The clinical diagnosis may be further confused by the introduction of adequate supragingival plaque control because the inflammatory reactions caused by the plaque in the soft tissue wall of the pocket are not likely to be manifested clinically as gingivitis. Thus inadequate subgingival plaque control can lead to continued loss of attachment, even without the presence of clinical gingival inflammation.

Bacteria are present in the gingival tissues in chronic and aggressive periodontitis cases.[13,17,43,48] Eradication of intragingival microorganisms may be necessary for a stable periodontal result.[20] Scaling, root planing, and even flap surgery may not eliminate intragingival bacteria in some areas.[18] These bacteria may recolonize the pocket and cause recurrent disease.

Bacteria associated with periodontitis can be transmitted between spouses and other family members.[2,64] Patients who appear to be successfully treated can become infected or reinfected with potential pathogens. This is especially likely in patients with remaining pockets.

Another possible explanation for the recurrence of periodontal disease is the microscopic nature of the dentogingival unit healing

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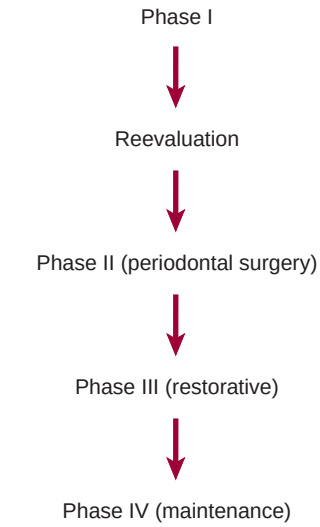

Figure 78-1 Incorrect sequence of periodontal treatment phases. Maintenance phase should be started immediately after the reevaluation of Phase I therapy.

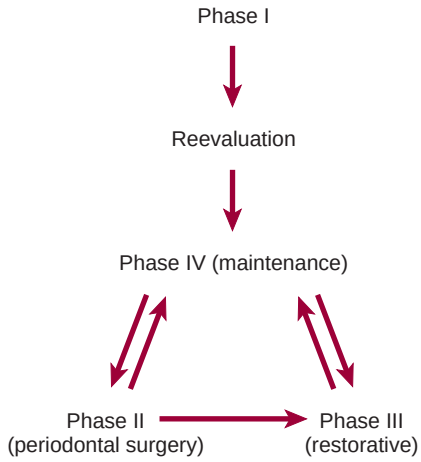

Figure 78-2 Correct sequence of periodontal treatment phases.

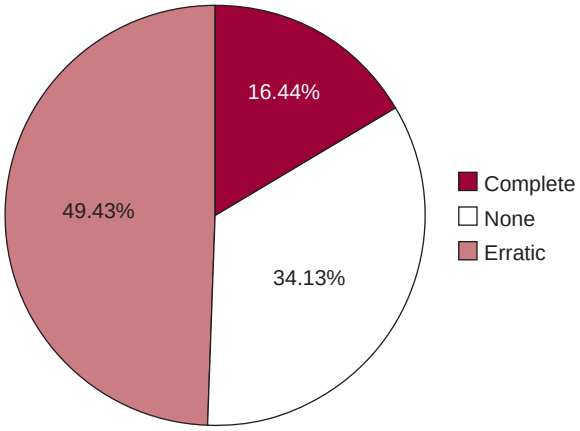

Figure 78-3 Compliance with maintenance therapy in 961 patients studied for 1 to 8 years. *(Modified from Wilson TG Jr, Glover ME, Schoen J, et al:* J Periodontol *55:468, 1984.)*

after periodontal treatment. Histologic studies have shown that after periodontal procedures, tissues usually do not heal by formation of new connective tissue attachment to root surfaces[14,57,58] but result in a long junctional epithelium. It has been speculated that this type of dentogingival unit may be weaker and that inflammation may rapidly separate the long junctional epithelium from the tooth. Thus treated periodontal patients may be predisposed to recurrent pocket formation if maintenance care is not optimal.

Subgingival scaling alters the microflora of periodontal pockets.[38,45,55] In one study, a single session of scaling and root planing in patients with chronic periodontitis resulted in significant changes in subgingival microflora.[38] Reported alterations included a decrease in the proportion of motile rods for 1 week, a marked elevation in the proportion of coccoid cells for 21 days, and a marked reduction in the proportion of spirochetes for 7 weeks.

Although pocket debridement suppresses components of the subgingival microflora associated with periodontitis, periodontal pathogens may return to baseline levels within days or months.[3,54] The return of pathogens to pretreatment levels generally occurs in approximately 9 to 11 weeks but can vary dramatically among patients.[3]

Both the mechanical debridement performed by the therapist and the motivational environment provided by the appointment seem to be necessary for good maintenance results. Patients tend to reduce their oral hygiene efforts between appointments.[7,71] Knowing that their hygiene will be evaluated motivates them to perform better oral hygiene in anticipation of the appointment.

In one study the proportion of spirochetes obtained in baseline samples of subgingival flora was highly correlated with clinical periodontal deterioration over 1 year.[31] However, subsequent reports in the same longitudinal study concluded that the arbitrary assignment of treated periodontitis patients to 3-month maintenance intervals appears to be as effective in preventing recurrences of periodontitis as assignment of recall intervals based on microscopic monitoring of the subgingival flora.[31,32] Microscopic monitoring was found not to be a reliable predictor of future periodontal destruction in patients on 3-month recall programs, presumably because of the alteration of subgingival flora produced by subgingival instrumentation.

In conclusion, *there is a sound scientific basis for recall maintenance because subgingival scaling alters the pocket microflora for variable but relatively long periods.*

MAINTENANCE PROGRAM

Periodic recall visits form the foundation of a meaningful long-term prevention program. The interval between visits is initially set at 3 months but may be varied according to the patient's needs.

Periodontal care at each recall visit comprises three parts (Box 78-1). The first part involves examination and evaluation of the patient's current oral health. The second part includes the necessary maintenance treatment and oral hygiene reinforcement. The third part involves scheduling the patient for the next recall appointment, additional periodontal treatment, or restorative dental procedures. The time required for a recall visit for patients with multiple teeth in both arches is approximately 1 hour,[49] which includes time for greeting the patient, setting up, and cleaning up.

Examination and Evaluation

The recall examination is similar to the initial evaluation of the patient (see Chapter 30). However, because the patient is not new to the office, the dentist primarily looks for changes that have occurred since the last evaluation. Analysis of the current oral

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BOX 78-1 Maintenance Recall Procedures

Part I: Examination
(Approximate time: 14 minutes)
Patient greeting
Medical history changes
Oral pathologic examination
Oral hygiene status
Gingival changes
Pocket depth changes
Mobility changes
Occlusal changes
Dental caries
Restorative, prosthetic, and implant status

Part II: Treatment
(Approximate time: 36 minutes)
Oral hygiene reinforcement
Scaling
Polishing
Chemical irrigation or site-specific antimicrobial placement

Part III: Report, Cleanup, and Scheduling
(Approximate time: 10 minutes)
Write report in chart.
Discuss report with patient.
Clean and disinfect operatory.
Schedule next recall visit.
Schedule further periodontal treatment.
Schedule or refer for restorative or prosthetic treatment.

hygiene status of the patient is essential. Updating of changes in the medical history and evaluation of restorations, caries, prostheses, occlusion, tooth mobility, gingival status, and periodontal and periimplant probing depths are important parts of the recall appointment. The oral mucosa should be carefully inspected for pathologic conditions (Figures 78-4 to 78-9).

Radiographic examination must be individualized,[5] depending on the initial severity of the case and the findings at the recall visit (Table 78-1). These are compared with findings on previous radiographs to check the bone height and look for repair of osseous defects, signs of trauma from occlusion, periapical pathologic changes, and caries.

Checking of Plaque Control

To assess the effectiveness of their plaque control, patients should perform their hygiene regimen immediately before the recall appointment. Plaque control must be reviewed and corrected until the patient demonstrates the necessary proficiency, even if additional instruction sessions are required. Patients instructed in plaque control have less plaque and gingivitis than uninstructed patients,[7,61,62] and the amount of supragingival plaque affects the number of subgingival anaerobic organisms.[19,56]

Treatment

The required scaling and root planing are performed, followed by an oral prophylaxis (see Chapter 45). Care must be taken not to instrument normal sites with shallow sulci (1 to 3 mm deep) because studies have shown that repeated subgingival scaling and root planing in initially normal periodontal sites result in

Figure 78-4 A, Hyperplastic gingivitis related to crown margins and plaque accumulation in a 27-year-old woman. **B,** Four months after treatment, there is significant improvement. However, some inflammation around crown margins still exists, which cannot be resolved without replacing the crowns.

TABLE 78-1 Radiographic Examination of Recall Patients for Supportive Periodontal Treatment*

Patient Condition/ Situation	Type of Examination
Clinical caries or high-risk factors for caries.	Posterior bite-wing examination at 12- to 18-month intervals.
Clinical caries and no high-risk factors for caries.	Posterior bite-wing examination at 24- to 36-month intervals.
Periodontal disease not under good control.	Periapical and/or vertical bite-wing radiographs of problem areas every 12 to 24 months; full-mouth series every 3 to 5 years.
History of periodontal treatment with disease under good control.	Bite-wing examination every 24 to 36 months; full-mouth series every 5 years.
Root form dental implants.	Periapical or vertical bite-wing radiographs at 6, 12, and 36 months after prosthetic placement, then every 36 months unless clinical problems arise.
Transfer of periodontal or implant maintenance patients.	Full-mouth series if a current set is not available. If full-mouth series has been taken within 24 months, radiographs of implants and periodontal problem areas should be taken.

Data from Matteson SR, Bottomley W, Finger H, et al: *The selection of patients for x-ray examinations: dental radiographic examinations,* DHHS Pub No FDA 88-8273, Washington, DC, 1987, US Department of Health and Human Services; and Wilson TG: *J Am Dent Assoc* 121:491, 1990.
*Radiographs should be taken when they are likely to affect diagnosis and patient treatment. The recommendations in this table are subject to clinical judgment and may not apply to every patient.

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Figure 78-5 **A,** Patient was 38 years old when these original radiographs were taken and was treated with a combination of surgical and nonsurgical therapy. This individual is a classic class C maintenance patient. **B,** Pretreatment photograph. Note the inflammation and heavy calculus deposits. **C,** Photograph taken 10 years after treatment. **D,** Radiographs taken 5 years after treatment. **E,** Radiographs taken 10 years after treatment. The radiographic appearance is as good as can be expected in such a severe case. Teeth #15 and #17 were extracted 8 years after treatment.

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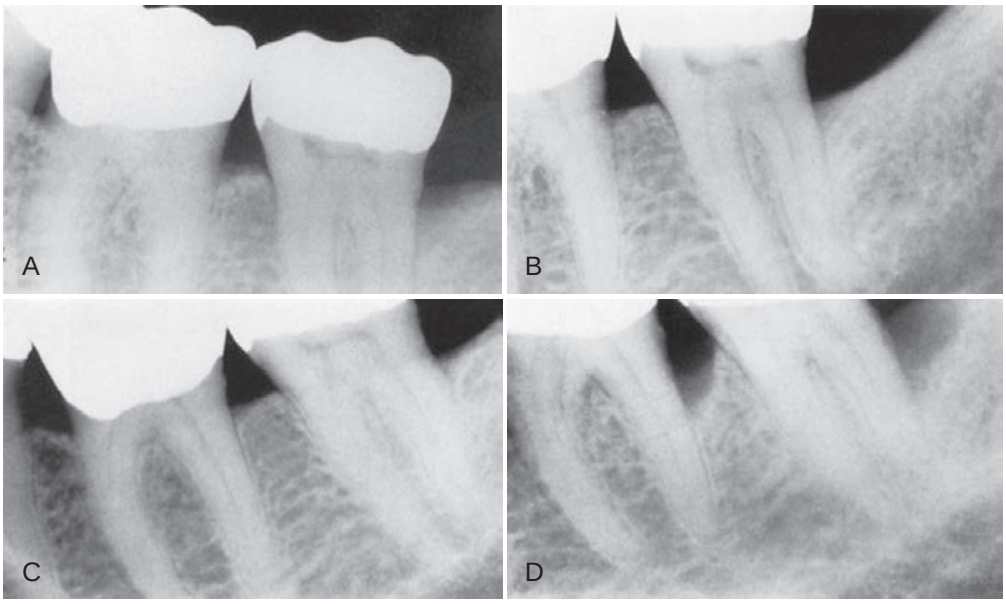

Figure 78-6 This series of radiographs clearly shows the importance of maintenance therapy. **A,** Original radiograph of a 58-year-old male. Note the deep distal bone loss on tooth #18 and the moderate distal lesion of tooth #19. Surgical treatment included osseous grafting. **B,** Radiograph 14 months after surgical therapy. The patient had recall maintenance performed every 3 to 4 months. **C,** Appearance 3 years after surgery, with regular recalls every 3 to 4 months. **D,** Appearance after 2 years without recalls (7 years after surgery). Note the progression of the disease on the distal surfaces of teeth #18 and #19.

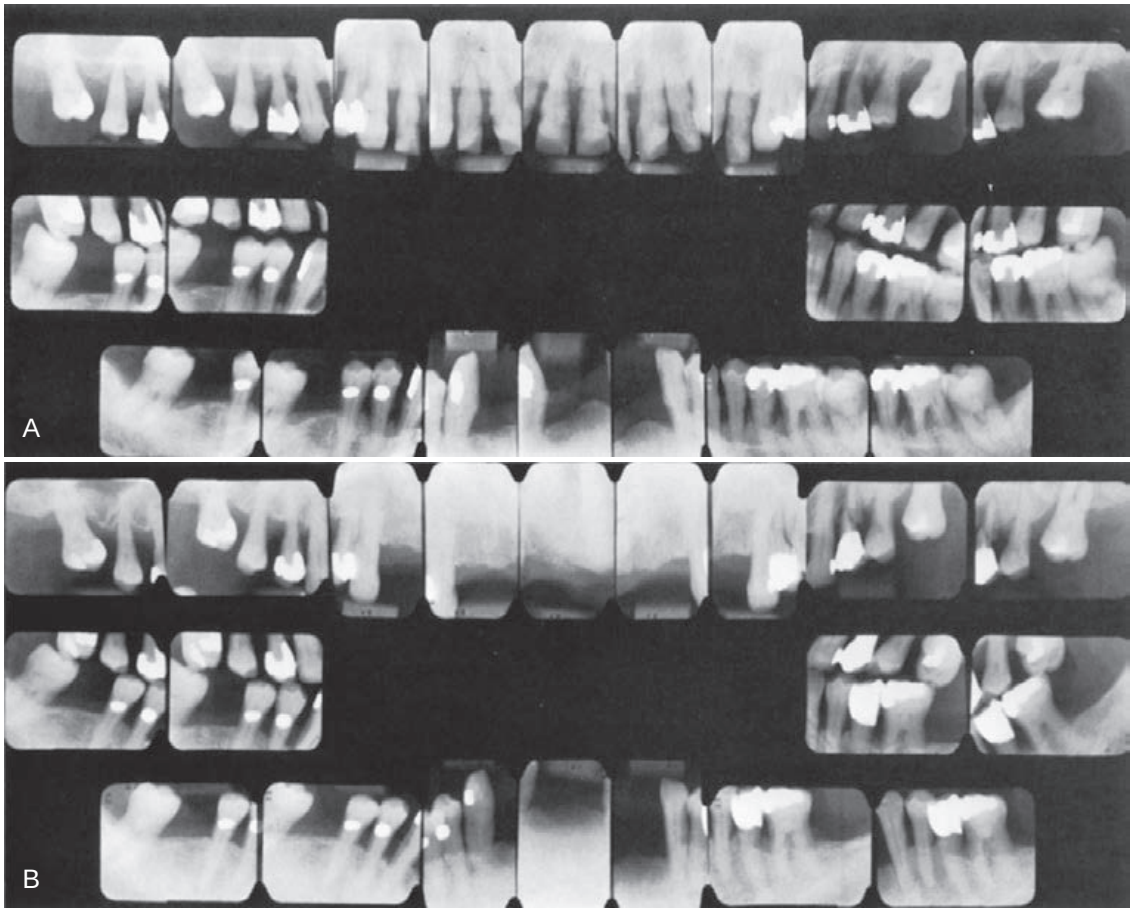

Figure 78-7 Advanced cases sometimes do better than expected when the patient complies with maintenance therapy. **A,** Initial radiographs showing a very advanced case. The maxillary arch had extractions and nonsurgical treatment. A plastic partial denture was placed and was expected to grow into a full denture within a few years. The mandibular arch was treated with periodontal surgery, and a permanent, metal and plastic, removable partial denture was placed. **B,** Radiographs taken 8 years later. The patient performed good oral hygiene and had 3-month recalls. Teeth #12 and #15 required extraction.

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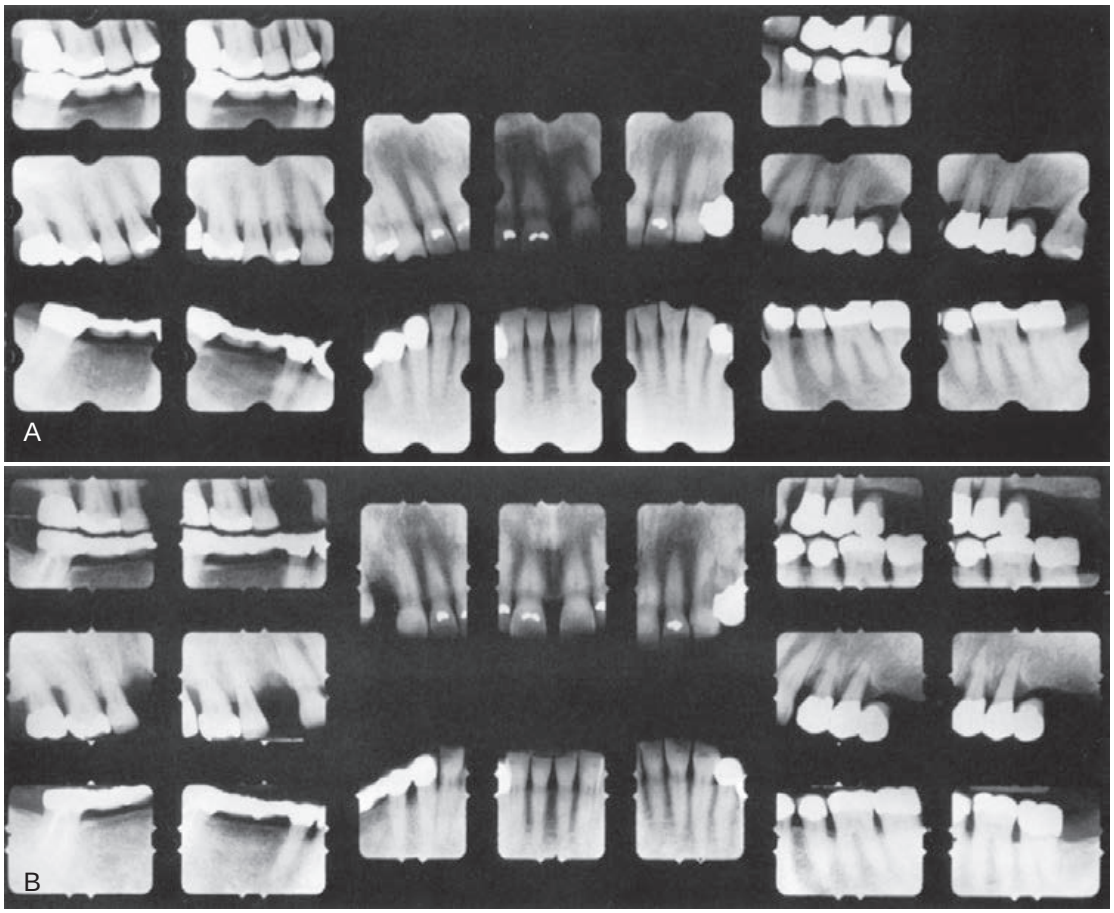

Figure 78-8 **A,** Initial radiographs. The patient was advised to have localized areas of periodontal surgery and periodontal recall every 3 months. However, the patient did not comply and only had dental cleanings once or twice yearly. **B,** Radiographs 4 years later. Note the loss of teeth #5 and #15 and the increased bone loss of several premolars and molars.

significant loss of attachment.[29] Irrigation with antimicrobial agents or placement of site-specific antimicrobial devices is performed in maintenance patients with remaining pockets.[3,30,34,49]

Recurrence of Periodontal Disease

Occasionally, lesions may recur, which often can be traced to inadequate plaque control on the part of the patient or failure to comply with recommended SPT schedules. It should be understood, however, that it is the dentist's responsibility to teach, motivate, and control the patient's oral hygiene technique, and the patient's failure is the dentist's failure. Surgery should not be undertaken unless the patient has shown proficiency and willingness to cooperate by adequately performing his or her part of therapy.[10,59,70]

Other causes for recurrence include the following:

1. Inadequate or insufficient treatment that has failed to remove all the potential factors favoring plaque accumulation (see Figure 78-4). Incomplete calculus removal in areas of difficult access is a common source of problems.
2. Inadequate restorations placed after the periodontal treatment was completed.
3. Failure of the patient to return for periodic checkups (see Figure 78-6). This may be a result of the patient's conscious or unconscious decision not to continue treatment or the failure of the dentist and staff to emphasize the need for periodic examinations.
4. Presence of some systemic diseases that may affect host resistance to previously acceptable levels of plaque.

A failing case can be recognized by the following:

1. Recurring inflammation revealed by gingival changes and bleeding of the sulcus on probing.
2. Increasing depth of sulci, leading to the recurrence of pocket formation.
3. Gradual increases in bone loss, as determined by radiographs.
4. Gradual increases in tooth mobility, as ascertained by clinical examination.

Cases that do not respond to adequate therapy or recur for unknown reasons are referred to as aggressive periodontitis (see Chapters 18 and 40).

The decision to re-treat a periodontal patient should not be made at the preventive maintenance appointment but should be postponed for 1 to 2 weeks.[15] Often the mouth looks much better at that time because of the resolution of edema and the resulting improved tone of the gingiva. Table 78-2 summarizes the symptoms of recurrence of periodontal disease and their probable causes.

CLASSIFICATION OF POSTTREATMENT PATIENTS

The first year after periodontal therapy is important in terms of indoctrinating the patient in a recall pattern and reinforcing oral hygiene techniques. In addition, it may take several months to evaluate accurately the results of some periodontal surgical procedures. Consequently, some areas may have to be re-treated because

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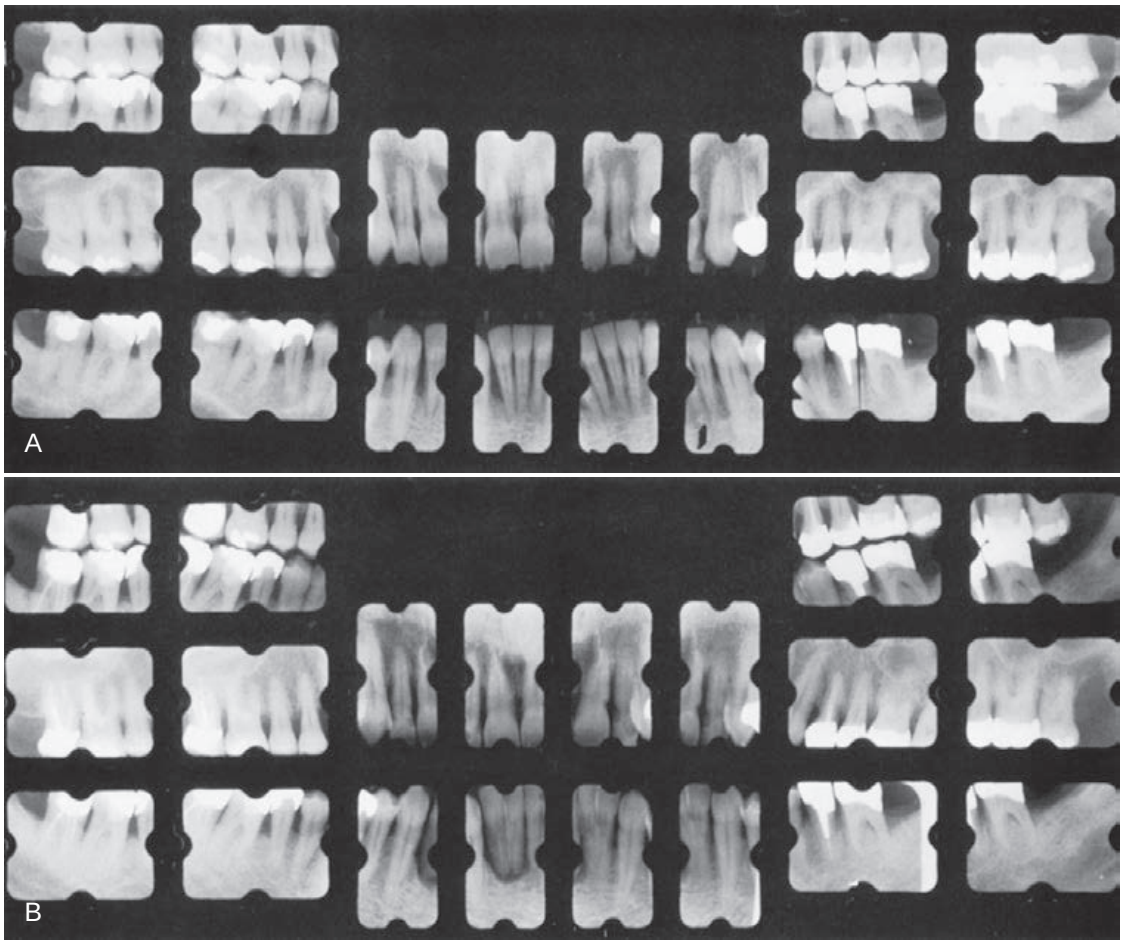

Figure 78-9 **A,** Initial radiographs. The patient was advised to have localized areas of periodontal surgery and periodontal recall every 3 months. However, the patient did not comply and had no treatment other than emergency care and occasional dental cleanings. **B,** Radiographs 7 years later. Note the advanced bone loss and caries on many teeth.

the results may not be optimal. Furthermore, the first-year patient often has etiologic factors that may have been overlooked and may be more amenable to treatment at this early stage. *For these reasons, the recall interval for first-year patients should not be longer than 3 months.*

The patients who are on a periodontal recall schedule are a varied group. Table 78-3 lists several categories of maintenance patients and a suggested recall interval for each. Patients can improve or may relapse to a different classification, with a reduction in or exacerbation of periodontal disease. When one dental arch is more involved than the other, the patient's periodontal disease is classified by the arch with the worse condition.

In summary, maintenance care is a critical phase of therapy. The long-term preservation of the dentition is closely associated with the frequency and quality of recall maintenance.

REFERRAL OF PATIENTS TO THE PERIODONTIST

Many periodontal patients can be well managed by the general dentist As more people retain their teeth throughout their lifetime and as the proportion of older people in the population increases, more teeth will be at risk of periodontal disease. Considerable research shows possible links between periodontal disease and systemic diseases, such as heart disease, stroke, and diabetes, and adverse pregnancy outcomes. Therefore the prevalence of patients requiring SPT is likely to increase in the future.

This expected increase in the number of periodontal patients will necessitate a greater understanding of periodontal problems and an increased level of expertise for the solution of such problems on the part of the general practitioner of dentistry. General dentists must know when co-management with a periodontist is indicated. Specialists are needed to treat particularly difficult periodontal cases, patients with systemic health problems, dental implant patients, and those with a complex prosthetic construction that requires reliable results.

The question of where to draw the line between the cases to be treated in the general dental office and those to be referred to a specialist varies for different practitioners and patients, and the American Academy of Periodontology has issued guidelines to help the general practitioner decide when co-management with a periodontist is indicated.[4] The diagnosis indicates the type of periodontal treatment required. If periodontal destruction necessitates surgery on the distal surfaces of second molars, extensive osseous surgery, or complex regenerative procedures, the patient is usually best treated by a specialist. On the other hand, patients who require localized gingivectomy or flap curettage usually can be treated by the general dentist.

It is immediately obvious that some patients should be referred to a specialist, whereas most patients clearly have problems that can

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TABLE 78-2 Symptoms and Causes of Recurrence of Disease

Symptom	Possible Causes
Increased mobility	Increased inflammation Poor oral hygiene Subgingival calculus Inadequate restorations Deteriorating or poorly designed prostheses Systemic disease modifying host response to plaque
Recession	Toothbrush abrasion Inadequate keratinized gingiva Frenum pull Orthodontic therapy
Increased mobility with no change in pocket depth and no radiographic change	Occlusal trauma caused by lateral occlusal interference, bruxism, high restoration Poorly designed or worn-out prosthesis Poor crown-to-root ratio
Increased pocket depth with no radiographic change	Poor oral hygiene Infrequent recall visits Subgingival calculus Poorly fitting partial denture Mesial inclination into edentulous space Failure of new attachment surgery Cracked teeth Grooves in teeth New periodontal disease Gingival overgrowth caused by medication
Increased pocket depth with increased radiographic bone loss	Poor oral hygiene Subgingival calculus Infrequent recall visits Inadequate or deteriorating restorations Poorly designed prostheses Inadequate surgery Systemic disease modifying host response to plaque Cracked teeth Grooves in teeth New periodontal disease

be treated by a general dentist. For a third group of patients, however, it will be difficult to decide whether treatment by a specialist is required. Any patient who does not clearly belong in the second of these categories should be considered a candidate for referral to a specialist.[4,42]

The decision to have the general practitioner treat a patient's periodontal problem should be guided by a consideration of the degree of risk that the patient will lose a tooth or teeth for periodontally related reasons or the risk of the periodontal disease contributing to their general health problems.

The most important factors in the decision are the extent and location of the periodontal deterioration. Teeth with pockets of 5 mm or more, as measured from the cementoenamel junction, may have a prognosis of rapid decline. The location of the periodontal deterioration is also an important factor in determining the risk of tooth loss. Teeth with furcation lesions may be at risk even when more than 50% of bone support remains. Therefore patients with strategically

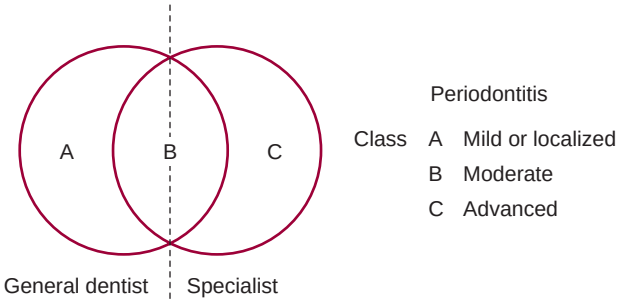

Figure 78-10 Scheme for determining which practitioner should perform periodontal maintenance in patients with different degrees of periodontitis.

important teeth that fall into these categories are usually best treated by specialists.

An important question remains: *Should the maintenance phase of therapy be performed by the general practitioner or the specialist?* This should be determined by the amount of periodontal deterioration present. Class A recall patients should be maintained by the general dentist, whereas Class C patients should be maintained by the specialist (see Table 78-3). Class B patients can alternate recall visits between the general practitioner and the specialist (Figure 78-10). The suggested rule is that the *patient's disease should dictate whether the general practitioner or the specialist should perform the maintenance therapy.*

TESTS FOR DISEASE ACTIVITY

Periodontal patients, even though they have received effective periodontal therapy, are at risk of disease recurrence for the rest of their lives.[25,26] In addition, many pockets in furcation areas may not have been eliminated by surgery. At present, the best way of determining areas that are losing attachment is to use a well-organized charting system.[47,68] Some computerized systems allow easy retrieval and comparison of past findings.

Comparison of sequential probing measurements gives the most accurate indication of the rate of loss of attachment.[6] A number of other clinical and laboratory variables have been correlated with disease activity.

At present, no accurate method of predicting disease activity exists, and clinicians rely on the information provided by combining probing, bleeding on probing, and sequential attachment measurements.[28,32,68] Patients whose disease is clearly refractory are candidates for bacterial culturing and antibiotic therapy in conjunction with additional mechanical therapy.

New methods will undoubtedly be developed in the future to help predict disease activity.[3] The clinician must be able to interpret whether a test may be useful in determining disease activity and future loss of attachment.[11] Tests should be adopted only when they are based on research that includes a critical analysis of the sensitivity, specificity, disease incidence, and predictive value of the proposed test.

MAINTENANCE FOR DENTAL IMPLANT PATIENTS

Patients with implants are susceptible to a form of bone loss called *periimplantitis*, and evidence suggests that such patients may be more prone to plaque-induced inflammation with bone loss than those with natural teeth[9,50,53,65] (see Chapter 77). Patients with

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TABLE 78-3 Recall Intervals for Various Classes of Recall Patients

Merin Classification	Characteristics	Recall Interval
First year	First-year patient: routine therapy and uneventful healing.	3 months
	First-year patient: difficult case with complicated prosthesis, furcation involvement, poor crown-to-root ratios, or questionable patient cooperation.	1-2 months
Class A	Excellent results well maintained for 1 year or more. Patient displays good oral hygiene, minimal calculus, no occlusal problems, no complicated prostheses, no remaining pockets, and no teeth with less than 50% of alveolar bone remaining.	6 months to 1 year
Class B	Generally good results maintained reasonably well for 1 year or more, but patient displays some of the following factors: 1. Inconsistent or poor oral hygiene 2. Heavy calculus formation 3. Systemic disease that predisposes to periodontal breakdown 4. Some remaining pockets 5. Occlusal problems 6. Complicated prostheses 7. Ongoing orthodontic therapy 8. Recurrent dental caries 9. Some teeth with less than 50% of alveolar bone support 10. Smoking 11. Positive family history or genetic test 12. More than 20% of pockets bleed on probing	3-4 months (decide on recall interval based on number and severity of negative factors)
Class C	Generally poor results after periodontal therapy and/or several negative factors from the following list: 1. Inconsistent or poor oral hygiene 2. Heavy calculus formation 3. Systemic disease that predisposes to periodontal breakdown 4. Many remaining pockets 5. Occlusal problems 6. Complicated prostheses 7. Recurrent dental caries 8. Periodontal surgery indicated but not performed for medical, psychologic, or financial reasons 9. Many teeth with less than 50% of alveolar bone support 10. Condition too far advanced to be improved by periodontal surgery 11. Smoking 12. Positive family history or genetic test 13. More than 20% of pockets bleed on probing	1-3 months (decide on recall interval based on number and severity of negative factors; consider re-treating some areas or extracting severely involved teeth)

periodontitis-associated tooth loss are at significantly increased risk of developing periimplantitis.[51,52]

The overall periodontal condition in partially edentulous implant patients can influence the clinical condition around implants.[12] The microflora of implants in partially edentulous patients differs from that in edentulous patients.[8] The implant microflora is similar to tooth microflora in the partially edentulous mouth. Periodontal and implant maintenance are linked because maintenance of a tooth microflora consistent with periodontal health is necessary to maintain implant microflora consistent with periimplant health.[8,60] Because periimplantitis is difficult to treat,[24] it is extremely important to treat periodontal disease before implant placement and to provide good supportive therapy with implant patients.

In general, procedures for maintenance of patients with implants are similar to those for patients with natural teeth,[3,21,27,36] with the following three differences:

1. Special instrumentation that will not scratch the implants are used for calculus removal on the implants.
2. Acidic fluoride prophylactic agents are avoided.
3. Nonabrasive prophy pastes are used.

During the phase after uncovering the implants, patients must use ultrasoft brushes, chemotherapeutic rinses, tartar control pastes, irrigation devices, and yarnlike materials to keep the implants and natural teeth clean. Patients often are reluctant to touch the implants but must be encouraged to keep the areas clean.

Special instruments should be used on the implants during recall appointments.[21,36] Metal hand instruments and ultrasonic and sonic tips should be avoided because they can alter the titanium surface.[22] Only plastic instruments or specially designed gold-plated curettes should be used for calculus removal because the implant surfaces can be easily scratched. The rubber cup with flour of pumice, tin oxide, or special implant-polishing pastes should be used on abutment surfaces with light, intermittent pressure.[35]

Although daily use of topically applied antimicrobials is advised,[36] acidic fluoride agents should not be used because they cause surface damage to titanium abutments.[44]

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SCIENCE TRANSFER

The most important factor that determines the long-term success of periodontal therapy is plaque control. No matter how skillful the surgeon, the initial postsurgical improvement of periodontal status quickly deteriorates if bacterial plaque deposition is not controlled. Patients with a history of periodontal disease should be seen for plaque control and any needed recall root planing at least every 3 or 4 months. All patients should have their plaque score monitored, and oral hygiene instruction should be upgraded when plaque scores increase. The use of antibacterial mouthwashes, such as chlorhexidine, on the toothbrush two to three times daily augments mechanical plaque removal and is an important adjunct to supportive periodontal treatment.

All patients need to have an ongoing recording of pocket depth, recession, and plaque status to detect any periodontal deterioration in its early stages so that appropriate treatment measures can be given.

When prosthetics must be unscrewed and removed for maintenance, it is best done in the office responsible for placing the prosthetics. Each time the prosthetic appliances are reattached, a slight change in the occlusion occurs. Time must be allowed for occlusal corrections.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 79

Results of Periodontal Treatment

Robert L. Merin

CHAPTER OUTLINE

PREVENTION AND TREATMENT OF GINGIVITIS
PREVENTION AND TREATMENT OF LOSS OF ATTACHMENT
Prevention of Loss of Attachment
Treatment of Loss of Attachment
TOOTH MORTALITY
SUMMARY

The prevalence of periodontal disease, the resulting high rate of tooth mortality, and the potential for multiple systemic health complications aggravated by chronic periodontitis raise an important question: Is periodontal treatment effective in preventing and controlling the chronic infection and progressive destruction of periodontal disease? Current concepts of evaluating health care require a scientific basis for treatment, referred to as **evidence-based therapy.** *Evidence is now overwhelming that periodontal therapy is effective in preventing periodontal disease, slowing the destruction of the periodontium, and reducing tooth loss.*

PREVENTION AND TREATMENT OF GINGIVITIS

For many years, the belief that good oral hygiene is necessary for the successful prevention and treatment of gingivitis has been widespread among periodontists. In addition, worldwide epidemiologic studies have confirmed a close relationship between the incidence of gingivitis and lack of oral hygiene.[5,6]

Löe and co-workers[17,36] provided conclusive evidence on the association between oral hygiene and gingivitis. After 9 to 21 days without performing oral hygiene measures, healthy dental students with previously excellent oral hygiene and healthy gingiva developed heavy accumulations of plaque and generalized mild gingivitis. When oral hygiene techniques were reinstituted, the plaque in most areas disappeared in 1 or 2 days, and gingival inflammation in these areas disappeared approximately 1 week after the plaque was removed. Thus gingivitis is reversible and can be resolved by daily, effective plaque removal.

A number of long-term studies have shown that gingival health can be maintained by a combination of effective oral hygiene maintenance and scaling procedures.*

A 3-year study was conducted on 1248 General Telephone workers in California to determine whether progression of gingival inflammation is reduced in an oral environment in which high levels of hygiene are maintained.[34,35] Experimental and control groups were computer-matched based on periodontal and oral hygiene status, past caries experience, age, and gender. During the study period, several procedures were instituted to ensure that the oral hygiene status of the experimental group was maintained at a high level. Subjects were given a series of frequent oral prophylaxis treatments combined with oral hygiene instruction. Subjects in the control group received no attention from the study team except for annual examinations. They were advised to continue their usual daily practices and accustomed visits for professional care. After 3 years, the increase in plaque and debris in the control group was four times as great as that in the experimental group. Similarly, gingivitis scores were much higher in control subjects than in the matching experimental group. Therefore *chronic marginal gingivitis can be controlled with good oral hygiene and dental prophylaxis.*

PREVENTION AND TREATMENT OF LOSS OF ATTACHMENT

Although periodontal therapy has been used for more than 100 years, it is only since the mid 1970s that a number of studies have been conducted to determine the effect of treatment on reducing the progressive loss of periodontal support for the natural dentition.

Prevention of Loss of Attachment

Löe et al[18,19] conducted a longitudinal investigation to study the natural development and progression of periodontal disease. The first study group, established in Oslo, Norway, in 1969, consisted of 565 healthy male nondental students and academicians between 17 and 40 years of age. Oslo was selected mainly because this city had an ongoing preschool, school, and postschool dental program

*References 1, 2, 8, 11, 12, 13, 20, 22, 34, and 35.

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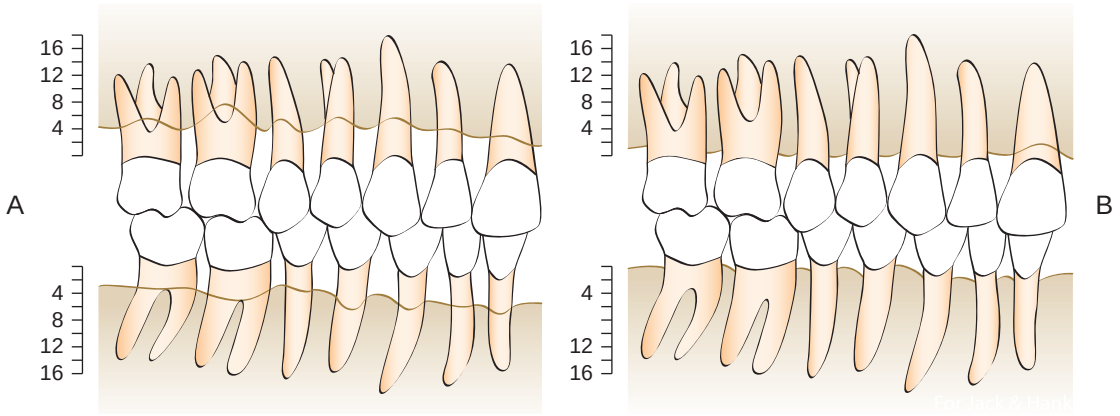

Figure 79-1 **A,** Mean periodontal support of teeth of Sri Lankan tea laborers at approximately 40 years of age. **B,** Mean periodontal support of teeth of Norwegian academicians at approximately 40 years of age. *(From Löe H, Anerud A, Boysen H, et al:* J Periodontol *49:607, 1978.)*

offering systematic preventive, restorative, endodontic, orthodontic, and surgical therapy on an annual recall basis for all children and adolescents, complete with a documented attendance record, for the previous 40 years. Members of the study population had experienced maximum exposure to conventional dental care throughout their lives. A second study group, established in Sri Lanka in 1970, consisted of 480 male tea laborers between 15 and 40 years of age. They were healthy and well built by local standards, and their nutritional condition was clinically fair. The workers had never been exposed to any programs relative to the prevention or treatment of dental diseases. Toothbrushing was unknown, and dental caries was virtually nonexistent.

The results of this study are interesting. As the members of the Norwegian group approached 40 years of age, the mean individual loss of attachment was slightly above 1.5 mm and the mean annual rate of attachment loss was 0.08 mm for interproximal surfaces and 0.10 mm for buccal surfaces. As the Sri Lankans approached 40 years of age, the mean individual loss of attachment was 4.50 mm, and the mean annual rate of progression of the lesion was 0.30 mm for interproximal surfaces and 0.20 mm for buccal surfaces. Figure 79-1 shows a graphic interpretation of the difference between the two groups. This study suggests that without interference, periodontal lesions progress continually and at a relatively even pace.

Further analysis of the Sri Lankan laborers showed that they were not all losing attachment at the same rate (Figures 79-2 and 79-3).[18] Virtually all gingival areas showed inflammation, but attachment loss varied tremendously. Based on interproximal loss of attachment and tooth mortality, three subpopulations were identified as individuals with "rapid progression" (RP) of periodontal disease (8%), individuals with "moderate progression" (MP) (81%), and individuals who exhibited "no progression" (NP) of periodontal disease beyond gingivitis (11%). At age 35, the mean loss of attachment in the RP group was 9 mm; in the MP group, 4 mm; and in the NP group, less than 1 mm. At the age of 45, the mean loss of attachment in the RP group was 13 mm and in the MP group, 7 mm. Therefore, under natural conditions and in the absence of therapy, 89% of the Sri Lankan laborers had severe periodontitis that progressed at much greater rates than in the Norwegian group.

In the previously discussed study of General Telephone workers in California, loss of attachment was measured clinically and alveolar bone loss measured radiographically.[33,34] After 3 years, the control group showed loss of attachment at a rate more than 3 times that of the matching experimental group during the same

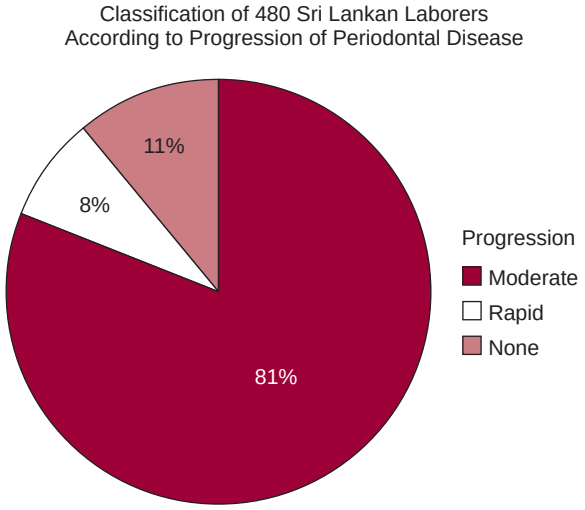

Figure 79-2 Progression of periodontal disease in an untreated population. *(Data from Löe H, Anerud A, Boysen H, et al:* J Clin Periodontol *13:431, 1986.)*

Mean Loss of Attachment at Various Ages (mm)

Age	Progression Group	
	Rapid	Moderate
35	9	4
45	13	7

Figure 79-3 Loss of attachment in untreated Sri Lankan laborers. *(Data from Löe H, Anerud A, Boysen H, et al:* J Clin Periodontol *13:431, 1986.)*

period (Figure 79-4). In addition, subjects who received frequent oral prophylaxis and were instructed in good oral hygiene practices showed less bone loss radiographically after 3 years than did control subjects. *It is clear that loss of attachment can be reduced by good oral hygiene and frequent dental prophylaxis.*

Treatment of Loss of Attachment

A longitudinal study of patients with moderate-to-advanced periodontal disease conducted at the University of Michigan showed that the progression of periodontal disease can be stopped for 3

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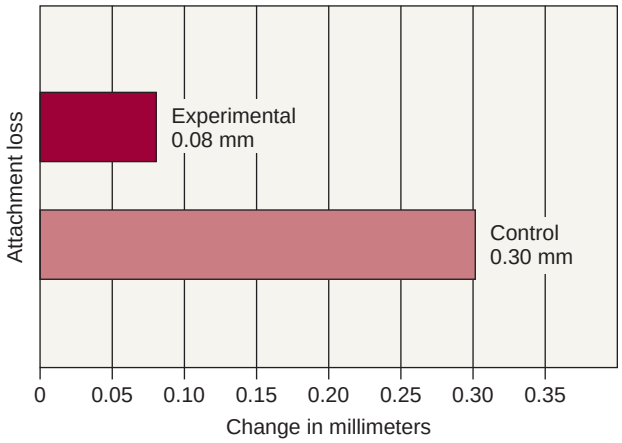

Figure 79-4 Change in mean attachment level from baseline to third-year examination for experimental and control groups. *(From Suomi JD, West JD, Chang JJ, et al:* J Periodontol *42:152, 1971.)*

years postoperatively regardless of the modality of treatment.[26–29] With long-term observations, the average loss of attachment was only 0.3 mm over 7 years.[26] These results indicated a more favorable prognosis for treatment of advanced periodontal lesions than previously assumed.

Another study was conducted in 75 patients with advanced periodontal disease to determine the effect of plaque control and surgical pocket elimination on the establishment and maintenance of periodontal health.[14] This study showed that no further alveolar bone loss occurred during the 5-year observation period. The meticulous plaque control practiced by the patients in this study was considered a major factor in the excellent results produced. After 14 years, results for 61 of the initial 75 individuals were reported.[13] Repeated examinations demonstrated that treatment of advanced forms of periodontal disease resulted in clinically healthy periodontal conditions and that this state of health was maintained in most patients and sites during the 14-year period. A more detailed analysis of the data, however, revealed that a small number of sites in a few patients lost a substantial amount of attachment. Approximately 43 surfaces in 15 different patients were exposed to recurrent periodontal disease of significant magnitude. The frequency of sites that lost more than 2 mm of attachment during the 14 years of maintenance was 0.8% to 0.1% per year.

Neither of these studies used a control group because failing to treat advanced periodontal patients cannot be justified for ethical reasons. However, in a study in private practice, an effort was made to find and evaluate patients with diagnosed moderate-to-advanced periodontitis who had not followed through with recommended periodontal therapy.[3] Thirty patients ranging in age from 25 to 71 years were evaluated after periods ranging from 18 to 115 months. All of these untreated patients had progressive increases in pocket depth and radiographic evidence of progressive bone resorption.

In a study of the progression of periodontal disease in the absence of therapy, two different populations were monitored.[16] One group of 64 Swedish adults with mild-to-moderate periodontal disease and one group of 36 American adults with advanced destructive disease were monitored but not treated for 6 years and 1 year, respectively. During the course of 6 years, 11.6% of all sites in the Swedish population (1.9% per year) showed attachment loss of greater than 2 mm, and the corresponding figure for the American population was 3.2% per year. Thus the frequency of sites with disease progression was 20 to 30 times higher in untreated groups of patients than in the treated and well-maintained groups described in the preceding discussion.[16] *Thus treatment is effective in reducing loss of attachment.*

TABLE 79-1 Average Loss of Teeth during a 5-year Period Compared with Normal Loss of Teeth in 1428 Men and Women Ages 20 through 59

	GRADE OF ORAL HYGIENE		
	Good	Fairly Good	Not Good
"Normal" loss of teeth*	1.1	1.4	1.8
Actual loss of teeth during 5-year period	0.4	0.6	0.9

From Lovdal A, Arno A, Schei O, et al: *Acta Odontol Scand* 19:537, 1961.
*Estimate based on data recorded at initiation of study period.

TABLE 79-2 Tooth Mortality after Treatment of Advanced Periodontitis in 104 Patients with 2604 Teeth Treated over a 10-Year Period

Teeth Lost*	Reason
2	Pulpal disease
3	Accidents
4	Prosthetic considerations
14	Various reasons; for example, one patient wanted a maxillary denture for cosmetic reasons
30	Periodontal
53	All reasons

Data from Ramfjord SP, Knowles JW, Nissle RR, et al: *J Periodontol* 44:66, 1973.
*2% of the teeth were lost during the study period. NOTE: United States health surveys conducted in the 1960s indicated that an average of 4.3 teeth were lost after age 35 in the general population.[9]

TOOTH MORTALITY

The ultimate test for the effectiveness of periodontal treatment is whether the loss of teeth can be prevented. Sufficient studies from both private practice and research institutions are now available to document that loss of teeth is reduced or prevented by therapy.

The combined effect of subgingival scaling every 3 to 6 months and controlled oral hygiene was evaluated over a 5-year period in 1428 factory workers in Oslo.[20] Tooth loss was significantly reduced in all patients. This study showed that frequent subgingival scaling reduces tooth loss even when oral hygiene is "not good" (Table 79-1).

The previously mentioned longitudinal study conducted at the University of Michigan included 104 patients with a total of 2604 teeth.[26–29] After 1 to 7 years of treatment, 53 teeth were lost for various reasons (Table 79-2). Approximately 32 teeth were lost during the first and second years after initiation of treatment. The remaining 21 teeth were lost in a random pattern over the next 6 years. Therefore the loss of teeth caused by advanced periodontal disease after treatment was minimal (1.15%).

Another study was undertaken to test the effect of periodontal therapy in cases of advanced disease.[14–15] The subjects were 75 patients who had lost 50% or more of their periodontal support (Figure 79-5). Treatment consisted of oral hygiene measures, scaling procedures, extraction of untreatable teeth, periodontal surgery, and prosthetic therapy if indicated. After completion of

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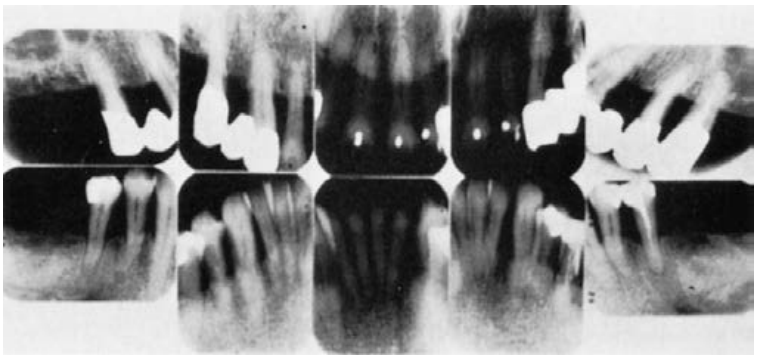

Figure 79-5 Radiographs taken 5 years after typical periodontal treatment. Note the advanced bone loss, despite the teeth retained in a healthy condition for the duration of the study. *(From Lindhe J, Nyman S:* J Clin Periodontol *2:67, 1975.)*

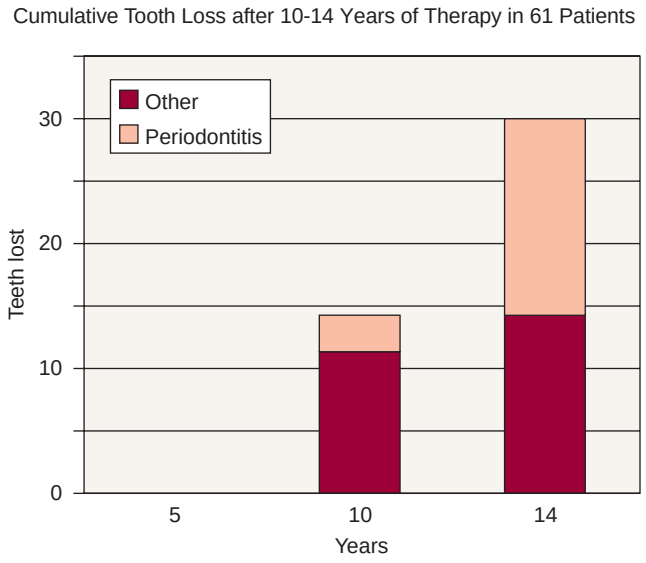

Figure 79-6 Tooth loss in treated patients with very advanced periodontal disease. *(Data from Lindhe J, Nyman S:* J Clin Periodontol *11:504, 1984.)*

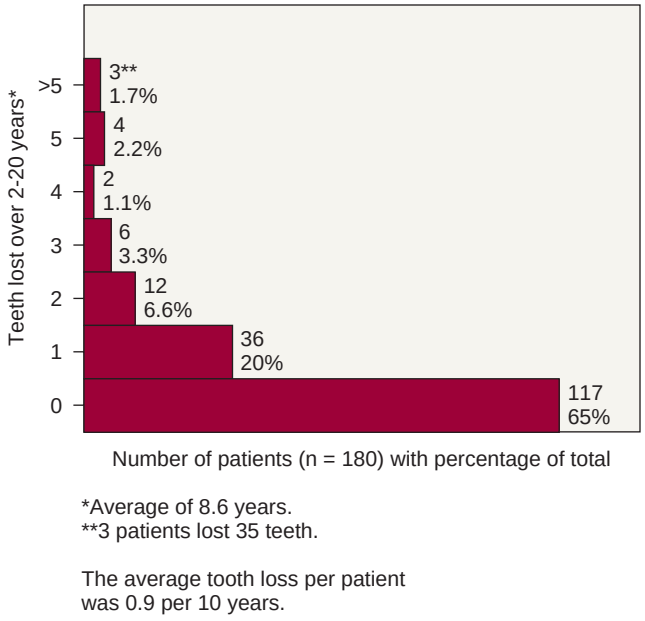

Figure 79-7 Tooth mortality. Average tooth loss per patient was 0.9 per 10 years. *(Modified from Ross IF, Thompson RH, Galdi M:* Parodontologie *25:125, 1971.)*

periodontal treatment, no patient showed further loss of periodontal support for the next 5 years. No teeth were extracted in the 5-year posttreatment period. Patients in this study were selected because of their capacity to meet high requirements of plaque control after repeated instruction in oral hygiene techniques; this fact does not detract from the validity of the study but tends to show the etiologic importance of bacterial plaque. The results indicate that periodontal surgery coupled with a detailed plaque control program not only temporarily cures the disease but also reduces further progression of periodontal breakdown, even in patients with severely reduced periodontal support.

After 14 years, 61 of the original patients were still in the study.[14] Recurrence of destructive periodontal disease in isolated sites of the dentition resulted in loss of a certain number of teeth during the observation period (Figure 79-6). In the 6 to 10 years after active therapy, one tooth in each of three different patients was lost, and during the final observation period (11 to 14 years), three teeth in one patient, two teeth in each of three patients, and one tooth in each of four patients had to be extracted because of recurrent periodontal disease. In addition, three teeth in each of three different patients and one tooth in each of five patients were extracted because of the development of extensive caries, periapical lesions, or other endodontic complications. During the entire course of the study, the total loss was 30 teeth (for all reasons) of 1330 teeth. The tooth mortality rate was therefore 2.3%.

Several studies in private practice have attempted to measure frequency of tooth loss after periodontal therapy. In one study, 180 patients who had been treated for chronic destructive periodontal disease were evaluated.[30–31] The average age of the patients before treatment was 43.7 years. A total of 141 teeth were lost. From the beginning of treatment to the time of the survey, the majority of patients lost no teeth (Figure 79-7). Three of 180 patients (1.7%) lost 35 teeth, approximately 25% of the teeth lost. Twelve additional patients lost 46 teeth, or 32.6% of the teeth lost. Many patients in the study had advanced alveolar bone loss, including extensive furcation involvements. However, only a relatively small number (141) of the teeth were lost in the study group of 180 patients between the beginning of periodontal treatment and the time of the study.

The teeth were lost for several reasons, including periodontal disease, caries, and other nonperiodontal causes. The length of time after treatment varied from 2 to 20 years, with an average of 8.6 years. Of considerable significance is the large number of teeth (81 teeth, or 57.5%) lost by a few patients (15 patients, or 8.4%). Even when this group is considered with the remaining 165 patients, the periodontal care helped to retain most teeth because the average tooth loss was slightly less than one tooth (0.9) over the 10 years after treatment.

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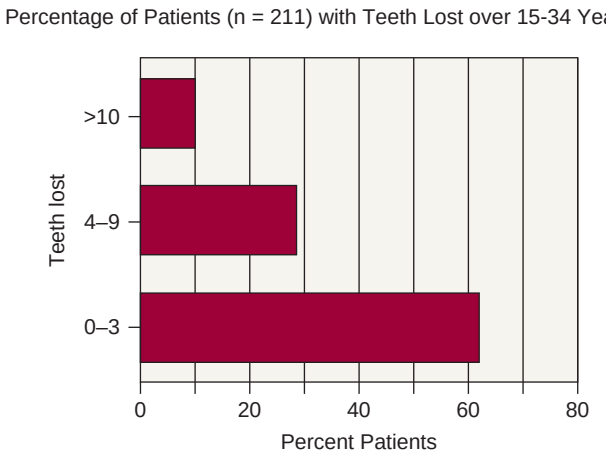

Figure 79-8 Tooth mortality 15 to 34 years after initiation of therapy (average of 22.2 years). Average tooth loss per patient was 1.6 teeth per 10 years. Compare with the same study population in Figure 79-7. As the treated population ages, the rate of bone loss appears to increase. *(Modified from Goldman MJ, Ross IF, Goteiner D:* J Periodontol *57:347, 1986.)*

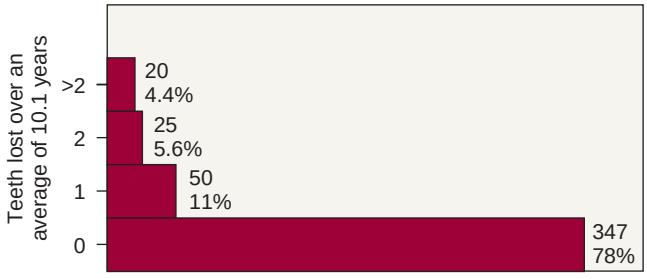

Figure 79-9 Tooth mortality in 442 periodontal patients treated over 10 years. *(Courtesy Dr. R.C. Oliver, Rio Verde, AZ.)*

In a follow-up study, the long-term results of periodontal therapy were evaluated after 15 to 34 years (average of 22.2 years).[4] The average tooth loss at this time was 1.6 teeth per 10 years. Patients were classified into three groups according to tooth loss. Approximately 62% had an average tooth loss of 0.45 per 10 years and were considered "well maintained"; 28% lost an average of 2.6 teeth per 10 years and were considered "downhill"; and 10% lost an average of 6.4 teeth per 10 years and were considered "extreme downhill" (Figure 79-8).

Another study included all patients in a practice who had been treated 5 or more years previously and had received regular preventive periodontal care since that time.[25] The 442 patients had an average of 10.1 years since treatment. Two-thirds of the patients were older than 40 years of age at the time of treatment. These patients had been seen every 4.6 months, on average, for their preventive periodontal care, which consisted of oral hygiene instruction and prophylaxis (Figures 79-9 and 79-10).

The total tooth loss resulting from periodontal disease was 178 of more than 11,000 teeth available for treatment. More important, 78% of the patients did not lose a single tooth after periodontal therapy, and 11% lost only one tooth. Considering that more than 600 teeth had furcation involvements at the time of the original treatment and that well over 1000 teeth had less than half the alveolar bone support remaining, the tooth loss was low. During the same average 10-year period after periodontal therapy, only 45 teeth were lost through caries or pulpal involvement. Even more surprising are the statistics over an average 10-year period for teeth with a less-than-optimal prognosis. Only 85 (14%) of a total of 601

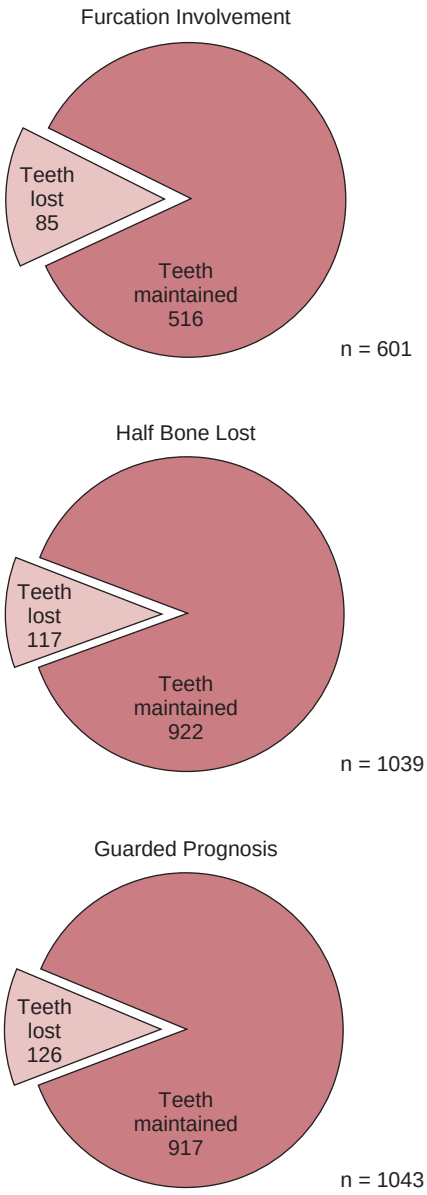

Figure 79-10 Loss of teeth with advanced periodontal disease over 10 years. *(Courtesy Dr. R.C. Oliver, Rio Verde, AZ.)*

teeth with furcation involvement were lost, and 117 (11%) of 1039 teeth with half or less of the bone remaining were lost. Of the 1043 teeth listed as having a "guarded prognosis" for any reason by the clinician performing the initial examination, only 126 (12%) were lost over this 10-year average period. The average tooth mortality rate was 0.72 tooth lost per patient per 10 years.

In a third study in private practice, 600 patients were followed for 15 to 53 years after periodontal therapy (Figures 79-11 and 79-12).[7] The majority (76.5%) had advanced periodontal disease at the start of treatment. There were 15,666 teeth present, for an average of 26 teeth per patient. During the follow-up period (average of 22 years), a total of 1312 teeth were lost from all causes. Of this number, 1110 were lost for periodontal reasons. The average tooth mortality rate per patient was 2.2 teeth; when this is converted to a 10-year rate, an average of one tooth was lost per 10 years in each patient. During this period of observation, 666 teeth with a questionable prognosis were lost out of a total of 2141. This means that 31% of the teeth with a questionable prognosis were

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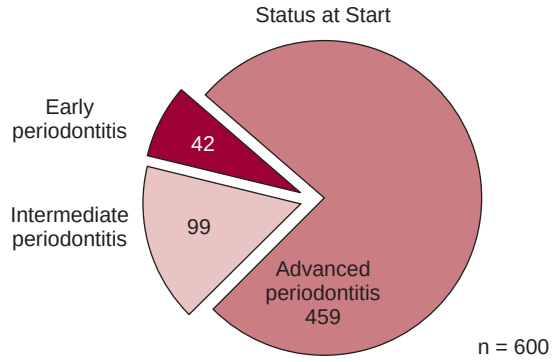

Figure 79-11 Status at the start of a study of 600 patients. *(Data from Hirschfeld L, Wasserman B:* J Periodontol *49:225, 1978.)*

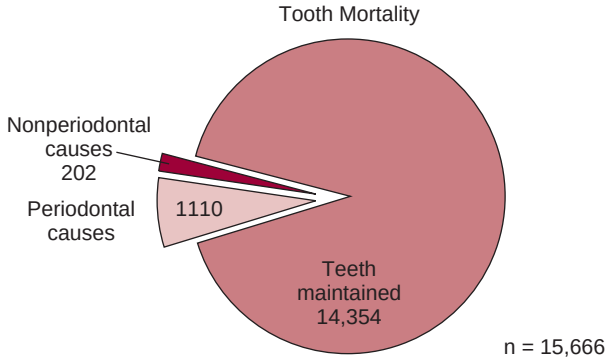

Figure 79-12 Loss of teeth in 600 patients over 15 to 53 years from nonperiodontal and periodontal causes. *(Data from Hirschfeld L, Wasserman B:* J Periodontol *49:225, 1978.)*

lost over 22 years of treatment. A total of 1464 teeth with furcation involvement were treated, and 31.6% were lost during the period of study. Approximately 83% of the patients lost fewer than 3 teeth over the 22-year average treatment period and were classified as "well maintained." The remaining 17% of the patients were divided into two groups: "downhill" (4 to 9 teeth lost) or "extreme downhill" (10 to 23 teeth lost). Thus 17% of the patients studied accounted for 69% of the teeth lost from periodontal causes. This study also showed that relatively few teeth are lost after periodontal therapy. In addition, relatively few of the teeth with guarded prognosis, including those with furcation involvement, are lost, and a small percentage of patients lose most of the teeth.

Three studies give insight into tooth mortality in untreated patients. The studies of Löe et al[19] in Sri Lankan laborers showed that after age 35, an average of 5 and 16 teeth were lost per 10 years in the "moderate progression" and "rapid progression" groups, respectively (Figure 79-13). In a previously discussed study in private practice,[3] an effort was made to find and evaluate patients with diagnosed moderate-to-advanced periodontitis who did not follow through with recommended periodontal therapy. Patients with untreated periodontal disease were losing teeth at a rate greater than 0.61 tooth per year (6.1 teeth per 10 years). A total of 83 teeth were lost in 30 patients, but the investigators excluded one patient who had lost 25 teeth. Including this patient would have increased the tooth loss in untreated patients to an even higher rate. In another study, reporting on patients with moderate-to-advanced periodontitis examined at the Department of Periodontology at the University of Kiel in Germany, Kocher et al[10] found a marked increase in tooth loss in the untreated patients compared with the treated patients when they were examined after 7 years.

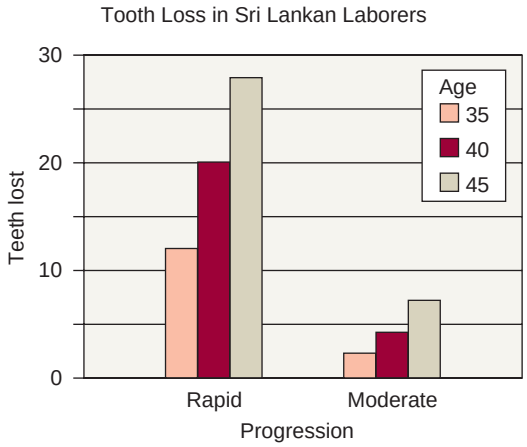

Figure 79-13 Tooth loss in a population with untreated periodontal disease. *(Data from Löe H, Anerud A, Boysen H, et al:* J Clin Periodontol *13:431, 1986.)*

TABLE 79-3 Tooth Mortality in Treated Periodontitis Patients

Study	Average Number of Teeth Lost per 10 Years with Periodontal Treatment*
Hirschfeld and Wasserman[7]	1.0
Kocher et al[10]	1.6
McFall[20]	1.4
Oliver[24]	0.72
Ross et al[30]	0.9
Goldman et al[14]	1.6
McLeod et al[21]	1.5

*Tooth mortality adjusted to 10 years by chapter author.

TABLE 79-4 Tooth Mortality in Untreated Periodontitis Patients

Study	Average Number of Teeth Lost per 10 Years without Periodontal Treatment*
Becker et al[13]	6
Kocher et al[10]	5
Löe et al[18] (moderate progression)	5
Löe et al[18] (rapid progression)	16

*Tooth mortality adjusted to 10 years by chapter author.

When Tables 79-3 and 79-4 are compared, it is obvious that tooth mortality is much greater in untreated groups.

SUMMARY

The prevalence of periodontal disease and the resulting high rate of tooth mortality have increased the need for effective treatment. Strong evidence now indicates that periodontal disease can contribute to numerous health problems, including pregnancy complications, heart disease, stroke, and diabetes.[23,24,32,33] Available treatment is effective in preventing periodontal disease and stopping the progression of bone destruction after periodontitis is present. In addition, overwhelming evidence suggests that

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Gingivitis can be reversed by using initial therapy coupled with adequate plaque control techniques by the patient and subsequent maintenance visits every 4 to 6 months. Untreated periodontitis can cause rapid progression of attachment loss in approximately 8% of patients, resulting in tooth loss and significant active periodontal disease by the age of 45. Moderate progression occurs in approximately 81% of patients, but even these will have an average of 7 mm loss of attachment by age 45. A small percentage of patients (about 11%) show little, if any, progression of gingivitis to periodontitis. Clinicians should use this information to apply more aggressive treatment to those that are most susceptible.

Treatment of even advanced periodontally involved teeth with a combination of nonsurgical and surgical therapy with appropriate plaque control by the patient and maintenance visits every 3 or 4 months in the dental office is remarkably successful. Tooth loss can be expected to be between 1% to 3% over decades of management even with initial periodontal disease levels being very dramatic. Importantly, just a small subset of patients are responsible for much of the tooth loss in treated populations.

If the clinical team and the patient are committed to periodontal health, the overall prognosis for successful periodontal therapy is excellent, and during treatment planning, a more optimistic approach to therapy is generally the best.

The variation in patient response to treatment is related to compliance and to different host immune responses and presence of highly pathogenic anaerobic bacteria in the biofilms. New reliable diagnostic techniques are needed to identify any at-risk patients, and clinicians should be ready to apply new breakthroughs in the assessment of immune capability and presence and numbers of putative periodontal bacteria.

periodontal therapy greatly reduces tooth mortality. Every dental practitioner should be familiar with the philosophy and techniques of periodontal therapy. Failure to diagnose and treat periodontal disease or to make periodontal treatment available to patients causes unnecessary dental problems and tooth loss and places the patient at risk for systemic health problems.

References can be found on the companion Expert CONSULT website at www.expertconsult.com.

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CHAPTER 80

Results of Implant Treatment

Perry R. Klokkevold

CHAPTER OUTLINE

DEFINING IMPLANT OUTCOMES
FACTORS THAT INFLUENCE IMPLANT OUTCOMES
Anatomic Location
Implant Design
Placement and Loading Protocols
Bone Augmentation
Risk Factors
ESTHETIC RESULTS AND PATIENT SATISFACTION
CONCLUSION

The landmark Goteborg study, as well as the replica study at the University of Toronto, set the stage for success and predictability expectations of root-form dental implants. The study, conducted over a 15-year period at the University of Goteborg, Sweden, by P.I. Brånemark and co-workers began in 1965 and concluded in 1980. The results, reported in several articles, defined the concept of osseointegration, described protocols for success, and shared clinical experiences. Perhaps the most significant article from the study, published in 1981, was a paper on osseointegrated implants in the treatment of the edentulous jaw.[2] Briefly stated, the Goteborg study included 2768 root-form implants placed into 410 edentulous jaws in 371 consecutive patients. The data were most often reported in subsets according to the three study phases (initial, developmental, and routine). Those cases treated in the routine period with standardized procedures and an observation time of 5 to 9 years were thought to reflect the potential of the method and were the basis of data reported in that historic publication. This subset consisted of 895 implants placed in 130 jaws. Implant survival was 81% in the maxilla and 91% in mandible. Prosthesis survival (continuous stability) was 89% in the maxilla and 100% in the mandible. The replica study, conducted at the University of Toronto, demonstrated that comparable results could be predictably achieved using the same implant design and treatment protocols.[5,72–74] Together, these studies demonstrated that implant survival of 81% or more and prosthesis survival of 89% or more could be expected in the edentulous patient.

In the decades since the landmark discovery of osseointegration and the documentation of its clinical effectiveness, clinicians have experienced tremendous success replacing missing teeth with endosseous, root-form dental implants in partially edentulous, as well as edentulous, patients.[1,46] Yet, despite the high level of success and long-term predictability, achieving success all the time, in every patient, and in each situation is not 100%. Complications and implant failures do occur.[2,12] Some implants fail to achieve osseointegration, some achieve osseointegration only to lose bone progressively over time leading to failure, and other implants rapidly lose bone and fail in a short time. Just as importantly, some implants may achieve and maintain osseointegration but fail because they do not meet the patient's or clinician's esthetic expectations.

The reporting of implant success varies widely in the literature, which makes defining an absolute implant success rate impractical. This chapter considers implant treatment results in light of the factors that influence implant survival and success. The intent is to outline important aspects that need to be considered in evaluating implant outcomes and to offer the reader guidelines for understanding published results.

DEFINING IMPLANT OUTCOMES

Implant outcomes are reported in a variety of ways in the literature. Various levels of implant success and failure are described in case reports, case series, retrospective studies, controlled studies, and prospective studies. The type of study and method of reporting is decided by the authors and often influenced by the information (data) collected and the study objectives. Each type of study or report has recognized limitations, but because of the tremendous variation that exists in the way individual investigators measure, interpret, and report implant outcomes, differences in the results from one study to another may not be obvious. Some implant outcomes are reported simply as the presence or absence of the implant(s) at the time of the last examination, regardless of whether the implant was in function, suffered from bone loss, or had other problems. This type of assessment is a measure of implant *survival* and should not be confused with implant *success.* In contrast to such an overly simplified assessment, some investigators report implant outcomes using very specific criteria to determine implant *success.*

Implant success is defined by specific criteria used to evaluate the condition and function of the implant. Criteria for implant success have been proposed in the literature but not used consistently. The problem is that a universally accepted definition of implant success has not been established. In the classic definition, Albrektsson et al[3] defined implant success as an implant with

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no pain, no mobility, no radiolucent periimplant areas, and less than 0.2 mm of bone loss annually after the first year of loading.[3] Bone loss in the first year was recognized but it was not defined or quantified as part of the success criteria until later in a separate definition by Roos.[57] The real challenge in comparing reported data from study to study is that authors use different criteria of success in their work. As a result, it is difficult or impossible to make comparisons between studies and drawing conclusions about implant success or failure from data reported in different studies is tenuous.

It is important to recognize that success rates are dramatically affected by variations in the criteria used to define them. In strict terms, if one considers "implant success" to be the outcome without any adverse effects or problems, then the treatment would be performed as planned, implants would remain stable and functioning without problems, periimplant tissues would be stable and healthy, and both the patient and the treating clinician(s) would be pleased with the outcome (Figure 80-1). If such strict criteria are used, implant success rates would be undoubtedly lower than those determined using less stringent criteria. Table 80-1 illustrates the powerful effect that small changes in success criteria have on the reported success rates. The data demonstrates that simply changing the criteria of success to include probing pocket depth (PPD) ≤5 mm to ≤6 mm changed the implant success rates from 52.4% to 62% and from 79.1% to 81.3% in patients with and without a history of periodontitis, respectively.[40] It also shows that implant survival (last column) is quite different than implant success.

"Implant survival," on the other hand, is simply defined as any implant that remains in place at the time of evaluation, regardless of any untoward signs, symptoms, or history of problems. Clearly, there is a difference between implants that are present, healthy, and functioning under an implant-retained restoration, implants that are present but suffering from periimplant bone loss, or implants that are present but not connected to any restoration and not functioning (Figure 80-2). Implants that are osseointegrated but not restored into function are referred to as "sleepers" and should not be considered successful merely because they are present and osseointegrated. To reiterate, implant survival and implant success are distinctly different outcome measures. As an example, consider the dramatic difference between survival and success rate for implants reported in a systematic review (21 studies included) of implants supporting fixed partial dentures; the reported 5-year implant survival rate was 95.4%, whereas the 5-year patient success rate,* defined as free of any complications, was only 61.3%.[55]

*Individual implant success rates were not reported or estimated.

Figure 80-1 Clinical example of implant success. A single implant was placed in the mandible to replace the lower second premolar (as planned). The implant integrated and was successfully restored in function. Both the patient and clinician(s) are pleased with the outcome. **A,** Clinical photograph of dentition in occlusion (from left side). **B,** Close-up clinical photograph of dentition in occlusion. Mandibular second premolar is an implant supported crown. **C,** Occlusal view of implant supported crown in the second premolar position. Notice that the replacement of the mandibular second premolar with an implant is a conservative treatment, obviating the need to prepare adjacent teeth. **D,** Periapical radiograph of the posterior mandibular teeth and implant in the second premolar position. Bone support is good and bone loss is minimal and consistent with expectations for this implant design.

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TABLE 80-1 Effect of Slight Modifications in Implant Success Criteria on Implant Success Rate Outcomes*

	PPD ≤5 mm No BOP BL <0.2 mm/year (%)	PPD ≤6 mm No BOP BL <0.2 mm/year (%)	PPD ≤5 mm No BOP (%)	PPD ≤6 mm No BOP (%)	Implant Survival (%)
Group A (history of periodontitis)	52.4%	62%	71.4%	81%	90.5%
Group B (periodontal health)	79.1%	81.3%	94.5%	96.7%	96.5%

Based on data from Karoussis IK, Salvi GE, Heitz-Mayfield LJ, et al: *Clin Oral Implant Res* 14:329–339, 2003.
PPD, Probing pocket depth; *BOP,* bleeding on probing; *BL,* bone loss.
*The initial success criteria at 10 years were set at PPD ≤5 mm, no BOP, and BL <0.2 mm annually. The implant success rates for group A (patients with a history of periodontitis) and group B (patients with periodontal health) using this initial criteria is listed in the first column. Notice how dramatically the success rates change when the criteria used to define success are modified to include PPD ≤6 mm (second column). The next two columns show the success rates for each group using PPD ≤5 mm and PPD ≤6 mm when the criteria are modified to omit bone loss as a determinant. The last column shows the implant survival rate for each group, which clearly demonstrates that survival rate is different than success rate.

Figure 80-2 Panoramic radiograph of an individual with an implant placed in the position of a missing maxillary right central incisor. The implant appears to be placed in a nonrestorable position. It is osseointegrated and could technically be counted as a "surviving" implant but must be considered a failure because it does not meet the intended goal and has not been restored into function.

Defining implant results in absolute terms is difficult and confusing. Implant survival, which is almost always reported in studies, may actually overestimate implant outcomes. Implant success, on the other hand, which is less often reported, could be a better measure of actual implant outcomes if specific success criteria were universally defined, accepted, and used. Currently, implant success is difficult or impossible to compare across studies because of differences in evaluation criteria used by investigators. Implant success in a single study (or series of studies that use the same success criteria) is meaningful only in the context of that study (or series). At present, the usefulness of implant success rates from multiple different studies is limited. Implant survival is important but is only an indicator of surviving implants and does not reveal whether implants are functioning or whether any problems were associated with them over time.

FACTORS THAT INFLUENCE IMPLANT OUTCOMES

Many factors influence implant outcomes, including available bone, implant design, placement and loading protocols, and host-related factors. Some of the factors thought to affect implant outcomes are briefly discussed here.

Anatomic Location

Osseointegration depends on the availability of an adequate quantity and quality of bone at the implant site(s). Areas with abundant bone volume in the desired location will be better than areas with deficient bone volume. Likewise, areas with good bone density provide more predictable outcomes than areas with poor bone density. The bone classification system described by Lekholm and Zarb[47] was intended to define bone sites that would have different levels of bone support for implants and that would likely impact survival and success. The quality of bone support is greatly influenced by the anatomic location, thus implant outcomes are sometimes categorized according to anatomic location. Extreme examples are the anterior mandible and the posterior maxilla. The anterior mandible typically consists of dense cortical bone, which offers great support and high bone-to-implant contact, whereas the posterior maxilla is often limited in volume because of alveolar resorption and sinus pneumatization and typically consists of a loose, trabecular structure and a thin cortical bone shell.

Implants placed in the posterior maxilla are less well supported compared to implants placed in the anterior mandible. Jaffin and Berman[34] demonstrated the significance of bone quality on implant survival in a report of 1054 Brånemark implants. Ninety percent of these implants were placed in type I, II, and III bone with only 3% failures. However, of the 10% that were placed in type IV bone, 35% failed.[34] Excluding those placed in the mandible, 23/52 implants placed in type IV bone in the maxilla failed for a dismal 56% survival rate (44% failed). They concluded that bone quality stood out as the single greatest determinant of implant loss.

Anatomic location appears to have a significant effect on implant outcomes. This is particularly true for the posterior maxilla. It should be recognized that the implants used in this study were machined surface implants and that current implants with altered microtopography are expected to perform better in the posterior maxilla. Not surprisingly, when all 1054 implants are considered together, the combined implant survival rate (93.9%) in this study falls within (or slightly higher than) the range of reported survival rates of implants in the Goteborg study (with similar implants).

Implant Design

One of the many factors thought to influence implant outcomes is implant design. Today, there are hundreds of companies that manufacture and market dental implants worldwide and the number continues to grow. Implant dimensions, geometries, and surface characteristics vary tremendously and continue to evolve as innovation and research findings pave the way to changes that are thought to improve outcomes. Very few of these implant designs have been studied and rely on being clinically similar to the other researched and approved implant designs without studies or documentation

to support their effectiveness. Thus, in most cases, it is impossible to appreciate the effect of particular implant design features on implant outcomes. Nonetheless, these new, different implant designs are being used and outcomes are being reported in studies that evaluate other aspects of treatment (i.e., not specific to the implant design) only to make comparisons and assessments more confusing and difficult.

Those studies that have documented the success of dental implants based on implant design characteristics have shaped some of the tenets for current implant standards, selection, and use. For example, many clinicians adhere to the premise that longer implants are better, threaded implants are better, rough-surfaced implants are better, etc. Over time, as changes in design and utilization evolved, some beliefs have been disproved, and continued advances and research will also refute other firmly held beliefs.

What are the best implant design characteristics? Is there an implant design that performs better than the rest? Actually, it is nearly impossible to determine which implant design characteristics are important because there are many variables to consider and implants are successful most of the time. Given the generally high success, it seems that subtle differences in implant design are probably not significant for the majority of patients and situations.[22,26] However, in compromised patients, inadequate sites, or challenging circumstances, certain implant designs may perform better than others. A prime example is the effect of implant surface characteristics. Implants with an altered surface microtopography (i.e., acid-etched, blasted, etc.) have been shown to enhance the bone-to-implant interface[42,43] and are thought to improve implant outcomes, especially in compromised sites.[66,67] Lower success rates have been associated with smooth surface (i.e., machined) implants.[35,56,60] Implant length is another consideration. Implant length is a factor that has long been held as important for success (i.e., longer is better), and many studies have supported this dogma,[61,62] but recent studies are challenging it.[29]

Placement and Loading Protocols

The traditional implant placement protocol required a healed edentulous ridge into which implants were placed and allowed to osseointegrate for a period of time without occlusal loading (see Chapter 71). The nonloading period after implant placement was empirically determined to be 3 to 4 months in the mandible and 6 months in the maxilla.[10] A strongly held belief at the time was that early loading would lead to higher failure rates.[11] In contrast to these early standards, some current protocols advocate dramatically different approaches, including implant placement immediately after tooth extraction and implant occlusal loading immediately or shortly after placement. Each of these approaches has distinct advantages, but they are also accompanied by challenges that have the potential to adversely affect implant outcomes.

Immediate Implant Placement. The immediate implant placement protocol describes the procedure in which an implant is placed in an extraction socket after tooth removal and socket debridement. This procedure, originally described by Schulte et al[64] and later by Lazzara,[45] has been reported with survival rates equivalent to that of implants placed into healed ridges.[14,49] The advantages of immediate placement include decreased surgery, cost, and healing time.[65] Immediate implant placement introduces additional risks of complications, including poor implant position, compromised esthetic outcome, and implant failure (see Chapter 77). Despite the increased risks, high long-term (1- to 16-year) survival rates of 96% have been reported for implants immediately placed into extraction sockets.[69]

Immediate Occlusal Loading. Brånemark established the concept of the osseointegrated dental implant as a predictable modality of treatment for the edentulous patient based on the empirically based requirement that the implant remain submerged and unloaded for a healing period of 3 to 6 months.[10] This original protocol, requiring the implant to remain stress free, was based on the concern that premature loading would cause micromotion of the dental implant leading to fibrous encapsulation and implant failure. However, studies have shown this assumption to be incorrect, demonstrating that immediately loaded implants can achieve success rates (>90%) similar to conventionally loaded dental implants.[15,18,27,31,38] The long-term predictability of immediately loaded implants requires strict surgical and prosthodontic protocols.[7]

Bone Augmentation

A common problem encountered in implant dentistry is insufficient bone quantity to allow implant placement. Deficiencies in alveolar bone result from developmental defects, periodontal disease, tooth loss, and trauma.[6,13,63] For most cases with alveolar ridge resorption, bone regenerative procedures are required to correct the defects before or simultaneous with implant placement (see Chapters 72 and 73). The obvious question is whether implants placed in sites that are reconstructed with bone augmentation procedures will achieve the same survival and success rates as implants placed in native bone sites.

The results of implants and bone augmentation procedures reported in the literature have been assessed by expert clinicians in several workshops.[16,28,32,33] One systematic review of literature (2003 Workshop on Contemporary Science in Clinical Periodontics), including 13 studies (guided bone regeneration) with 1741 patients and 5 studies (distraction osteogenesis) with 92 patients, found that survival rates of dental implants in augmented bone achieved a high level of predictability that was similar to implants placed in natural bone.[28] A more recent systematic review of the literature (the 2008 Consensus Report of the Sixth European Workshop on Periodontology) pointed out that the bone augmentation procedures can fail and that implants placed in these areas do not enjoy the high long-term survival rates of dental implants placed in pristine sites.[68] The 2003 systematic review did not include an assessment of whether bone augmentation procedures failed, rather it intentionally focused on implants placed in sites that were successfully treated with bone augmentation. More research is needed to determine the long-term performance of dental implants placed in augmented bone and the clinical benefits of bone augmentation with respect to alternative treatments (i.e., the use of short implants).

Risk Factors

In general, most patients enjoy a similar rate of survival and success with dental implants. It is a minority of patients who experience implant failure. In addition to factors previously discussed, host-related factors may adversely affect healing, osseointegration, and maintenance of dental implants. Smoking, diabetes, and periodontitis have been identified as "risk factors" that may adversely affect implant outcomes. In a systematic review of the literature, Klokkevold and Han[41] evaluated the influence of smoking, diabetes, and periodontal disease on implant outcomes and found that smoking has an adverse affect on implant survival and success, with the effects being more pronounced in areas of loose trabecular bone (i.e., the posterior maxilla). The review also suggested that type 2 diabetes may have an adverse effect on implant survival rates, but the limited number of included studies did not permit a definitive

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conclusion.[41] Finally, their review also concluded that while patients with a history of treated periodontitis did not show any decrease in implant survival, they did experience more implant complications and lower success rates.[41]

Smoking. Above all other risk factors, smoking has been shown to have a significant negative impact on implant survival and success. In a study of 2194 implants, Bain and Moy reported a significantly greater rate of failure in smokers (11.28%) compared to nonsmokers (4.76%).[8] De Bruyn and Colleart reported an early failure rate of 9% in smokers compared to 1% in nonsmokers.[17] Two other studies, one of 10 years[23] and the other with follow-up ranging from 6 months to 21 years,[53] concluded that smoking was a definite risk factor for implant survival.[19,20]

Diabetes. The role of diabetes mellitus as a risk factor for implant outcomes is less clear. Although it is expected that a metabolic disease such as diabetes would have an adverse affect on bone healing[70] and tissue support for implants, the research has not definitively determined that diabetes has a negative impact on implant survival or success.[4] Moy and co-workers[53] reported a significantly lower patient* success rate (68.7%) for patients with diabetes compared to the patient success rate (85.1%) for the entire study population (1140 patients with 4680 implants). However, this low success rate (high failure rate) may be an overestimate of the actual implant failure rate for patients with diabetes because it counts the number of patients with implant failures regardless of how many implants they had (i.e., implants that may have been successfully inserted and maintained in these patients are not counted). The number of patients with diabetes included in the study was 48/1140 or 4.2% of the total. Of these, 15 patients experienced implant failure(s). Conversely, in a systematic review of the literature (33 studies), Javed and Romanos found that patients with good metabolic control (hemoglobin A1c in normal range) achieve similar success with osseointegrated implants as patients without diabetes.[36] Dowell and co-workers[21] also found similar success for implants placed in patients with controlled diabetes.

Periodontitis. There are a limited number of studies available to assess the prognosis of implant treatment in patients with a history of periodontitis.[39] Most of these studies suggest that implants are equally successful in patients with a past history of chronic periodontitis. Short-term studies demonstrate 90% to 100% implant survival in patients with a history of chronic periodontitis.[50,52] Long-term studies report 90% to 97% implant survival rates for patients with a history of chronic periodontitis.[40,48,58,71] Short-term implant survival rates for patients treated for aggressive periodontitis are 95% to 100%.[50,51] One long-term study reported an 88.8% implant survival over 5 years for patients treated for aggressive periodontitis.[52] Implant survival in patients with a history of periodontitis appears to be highly predictable. However, the lack of long-term studies to support implant survival in patients treated for aggressive periodontitis leaves the prognosis in these patients open to question.[39]

Long-term studies evaluating implant treatment in periodontally compromised patients suggest that they may experience more periimplant problems.[40] When these patients are followed for extended periods of time, there appear to be more complications (i.e., periimplantitis) associated with implants compared to periodontally healthy patients. In a 10-year prospective study of patients with or without a history of chronic periodontitis, biologic complications (i.e., periimplantitis) was higher for those with a history of chronic periodontitis (28.6%) compared to those with periodontal health (5.8%).[40] This controlled study by Karoussis and co-workers[40] found a statistically significant difference in mean periimplant bone loss between patients with a history of chronic periodontitis and periodontally healthy patients. These periimplant problems may be attributed to a continuous increase in the percentage of implants exhibiting probing pocket depths ≥4 mm over time.[24]

*Individual implant survival or implant success rates were not reported.

ESTHETIC RESULTS AND PATIENT SATISFACTION

The ultimate test for implant outcomes is achieving natural appearing, as well as optimally functional, implant-supported tooth replacements. Proper tooth dimensions, contours, and ideal soft tissue support are key factors for successful esthetic outcomes.[44] If crown form, dimension, and shape and gingival harmony around the implants are not ideal, the patient may consider the implant restoration unacceptable because the result does not represent a natural dental profile (Figure 80-3). For some patients, such as those with severe alveolar deficiency, an ideal esthetic outcome may be impossible because reconstructive surgical procedures are complex, require extensive time, and remain unpredictable. For others, a "less than ideal" esthetic outcome may be perfectly acceptable (see Chapter 74).

Esthetic problems and patient dissatisfaction happen when results are inferior to what was expected. Patient satisfaction with the esthetic outcome of the implant prosthesis varies from patient to patient, depending on a number of factors. The risk for esthetic failure is increased for patients with high esthetic expectations. The risk is also higher when patients present with patient-related risk factors such as a high smile line, thin periodontal soft tissues, and compromised bone support. Patient perceptions and desires greatly influence and determine how well an individual patient accepts the implant outcome.

Figure 80-3 Clinical photograph of maxillary anterior fixed restoration supported by two malpositioned implants in the central incisor positions. The patient is dissatisfied with the esthetic outcome. The left implant is positioned between the central and lateral incisor and angled toward the facial surface at a level above the gingival margin. A tooth-colored material was used to mask the exposed framework in the gingival area.

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SCIENCE TRANSFER

Successful implant therapy has many varied criteria. In some studies, implant survival was considered a success and in these cases, success rates were over 90%. However, in contrast to a tooth, an implant can lose a high proportion of its bone support, but if there is still osseointegration at the apical part of the implant, it may remain in function without symptoms. If the presence of unfavorable responses to implants, such as bone loss, periimplantitis, and pocket depth, are considered, the success rates will be lower than that associated with survival.

Clinicians should be aware that unfavorable responses and implant loss are more common in the type IV bone found in the posterior maxilla compared to the denser bone seen in the anterior mandible. Implants placed in bone augmentation sites have an increased complication rate compared to implants placed in native bone, but the survival rates are similar.

Smoking and uncontrolled diabetes are associated with higher failure rates and a higher incidence of complications, thus these patients need to get control of their diabetes and stop smoking to optimize implant therapy.

New generation implants with roughened surface topology have better osseointegration than the machined surface implants, and clinicians can expect higher success and higher survival using a variety of improved surface technologies.

TABLE 80-2 Patient Satisfaction with Implant Treatment*

Implant Experience	Highly Satisfied or Satisfied (%)
Function/chewing	97
Phonetics	96
Esthetics	97
Oral hygiene ease	93
Complete fulfillment	92
Do treatment again	94
Recommend to friend or relative	89
Reasonable/justified cost	87

Based on data from Pjetursson BE, Karoussis I, Burgin W, et al: *Clin Oral Implants Res* 16:185–193, 2005.

*This table lists the percentage of patients subjectively responding with highly satisfied or satisfied to survey questions about their implant experience. More than 90% responded favorably and felt that implant treatment was positive.

Although infrequently reported, the "esthetic success" and "patient satisfaction" of implant therapy needs to be included when considering the results of implant therapy. There have been a number of proposed methods to evaluate esthetic results, yet the reporting of esthetic parameters in the scientific literature is scarce.[9] There is a restorative index that appraises the "white" esthetics of the final restoration,[37] a soft tissue or "pink" esthetic score that considers soft tissue parameters,[30] and an esthetic index that uses a combination of pink and white esthetic scores focusing on the visible part of the implant restoration.[8] These indices are aimed at quantifying the esthetic result, which could then present an objective method of judging "implant esthetic success."

In a survey of patient satisfaction, more than 90% were completely satisfied both in terms of function and esthetics.[54] The study consisted of a questionnaire given to 104 patients 5 to 15 years (mean = 10.2 years) after implant placement to assess their subjective perception of implant treatment. Of these patients, 48% were treated with single implant crowns and 52% were treated with fixed partial dentures. The survival rate for all implants was 93%. A great majority of patients had favorable responses to the questions regarding function, esthetics, hygiene and cost. Table 80-2 lists the percentage of patients responding with highly satisfied or satisfied to each category. Comparing chewing comfort for teeth or implants, 72.1% perceived no difference, 17.3% felt more secure chewing on teeth, and 7.7% felt more secure chewing on implants.

In another survey study, patient satisfaction with implant-supported prostheses in totally edentulous jaws was evaluated.[59] Patient experience with an implant-supported prosthesis was over a period of 10 years. Most of the 135 patients (97%) reported overall satisfaction with treatment. Chewing satisfaction was reported as good or very good by all but one patient (99.3%). Improved lifestyle and greater self-confidence in public was reported by 75% and 82% of patients, respectively.

A systematic review of the literature including all randomized controlled trials published in English or French up to April 2007 comparing mandibular conventional dentures and implant overdentures in adult edentulous patients identified eight publications for metaanalysis.[25] The study reported that patients were more satisfied with implant overdentures when compared with conventional mandibular dentures. However, there was a lack of evidence to show a patient's perception on the impact of mandibular implant overdentures on general health.

CONCLUSION

The utilization of dental implants to replace missing teeth is highly predictable, advantageous, and beneficial for patients. Because of variations in implant design, study protocols, and populations studied, results are difficult to compare and an absolute definition of implant success is elusive. The results of implant treatment are reported using a wide range of criteria from simply being present (survival) to being in function without complications (success). It is challenging to compare the results of one study to another because of the number of variables that continue to change. Clinical research suggests that certain risk factors may decrease success rates for some patients. Understanding what is being reported in the literature will help readers to appreciate implant treatment results.

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