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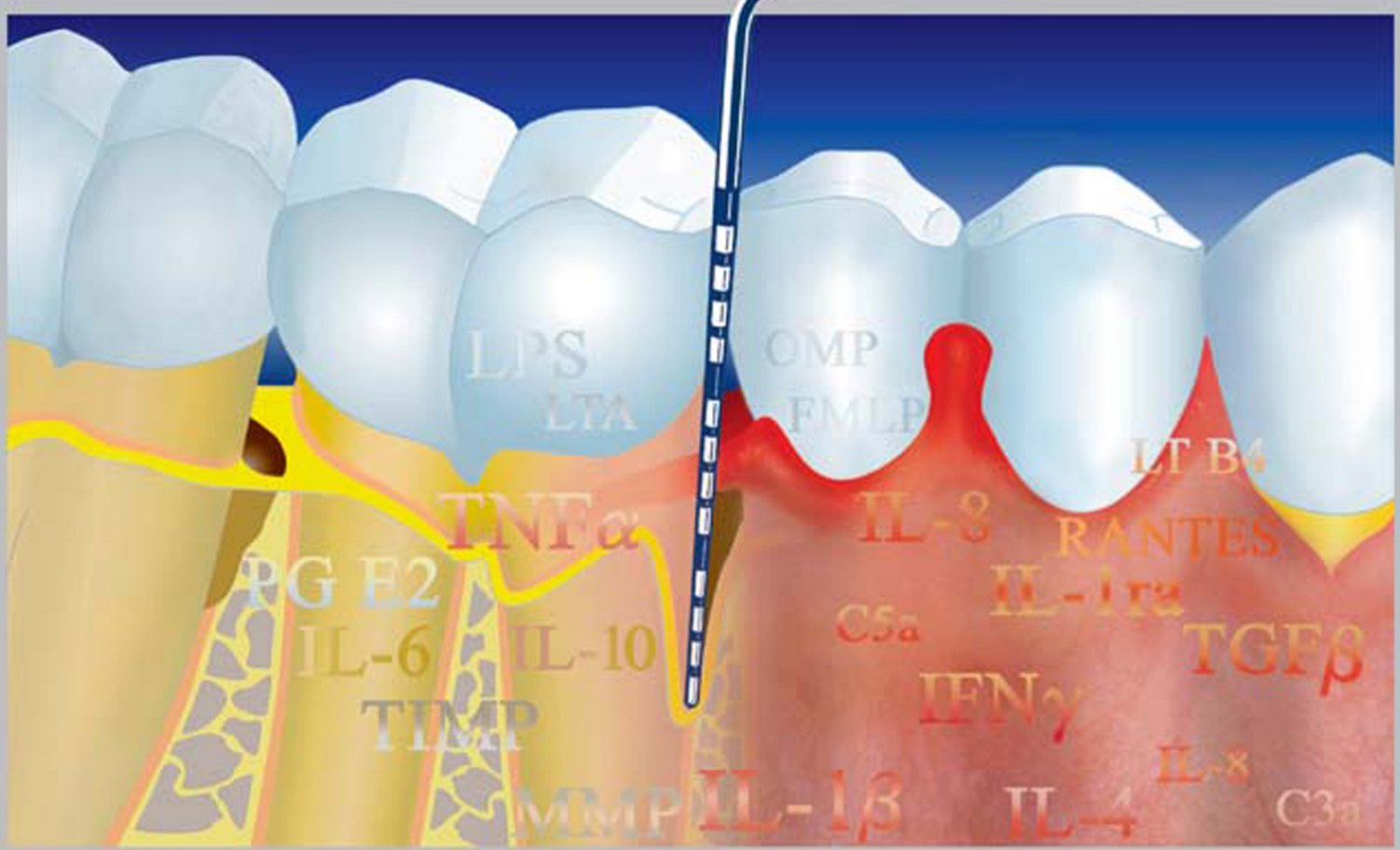
Editors:
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Periodontology

Herbert F. Wolf
Edith M. & Klaus H. Rateitschak
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Forewords by Samuel B. Low and Maurizio S. Tonetti

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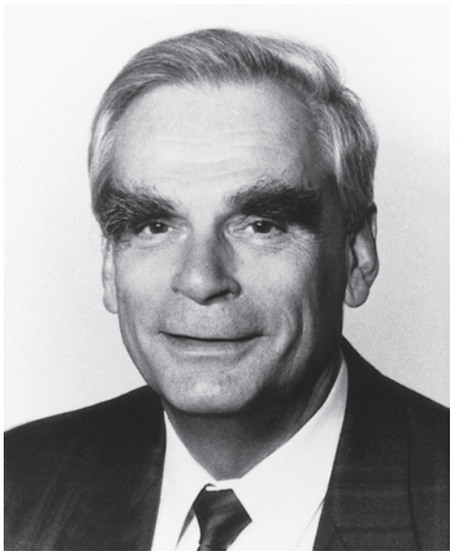
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Klaus H. Rateitschak
in memoriam

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3rd revised and expanded edition
1870 illustrations

Thieme
Stuttgart · New York

http://freedentaleducation.blogspot.com

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همیار دندانسازان و دندانپزشکان

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Library of Congress Cataloging-in-Publication Data

Periodontology, English
Color Atlas of Dental Medicine /
editors, Klaus H. Rateitschak and
Herbert F. Wolf; forewords by Roy
C. Page and Hubert E. Schroeder.—
2nd ed. Adaption of: Parodontologie,
2nd, 1989.
Rev. ed. of: Color Atlas of Periodontology /
Klaus H. Rateitschak ... [et al.] 1985.
Bibliography: p. Includes Index.
Contents: v. I. Periodontolgy /
Klaus H. Rateitschak ... [et al.]
1. Dentistry—Atlases.
I. Rateitschak, Klaus H. II. Color
Atlas of Periodontology, III. Title.
[DNLM; 1. Periodontics—Atlases.
WU 17 P257c]
RK287.P3713 1989
617.6'320754-dc20
DNLM/DLC
for Library of Congress

1st German edition 1984
1st English edition 1985
1st French edition 1986
1st Spanish edition 1987
1st Japanese edition 1987
1st Italian edition 1988

2nd German edition 1989
2nd English edition 1989
2nd Spanish edition 1991
2nd French edition 1991

3rd German edition 2004

© 1985, 2005 Georg Thieme Verlag,
Rüdigerstraße 14, 70469 Stuttgart,
Germany
http://www.thieme.de
Thieme New York, 333 Seventh Avenue,
New York, NY 10001 USA
http://www.thieme.com

Typesetting by primustype Hurler GmbH,
Notzingen
Printed by K. Grammlich, Pliezhausen

ISBN 3-13-675003-9 (GTV)
ISBN 0-86577-902-3 (TNY) 1 2 3 4 5

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Preface to the Third Edition

This Third Edition of the *Color Atlas of Periodontology* now emerges, more than 13 years after the Second. The reasons for the long delay are many, but the saddest was the passing of our colleague and friend, and co-author Edith's husband, Klaus Rateitschak, on September 30, 2002. We dedicate this Third Edition to him, an edition on which he worked intensively, up to the very end, and in which he found immense joy and satisfaction in the almost innumerable innovations that the 1990s brought to our profession, while launching it into the 21st Century.

The turn of the century presented to us the opportunity to freshly analyze what had been achieved, and to define new goals for the future. Dentistry and especially periodontology received fresh and fervent impetus to integrate new and relevant scientific and clinical knowledge into our daily professional practices, including even total paradigm shifts. These facts in themselves presented sufficient basis for us to totally revise the Second Edition, maintaining of course those concepts and treatment modalities that have stood the test of time, but incorporating exciting new findings that have immediate clinical applicability.
The most noble task of a dentist—also the watchword of our now departed colleague—is to maintain the health and well-being of the patient. This translates in our profession into the prevention of oral diseases.

However, if, despite our best efforts in prevention, damage to tooth structure and the periodontium occur, treatment of the periodontal condition must be the fundamental endeavor, even before any further dental therapy. Each and every restorative effort—from the smallest filling through removable partial dentures and to complex fixed bridge constructions, regardless of whether these are tooth-borne or implant-borne—must be undertaken only when the supportive periodontal tissues are inflammation-free and healthy.
Inflammatory periodontal diseases today represent some of the most widespread health problems in the world. The effects of periodontal diseases upon systemic health are becoming clearer almost daily. To ignore these now obvious associations is quickly becoming unacceptable.

Because of this fact, the following chapters or sections have been rearranged, expanded, sometimes shortened, or in many cases completely rewritten:

- An introduction for the "layperson," the patient
- Etiology—Dental plaque as a "biofilm"; the periodontopathic microorganisms
- Pathogenesis—Host response, risk factors
- Oral pathological changes in gingiva and periodontium
- Oral manifestations of HIV-disease. Treatment possibilities
- Gingival recession—"Prevention before surgical treatment"
- The "systemic pre-phase" of periodontal therapy
- Non-surgical, "closed" periodontal therapy—Promising new techniques
- Pharmacologic strategies for the treatment of periodontitis
- Surgery—Principles of access flaps for "open" root debridement
- Regenerative and resective surgical methods
- Furcation involvement—Classifications and treatment options
- Esthetic surgery—Mucoplastic operations
- Risk-managed maintenance therapy—Success and failure
- Periodontics and prosthetics—Standard and esthetic possibilities
- Ridge augmentation using soft tissue
- Indication for implants in the periodontally treated dentition
- Periodontal tissues in the elderly
- Comprehensive classification of periodontal diseases—Original 1999 text, AAP/EFP

As always before, every effort was made in this Third Edition to fully integrate the text and the illustrations, to make it pleasant (or at least easier) for the reader as she/he studies or simply peruses the book. Nevertheless, the comprehensive nature and thus the very weight of this new treatise, this Third Edition, have essentially removed it from the ranks of a bedside reader. For this we apologize!

Herbert F. Wolf, Zürich
Edith M. Rateitschak-Plüss, Basle
Thomas M. Hassell, Flagstaff

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Foreword to the Third Edition—I

Samuel B. Low, D. D. S., M. Ed.

Professor of Periodontology
College of Dentistry, University of Florida

Diagnosis and treatment of the diseases of the periodontium remain as one of the critical components of the practice of dentistry. However, implementation of periodontal therapy as a component of comprehensive oral care remains today a significant challenge. One major aspect of understanding contemporary concepts of periodontology is locating an appropriate resource that contains the time-proven fundamentals of therapy while providing the recipient with cutting edge, new information. The dental practitioner community continues to seek clarity in a discipline that moves into the 21st Century with lightening speed. Moreover, today's clinician—whether it be the dental student or the seasoned dentist—desires information in a format that is time-effective, not verbose, and visually appealing. This Third Edition of the *Color Atlas of Periodontology* bears a title that could be considered by some to be a misnomer, for it is far from merely a collection of illustrations, diagrams, and clinical photographs. There exists also a more than adequate text, whereby passages and illustrations blend together in a synergistic fashion and with harmony.

In recent decades, periodontology has benefited from a mainstay of infusion of clinical and laboratory science into a discipline whose goal is *retaining* the dentition. This Third Edition contains very comprehensive sections in the area of microbiology, and more importantly host defense mechanisms in the subject of pathogenesis. When one peruses these respective chapters, it becomes evident that the text is illustrated in such a clear manner that what can be difficult in comprehension can be readily grasped in understanding. Terminology and definitions are current and in alignment with today's published concepts. Serious attention is given to data collection and diagnosis. There are accurate descriptions of current diagnostic tests, whether they are from a microbiologic venue or in a host response category. What is particularly gratifying is a strong section in oral pathological alterations of the periodontium. The current and future scope of periodontology has as its domain *all* of the oral cavity, and especially as it relates to the patient's systemic health.

However, the most comprehensive section is that of therapy, where all phases of surgical and non-surgical treatment are covered in all facets. While most authors would abhor the similarities of a "cook book," the practitioners who desire to expand their repertoire will be delighted with the attention to detail. And while the accepted concept is that textbooks are already "history," the therapy sections defy this concept, including areas involving mucogingival procedures that are very current, even progressive. Few texts can compare with the quality of illustrations superimposed upon the truly excellent intraoral photography. One also notes that all procedures are presented with objectivity and with no predilection to any particular philosophy. References to products are handled in a manner that allows the clinician to make unbiased choices.

Today's clinician must be knowledgeable in esthetic dentistry. The reader will find a seamless integration of periodontal concepts with the discipline of prosthodontics. Well documented cases provide a clear understanding of what can be achieved in the perio/restorative esthetic environment. For the restorative dentist, step-by-step procedures are presented in the areas of basic biologic width, enhancement of interdental papillae, and dealing with alveolar ridge defects.

In summary, few practitioners will find a more all-inclusive text on periodontology that will appeal to all levels of clinicians, dental students, and periodontics graduate students. The format is very user-friendly, with intent to provide the reader with a comprehensive reference in a time-effective manner. I suggest this Third Edition as required reading for all who are engaged in rendering periodontal therapy.

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Foreword to the Third Edition—II

Maurizio S. Tonetti, D. M. D., Ph. D., M. M. Sc., F. D. S., R. C. P. S.

Professor of Periodontology
Eastman Dental Institute, University College London

The *Color Atlas of Periodontology*, first published in 1984, has come to its third, thoroughly revised and extensively expanded edition, being a cherished classic in the library of practitioners, students, and teachers of this discipline. Having used the First Edition as one of the reference textbooks during my specialist training, and having "borrowed" many diagrams from the Second Edition to illustrate difficult concepts to my students, it is with particular pleasure that I write this Foreword to the new edition of the *Color Atlas of Periodontology*.

This new edition represents both an element of evolution and of revolution with respect to the previous ones. Evolution since the authors have expanded and brought to perfection what has rendered the *Atlas* a true classic: The clarity of a synthetic up-to-date discussion of the critical topics in periodontology based on text, diagrams, and generous, detailed illustration. It is the ideal media to capture both theoretical and clinical concepts in an effective, time-efficient manner. The *Atlas* continues to be a didactic masterpiece making both scientific discovery and clinical progress accessible.

The *Atlas* is also an element of revolution since the authors have tried to implicate in clinical practice the paradigm changes that have originated from the last 20 years of fundamental and clinical research. It is this attempt that makes this book particularly attractive: As progress in the science of periodontology steamed along, the gap between this and the reality of practice has grown ever wider. The *Atlas* offers students and clinicians the opportunity to reflect on the profound changes that have occurred and illustrates concise and practical approaches to *use* the new and emerging knowledge for the benefit of periodontal patients today.

Periodontology has reached a critical turning point: A better understanding of the etiology and pathogenesis of the different forms of periodontitis and the recognition of the critical role played by individual susceptibility traits have laid the foundations for new, more effective treatment strategies. Better diagnosis and risk assessment, new treatment alternatives, an unprecedented body of scientific evidence in support of what we should do (and perhaps more importantly what we should no longer do) will be useless if they do not find their way into daily clinical practice.

It is therefore my wish that the readers of this work will be open to the changes in their daily practice that current research in periodontology has to offer.

My thanks go the Wolf/Rateitschak/Hassell team for another masterful contribution.

Bravo and Encore!

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Acknowledgments

First and foremost, we thank our families and our friends for their unflagging encouragement and support, and for their deep understanding and even deeper patience over more than a decade of work as this Third Edition evolved and became a reality. I, Herbert Wolf, personally take this opportunity to thank Klaus "Pascha" Rateitschak who, years before his death, honored me with the knowledge that I would be first author on this completely revised Third Edition of our *Atlas*. Pascha and I became friends and collaborators in the early 1960s, when we started our careers under the mentorship of Dr. Hans R. Mühlemann, scientist and teacher at the University of Zurich.

The creation of this book, which became ever more comprehensive and detailed, was made possible only through the collaboration and assistance of the coworkers in Dr. Wolf's private dental practice (Zurich, Switzerland), our colleagues in the Center for Dental Medicine (Basle, Switzerland), and the faculty, staff, and students at the College of Health Professions, Northern Arizona University (Flagstaff, AZ). In our work, we were helped greatly by contributions from other universities and many private dental and periodontal practices. Contributions of clinical cases and photographs are acknowledged in detail on page 524.
We also thank especially *Dr. Samuel Low* (Gainesville, Florida) and *Dr. Maurizio Tonetti* (London, England) for their generous, original, and objective new forewords to this Third Edition.

Our thanks go to our many colleagues worldwide for their participation as contributors, assistants, photographers, therapists, problem-solvers, and "critics": *Andreas Adler, Basle—Sandra and Christian Augustin-Wolf, Zurich—Christine Baca, Flagstaff—Manuel Battegay, Basle—Jean-Pierre Ebner, Basle—Daniel Edelhoff, Aachen—Joachim Hermann, Zurich—Markus Hürzeler, Munich—Marco Imoberdorf, Zurich—Thomas Lambrecht, Basle—Niklaus Lang, Berne—Samuel Low, Gainesville—Pascal Magne, Geneva—Michael Marxer, Lucerne—Carlo Marinello, Basle—Martin Meyer, Zurich—Jürg Meyer, Basle—Andrea Mombelli, Geneva—Rainer Oberholzer, Suhr—Lucca Ritz, Basle —Hubert Schroeder, Zurich—Ulrich Saxer, Zurich—Peter Schüpbach, Horgen—Stephan Studer, Zurich—Maurizio Tonetti, London—Anton Wetzel, St. Gallen—Lennart Wieslander, Basle—Jakob Wirz, Winterthur—Nicola Zitzmann, Basle.*

All of the graphic illustrations, schematic diagrams, and tables conceived by Herbert Wolf were realized on the computer with great competence (and understanding for the authors' unending litany of desires) by *Joachim Hormann*, Graphic Design Co., Stuttgart. He deserves our special thanks. Also *so* worthy of our thanks and praise are Ms. *Censeri Abare* (Gainesville, FL) and Ms. *Heidi Hamberger* (Basle), who prepared the original typescript for this book and then also performed countless corrections and modifications of innumerable drafts, always with precision and with patience.

Since the initiation of the First Edition of this *Atlas* (1978), *Dr. Christa Durach* and photographer *Dieter Isch* have stood by our side faithfully and with critically important contributions. Our deepest thanks go to these two outstanding individuals.

The enormous costs for all of the color illustrations were born by the authors and by our publisher, and the following commercial entities made significant financial contributions:

Procter & Gamble Co., Trisa Co., Deppeler Co., Gaba Co., Lever Co., and the *Walter-Fuchs Foundation.*

Our text and illustrative materials were prepared for final printing in the *Kaltnermedia* Co. in *Bobingen*, by *Martin Maschke, Markus Christ,* and *Angelika Schönwälder*, who performed brilliantly with understanding, know-how, and enduring patience. Despite the ever-present time pressure, *Grammlich* Co., *Pliezhausen,* carried out the work with precision and persistence.

Once again, in this Third Edition, we have been immensely grateful for the excellent guidance and support provided to us by Thieme Medical Publishers, Stuttgart and New York. Special thanks are due to *Dr. Christian Urbanowicz, Markus Pohlmann, Karl-Heinz Fleischmann* and his successor *Rolf Zeller*. These individuals put every ounce of energy, dedication, and personal creativity into this book, and always exhibited understanding (and patience!) for the authors' numerous and often "impossible" wishes.

Herbert F. Wolf
Edith M. Rateitschak-Plüss
Thomas M. Hassell

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Table of Contents

Fundamentals

1 Introduction
1 Periodontal Diseases
4 The Clinical Course of Untreated Periodontitis
4 Periodontitis—Concepts of Therapy

7 Structural Biology
8 Gingiva
10 Epithelial Attachment
12 Connective Tissue Attachment
14 Root Cementum
16 Osseous Support Apparatus
18 Blood Supply of the Periodontium
19 Innervation of the Periodontium
20 The Coordinated Functions of the Periodontal Structures

21 Etiology and Pathogenesis
22 Periodontitis—A Multifactorial Disease

23 Microbiology
24 Biofilm—Plaque Formation on Tooth and Root Surfaces
25 Supragingival Plague
26 Natural Factors Favoring Plaque Retention
27 Iatrogenic Factors Favoring Plaque Retention
28 Subgingival Plaque
29 Bacterial Invasion Into Tissue?
30 Classification of Oral Microorganisms
31 Cell Walls of Gram-Positive and Gram-Negative Bacteria
32 Periodontitis—Classical or Opportunistic Infection?
33 Putative Periodontopathic Bacteria
34 Virulence Factors
34 Virulence Transfer
36 Marker Bacteria in Periodontitis
37 Pathogenic "Single Fighters" vs. Pathogenic Complexes?
38 Endotoxins—Lipopolysaccharide (LPS)
38 Interaction Between LPS and the Host

39 Pathogenesis—Reactions and Defense Capabilities of the Host
40 New Concepts of Pathogenesis – Therapeutic and Diagnostic Consequences
41 Host Response—Mechanisms and "Participants"
42 Non-specific, Congenital Immunity—the First Line of Defense
43 Specific, Acquired Immunity—the Second Line of Defense
44 Components of the Immune System—Summary
45 Interactions Between Non-specific and Specific Immunity
46 Regulatory Cell Surface Molecules: Markers, Receptors
47 Cytokines
49 Eicosanoids—Prostaglandins and Leukotrienes
50 Enzymatic Mechanisms—Matrix Metalloproteinases
51 Risk for Periodontitis—the Susceptible Host
52 Genetic Risk Factors—Diseases, Defects, Variations
54 Alterable Risk Factors, Modifying Co-Factors
55 Pathogenesis I—Initial Inflammatory Reactions
56 Pathogenesis II—Histology
58 Pathogenesis III—Molecular Biology
60 Attachment Loss I—Destruction of Connective Tissue
61 Attachment Loss II—Bone Resorption
62 Pathogenesis—Clinical Features: From Gingivitis to Periodontitis
63 Cyclic Course of Periodontitis
64 Periodontal Infections and Systemic Diseases
65 Etiology and Pathogenesis—Summary

67 Indices
70 Papilla Bleeding Index—PBI
71 Periodontal Indices
72 Community Periodontal Index of Treatment Needs—CPITN
73 Periodontal Screening and Recording—PSR

74 Epidemiology
74 Epidemiology of Gingivitis
75 Epidemiology of Periodontitis
76 WHO Studies

Disease Entities and Diagnosis

77 Types of Plaque-associated Periodontal Diseases
77 Gingivitis—Periodontitis
78 Classification of Periodontal Diseases—Nomenclature

79 Gingivitis
80 Histopathology
81 Clinical Symptoms
82 Mild Gingivitis
83 Moderate Gingivitis
84 Severe Gingivitis

85 Ulcerative Gingivitis/Periodontitis
86 Histopathology
87 Clinical Symptoms—Bacteriology
88 Ulcerative Gingivitis (NUG)
89 Ulcerative Periodontitis (NUP)
90 Ulcerative Gingivoperiodontitis—Therapy

91 Hormonally Modulated Gingivitis
93 Severe Pregnancy Gingivitis—Gravid Epulis
94 Pregnancy Gingivitis and Phenytoin

95 Periodontitis
96 Pathobiology—The Most Important Forms of Periodontitis
96 Type II
96 Type III B
97 Type III A
97 Type IV B
98 Pathomorphology—Clinical Degree of Severity
99 Pockets and Loss of Attachment
100 Intra-alveolar Defects, Infrabony Pockets
102 Furcation Involvement
104 Histopathology
105 Additional Clinical and Radiographic Symptoms
108 Chronic Periodontitis—Mild to Moderate
110 Chronic Periodontitis—Severe
112 Aggressive Periodontitis—Ethnic Contributions?
114 Aggressive Periodontitis—Acute Phase
116 Aggressive Periodontitis—Initial Stage
118 Prepubertal Periodontitis—PP (Aggressive Periodontitis)

119 Oral Pathologic Alterations of Gingiva and Periodontium*
120 Primarily Gingival Alterations (Type I B)
120 Gingival and Periodontal Alterations (Type IV A/B)
121 Phenytoin-Induced Gingival Overgrowth
122 Dihydropyridine-induced Gingival Overgrowth
123 Cyclosporine-induced Gingival Overgrowth
124 Gingival Hyperplasia Following Combined Drug Therapies
125 Benign Tumors—Epulis
126 Benign Tumors—Fibrosis, Exostosis
127 Malignant Tumors
128 Gingivosis / Pemphigoid
128 Pemphigus vulgaris
129 Lichen planus: Reticular and Erosive
130 Leukoplakia, Pre-Cancerous Lesions—Oral Granulomatoses
131 Herpes—Herpetic Gingivostomatitis
132 Periodontitis with Systemic Diseases (Type IV)—Diabetes Type I and Type II
134 Periodontitis Associated with Systemic Diseases (Type IV B) Down Syndrome, Trisomy 21, "Mongolism"
136 Pre-pubertal Periodontitis Associated with Systemic Disease Papillon-Lefèvre Syndrome (Type IV B)
138 Papillon-Lefèvre Syndrome—"An Exception for Every Rule"

139 HIV Infection—AIDS
140 HIV Disease—Epidemiology
141 Classification and Clinical Course of HIV Disease
142 Oral Manifestations of HIV Disease
143 Bacterial Infections in HIV
144 Fungal Infections
145 Viral Infections
146 Neoplasms
147 HIV-Associated Lesions of Unknown Etiology
148 Invasion and Replication of the HI Virus— Hurdles for Systemic Medical Treatment
149 Treatment of the HIV Patient—Pharmacologic Aspects
150 HIV—Treatment of Opportunistic Infections
150 Infection Prevention and Post-exposure Prevention—The Dental Team
151 Treatment of HIV-Periodontitis

155 Gingival Recession
156 Fenestration and Dehiscence of the Alveolar Bone
157 Clinical Symptoms
158 Recession—Localized
159 Recession—Generalized

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160 Clinical Situations Resembling Recession
161 Recession—Diagnosis
162 Measurement of Recession (Jahnke)
162 Classification of Recession (Miller)
164 Consequences of Recession

165 **Data Collection—Diagnosis—Prognosis**
166 Data Collection—Examinations
167 General Patient Health History
167 Special Patient Health History
168 Classic Clinical Findings
169 Pocket Probing—Probing Depth, Clinical Attachment Loss
170 Pocket Probing—Periodontal Probes
171 Pocket Probing Depths—Interpretation of the Measured Values
172 Furcation Involvement—Horizontal and Vertical Furcation Invasion
174 Tooth Mobility—Functional Analysis
176 Radiography

178 **Additional Diagnosis—Tests**
179 **Microbial Diagnosis—Test Methods**
180 Microbial Pocket Diagnosis—Dark Field and Phase Contrast Microscopy
181 Microbial Pocket Diagnosis—Cultures
182 New Diagnostic Tests—Evaluation
183 Molecular Biological Tests
184 Bacterial Probe Test—Practical IAI PadoTest
185 DNA/RNA Probe Tests—IAI PadoTest 4·5
186 Immunological Tests—Antigen-Antibody Reactions
187 Enzymatic Bacterial Tests—BANA Test

188 **Tests of the Host Response—Risks**
189 Genetic Risk—Test for IL-1 Gene Polymorphism
190 IL-1 Gene Test—Technique, Evaluation
191 Risk Factor IL-1-positive Genotype—Additional Risk Factors
192 Poor Oral Hygiene as a Risk Factor—Bleeding on Probing (BOP)
193 Periodontal Risk Assessment—Individual Risk Profile
194 Diagnostic Data Collection—Periodontal Charting, I and II
195 Computer-Enhanced Charting—the Florida Probe System
196 Diagnosis
197 Prognosis

198 **Prevention—Prophylaxis**
198 Definition: Prevention—Prophylaxis
199 Prevention of Gingivitis and Periodontitis

Therapy

201 **Treatment of Inflammatory Periodontal Diseases**
202 Therapeutic Concepts and Techniques
203 Therapy—Problems
204 Periodontitis—Therapeutic Goals, Therapeutic Outcomes

205 **Periodontal Wound Healing**
206 Wound Healing and Regeneration—Possibilities
208 Treatment Planning—Sequence of Treatment
Pre-phase—Systemic Health, Oral Hygiene
Phase 1—Causal, Antimicrobial, Anti-infectious
Phase 2—Surgical, Corrective
Phase 3—Preventive, Anti-infectious, "Life Long"
210 General Course of Therapy—Individual Planning

211 **Systemic Pre-phase**
212 Evaluation—Can the Patient be Safely Treated?
213 Bacteremia—Endocarditis Prophylaxis
214 Dental Procedures Carrying the Risk of Bacteremia
215 Diabetes mellitus (DM)—Risk Factor for Periodontitis
216 Smoking—An Alterable Risk Factor

217 **Emergency Treatment**

221 **Phase 1 Therapy**
222 Case presentation—Motivation—Information

223 **Initial Treatment 1—**
Oral Hygiene by the Patient
224 Motivation—Gingival Bleeding
225 Plaque Disclosing Agents
226 Toothbrushes
228 Toothbrushing Technique
229 The Solo Technique—A Different Way to Brush Your Teeth
230 Electric Toothbrushes
231 Interdental Hygiene
234 Dentifrices
235 Chemical Plaque Control—"Soft Chemo" Prevention
236 Irrigators
237 Oral Hygiene for Halitosis
238 Possibilities, Successes and Limitations of Oral Hygiene

239 **Initial Therapy 1—**
Creating Conditions that Enhance Oral Hygiene
240 Supragingival tooth cleaning—Power-driven Instruments ...
241 ... and their Use
242 Supragingival Tooth Cleaning—Hand Instruments, Prophy Pastes ...
243 ... and their Use
244 Creating of Conditions that Enhance Oral Hygiene—Removal of Iatrogenic Irritants
246 Correction of Iatrogenic Irritants—Bridge Pontics
247 Removal of Natural Plaque-retentive Areas—
Odontoplasty of Grooves, Depressions, Irregularities
248 Reduction of Natural Plaque-retentive Areas—Crowding:
Morphologic Odontoplasty
249 Treatment of Plaque-elicited Gingivitis
252 Gingivitis Treatment

253 **Phase 1 Therapy**
253 Definitions
254 Non-Surgical, Anti-Infectious Therapy—Goals of Treatment
255 Antimicrobial Therapy—Combating the Reservoir
256 Root Planing—With or without Curettage?
257 Closed Therapy—Indication, Instrumentation
258 Hand Instruments for Scaling and Root Planing—Curettes
259 Powered Instruments for Debridement
260 Gracey Curettes—Areas of Use
262 Hand Instruments for Special Problems—Curettes
263 Practical Scaling Technique with Gracey Curettes—Systematic Approach
268 Instrument Sharpening
269 Manual Sharpening of Hand Instruments
270 Automated Sharpening
271 Subgingival Debridement—Closed Root Cleaning
276 Closed Therapy in Quadrant 1 ...
277 ... and in the Rest of the Dentition?
278 Limitations of Closed Therapy
280 Possibilities and Limitations of Closed Therapy ...

281 **FMT—"Full Mouth Therapy"**
282 FMT—Instrumental/Mechanical and ...
283 ... Pharmacologic Therapy
284 FMT—Radiographic Results
285 FMT—Statistical/Numerical Results

287 **Medication**
287 Anti-Infectious Supportive Therapy—Antibiotics in Periodontitis Therapy
288 Decision-Making Criteria—When to Use Antibiotics?
290 Antibiotics—Bacterial Sensitivity and Resistance
291 Systemic Versus Local (Topical) Antimicrobial Therapy
292 Local (Topical) Antimicrobial Therapy—"Controlled Release Drugs" (CRD)
294 Host Response/Reaction—Modulating Substances

295 **Phase 2 Therapy**
Periodontal Surgery—Corrective Phase
296 Purposes and Goals of Periodontal Surgery
297 Patient Selection
298 Factors that Influence the Treatment Result
299 Methods of Periodontal Surgery and their Indications
300 Principles of Various Treatment Modalities—Advantages and Disadvantages
302 Pre-operative Treatment—Post-operative Care

303 **Flap Procedures—Open Treatment**
304 Flap Design—Incisions
305 Instruments for Flap Surgery
306 Needles and Suture Materials
307 Sutures and Knots
308 Frequently Used Suture Placements

309 **"Access Flap" Surgery, Open Flap Debridement (OFD)—**
Modified Widman Flap (MWF)
310 Principles of the Modified Widman Procedure—Ramfjord Technique
312 Principles of the MWF—Occlusal View
313 "Access Flap"/Modified Widman Procedure—Case Presentation
317 "Access flap"/Modified Widman Procedure
318 Long-term Results Following Various Treatment Modalities
319 Wedge Excision Distal to a Lone-standing Tooth—Principles
320 Wedge Excision—Most Distal Tooth

323 **Regenerative Methods**
324 Intra-Alveolar Pockets—Anatomy of the Defect
326 Bone Regeneration without "Transplantation"
327 Filling Materials for Intra-Alveolar Pockets—Transplants/Implants
328 Instruments for Harvesting Autogenous Bone ...
329 ... and Their Use
330 Implantation (Transplantation?) of Autogenous Bone
332 Bone Replacement Materials—"Fillers"

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334 Alloplastic Implant—Combined Surgical Procedure
337 Combined Procedure—Intra-Osseous Implant
338 Guided Tissue Regeneration—GTR
340 Selection of Membranes and Barriers
341 GTR Using Non-resorbable Membranes
345 Individual Immediate Membrane—Atrisorb/Atrigel Technology
346 GTR with Membrane and Filler—"Atrisorb Free Flow," "Bio-Oss Collagen"
348 GTR with Filling Material and a Membrane—"Bio-Gide" and "Bio-Oss Collagen"
351 Regeneration Using Proteins, Growth Factors and Differentiation Factors
352 Flap Surgery Combined with Enamel Matrix Proteins—Emdogain
354 Regeneration Using Emdogain
354 Regenerative Therapy—Quo Vadis?

355 **Resective Methods**
Pocket Elimination—Osseous Surgery
356 Instruments for Osseous Surgery ...
357 ... and Their Use
358 Evaluation of Various Resective Methods
359 Resective Therapy
Apical Flap Repositioning—Pocket Elimination
366 Combined Flap Surgical Techniques

367 **Gingivectomy and Gingivoplasty**
368 Instruments for Gingivectomy/Gingivoplasty ...
369 ... and Their Use
370 Tissue Dressings and Tissue Adhesives
371 Gingivectomy/Gingivoplasty
377 GV/GP, Maxilla: Facial and Palatal
378 GV/GP—Minor Surgical Procedures, Corrective Operations
Exposing the Margins of Restorations and Cavity Preparations
379 GV/GP—Phenytoin-Induced Gingival Overgrowth
380 Limitations of GV/GP—Cyclosporine-Induced Gingival Overgrowth

381 **Furcation Involvement—Furcation Treatment**
382 Embryologic Development of Multi-Rooted Teeth—Furcation
383 Furcation Involvement—Classifications
384 Treatment Planning—Problems—Long-Term Results
385 Furcation Treatment—Aids for Decision Making
386 Therapeutic Possibilities for Various Cases
388 Furcation Involvement (F1) in the Mandible—Odontoplasty and Scaling
389 Furcation Involvement, F2, in the Maxilla—Furcationplasty
390 Furcation Involvement, F2, in the Mandible—GTR Technique
392 Furcation Involvement, F3, in the Mandible—Hemisection with Extraction
394 Furcation Involvement, F3, in the Maxilla—Root Resection with Reconstruction
396 Maxillary Furcation Involvement, F3—Trisection with Maintenance of all Roots

397 **Mucogingival, Plastic Surgery**
398 Mucogingival Problems ...
399 ... and Possibilities for Resolution
400 Frenotomy—Frenectomy
401 Free Gingival Graft—Autogenous Transplant—with Epithelium
402 Instruments for Harvesting the Transplant ...
403 ... and Their Use
404 Free Gingival Graft—Thickness and Shape
405 Free Gingival Graft—Halting Recession
410 FGG—Wound Healing, Clinically ...
411 ... and via Fluorescence Angiography
412 Advantages and Disadvantages of FGG

413 **Covering Areas of Recession**
414 Shape of the Defect—Selection of Surgical Technique
415 Pedicle Flap Techniques
416 Direct Coverage with a Free Gingival Graft
418 Coronal Repositioning after Free Gingival Grafting—2-Stage Surgical Procedure

419 **Covering Areas of Recession with a Connective Tissue Graft**
421 Harvesting Graft Tissue from the Palate
422 Harvesting the Graft from the Palate—Schematic and Clinical
424 Additional Methods for Harvesting Connective Tissue Grafts
425 Free Connective Tissue Graft—Nelson Technique
429 Free Connective Tissue Graft—Nelson Technique
430 Connective Tissue Graft ... and Dealing with Complications

435 **Covering Areas of Recession via "Guided Tissue Regeneration"—GTR**
437 Covering Gingival Recession Using Resorbable Membrane
Treating Recession after Orthodontic Therapy
440 Covering Areas of Recession on Several Teeth—Possibilities
441 Coronally Repositioned Flaps and Papilla Rotation
442 Esthetic Improvement through Mucogingival Surgery and Prosthetic Restoration
444 Summary—Mucogingival, Plastic Surgery

445 **Periodontics—Endodontics**
446 Class I—Primarily Endodontic Problem
446 Class II—Primarily Periodontal Problem
447 Class III—Combined Perio-Endo Problems

449 **Phase 3 Therapy**
Periodontal Maintenance Therapy—Recall
450 Recall in the Dental Practice—Recall Effect
451 Recall—Continuous Risk Management
452 The "Recall Hour"—Practical Periodontal Maintenance Therapy
454 Dentist and Dental Hygienist—The "Preventive Team"
454 Auxiliary Personnel and Treatment Needs
455 Failures—Lack of Periodontal Maintenance Therapy
456 Negative Results of Therapy
458 Hypersensitive Dentin

Supporting Therapies

459 **Function—Functional Therapy**
459 Normal Function
460 Physiologic Tooth Mobility
461 Occlusal Periodontal Trauma
462 Occlusal Bite Guard—The Michigan Splint

463 **Orthodontics**
464 Maxillary Anterior Segment Space Closure Following Periodontal Therapy
466 Uprighting the Mandibular Second Molar
468 Maxillary Anterior Esthetic Correction Following Periodontitis Treatment
470 Treatment of the Malpositioned Canine

471 **Splinting—Stabilization**
473 Temporary Splinting
474 Semi-Permanent Splinting—Anterior Area
475 Permanent Splinting—Adhesive Technique
476 Prosthetic Stabilization—Long-Term Temporary

477 **Perio-Prosthetics 1**
Standard Techniques
478 Temporaries—Removable or Fixed?
479 Adhesive Bridge in the Mandible—Long-Term Temporary
480 Fixed Temporary—Problem Zones
481 Fixed Acrylic Temporary—Practicality
482 Fixed, Metal-Reinforced Long-Term Temporary
484 Definitive Fixed Reconstruction—Total Bridgework
486 Molar Loss—What Now?
487 Telescope ("Coping") Bridgework with Few Remaining Teeth
488 Cast Framework Prostheses—"Social Solution"

489 **Perio-Prosthetics 2**
Supplemental Measures, Esthetics
489 Goals, Problems and Solutions in Esthetic Perio-Prosthetics
490 Crown Margins—Biologic Width—Dentogingival Complex (DGC)
491 Esthetic Width—Umbrella Effect—Transparency
492 Smile Line—Red-White Proportions
493 Crown Lengthening—Principles
494 Surgical Crown Lengthening—Step-by-Step
496 Papilla Loss—Classification, Rules
497 Papilla Loss—Prosthetic Rehabilitation with Veneers
500 Papilla Loss—Prosthetic Treatment Using Crowns
502 Edentulous Arch Segments—Pontics
503 Alveolar Ridge Defects—Classifications
504 Alveolar Ridge Defect—Prosthetic Solution
505 Alveolar Ridge Defects—Surgical Corrections—Methods
506 Ridge Augmentation Using Partially Epithelialized Connective Tissue Graft
509 Ridge Defect—Build-up with Connective Tissue Graft—Summary

511 **Dental Implants—Implant Therapy**
512 Determinative Diagnostic Criteria
513 Therapeutic Concepts—Therapeutic Results
514 Recall—Management of Implant Problems

Appendices

515 **Geriatric Periodontology?—The Periodontium in the Elderly**
518 Age-Related Changes—Influence Upon Treatment Planning

519 **Classification of Periodontal Diseases**
519 New Classification of Periodontal Diseases (1999)
522 Changes—Comparing the 1989 and 1999 Classifications

523 Recall—"Personalized Periodontology"

524 **Acknowledgments for Figures**
525 **References**
528 **Index**

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Introduction

"Periodontology" is the study of the tooth-supporting tissues, the "periodontium." The periodontium is made up of those tissues that surround each tooth and which anchor each tooth into the alveolar process (Latin: para = adjacent to; Greek: odus = tooth).
The following soft and hard tissues constitute the structure of the periodontium:

- Gingiva
- Root Cementum
- Periodontal Ligament
- Alveolar Bone

The structure and function of these periodontal tissues have been extensively researched (Schroeder 1992). Knowledge of the interplay between and among the cellular and molecular components of the periodontium leads to optimum therapy, and also helps to establish the goals for future intensive research.

Periodontal Diseases

Gingivitis – Periodontitis

There are numerous diseases that affect the periodontium. By far the most important of these are plaque-associated gingivitis (gingival inflammation without attachment loss) and periodontitis (inflammation-associated loss of periodontal supporting tissues).

- *Gingivitis* is limited to the marginal, supracrestal soft tissues. It is manifested clinically by bleeding upon probing of the gingival sulcus, and in more severe cases by erythema and swelling, especially of the interdental papillae (Fig. 3).
- *Periodontitis* can develop from a pre-existing gingivitis in patients with compromised immune status, the presence of risk factors and pro-inflammatory mediators, as well as the presence of a predominately periodontopathic microbial flora. The inflammation of the gingiva may then extend into the deeper structures of the tooth-supporting apparatus. The consequences include destruction of collagen and loss of alveolar bone (attachment loss). The junctional epithelium degenerates into a "pocket" epithelium, which proliferates apically and laterally. A true periodontal pocket forms. Such a pocket is a predilection site and a reservoir for opportunistic, pathogenic bacteria; these bacteria sustain periodontitis and enhance the progression of the disease processes (Fig. 4).

Gingival Recession

Gingival recession is not actually a "disease," but rather an anatomic alteration that is elicited by morphology, improper oral hygiene (aggressive scrubbing), and possibly functional overloading.

- Teeth are not lost due to classical gingival recession, but patients may experience cervical hypersensitivity and esthetic complications. If gingival recession extends to the mobile oral mucosa, adequate oral hygiene is often no longer possible. Secondary inflammation is the consequence.

  In addition to classical gingival recession, apical migration of the gingiva is often observed in patients with long-standing, untreated periodontitis, and it may be a consequence of periodontitis *therapy* in elderly patients ("involution"; Fig. 2).

These three periodontal disorders – gingivitis, periodontitis, gingival recession – are observed world-wide; they affect almost the entire population of the earth to greater or lesser degree. In addition to these common forms of oral pathology, there are many less frequently encountered diseases and defects of the periodontal tissues. All of these diseases were comprehensively classified at an international World Workshop in 1999 (see Appendix, p. 519).

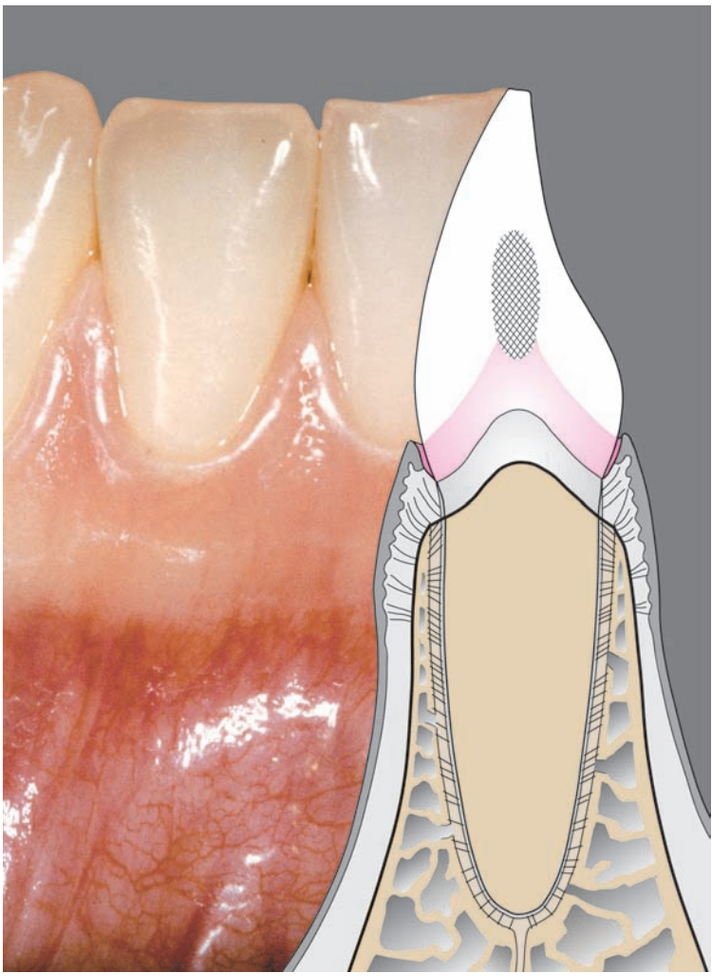
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1 Healthy Periodontium

The most important characteristic of the periodontium is the special connection between soft and hard tissues:

- In the marginal region, one observes the inflammation-free gingiva, which provides the epithelial attachment to the tooth by means of its junctional epithelium (pink collar). This connection protects the deeper-lying components of the periodontium from mechanical and microbiologic insult.
- Subjacent to the junctional epithelium, one observes the supracrestal fibers, which serve to connect the tooth with the gingiva, and also the periodontal ligament fibers in the region of the alveolar bone, which insert into the bone and the cementum of the root surface.

Prevention of disease: Maintaining the health of the periodontium is the highest goal in periodontology, and should also be the patient's goal. It is achieved by optimum, purely mechanical oral hygiene. Disinfectant mouthwashes may enhance mechanical hygiene.



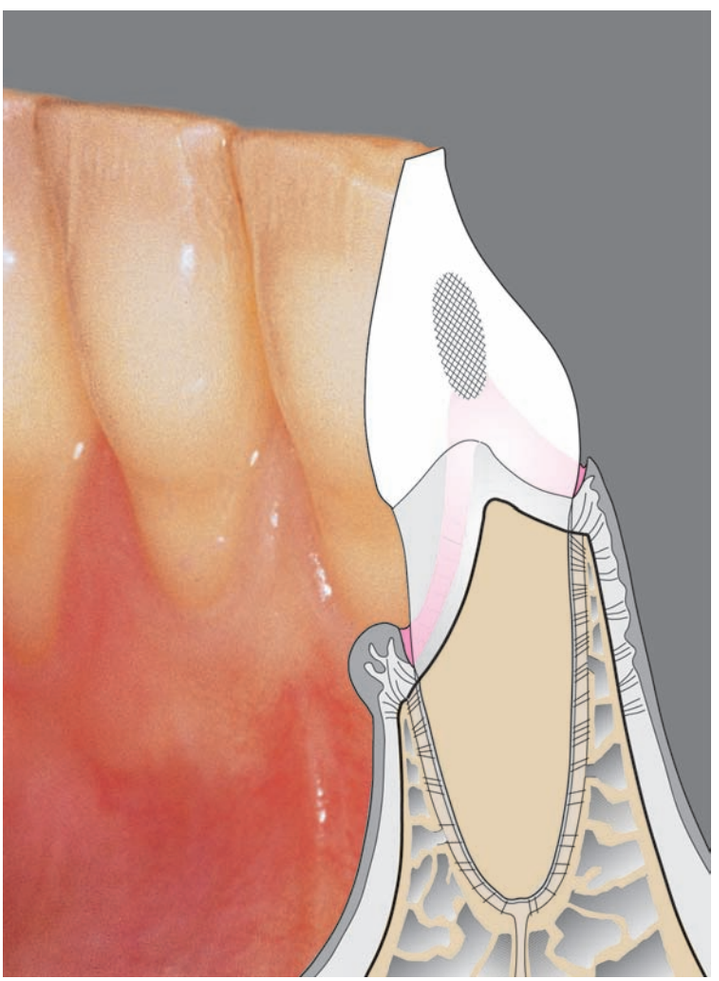
Healthy Periodontium

2 Gingival Recession

The main characteristic of this condition, which is often esthetically objectionable to patients, is an inflammation-free apical migration of the gingival margin. A morphological prerequisite is generally a facial bony lamella that is either extremely thin or entirely lacking. Gingival recession can be initiated and propagated by improper traumatic tooth brushing (horizontal scrubbing), and functional overloading may also play a role (?). Thus gingival recession cannot be classified as a true periodontal disease.

The best way for a patient to prevent gingival recession is by using an adequate but gentle oral hygiene technique (vertical-rotatory brushing or use of a sonic toothbrush).

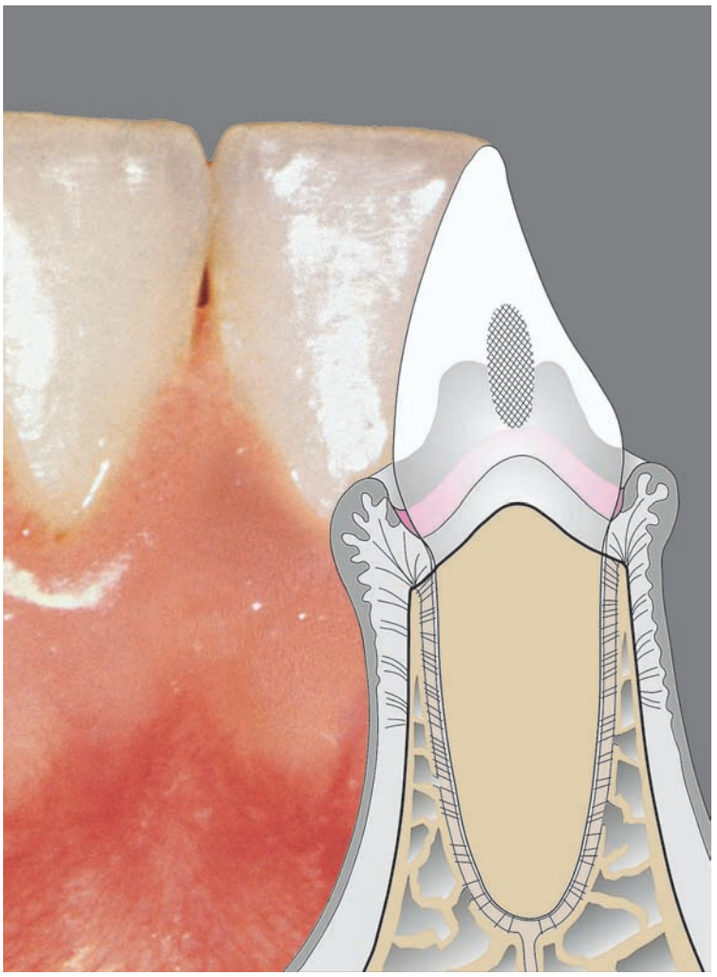
Treatment: Incipient or progressing gingival recession can be halted by altering the patient's oral hygiene techniques; in severe cases, mucogingival surgery may be employed to stop the progression or re-cover the exposed root surfaces.



Gingival Recession

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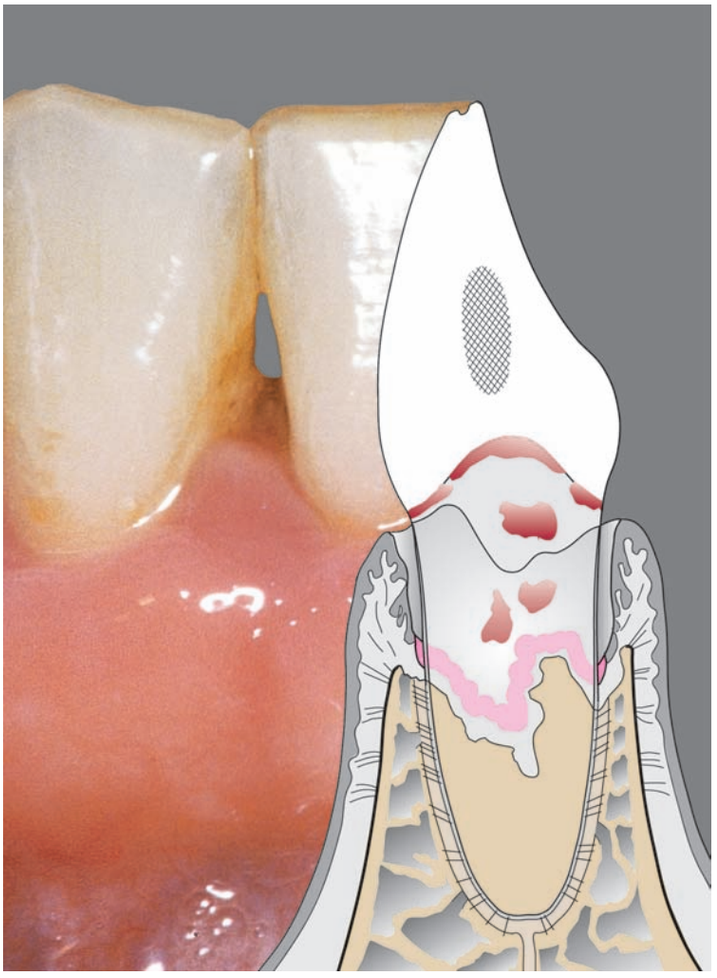
Gingivitis



3 Gingivitis
Gingivitis is characterized by plaque-induced inflammation of the papillary and marginal gingivae. Clinical symptoms include bleeding on probing, erythema, and eventual swelling. Gingivitis may be more or less pronounced depending upon the plaque—a biofilm—(quantity/quality) and the host response. Deeper lying structures (alveolar bone, periodontal ligament) are not involved. Gingivitis *may* be a precursor to periodontitis, but this does *not* always occur.

Treatment: Gingivitis can be completely controlled simply through adequate plaque control. Following initiation or improvement of oral hygiene procedures, coupled with professional plaque and calculus removal, complete healing can be expected. Nevertheless, freedom from inflammation, e. g., absence of bleeding on probing, will be impossible to achieve if the patient is not capable of maintaining a high standard of oral hygiene over the long term, or is not willing to do so (compliance!).

Periodontitis



4 Periodontitis
At the gingival margin, the characteristics of periodontitis are similar to those of gingivitis, but the inflammatory processes extend further, into the deeper-lying periodontal structures (alveolar bone and periodontal ligament). True periodontal pockets are formed and connective tissue attachment is lost. Loss of hard and soft tissues is usually localized and not generalized.
Periodontitis may be classified as *chronic* (Type II) or *aggressive* (Type III), with varying degrees of severity. Approximately 90 % of all cases are characterized as “chronic periodontitis” (p. 108, 519).

Treatment: Most cases of periodontitis can be treated successfully. However, the required therapeutic endeavor can vary enormously from case to case. The treatment effort may be relatively small in early stages of periodontitis. Mechanical treatment remains today in the foreground. In special cases, topical and systemic medications may be used as supportive therapy.

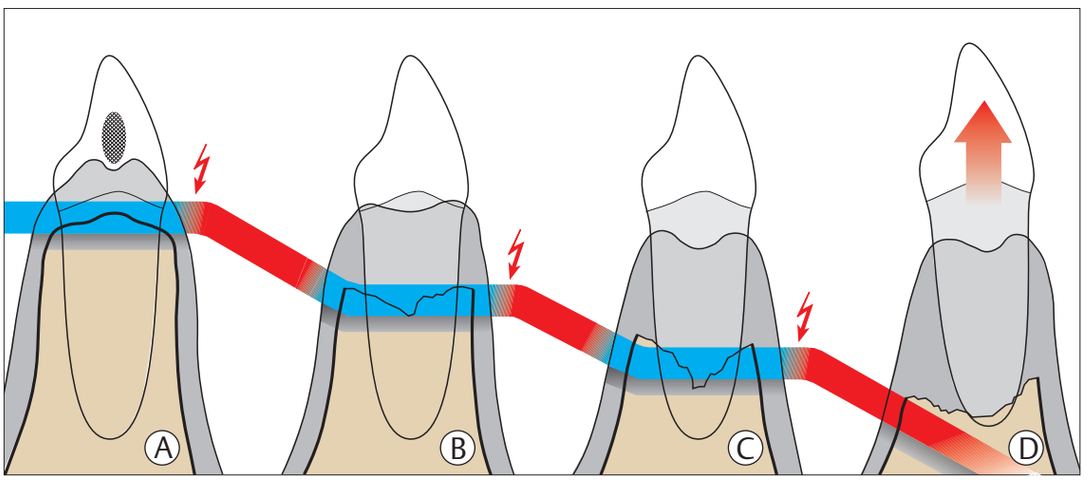
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The Clinical Course of Untreated Periodontitis

Periodontitis is usually a very slowly progressing disease (Locker & Leake 1993; Albandar et al. 1997), which in severe cases—particularly when untreated—can lead to tooth loss. Enormous variation in the speed of progression of periodontitis is observed when one differentiates between individual patients. In addition to the quantity and composition of the bacterial plaque, individually varying influences also play important roles: the systemic health of the patient, the patient's genetic constitution, psychically influenced immune response status, ethnic and social factors, as well as risk factors such as smoking and stress (p. 22, Fig. 41). All of these circumstances can influence the onset and the speed at which the disease process accelerates in different patient age groups.

Not all teeth or individual surfaces of teeth are equally susceptible (Manser & Rateitschak 1996):

- Molars are the most endangered
- Premolars and anterior teeth are less susceptible
- Canines are the most resistant.



5 Clinical Course of Untreated Periodontitis
In the aggressive forms of periodontitis (pp. 95, 97), the manifestation of tissue loss on individual teeth occurs in successive acute phases rather than in a gradual, chronic progression. Phases of progression and quiescence alternate. Destructive phases may occur rapidly one after another, or longer quiescent phases may be in evidence.

Red Acute phase/destruction
Blue Phase of quiescence

Periodontitis—Concepts of Therapy

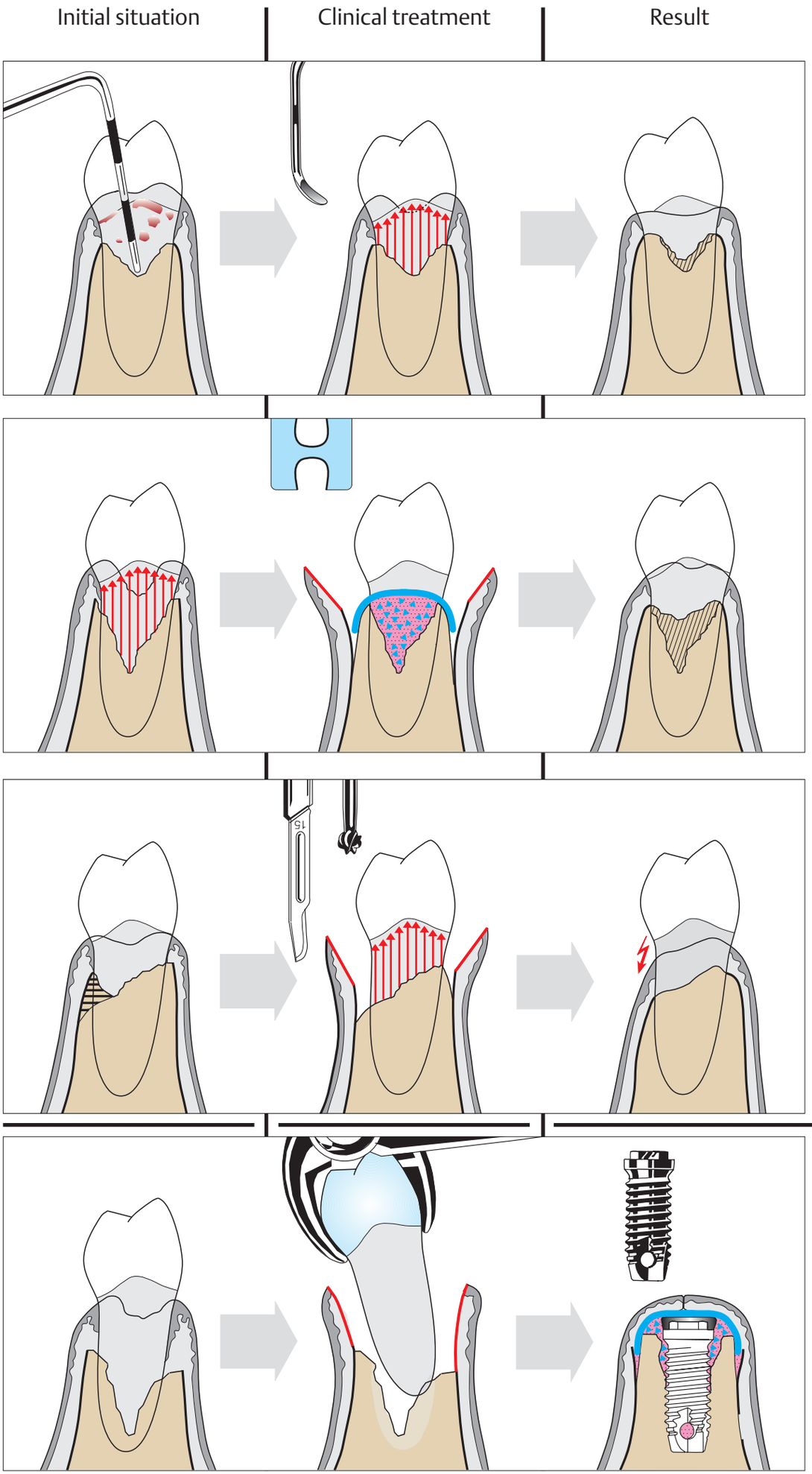
The primary goal is *prevention* of periodontal diseases, and the secondary goal is to enhance the healing of existent periodontitis, as far as possible toward complete *Restitutio ad integrum.* Clinical and basic research is targeted today toward realization of these goals in the near future.

At the present time, proven therapeutic concepts are available for the elimination of inflammation and the cessation of the progression of disease. In addition, to a certain degree, it is possible today to regenerate lost periodontal attachment (GTR, p. 338). The following treatment modalities are available to the periodontal therapist:

1. Closed or open root planing ("causal therapy," the "gold standard")
2. Regenerative surgical therapies
3. Resective surgical therapies
4. Alternatives: extraction or dental implants?

1. *Root planing* is the *Conditio sine qua non* in all periodontal therapy. This is pure *causal therapy,* during which the causative "biofilm" (plaque) and subgingival calculus are removed. If the pockets are shallow and the morphological relationships simple (single-rooted teeth), treatment can be performed *closed,* but in advanced cases, *open* treatment with direct visual access following reflection of a tissue flap is often preferable (e.g., modified Widman procedure). The result of such treatment is usually healing in the sense of "repair" (p. 206). A long junctional epithelium forms.
2. *Regenerative therapeutic methods* ("guided tissue regeneration" [GTR]; autogenous, alloplastic implants) have taken on increased significance in recent years. These procedures are constantly being further developed, and in the future they may be enhanced by the use of growth and differentiation factors.
   Regenerative therapy can lead to the actual re-formation of significant periodontal tissues.
3. *Radical surgery* for the elimination of periodontal pockets has lost some its popularity in recent years, although the results are generally more predictable and the tendency toward recurrence is low.
4. In cases of complex, severely advanced periodontitis, e.g., with severe furcation involvement in the molar region, the dentist may consider tooth extraction and *replacement with a root-form implant* instead of resective or regenerative periodontal surgery. Even in such cases, periodontal treatment of the remaining dentition must be performed, as well as optimum plaque control by the patient and the creation of an adequate amount of bone for the implant.

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Treatment Possibilities

6 "Closed" or "Open" Root Planing

Left: 6 mm bony pocket. The root surface is covered with plaque and calculus (brown).

Middle: The root surface is thoroughly cleaned using curettes, sonic or ultrasonic instruments either closed or open (surgical flap reflection).

Right: In the deepest regions of the pocket, some new bone formation may occur (hatched area).

7 Guided Tissue Regeneration (GTR)—Surgical

Left: 8 mm bony pocket.

Middle: Following flap reflection and planing of the root surface, the defect is covered with a membrane barrier. This prevents "repair" in the form of a long junctional epithelium. The bony crater can be filled with autogenous bone or an artificial bone substitute.

Right: Enhanced regeneration of all periodontal tissues may ensue.

8 Radical Surgical Procedure

Left: 7–8 mm bony pocket.

Middle: The bony pocket is eliminated by means of osteoplasty or actual ostectomy (e. g., lingual "ramping," p. 357)

Right: The pocket has been eliminated; an exposed cervical area remains suprajacent to the healthy gingiva (arrow)!

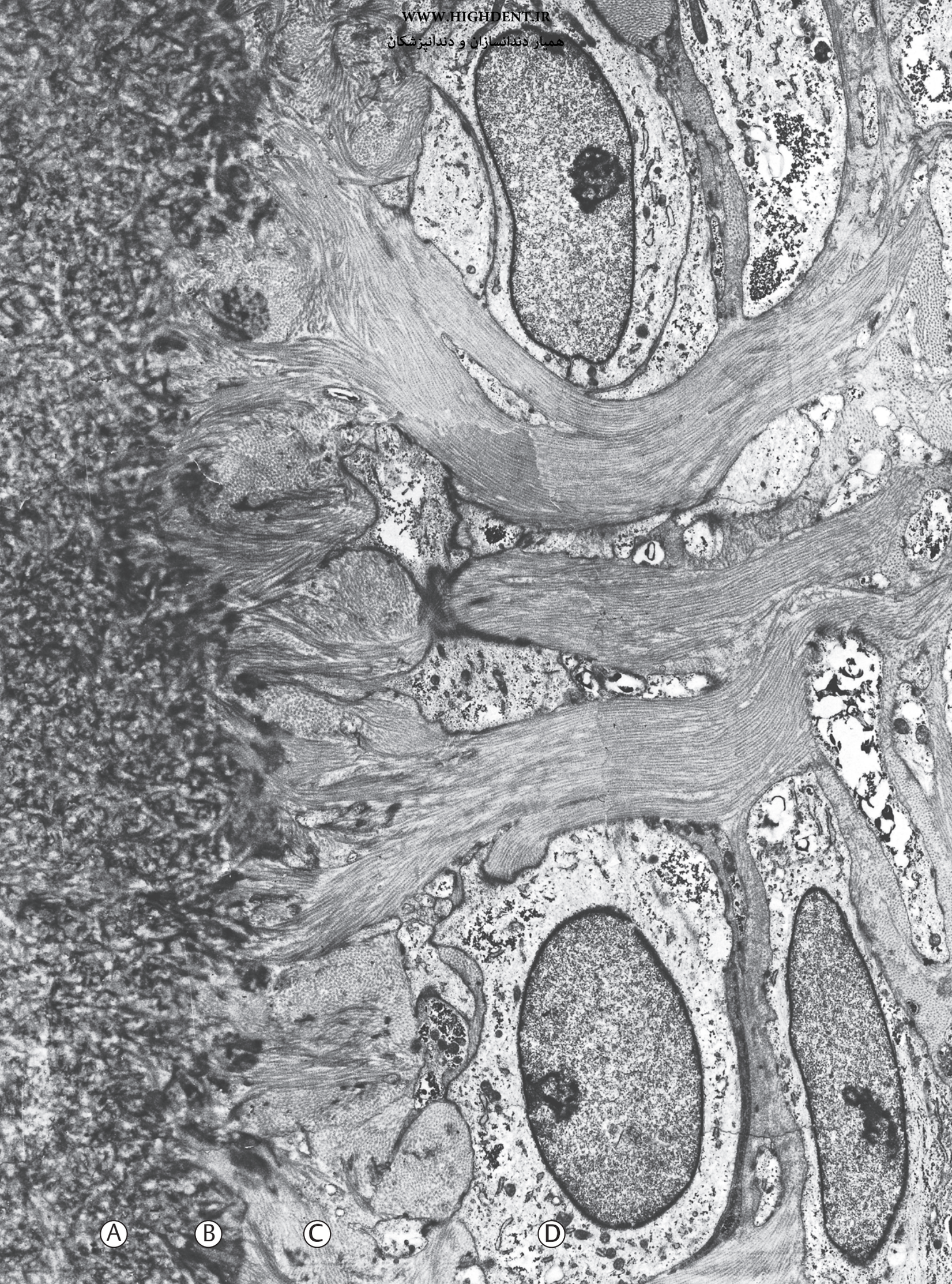
9 Dental Implant

Left: The pronounced attachment loss rendered successful treatment questionable even with extensive therapy. In such cases, especially in molars with pronounced furcation defects (F3, p. 383), implant therapy should be considered.

Middle: Tooth extraction.

Right: The implant is covered by mucosa. Osseous regeneration occurs beneath a membrane.

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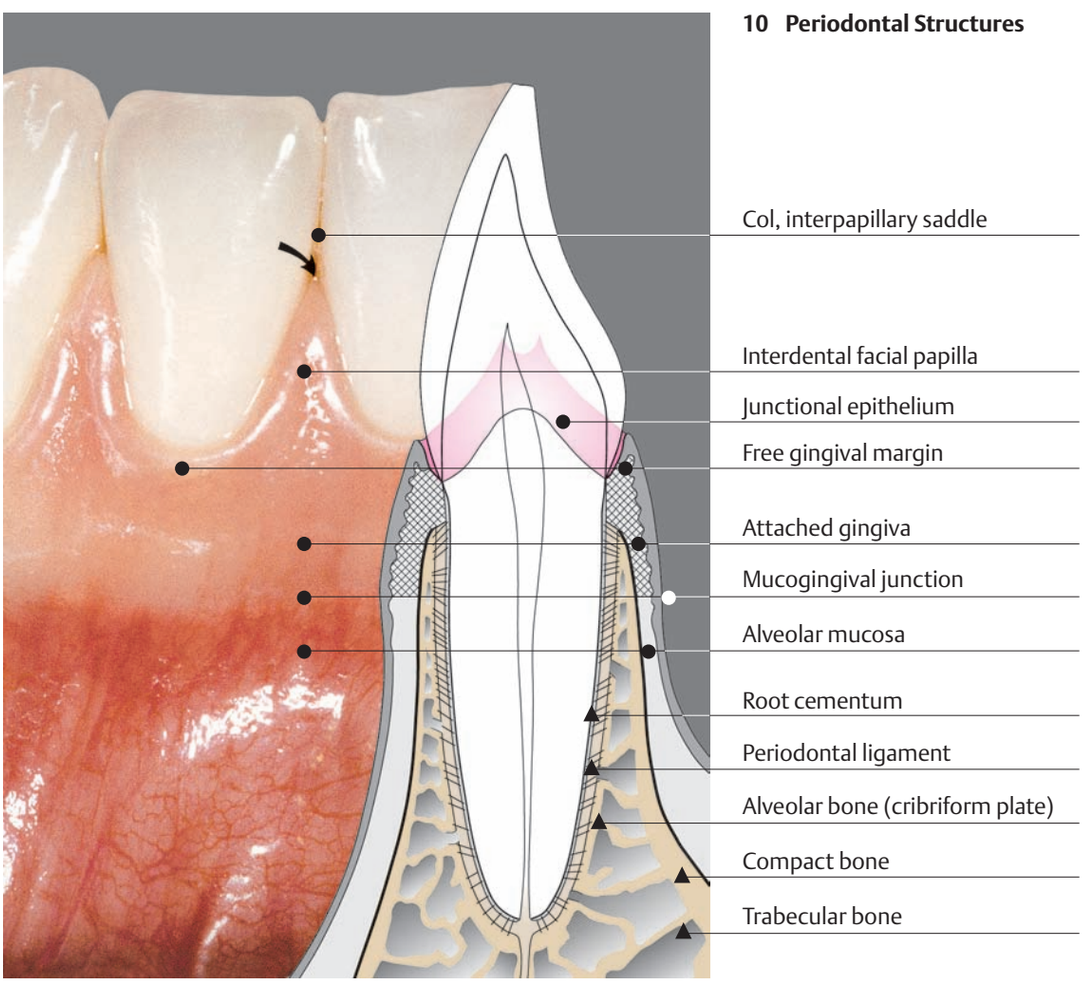
Structural Biology

"Structural biology" is a general term referring to the classical macromorphology and histology of tissues, as well as their function, including the biochemistry of the cells and the intercellular substances.

Basic knowledge of the normal structural biology of periodontal tissues and their dynamics (mediator-guided homeostasis, "turnover") is a prerequisite for full understanding of pathobiological changes in the periodontium, which can involve adaptations of the normal structures or an imbalance of otherwise normal functions (Schroeder 1992).

The term "periodontium" encompasses four different soft and hard tissues: gingiva, root cementum, alveolar bone, and the periodontal ligament, which attaches root cementum to bone. Each of these four tissues can be further differentiated in terms of structure, function and localization.

10 Periodontal Structures



Left Side:

Transmission electron photomicrograph (TEM) of root formation in humans (ca. 6-year-old)
This TEM depicts the growing demarcation between dentin, cementum and periodontal ligament during root formation. Initial mineralization of the "cementoid" directly apposed to the dentin, with penetrating collagen fibers and fibroblast-like cementoblasts, which are involved in the formation of acellular exogenous fiber cementum.

A Dentin
B Cementoid
C Radiating collagen fibers
D Cementoblast (fibroblast-like) building acellular exogenous fiber cement

Courtesy *D. Bossardt, H. Schroeder*

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Gingiva

The gingiva is one portion of the oral mucosa. It is also the most peripheral component of the periodontium. Gingiva begins at the mucogingival line, and covers the coronal aspect of the alveolar process. On the palatal aspect, the mucogingival line is absent; here, the gingiva is a part of the keratinized, non-mobile palatal mucosa.

The gingiva ends at the cervix of each tooth, surrounds it, and forms there the epithelial attachment by means of a ring of specialized epithelial tissue (junctional epithelium; p. 10). Thus the gingiva provides for the continuity of the epithelial lining of the oral cavity.

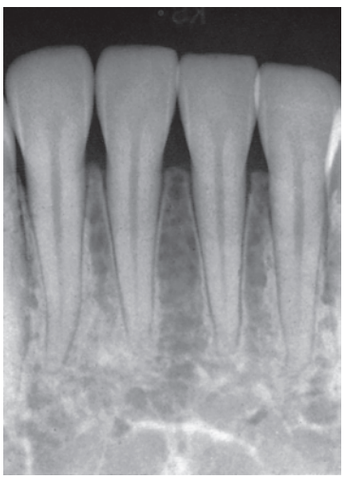
The gingiva is demarcated clinically into the *free marginal* gingiva, ca. 1.5 mm wide; the *attached* gingiva, which may be of varying width; and the *interdental* gingiva.

Healthy gingiva is described as "salmon" pink in color; in Blacks (seldom also in Caucasians) the gingiva may exhibit varying degrees of brownish pigmentation. Gingiva exhibits varying consistency and is not mobile upon the underlying bone. The gingival surface is keratinized and may be firm, thick and deeply stippled ("thick phenotype"), or thin and scarcely stippled ("thin phenotype"; Müller & Eger 1996, Müller et al. 2000).

11 Healthy Gingiva
The free gingival margin courses parallel to the cementoenamal junction. The facial interdental papillae extend to the contact area of adjacent teeth. A gingival groove can be observed in some areas, demarcating the free gingival margin from the attached gingiva.

Right: The radiograph depicts normal interdental septa. In the original radiograph, the crest of the alveolar bone was observed ca. 1.5 mm apical to the CEJ.



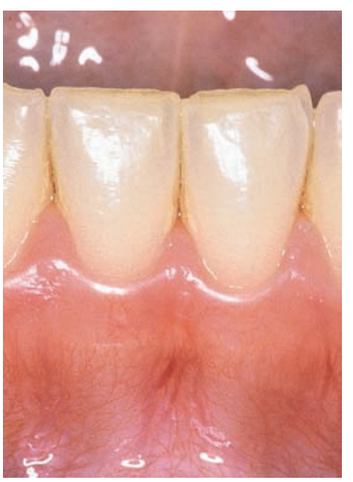


12 Variations in Consistency of Healthy Gingiva
Left: Firm, fibrous gingiva = "thick" phenotype.

Right: Delicate, scarcely stippled gingiva = "thin" phenotype. The subjacent bone covering the roots is clearly visible.

Thicker gingiva provides better conditions for treatment and wound healing (blood flow; stable position of the gingival margin).

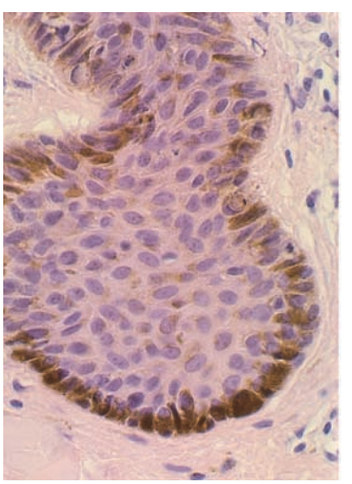




13 Healthy, Pigmented Gingiva
Note the symmetrical pigmentation of the attached gingiva in this 16-year-old African female.

Right: This pigmentation results from the synthesis of melanin by melanocytes located in the basal layer of the epithelium. The melanocytes in this histologic section appear as brown spots.





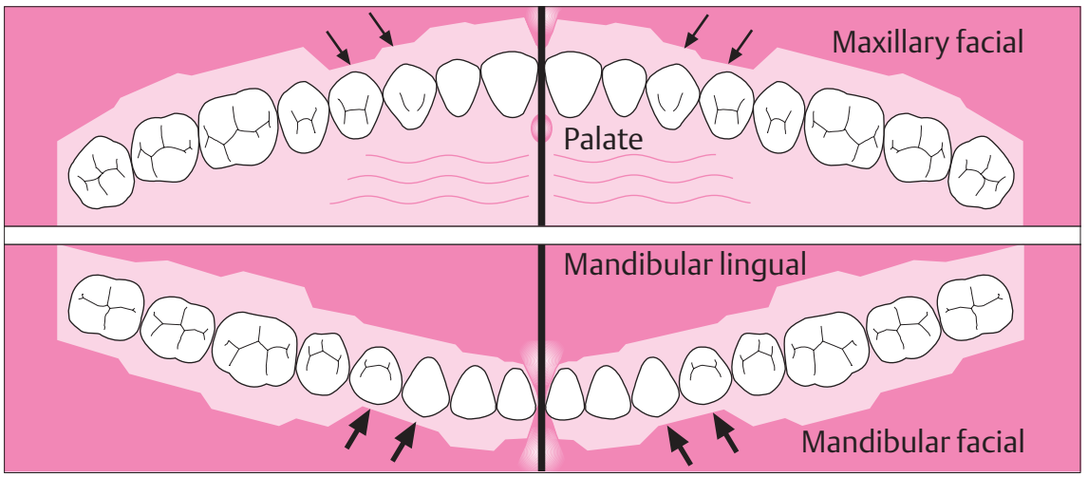
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Gingival Width

The attached gingiva becomes wider as a patient ages (Ainamo et al. 1981). The width varies between individuals and among various groups of teeth in the same person. Although it was once believed that a minimum width of attached gingiva (ca. 2 mm) is necessary to maintain the health of the periodontium (Lang & Löe 1972), this concept is not accepted today. However, a wide band of attached gingiva does offer certain advantages in the case of periodontal surgery, both therapeutically and esthetically.

Col—Interpapillary Saddle

Apical to the contact area between two teeth, the interdental gingiva assumes a concave form when viewed in labiolingual section. The concavity, the "col," is thus located between the lingual and facial interdental papillae and is not visible clinically. Depending on the expanse of the contacting tooth surfaces, the col will be of varying depth and breadth. The epithelium covering the col consists of the marginal epithelia of the adjacent teeth (Cohen 1959, 1962; Schroeder 1992). The col is not keratinized. In the absence of contact between adjacent teeth, the keratinized gingiva courses uninterrupted from the facial to the oral aspect.



14 Mean Width of Attached Gingiva

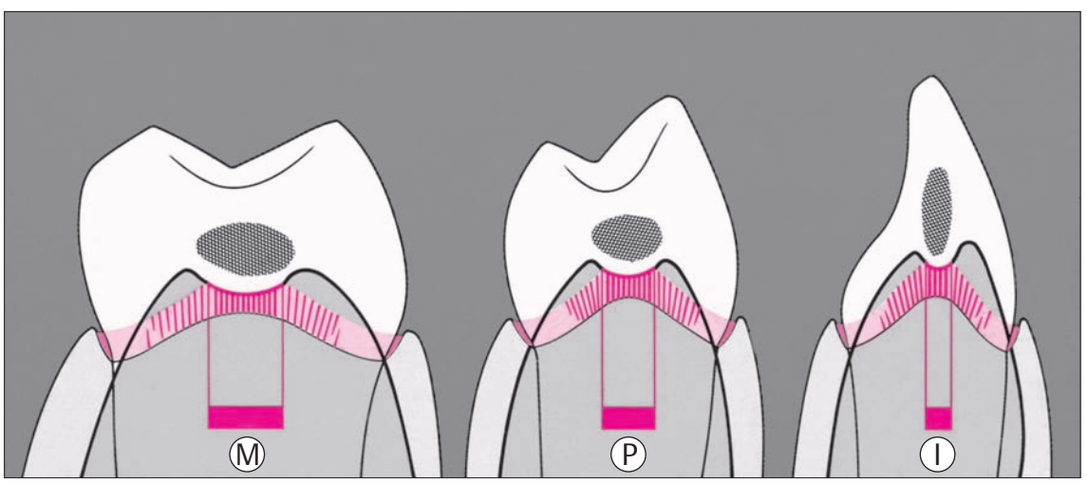
- In the *maxilla*, the *facial* gingiva in the area of the incisors is wide, but narrow around the canines and first premolars. On the *palatal* aspect, the marginal gingiva blends without demarcation into the palatal mucosa.
- In the *mandible*, the *lingual* gingiva in the area of the incisors is narrow, but wide on the molars. On the *facial*, the gingiva around the canines and first premolars is narrow (arrow), but wide around the lateral incisors.



15 Variability of Gingival Width

Width of attached gingiva can vary dramatically. The three patients depicted here, all about the same age, exhibit gingival width varying from 1 to 10 mm in the mandibular anterior area.

Right: After staining the mucosa with iodine (Schiller or Lugol solution) the mucogingival line is easily visible because the non-keratinized alveolar mucosa is *iodine-positive* while the keratinized gingiva is not (cf. p. 161).



16 Col—Interpapillary Saddle

The col consists essentially of a connection between the junctional epithelia (JE; p. 10) of any two adjacent teeth. Tooth morphology, width of the tooth crowns and relative tooth position will determine the orofacial and the coronoapical extent of the contact surfaces (hatched) and their breadth (2–7 mm, red bar), as well as the depth (1–2 mm) of the interpapillary saddle.

I	Incisor
P	Premolar
M	Molar

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Epithelial Attachment

Junctional Epithelium—Epithelial Attachment—Gingival Sulcus

The marginal gingiva attaches to the tooth surface by means of the junctional epithelium, an attachment that is continuously being renewed throughout life (Schroeder 1992).

Junctional Epithelium

The junctional epithelium (JE) is approximately 1–2 mm in coronoapical dimension, and surrounds the neck of each tooth. At its apical extent, it consists of only a few cell layers; more coronally, it consists of 15–30 cell layers. Subjacent to the sulcus bottom, the JE is about 0.15 mm wide.

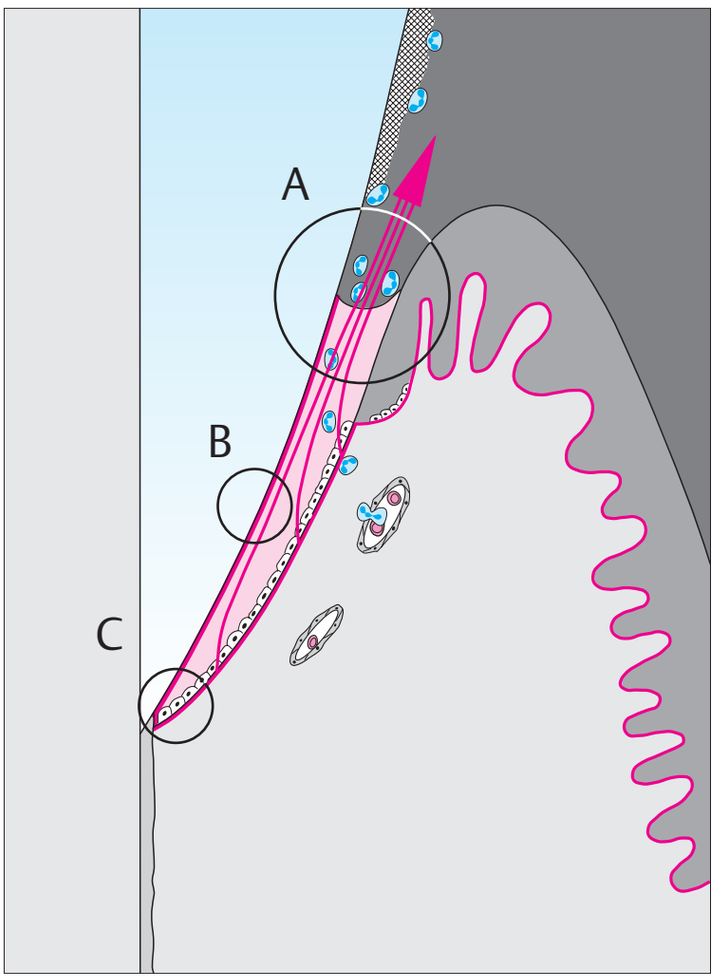
The junctional epithelium consists of two layers, the basal (mytotically active) and the suprabasal layer (daughter cells). It remains undifferentiated and does not keratinize. The basal cell layer interfaces with the connective tissue via hemidesmosomes and an external basal lamina. Healthy JE exhibits no rete ridges where it contacts the connective tissue. JE turnover rate is very high (4–6 days) compared to oral epithelium (6–12 days, Skougaard 1965; or up to 40 days, Williams et al. 1997).

17 Junctional Epithelium and Gingiva in Orofacial Section

The gingiva consists of three tissues:

- Junctional epithelium
- Oral epithelium
- Lamina propria (connective tissue)

The *junctional epithelium* (JE) assumes a key role in maintenance of periodontal health: it produces the *epithelial attachment* and therefore creates the firm connection of soft tissue to the tooth surface. It is quite permeable, and thus serves as a pathway for diffusion of the metabolic products of plaque bacteria (toxins, chemotactic agents, antigens, etc.). There is also diffusion in the opposite direction, of host defense substances (serum exudates, antibodies, etc.). Even when the gingivae do not appear inflamed clinically, the JE is constantly transmigrated by polymorphoneuclear leukocytes (PMNs) moving towards the sulcus (p. 55, Fig. 109). The red arrows depict the migration of daughter cells from the basal layer toward the gingival sulcus. The circled areas A-C are depicted in detail on page 11.



Structure of the Junctional Epithelium (JE)

Height: 1–2 mm (p. 490)
Coronal width: 0.15 mm

A Gingival sulcus (GS)
Histologic
– Width: 0.15 mm
– Depth: 0–0.5 mm
Clinical
– Depth: 0.5–3 mm (dependant upon penetration of the probe into the junctional epithelium; Fig. 378)

B Epithelial attachment
– Internal basal lamina (IBL) Thickness: 35–140 nm (1 nm = 10^{-9} m)
– Hemidesmosomes

C Apical extent of the junctional epithelium

Epithelial Attachment

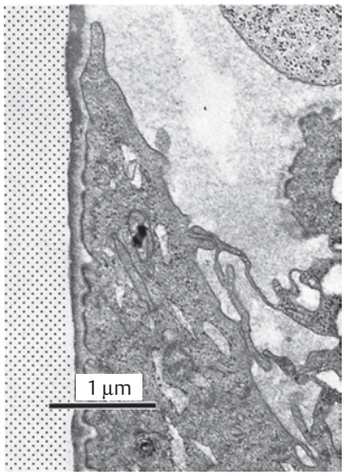
The epithelial attachment to the tooth is formed by the JE, and consists of an *internal basal lamina* (IBL) and *hemidesmosomes*. It provides the epithelial attachment between gingiva and tooth surface. This can be upon enamel, cementum or dentin in the same manner. The basal lamina and the hemidesmosomes of the epithelial attachment are structural analogs of their counterparts comprising the interface between epithelium and connective tissues.

All cells of the JE are in continual coronal migration, even those cells in immediate contact with the tooth surface. Such cells must continually dissolve and reestablish their hemidesmosomal attachments. Between the basal lamina and the tooth surface, a 0.5–1 µm thick "dental cuticle" is observed; this is possibly a serum precipitate or a secretion product of the junctional epithelial cells.

Gingival Sulcus

The sulcus is a narrow groove surrounding the tooth, about 0.5 mm deep. The bottom of the sulcus is made up of the most coronal cells of the junctional epithelium, which are sloughed (exfoliated) in rapid succession. One lateral wall of the sulcus is made up of the tooth structure, the other wall is the oral sulcular epithelium (OSE; Schroeder 1992).

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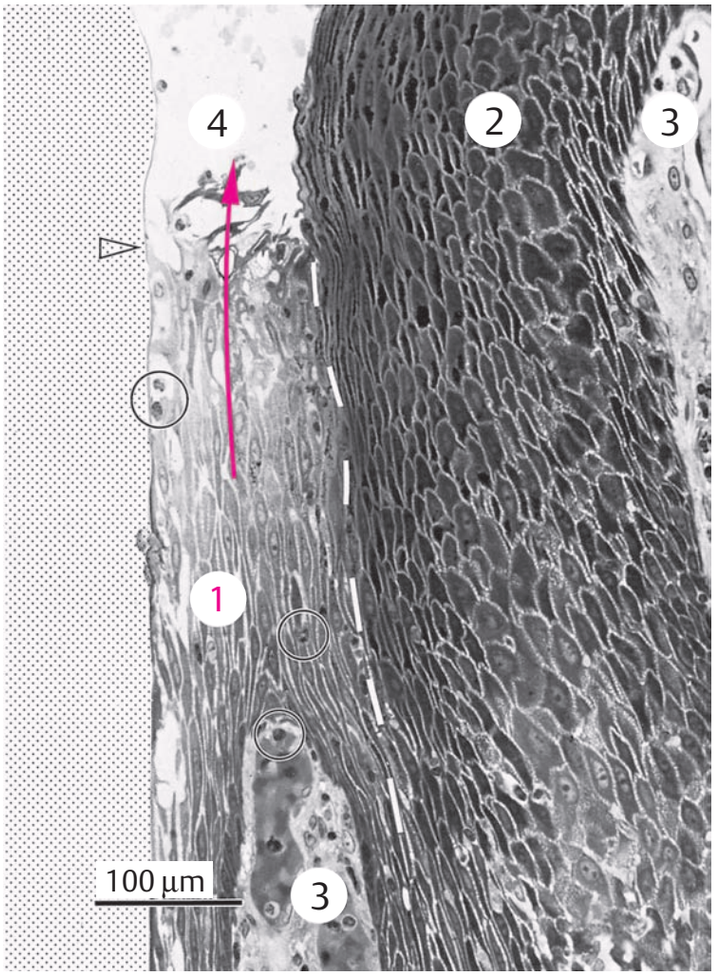


1 Junctional Epithelium JE

2 Oral Sulcular Epithelium OSE

3 Connective Tissue CT

4 Gingival Sulcus GS

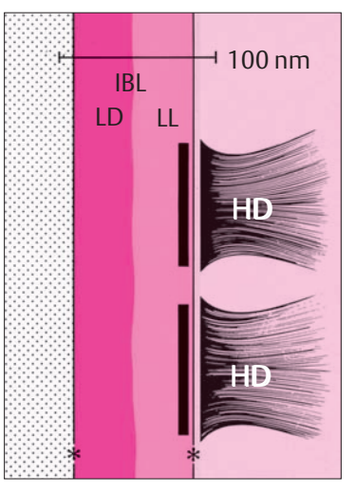


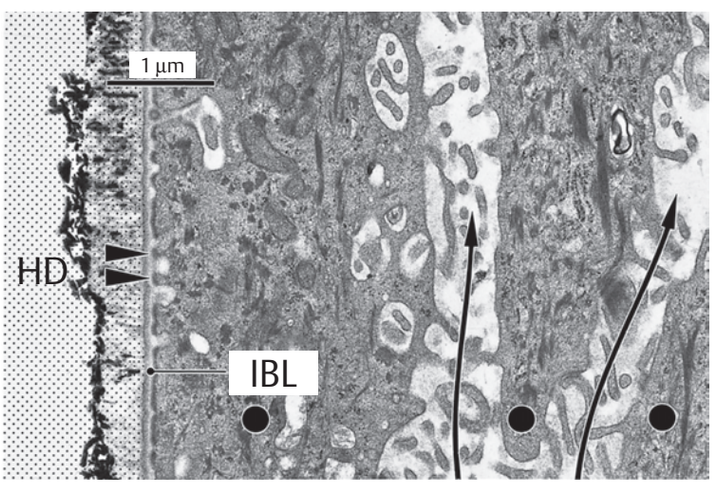
18 Gingival Sulcus and Junctional Epithelium

The junctional epithelial cells (**1**) are oriented parallel to the tooth surface and are sharply demarcated (broken line) from the more deeply staining cells of the oral sulcular epithelium (**2**). All of the daughter cells that emanate from the entire 1–2 mm length of the basal layer of the junctional epithelium must transmigrate the exceptionally narrow (100–150 µm) sulcus bottom (red arrow). Note the polymorphonuclear leukocytes (circled), which emigrate from the venule plexus in the subepithelial connective tissue (**3**) without altering it in any way.

Left: In the enlargement, a portion of the most coronal JE cells (cf. empty black arrow in lower power view) is shown still manifesting hemidesmosomes and an internal basal lamina attached to the enamel surface.

Courtesy *H. Schroeder*

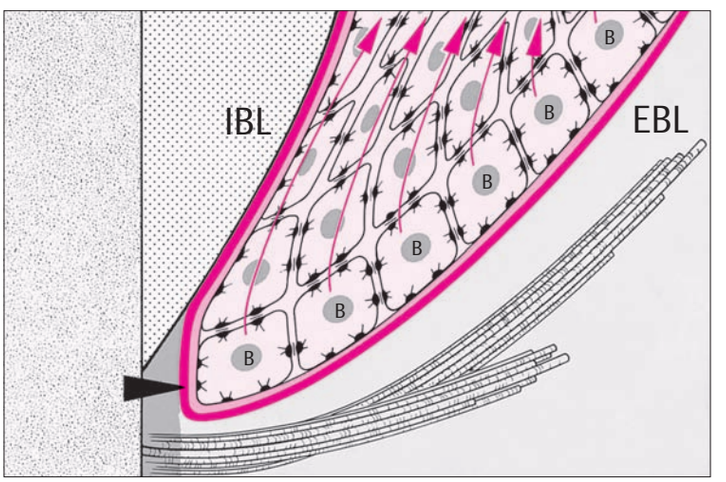




19 Internal Basal Lamina and Hemidesmosomes

Each JE cell adjacent to the tooth forms hemidesmosomes (**HD**) that enable these cells to attach to the internal basal lamina (**IBL**) and ultimately to the surface of the tooth. Remnants of enamel crystals are visible at the left. The long arrows indicate intercellular spaces between three JE cells (●).

Left: The basal lamina is comprised of two layers: the Lamina lucida (**LL**) and the Lamina densa (**LD**).



20 Apicalmost Portion of the Junctional Epithelium

In a young, healthy patient, the JE ends apically at the cemento-enamel junction. Daughter cells of the cuboidal basal cells (**B**) migrate toward the sulcus (red arrows). If a JE cell comes into contact with the tooth surface, it establishes the attachment mechanism described above. The internal basal lamina (**IBL**) is continuous with the external basal lamina (**EBL**) around the apical extent of the JE (black arrowhead).

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Connective Tissue Attachment

Gingival and Periodontal Fiber Apparatus

The fibrous connective tissue structures provide the attachment between teeth (via cementum) and their osseous alveoli, between teeth and gingiva, as well as between each tooth and its neighbor. These structures include:

- Gingival fiber groups
- Periodontal fiber groups (periodontal ligament)

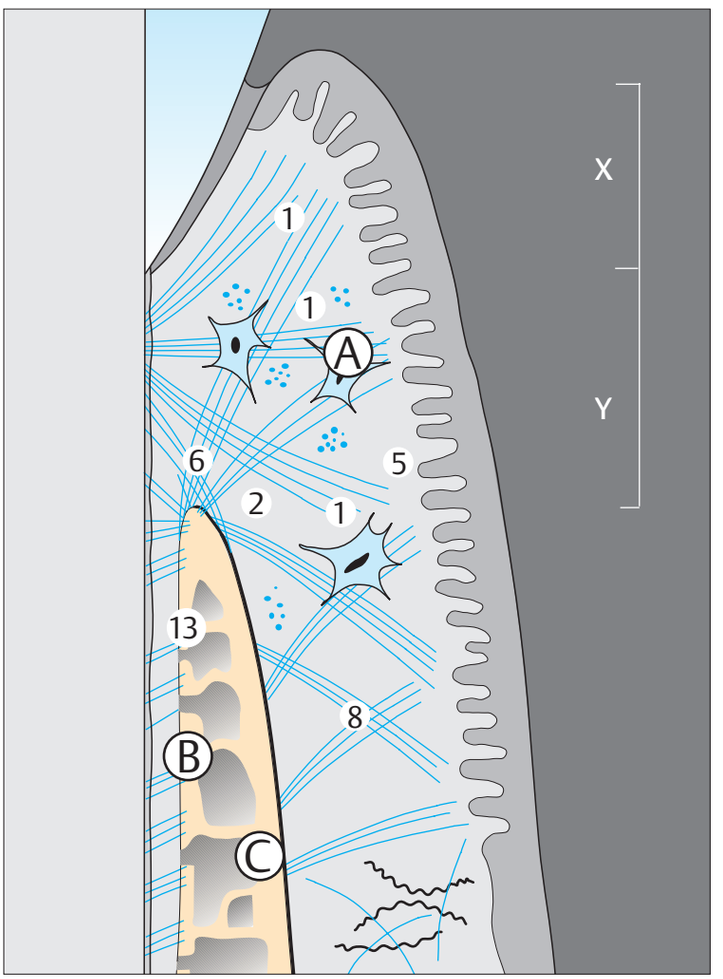
Gingival Fiber Groups

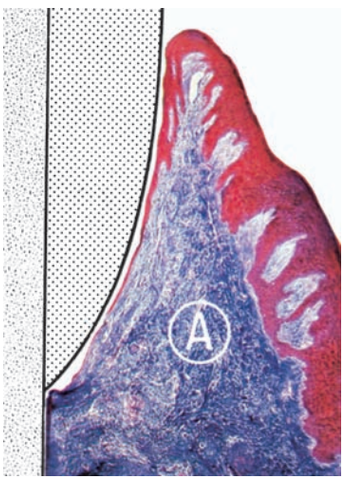
In the supra-alveolar area, collagen fiber bundles course in various directions. These fibers give the gingiva its resiliency and resistance, and attach it onto the tooth surface subjacent to the epithelial attachment. The fibers also provide resistance to forces and stabilize the individual teeth into a closed segment (Fig. 22). The periosteogingival fibers are also a component of the gingival fiber complex. These connect the attached gingiva to the alveolar process.

21 Localization and Orientation of Gingival and Periodontal Ligament Fiber Bundles (see also Fig. 22)
In the supra-alveolar region, within the free marginal gingiva and partially also the attached gingiva, the connective tissue compartment is composed primarily of collagen fiber bundles (**A**). These splay from the cementum of the root surface into the gingiva. Other fiber bundles course more or less horizontally within the gingiva and between the teeth, forming a complex architecture (Fig. 22). In addition to collagen fibers, one may also observe a small number of reticular (argyrophilic) fibers.
The periodontal ligament space (**B**) in adults is ca. 0.15–0.2 mm wide. About 60 % of the space is occupied by collagen fiber bundles. These fibers traverse from cementum to alveolar bone (**C**).

Right: Marginal gingiva. Fiber-rich connective tissue (**A**, blue), junctional epithelium and oral epithelium (reddish brown).

Histology courtesy *N. Lang*





A Gingival Fibers

B Periodontal Ligament Fibers

C Alveolar Bone

X Sulcus and Junctional Epithelium

Y Connective Tissue Attachment

X+Y **“Biologic Width” (BW)** (p. 490)

Periodontal Fiber Groups, Periodontal Ligament

The periodontal ligament (PDL) occupies the space between the root surface and the alveolar bone surface. The PDL consists of connective tissue fibers, cells, vasculature, nerves and ground substance. An average of 28,000 fiber bundles insert into each square millimeter of root cementum!

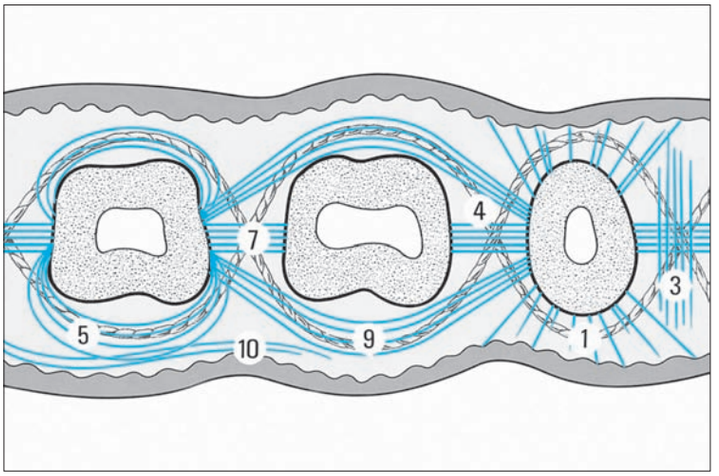
The building block of a fiber bundle is the 40–70 nm thick collagen fibril. Many such fibrils in parallel arrangement make up a collagen fiber. Numerous fibers combine to form collagen fiber bundles. These collagen fiber bundles (Sharpey’s fibers) insert into the alveolar bone on one end and into cementum at the other (Feneis 1952). The most ubiquitous cells are fibroblasts, which appear as spindle-shaped cells with oval nuclei and numerous cytoplasmic processes of varying lengths. These cells are responsible for the synthesis and break-down of collagen (“turnover”).
Cells responsible for the hard tissues are the cementoblasts and osteoblasts. Osteoclastic cells are only observed during phases of active bone resorption. Near the cementum layer, within the PDL space, one often observes string-like arrangements of epithelial rest cells of Malassez.

The periodontal ligament tissues are highly vascularized (p. 18) and innervated (p. 19).

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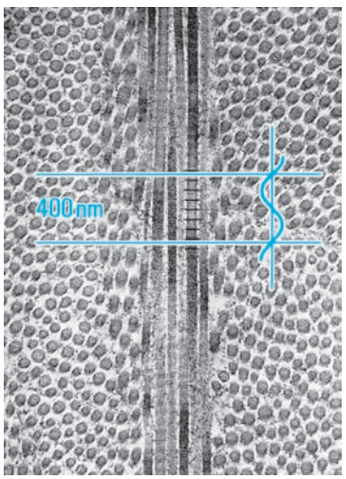
Course of the gingival fiber bundles (see also Fig. 21)

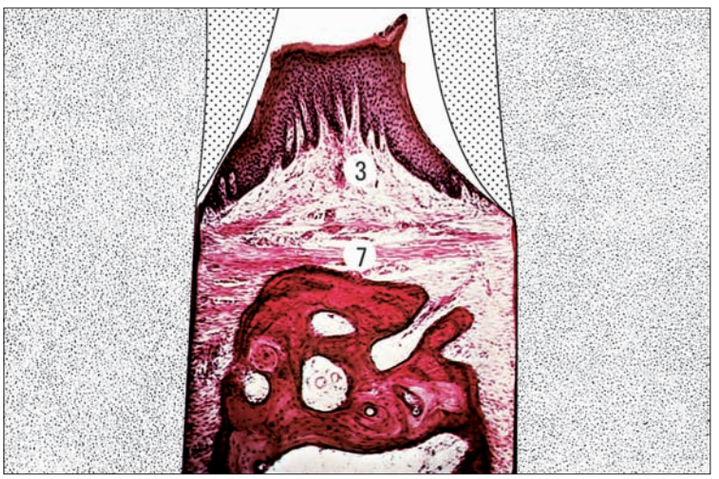
1 Dentogingival
– Coronal
– Horizontal
– Apical
2 Alveologingival
3 Interpapillary
4 Transgingival
5 Circular, semicircular
6 Dentoperiosteal
7 Transseptal
8 Periosteogingival
9 Intercircular
10 Intergingival



Gingival Fibers

22 Fiber Apparatus in Horizontal Section
The course of the most important supracrestal (gingival) fiber bundles is depicted. The connections between teeth and gingiva as well as between individual teeth are clearly shown.

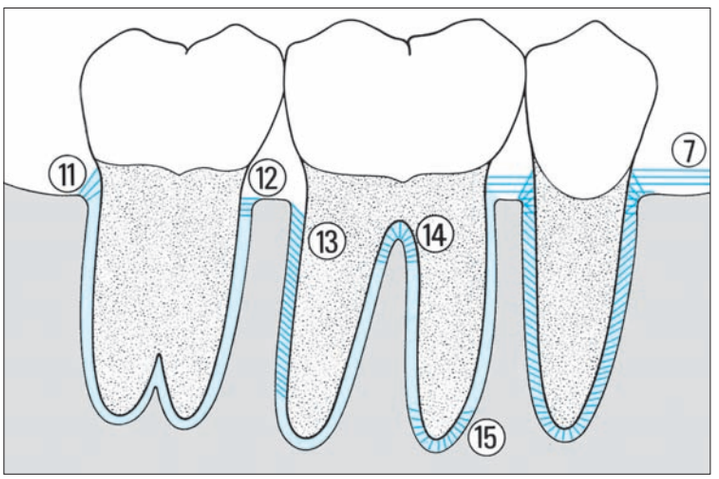




23 Fiber Bundles Viewed in Mesiodistal Section
In the interdental area, the transseptal fiber bundles (**7**) extend supragingivally from one tooth to its neighbor. The fibers stabilize the arch in its mesiodistal dimension (courtesy N. Lang).
Left: The basic element of the fiber bundle is the collagen fibril (cut in cross and longitudinal sections); such fibrils are secreted by fibroblasts, and exhibit a regular 64 nm banding pattern (compare the wavelength of blue light, 400 nm). TEM courtesy *H. Schroeder*

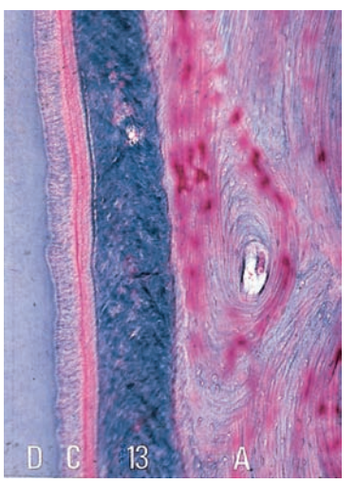
Course of the periodontal fiber bundles

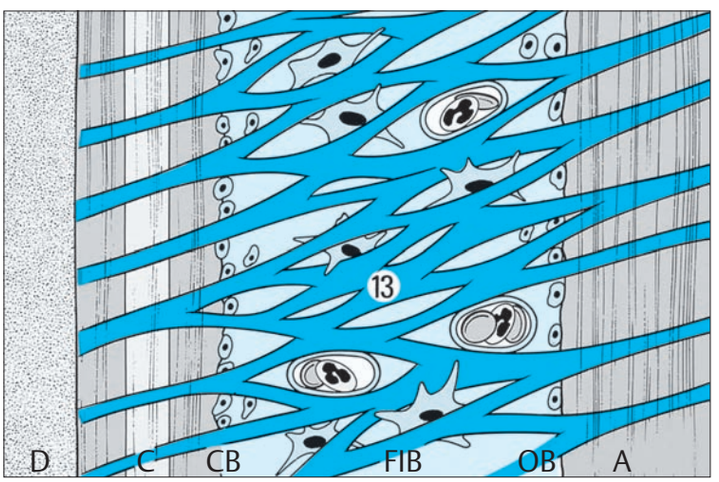
11 Crestal
12 Horizontal
13 Oblique
14 Interradicular
15 Apical



Periodontal Fibers

24 Fiber Apparatus in Mesiodistal Section
The anchoring of a tooth in the alveolar bone is accomplished via the dentoalveolar fibers of the periodontal ligament (PDL). Occlusal forces are absorbed primarily by the oblique fibers, which course from bone to cementum (**13**). The remaining fiber bundles (**11**, **12**, **14**, **15**) counteract tipping and rotating forces.





25 Periodontal Ligament—Details
Collagen fiber bundles (**13**) are intertwined. Osteoblasts (**OB**) line the surface of the bone; cementoblasts (**CB**) line the cementum surface; numerous fibroblasts (**FIB**) occupy the PDL space.

Left: The histologic section (Azan, x50) depicts the fiber-rich periodontal ligament (**13**) and its relationship to cementum (**C**) and bone (**A**). **D** = Dentin.

Histology courtesy *N. Lang*

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Root Cementum

Types of Cementum

From a purely anatomic standpoint, root cementum is part of the tooth, but also part of the periodontium. Four types of cementum have been identified (Bosshardt & Schroeder 1991, 1992; Bosshardt & Selvig 1997):

1 Acellular, afibrillar cementum (AAC)
2 Acellular, extrinsic-fiber cementum (AEC)
3 Cellular intrinsic-fiber cementum (CIC)
4 Cellular mixed fiber cementum (CMC)

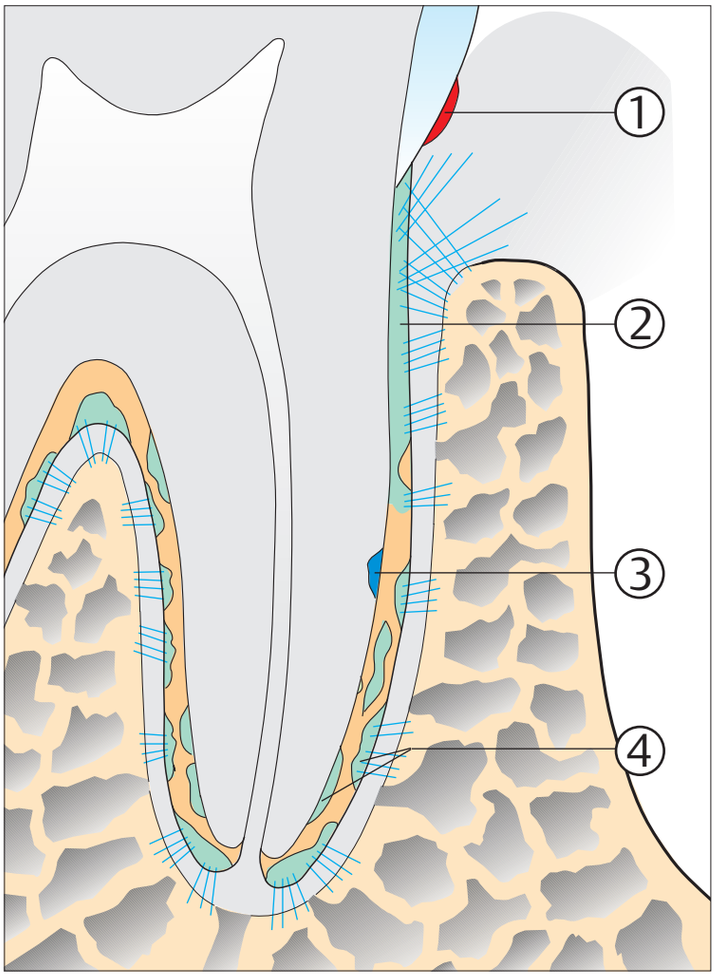
AEC and CMC are the most important types of cementum.

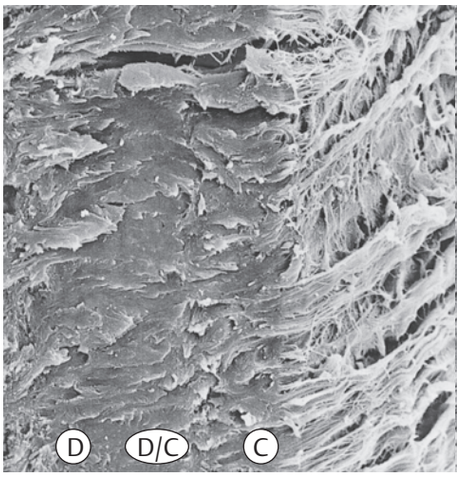
Cementum-forming Cells

Fibroblasts and cementoblasts collaborate in the formation of cementum. *Periodontal ligament fibroblasts* secrete acellular extrinsic cementum. *Cementoblasts* secrete cellular intrinsic cementum, and a portion of the cellular mixed fiber cementum, and probably also acellular afibrillar cementum. *Cementocytes* evolve from the cementoblasts, which become entrapped in cementum during cementogenesis. As a result, cementocytes are observed within cellular mixed fiber cementum and frequently in cellular intrinsic cementum (see also Cementum Formation and Healing, p. 206).

26 Types of Cementum—Structure, Localization and Development

1 **Acellular, Afibrillar Cementum** (AAC; red) AAC is formed at the most cervical enamel border following completion of pre-eruptive enamel maturation, and sometimes also during tooth eruption. It is probably secreted by cementoblasts.
2 **Acellular, Extrinsic-fiber Cementum** (AEC; green) AEC forms both pre- and post-eruptively. It is secreted by fibroblasts. On the apical portions of the root, it comprises a portion of the mixed-fiber cementum.
3 **Cellular, Intrinsic-fiber Cementum** (CIC; blue) CIC is formed both pre- and post-eruptively. It is synthesized by cementoblasts, but does not contain extrinsic Sharpey's fibers.
4 **Cellular, Mixed-fiber Cementum** (CMC; orange/green) CMC is formed by both cementoblasts and fibroblasts; it is a combination of cellular intrinsic-fiber cementum and acellular extrinsic-fiber cementum.





See p. 15, Fig. 29, left

Acellular Extrinsic Cementum (AEC)

The AEC is primarily responsible for the anchorage of the tooth in the alveolus. It is found in the cervical third of all deciduous and permanent teeth. The AEC consists of tightly packed and splaying fiber bundles (Sharpey's fibers), which are embedded in the calcified cementum.

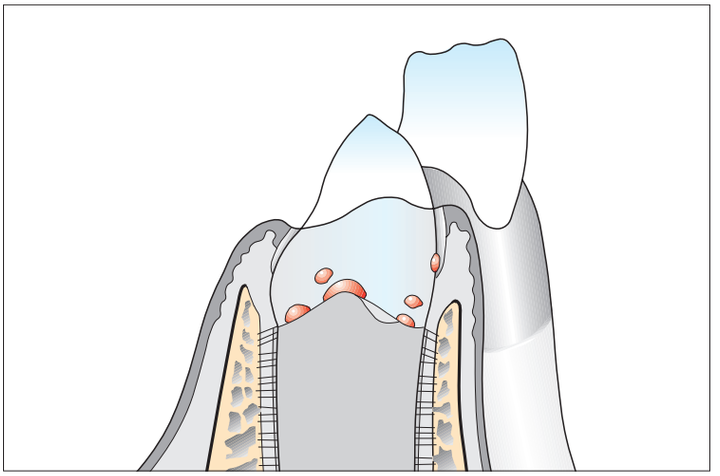
The collagenous structures of cementum and dentin intertwine with each other during root formation and before calcification. This phenomenon explains the tight connection between these two hard tissues.

AEC is the type of cementum that is desired following regenerative periodontal surgical procedures.

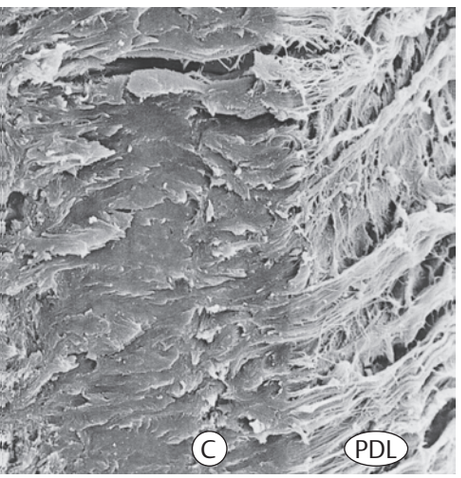
Cellular Mixed-fiber Cementum (CMC)

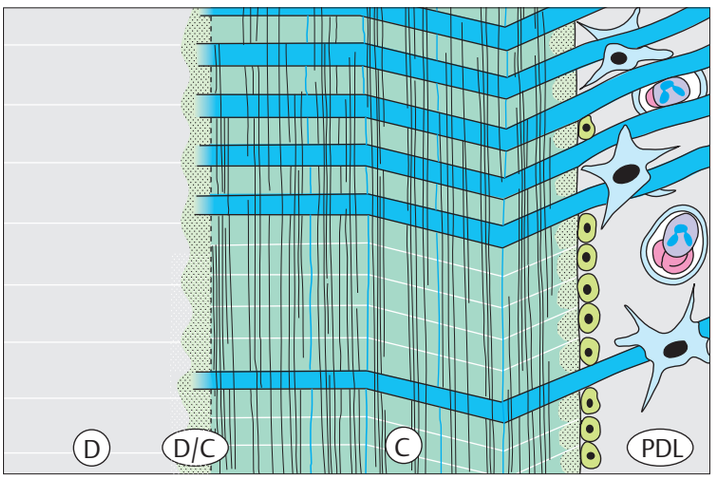
The CMC is also of importance for the anchorage of the tooth in its alveolus. But it is only the acellular extrinsic-fiber cementum portion (AEC) within the mixed cementum, into which the Sharpey's fibers secreted by fibroblasts insert and therefore affix the tooth. CMC is layered vertically but also horizontally to the root surface. The portions secreted by cementoblasts contains high numbers of cementocytes (Fig. 30, left). The CMC is also tightly affixed to the dentin because of the intertwining of the collagen fiber bundles during tooth formation. The CMC "grows" faster than AEC.

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27 Acellular, Afibrillar Cementum (AAC)
AAC is observed only at the cervical region of the tooth, at the cementoenamel junction. It appears as small "islands" upon the enamel and sometimes also on the marginal areas of the root. AAC is formed during tooth eruption, when the reduced enamel epithelium partially dissolves as the enamel surface comes into contact with connective tissue.





28 Acellular, Extrinsic-fiber Cementum (AEC)
AEC is localized to the coronal third of the root (**C**), and exhibits a horizontal fiber structure (blue). Variations in the directionality may occur when the tooth encounters positional changes during cementum formation.

Left and p. 14: Note the intense intertwining and connections between dentin (**D**) and cementum (**C**), as well as of cementum and periodontal ligament (**PDL**).

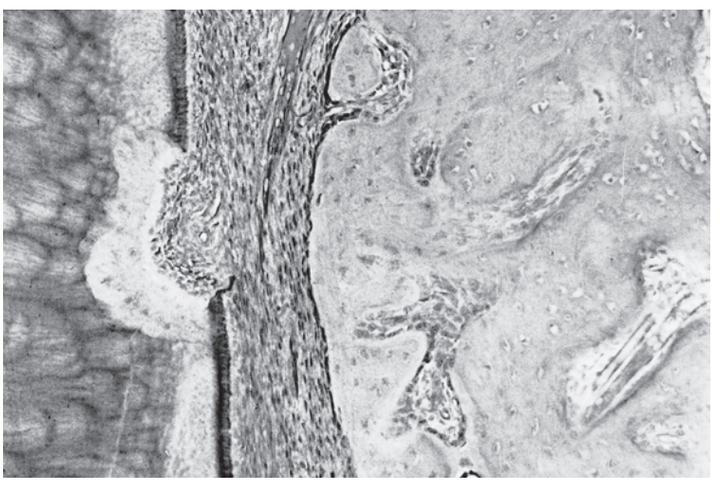
Abbreviations

D Dentin

D/C Dentin-Cementum interface

C Cementum
C2 AEC
C4 CMC

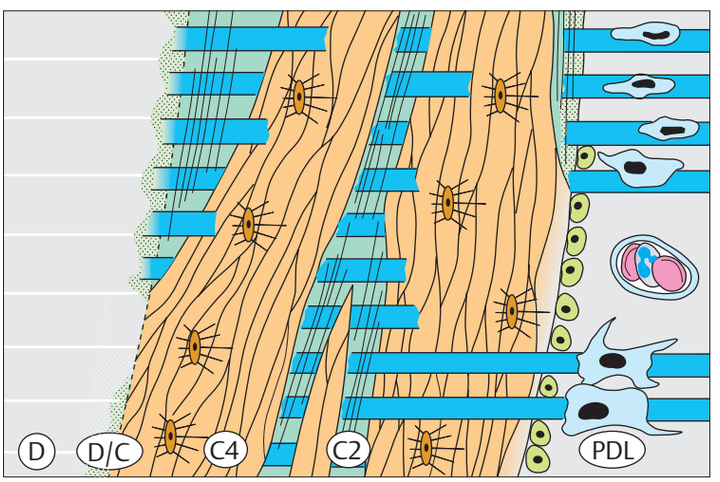
PDL Periodontal ligament



29 Cellular, Intrinsic-fiber Cementum (CIC)
CIC is usually a component of mixed-fiber cementum. It is localized to the middle, apical and furcal regions of the roots and usually contains embedded cementocytes.

As shown in this figure, CIC is also "reparative" cementum, i. e., it can repair resorption areas and root fractures.





30 Cellular, Mixed-fiber Cementum (CMC)
CMC is found in the apical portion of the root and in the furcation region. It represents a mixture of acellular extrinsic-fiber cementum (**C2**) and cellular intrinsic-fiber cementum (**C4**).

Left: "Medusa"-like cementocytes in the apical region of a multirooted tooth.

Adapted from *D. Bosshardt, H. Schroeder*

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Osseous Support Apparatus

Alveolar Process—Alveolar Bone

The *alveolar processes* of the maxilla and the mandible are tooth-dependent structures. They develop with the formation of and during the eruption of the teeth, and they atrophy for the most part after tooth loss. Three structures of the alveolar process may be discriminated:

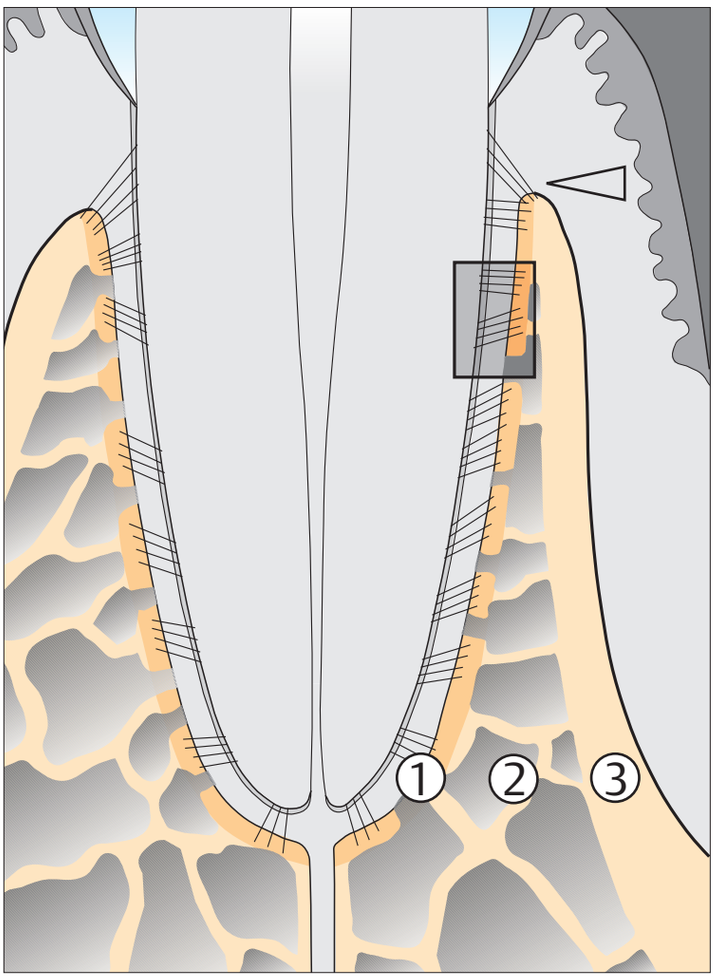
- Alveolar bone proper
- Trabecular bone
- Compact bone

Compact bone covers and contains the alveolar processes. At the entrance to the alveoli, the alveolar crest, it blends into the cribriform plate, the alveolar bone proper, which forms the alveolar wall and is approximately 0.1–0.4 mm thick. It is perforated with numerous small canals (Volkmann canals) through which vessels as well as nerve fibers enter into and exit the periodontal ligament space. The *trabecular bone* occupies the space between compact bone and alveolar bone proper. The distance between the marginal gingiva and the alveolar crest is referred to as the "biologic width" of 2–3 mm (Gargiulo et al. 1961, see p. 490).

31 Osseous Support Apparatus
The tooth-supporting alveolar process consists of the alveolar bone (**1**), trabecular bone (**2**), and compact bone (**3**). Alveolar bone and compact bone join at the margin to form the alveolar crestal bone (arrow). In this region, the alveolar process is often extremely thin, especially on the facial aspect, and unsupported by trabecular bone (Fig. 36).

Right: Histologic section (HE, x10) through the periodontium (the location is indicated by the rectangle superimposed in the main figure). On the right side of the picture, the alveolar bone with its osteons and a Haversian canal is clearly visible. Bundle bone has been deposited adjacent to the structures on the periodontal aspect.
The periodontal ligament is cell-rich, and exhibits a thin layer of cementum-forming fibroblasts along the acellular, extrinsic-fiber cementum (left).

Histology courtesy *H. Schroeder*





1 Alveolar Bone
Synonyms:

Anatomically
– Alveolar Wall
– Cribriform Plate

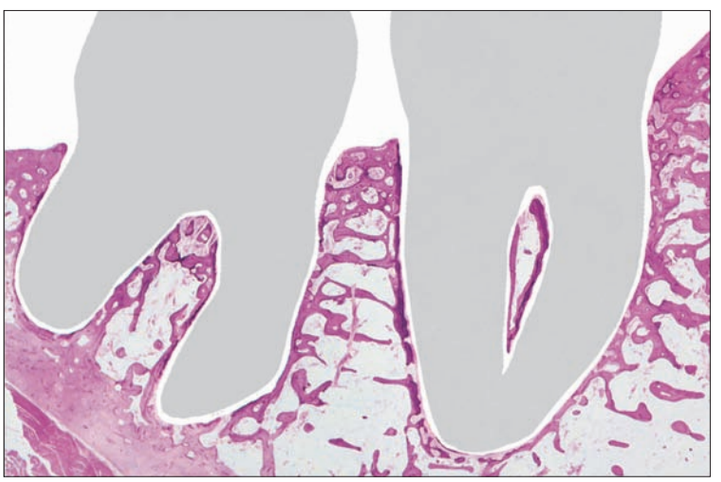
Radiographically
– Lamina dura

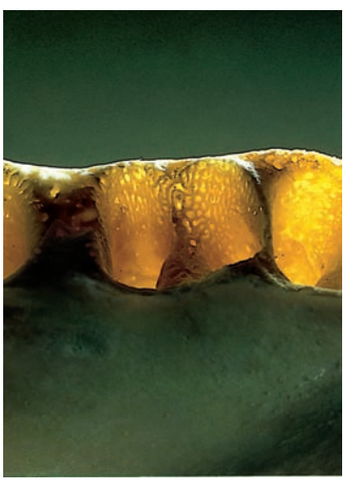
2 Trabecular Bone

3 Compact Bone

32 Mandibular Alveolar Process in Sagittal Section
In this histologic section (H and E, x1) the elegant structure of the trabecular bone and the more or less large marrow spaces are visible. The *alveolar bone proper* is depicted only as a very thin, often partially broken line.

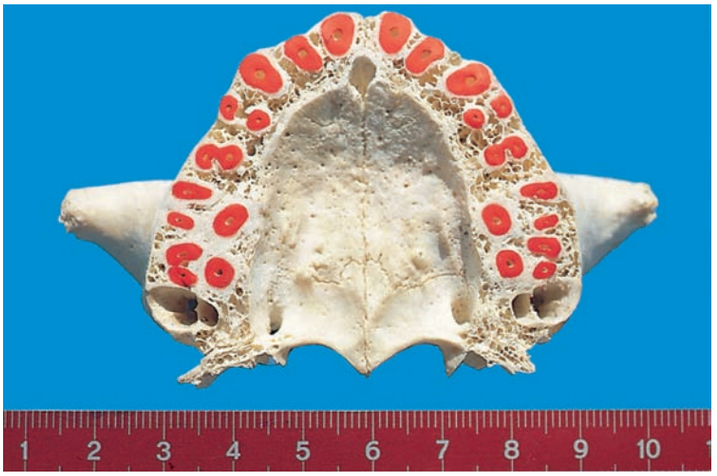
Right: In this transilluminated bone preparation, it becomes clear that the alveolar bone is perforated by numerous small holes, as in a sieve (cribriform plate).





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Maxilla



Maxillary Bone

33 Maxillary Alveolar Process in Horizontal Section
The section is through the alveolar process and tooth roots at about the midpoint. With the exception of the molar areas, the bone is thicker on the oral surface than on the facial. The trabecular bone is variably thick. Clearly visible is the varying mass of the bony interdental and interradicular septa, as well as the varying shape of the root cross sections.





34 Maxilla—Frontal Section
This section was cut through the plane of the canine, and it shows the relationship of the root to the nasal sinus. Clearly visible is the very thin bony layer on the facial surface of the root (Fig. 36).

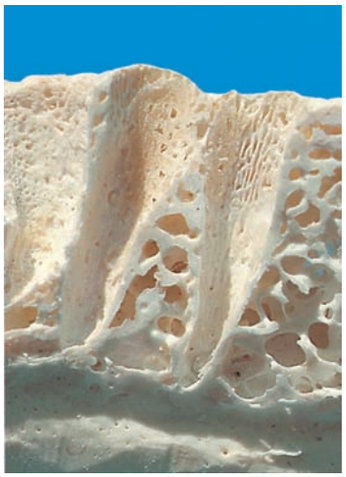
Left: The sagittal section shows how the root tips of premolars and molars sometimes extend into the maxillary sinus. The alveolar bone may actually border directly on the sinus mucosa.

Mandible



Mandibular Bone

35 Mandibular Alveolar Process in Horizontal Section
The section represents a cut approximately halfway down the tooth root. In contrast to the maxilla, the orofacial width of the mandibular alveolar process is considerably less. All roots exhibit an “hourglass” profile (proximal concavities).





36 Orofacial Sections through the Mandible
From right to left, an incisor, a canine, a premolar and two molars were sectioned. Impressive is the thin, tapered bony lamella on the facial aspect; it is impossible to distinguish between compact bone and alveolar bone proper.

Left: Sagittal section through the alveolar process and alveoli. Especially in the molar area, the alveolar bone is traversed by numerous Volkmann canals.

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Blood Supply of the Periodontium

All periodontal tissues, but especially the periodontal ligament, have a copious blood supply even in the healthy state. This is due not only to the high metabolism of this cell- and fiber-rich tissue, but also to the peculiar mechanical/functional demands on the periodontium. Occlusal forces are resisted not only by the periodontal ligament and the alveolar process, but also by means of the tissue fluid and its transfer within the periodontal ligament space (*hydraulic pressure distribution,* dampening).

The most important afferent vessels for the alveolar process and the periodontium are:

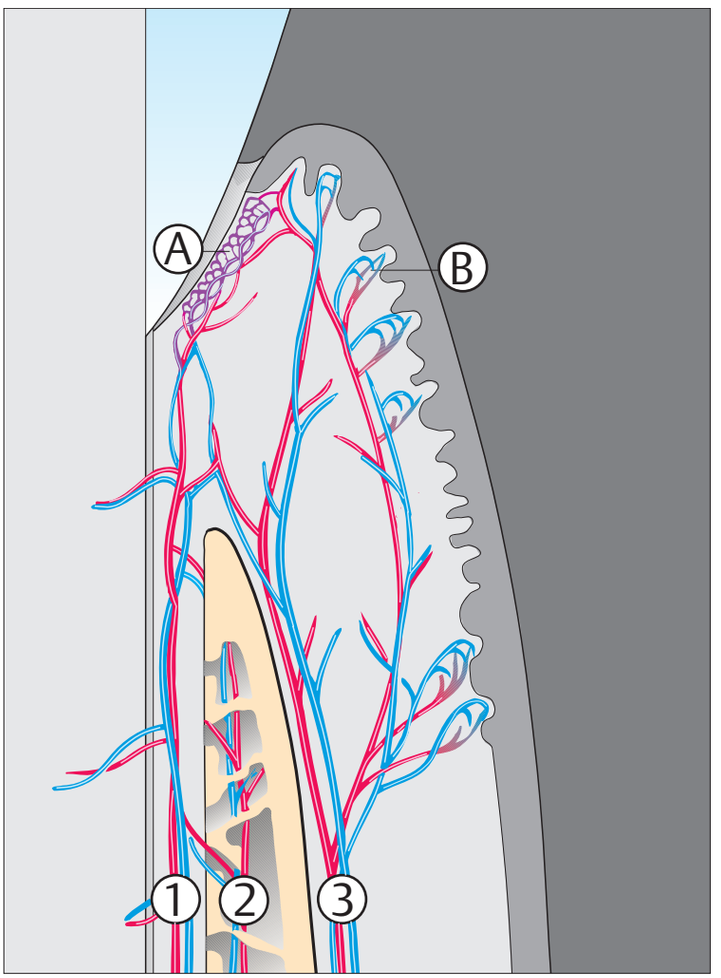
- In the *maxilla*, the anterior and posterior alveolar arteries, the infraorbital artery and the palatine artery
- In the *mandible*, the mandibular artery, the sublingual artery, the mental artery, and the buccal and facial arteries.

Lymph vessels follow for the most part the blood vascular tree.

37 Diagram of Periodontal Blood Supply
The periodontal ligament (**1**), the alveolar process (**2**) and the gingiva (**3**) are supplied by three vascular sources. These vessels exhibit frequent anastomoses.
Within the periodontal ligament, the vascular network is especially dense. Adjacent to the junctional epithelium, these vessels splay into a very dense vascular system, the capillary-venule plexus (**A**). This plexus is of great significance for host defense against infection (p. 55).
The oral epithelium contacts the subjacent connective tissue through a series of rete ridges. Each rete ridge of connective tissue contains *capillary loops* (**B**).

Right: In this section beneath the JE, a dense *vascular plexus* (**X**) is observed even in health. Above the white arrows, one observes the most marginal vascular loops in the area of the adjacent, nonkeratinized oral sulcular epithelium (**OSE**).

Courtesy *J. Egelberg*





Blood Supply Pathways

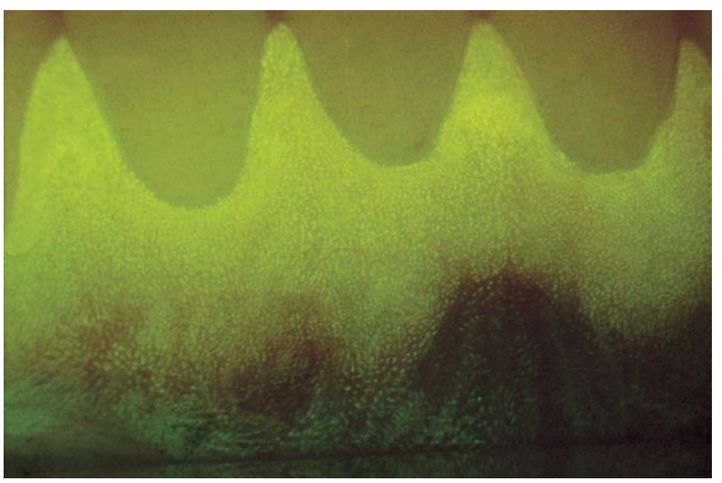
1 Periodontal
2 Alveolar
3 Supraperiostal/mucogingival

A Post-capillary Venous Plexus

B Sub-epithelial Capillary Loops

38 Fluorescence Angiography—Vascular Loops Subjacent to the Oral Epithelium
Following intravenous injection of 2 ml of sodium fluorescein solution (20 %), the vessels (capillaries) subjacent to the oral epithelium can be rendered visible by UV light.
Some small vascular loops are visible in the connective tissue rete pegs (**B** in Fig. 37).

Courtesy *W. Mörmann*



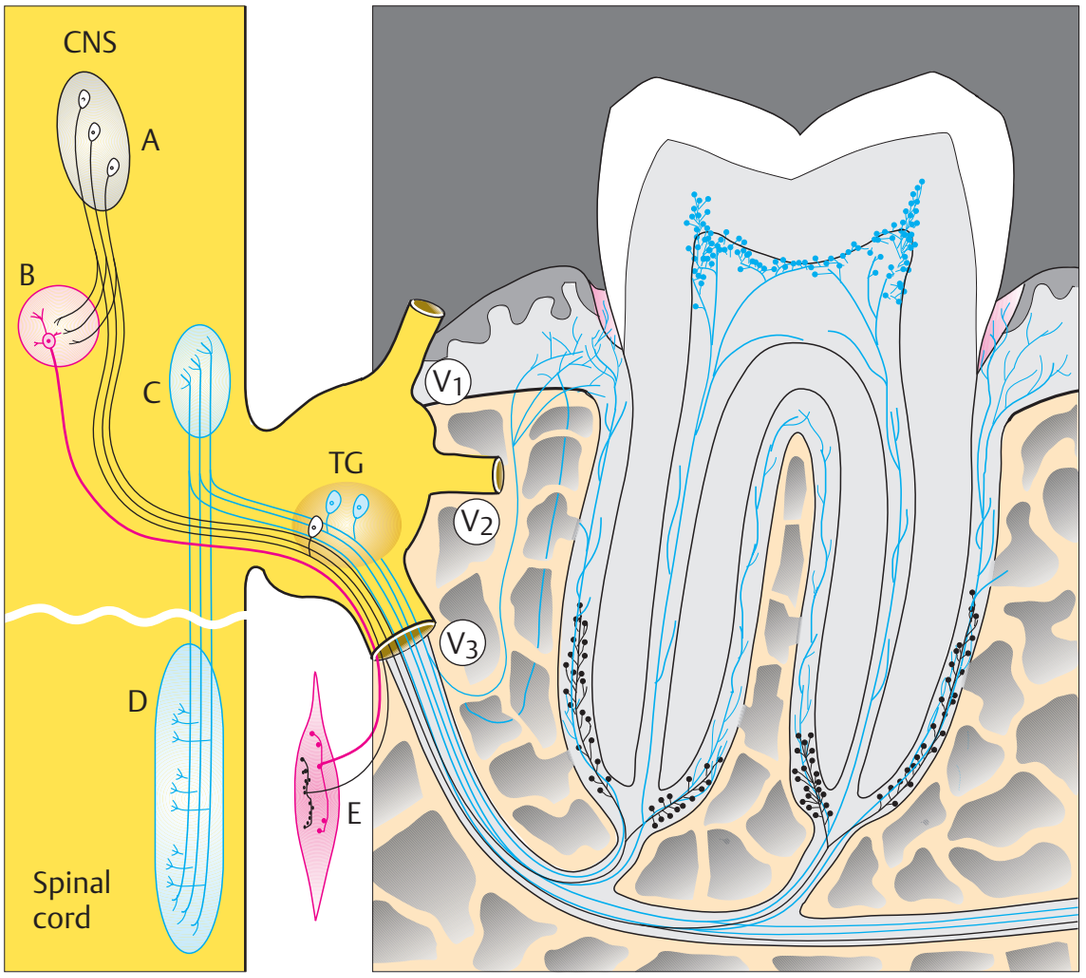
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Innervation of the Periodontium

The sensory innervation of the maxilla occurs via the second branch of the trigeminal nerve, and that of the mandible via the third branch. The following description of the neural distribution within the periodontal structures is based upon investigations by Byers (1985), Linden et al. (1994) and Byers & Takeyasu (1997).

The periodontium, especially the gingiva and periodontal ligament, contains "Ruffini-like" *mechanoreceptors* and *nociceptive nerve fibers,* in addition to the ubiquitous branches of the sympathetic nervous system.

The functions of these innervations are coordinated with those of the dental pulp and the dentin. The stimulus threshold of the mechanoreceptors, which react to tactile (pressure) stimulus, as well as to the stretching of the periodontal ligament fibers, is very low. In contrast, the pain-sensing nociceptive nerve endings have a relatively high threshold. It is via these two separate afferent systems that "information" about jaw position, tooth movements, speech, tooth contact during swallowing and chewing, minor positional alterations (physiologic tooth mobility), pain during unphysiologic loading, as well as injuries are



39 Innervation of a Mandibular Molar

Innervation of gingival and periodontal structures is via the mandibular nerve, the third branch of the trigeminal nerve.

Modified from *M. Byers*

A Mesencephalic sensory neurons of the trigeminal nerve
B Motor nucleus of the trigeminal
C Sensory nucleus of the trigeminal
D Spinal sensory trigeminal nucleus
E Fibers of the masticatory musculature

TG Trigeminal ganglion (Gasserian ganglion) with its three branches:

V_1 Ophthalmic
V_2 Maxillary
V_3 Mandibular

CNS Central Nervous System

transmitted. In this way, various mechanoreceptors transmit "conscious reactions" via trigeminal ganglia to the sensory nucleus of the trigeminal in the central nervous system, while unconscious reflexes transmit to mesencephalic sensory neurons. These various receptors are localized in varying regions of the periodontal structures: At the level of the middle of the root, one finds more receptors for up-take of "conscious reactions," whereas in the apical region there are more receptors for the unconscious reflexes whose signals transmit to the mesencephalic sensory neurons.

The junctional epithelium as well as the epithelia of the free and attached gingiva, neither of which are vascularized, are served by a dense network of nociceptive and tactile nerve endings. The same is true for the subepithelial, supracrestal gingival connective tissue.

Somatosensory perception in certain gingival diseases (e.g., ulcerative gingivoperiodontitis), as well as pressure and pain sensation during probing of the healthy gingival sulcus or periodontal pockets are the clinical manifestations of the innervation of gingival tissues.

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The Coordinated Functions of the Periodontal Structures

Turnover—Adaptation—Defense—Healing

Within the healthy periodontium, there is a constant *turnover* of all tissues, except cementum. The term *tissue homeostasis* was coined to characterize the process by which the composition of the various structures, i.e., the balance of their volumes, their integrity vis-à-vis each other, and their mutually interwoven functions (Williams et al. 1997). The apposition and/or resorption of the various tissues can vary even in the healthy condition, depending upon several factors; for example, the periodontal tissues undergo a process known as *adaptation*, or in cases of reduced occlusal loading (afunction, hypofunction) or increased loading (hyperfunction, parafunction). This concept is not limited only to an adaption to masticatory forces, but is broader in the sense of all "insults" that the periodontal tissues may encounter, including the always present—although in very differing degrees—infection.

Host defense against all "attacks" refers primarily to the immune system (p. 41), but also to the healthy tissue. Disease (periodontitis) will ensue if the demands upon the tissues are larger than their capability to reactively adapt.

The adaptability of the tissues, i.e., their ability to vary the rate of turnover in response to various mediators (e.g., cytokines) also plays an important role in *healing*, for example following injury or after mechanical periodontal therapy (scaling and root planing—S/RP).

Primary Functions of the Periodontal Tissues

Epithelium

The epithelium of the attached gingiva (OE, oral epithelium) is referred to as masticatory mucosa. Like the palatal mucosa, it is keratinized. Keratinization is a mechanism for *protection against all mechanical challenges, but also against thermal, chemical and infectious attack*.

The turnover rate of the gingival epithelium has been variously reported between 6 (Schroeder 1992) and 40 days (Williams et al. 1997). Probably it is influenced by "chalones" (substances which inhibit mitosis) on the one hand and by cytokines (Fig. 95), e.g., epidermal growth factor (EGF) and transforming growth factor (TGF-b) on the other.

The junctional epithelium exhibits a faster turnover rate than gingival epithelium. Cell division occurs in the basal cell layer. All of the daughter cells migrate in the direction of the gingival sulcus, where they are rapidly sloughed. Through this constant flow of junctional epithelial cells, sulcus fluid, and active migration of granulocytes (PMN), the sulcus is effectively cleared of invading bacteria and their metabolic products. In addition to immunocompetent cells, it is also primarily the dynamics of the resident tissue cells and the coronally streaming flow of tissue fluid that is responsible for the *prevention of infection* and therefore also for the maintenance of health of the marginal periodontal structures.

Gingival Connective Tissue

The circumstance for the periodontal connective tissue is similar to that of the epithelial structures. It has a turnover rate of only a few days, orchestrated by cytokines and growth factors (platelet-derived growth factor/PDGF; fibroblast growth factor/FGF, among others). In this matrix, it is the fibroblasts that are responsible for the synthesis and breakdown of collagen matrix. The matrix metalloproteinases (Fig. 102) that are responsible for the breakdown of collagen depend upon divalent cations (e.g., Zn^{+2}). The balance between synthesis and breakdown can be "shifted" to a certain degree toward synthesis in the presence of pathogenic influences. However, if the pathogenic insult is too great, the process of increased resorption (or reduced synthesis?) can occur and lead ultimately to tissue destruction.

Periodontal Bone

Bone synthesis and resorption, especially the bone loss characteristic of periodontitis, is covered in detail in the chapter "Etiology and Pathogenesis" (pp. 60–61).

Cementum

In contrast to epithelium, connective tissue and bone, cementum is not subject to continuous turnover. Throughout life, cementum tends to increase in thickness due to apposition. Local resorption observed as resorption lacunae may result from trauma, orthodontic forces, or they may be idiopathic. Such defects are often "repaired" by synthesis of cellular intrinsic-fiber cementum.

Summary

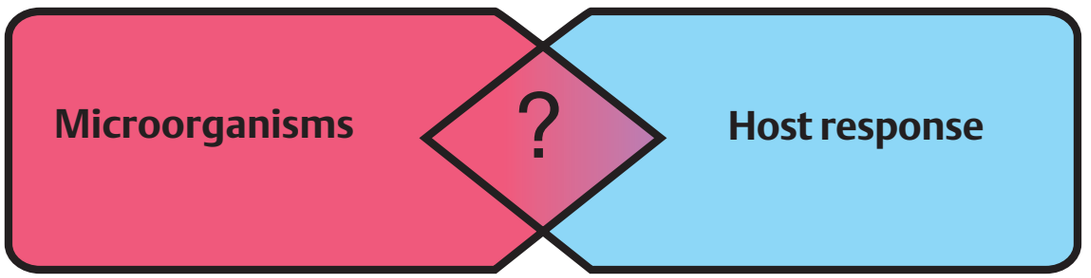
The healthy periodontal structures have the capacity to mount a certain defense potential even before the actual immune response is mounted; the latter, of course, also introduces certain destructive elements into the milieu.

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Etiology and Pathogenesis

The most common diseases of the tooth-supporting apparatus are plaque-induced, usually chronic, inflammatory alterations in the gingiva and the subjacent periodontal structures.
Gingivitis may persist for many years without progressing to periodontitis. With good oral hygiene and effective professional removal of plaque and calculus, gingivitis is completely reversible.
Periodontitis usually develops out of a more or less pronounced gingivitis. Periodontitis is only partially reversible (see periodontal healing, p. 205; regenerative therapies, p. 323).

The reasons why gingivitis develops into periodontitis (or does *not*) are still incompletely understood. As with all *infections*, it appears that the proliferation of pathogenic microorganisms, their toxic potency, their capacities to invade tissues, and above all the individual host response to such infection are the determining factors (p. 55; Kornman et al. 1997, Page & Kornman 1997, Salvi et al. 1997).



40 Bacterial Attack vs. Defense Reactions by the Host
The virulence of bacteria, their capability to invade into tissues, their number and composition, as well as their metabolic products elicit reactions by the host (immune response). Whether periodontal disease will occur, and its severity, depends only partially upon the bacterial insult. Transition from gingivitis to periodontitis involves factors such as host response, risk factors, and local factors.

An absolutely plaque-free condition in the oral cavity, with prevention of any biofilm formation on tooth surfaces is unachievable, and illusion, and probably even unphysiologic. Nevertheless, gingival and periodontal health can be maintained if the accumulation of plaque is small and if the biofilm contains only weakly virulent organisms (gram-positive, facultative anaerobes), and if an effective host response is mounted.
If the bacterial flora takes on periodontally pathogenic characteristics (e.g., certain gram-negative microorganisms), the result will be inflammation and specific immunologic responses; these responses may represent not only defense mechanisms, but—especially in long-term chronic infection—also destructive potential (cytotoxic, immunopathologic; p. 34).
Inflammation-inducing products of bacteria include enzymes, antigens, toxins, and "signal" substances that activate macrophages and T-cells (Birkedal-Hansen 1998). It is likely that bacterial enzymes, other metabolic products and toxins can directly elicit injury to the periodontal tissues even *without* immediate host response (inflammation). Bacterial products including hyaluronidase, chondroitin sulfatase, proteolytic enzymes, as well as cytotoxins in the form of organic acids, ammonia, hydrogen sulfide and endotoxins (e.g., lipopolysaccharides, LPS) have been demonstrated in periodontal tissues.

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Periodontitis—A Multifactorial Disease

In recent years, the conceptual view concerning the etiology of periodontitis has evolved. Early on, it was the bacteria that were viewed as the determining factor. Certain pathogenic microorganisms were shown to be associated with various forms of periodontal disease, as well as the speed of progression. However, the existence and distribution of pathogenic bacteria did not always correlate with the inception and clinical progression of periodontitis. Furthermore, it was demonstrated that the presence of pathogenic bacteria in a periodontal pocket is not necessarily the *cause* of that pocket; rather, it seemed much more important that the pocket milieu presents a favorable environment for the existence and proliferation of pathogenic organisms. The stage would then be set—like a vicious cycle—for the progression of the disease processes (Mombelli et al. 1991).

Nevertheless, the old adage *"no bacteria = no periodontitis"* still holds true, but on the other hand it is also a fact that bacteria, including periodontopathic bacteria, do not without exception cause periodontitis.

41 Etiology of Periodontitis—Interaction between Dental Plaque and the Host

Bacteria

1 The primary etiologic factor for the existence of periodontitis is pathogenic microorganisms within the subgingival biofilm.

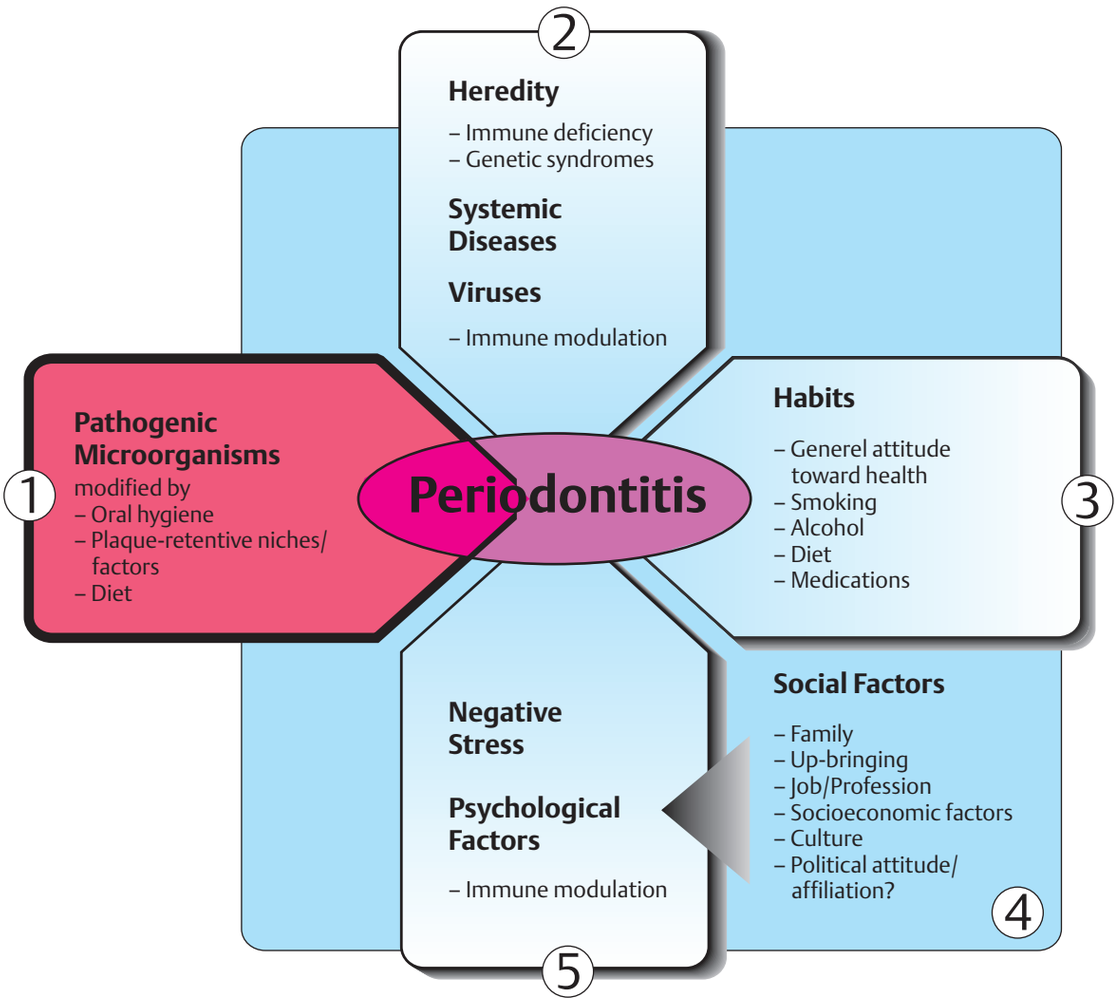
Host

2 The genetically determined non-specific and specific immune responses, as well as systemic syndromes and diseases influence the existence and the clinical course of periodontitis.

3 "Habits" and the patient's own approach to general health will influence plaque formation and host immune response, both systemically and particularly with regard to oral health.

4 Social circumstances influence the systemic and psychic well being of the patient. Problems in the socioeconomic arena lead to negative stress.

5 Psychic burdens and stress influence the immune status.



In addition to specific microorganisms, diverse *host factors* are critical for the development of periodontitis from a preexisting gingivitis (cf. Fig. 41, modified from Clarke & Hirsch 1995). Such factors include the immune responses triggered by pathogens, and these are well understood today. Such defense reactions may be disproportional to the insult, resulting in immunopathologic tissue injury.

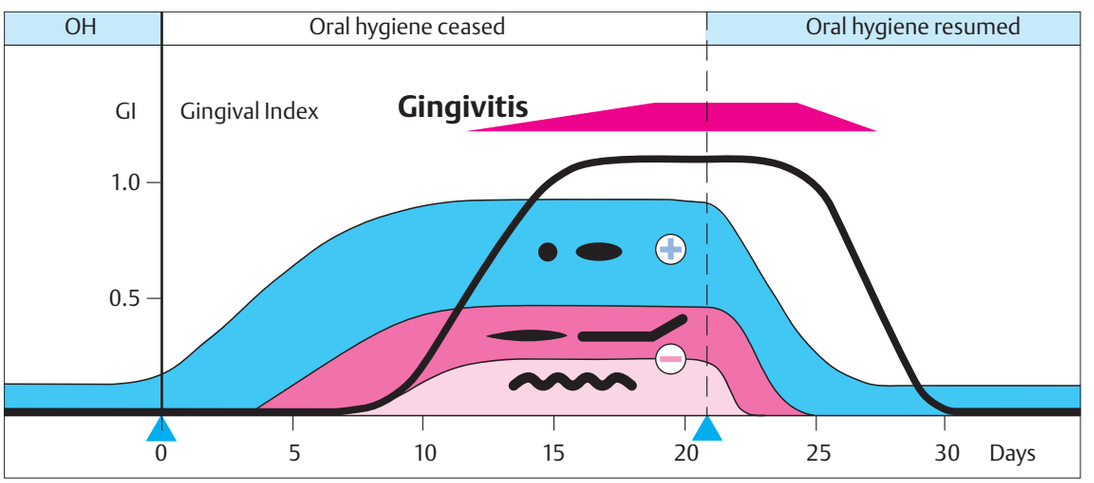
Recently, however, in addition to the genetically determined immune reactions, a great number of other individual risk factors have been identified, which may be responsible for the initiation and the degree of severity of the clinical course of periodontitis (p. 51).

Of the risk factors listed in Figures 41 and 104, only a few are capable of damaging the periodontium directly (e. g., smoking); of much greater importance is the influence of such factors on the patient's own immune system. The delicate balance between "attack/destruction" (bacteria) and defense (host response) is disturbed. It is only logical to assume that the most severe, early-onset and aggressive forms of periodontitis will occur when particularly virulent bacteria are present in a weak (immunodeficient) host.

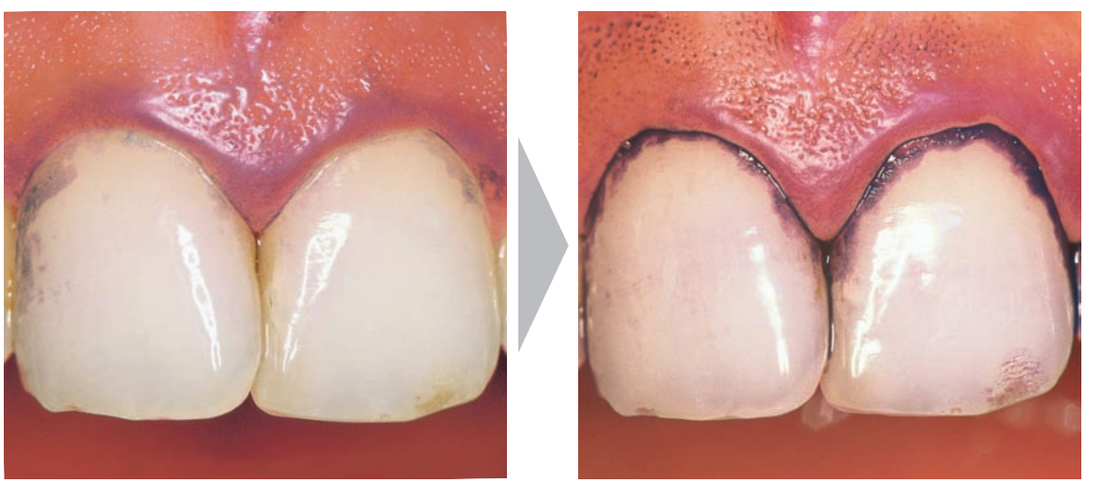
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Microbiology

Bacteria are present throughout life in a myriad of sites on and in the human body. The bacteria may be beneficial for the host, or of no consequence (commensal), or injurious. In the oral cavity, over 530 different species of microorganisms have so far been identified; fortunately, for the most part these organisms remain in ecological balance and do not cause disease. Certain facultatively pathogenic ("opportunistic") bacteria are occasionally observed in high numbers, e. g., in cases of disease (periodontitis, mucosal infection). It remains unclear whether these bacteria alone represent the cause of the diseases, or whether they simply find favorable living conditions in the disease milieu. *Non-specific* supragingival plaque (mixed flora) will elicit gingivitis within ca. seven days. If the plaque is removed, gingivitis regresses in a short period of time ("reversibility"). On the other hand, for the various forms of periodontitis, especially the aggressive, rapidly-progressing forms, *specific* bacteria are associated.



42 Experimental Gingivitis
In 1965, *Löe et al*. published the classic experimental proof of the *bacterial etiology of gingivitis*. In plaque- and inflammation-free subjects, plaque begins to accumulate if all oral hygiene is ceased. For the first few days, this plaque is composed of gram-positive cocci and rods, then later of filamentous organisms and finally spirochetes (gram-negative). Within a few days, a mild gingivitis ensues. If the plaque is removed, the gingiva quickly returns to a state of health.



43 Healthy Gingiva and Initial Gingivitis
Left: "Almost" clean central incisors. An extremely thin biofilm is compatible with clinically healthy gingiva. A few bacteria may even serve to maintain the "memory" of the immune system (p. 55).

Right: Plaque accumulation following seven days without oral hygiene. Gingivitis has developed. The proliferation and relative increase in numbers of gram-negative microorganisms are responsible for the gingivitis.

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Biofilm—Plaque Formation on Tooth and Root Surfaces

The oropharynx is an open ecosystem wherein bacteria are always present; bacteria attempt to colonize in all favorable locations. Most bacteria, however, can only persist after the formation of a biofilm upon desquamation-free surfaces, i.e., hard substances (tooth and root surfaces, restorative materials, implants, prostheses etc.). In the presence of healthy dental and gingival relationships, there is a balance between the additive and retentive mechanisms of biofilms vis-à -vis the abrasive forces that tend to reduce biofilm formation, e.g., self-cleansing by the cheeks and tongue, diet and mechanical oral hygiene measures.

The existence of a biofilm results within a matter of hours or days, in the phases described below (Darveau et al. 1997, Descouts & Aronsson 1999, Costerton et al. 1999).

The establishment and stabilization of bacteria within a biofilm are important not only for the etiology of periodontitis, but also for adjunctive systemic and topical medicinal treatment for periodontitis (p. 287): Biofilm bacteria imbedded within a matrix of extracellular polysaccharides are more than 1,000 times less sensitive to antimicrobials (e.g., antibiotics) than free-floating ("planktonic") bacteria.

44 Dental Plaque—Development

Within minutes after completely cleansing the tooth surface, a *pellicle* forms from proteins and glycoproteins in saliva.

A *Association*: Through purely physical forces, bacteria associate loosely with the pellicle.

B *Adhesion*: Because they possess special surface molecules (adhesins) that bind to pellicle receptors, some bacteria become the "primary colonizers," particularly streptococci and actinomyces. Subsequently, other microorganisms adhere to the primary colonizers.

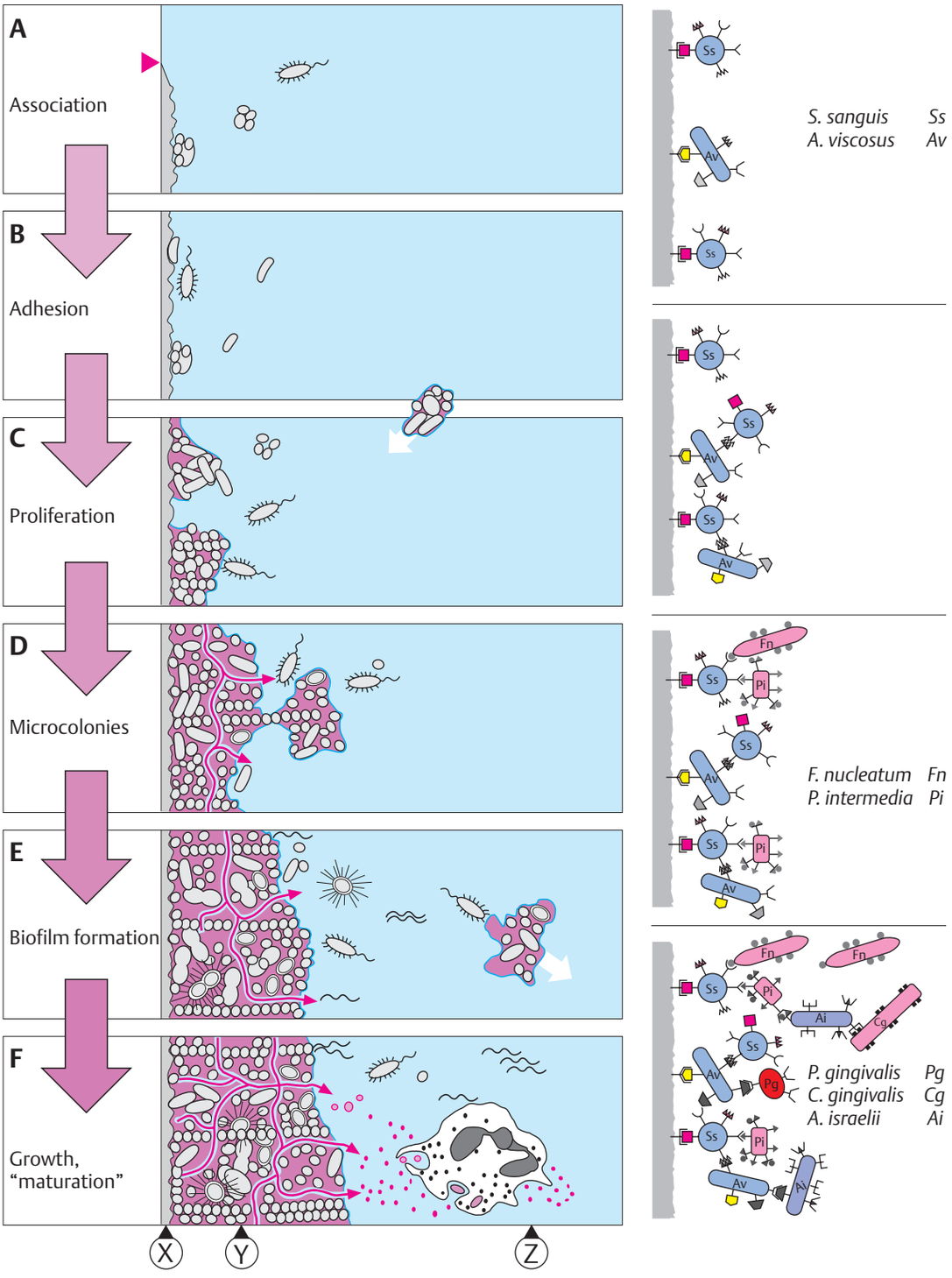
C Bacterial *proliferation* ensues.

D *Microcolonies* are formed. Many streptococci secrete protective extracellular polysaccharides (e.g., dextrans, levans).

E *Biofilm ("attached plaque"):* Microcolonies form complex groups with metabolic advantages for the constituents.

F *Plaque growth—maturation*: The biofilm is characterized by a primitive "circulatory system." The plaque begins to "behave" as a *complex organism*! Anaerobic organisms increase. Metabolic products and evulsed cell wall constituents (e.g., lipopolysaccharides, vesicles) serve to activate the host immune response (p. 38). Bacteria within the biofilm are protected from phagocytic cells (PMN) and against exogenous bacteriocidal agents.

X Pellicle
Y Biofilm—"Attached Plaque"
Z Planktonic Phase



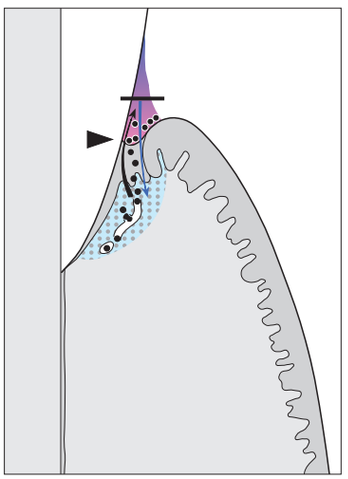
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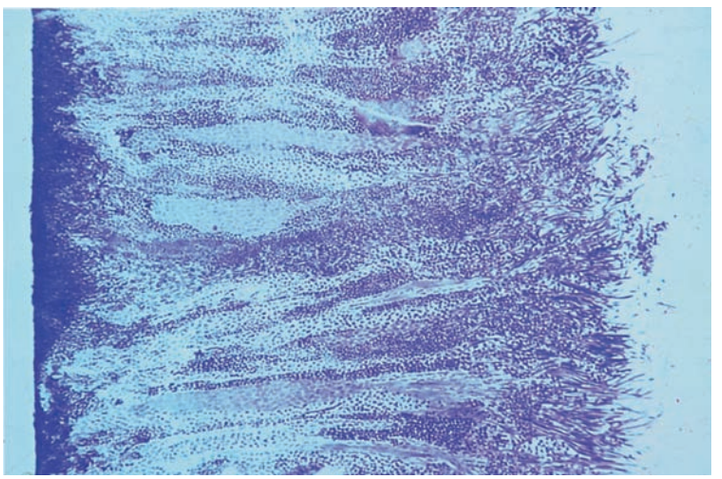
Supragingival Plague

... and its initial subgingival expansion

The first bacteria that accumulate *supragingivally* on the tooth surface are mostly gram-positive (*Streptococcus* sp, *Actinomyces* sp.). In the course of the following days, gram-negative cocci as well as gram-positive and gram-negative rods and the first filamentous forms begin to colonize (Listgarten et al. 1975, Listgarten 1976). By means of a variety of *metabolic products*, the bacterial flora provoke the tissue to an increase in exudation and to migration of PMN leukocytes into the sulcus ("leukocyte walls" against the bacteria).

The increase in PMN diapedesis and the flow of sulcus fluid lead to initial disintegration of the junctional epithelium. This makes it possible for bacteria to more easily invade between the tooth and the junctional epithelium, and invade the subgingival area (gingivitis, gingival pocket formation). In the total absence of oral hygiene, plaque formation and an initial host defensive response within gingival tissue occur. With optimum—including interdental—oral hygiene, the formation of biofilm is repeatedly disrupted and gingival health is maintained.



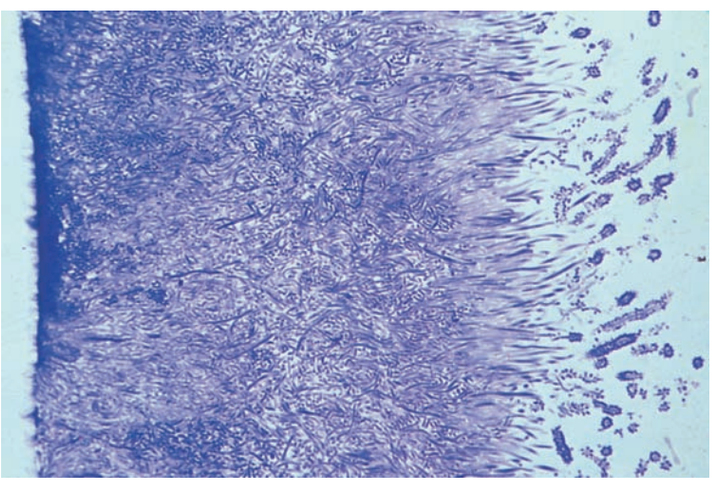


45 One-week-old Plaque—Interactions

Note the thick zone of early colonizers on the enamel surface and the column-like structures that result from rapid proliferation of streptococci. On the plaque surface, one observes rods and filaments.

Left: Interaction between host and plaque. Chemotactically regulated immigration of polymorphonuclear granulocytes (PMN, arrow). The black horizontal line indicates the level from which this sample of plaque was taken.



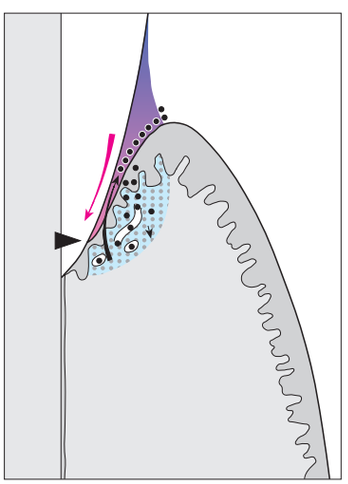


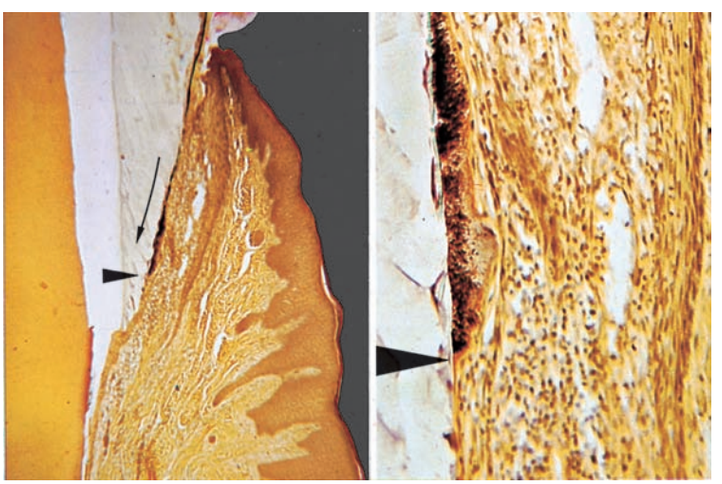
46 Three-week-old Plaque

The composition of the supragingival plaque has changed markedly. Filamentous organisms now predominate. Conspicuous forms resembling "corn cobs" are observed at the plaque surface.

Left: In this transmission electron photomicrograph, the structure of such a "corn cob" is revealed. At the center is a gram-negative filamentous organism (**F**), surrounded by gram-positive cocci (**C**).

Histology and TEM courtesy *M. Listgarten*





47 Expansion of Supragingival Plaque—Gingival Pocket

Middle and Right: Weakening of the epithelial attachment to the tooth permits apical migration of gram-positive plaque bacteria in a thin layer between the tooth and the junctional epithelium (thin arrow). Gram-negative bacteria colonize subsequently, and a *gingival* pocket forms (Fig. 150).
Histology courtesy *G. Cimasoni*

Left: Schematic representation of the interaction between plaque and host tissue.

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Natural Factors Favoring Plaque Retention

The formation of a plaque biofilm can be enhanced by natural retention factors, which can also render biofilm removal by means of oral hygiene more difficult. These retention factors include:

- Supra- and subgingival calculus
- Cementoenamel junctions and enamel projections
- Furcation entrances and irregularities
- Tooth fissures and grooves
- Cervical and root surface caries
- Crowding of teeth in the arch.

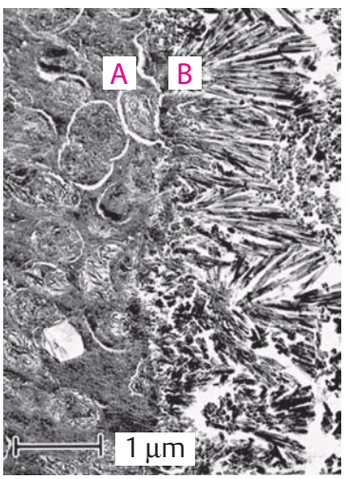
By itself, *calculus* is not pathogenic. However, its rough surface presents a retention area for vital, pathogenic bacteria. At the microscopic level, the *cementoenamel junction* is very irregular, and offers retentive roughness. Enamel projections and "pearls" also inhibit soft tissue attachment. *Furcation entrances, fissures,* etc. are retentive niches for plaque. *Carious lesions* represent a huge bacterial reservoir. *Crowding of teeth* reduces self-cleansing and renders oral hygiene more difficult.

48 Supragingival Calculus
Lingual surfaces of mandibular incisors and buccal surfaces of maxillary molars near the orifices of salivary ducts often exhibit massive accumulations of supragingival calculus.

Right: TEM of old supragingival calculus. Calcified plaque (**A**) close to the tooth surface. Note the accumulation of cell-free hexagonal monocrystals (**B**) upon the calcified plaque.

Courtesy *H. Schroeder*

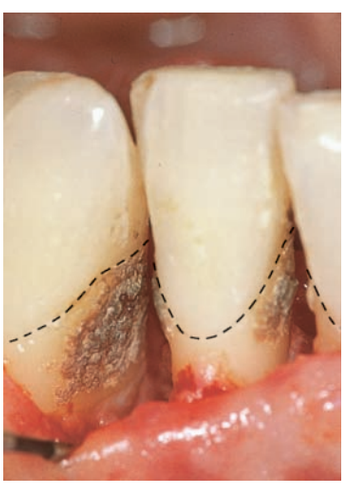




49 Subgingival Calculus
In this patient with long-standing periodontitis, the gingiva has receded. Calculus that was formerly subgingival is now supragingival.

Right: Subgingival calculus is observed clinically after reflecting the gingival margin. Subgingival calculus is usually dark in color (Fe minerals) and harder than the more loosely structured supragingival calculus (calcium phosphates). The cementoenamel junction is indicated by the dashed line.





50 Crowding, Enamel Projection (enamel "pearl")
The lingually displaced mandibular incisors do not benefit from the natural self-cleansing action of the lower lip. Oral hygiene is also rendered more difficult.

Right: The furcation on this molar is filled by a projection of enamel that ends in a bulbous pearl, extending into the interradicular area. When a pocket forms in such an area, plaque control is particularly difficult.





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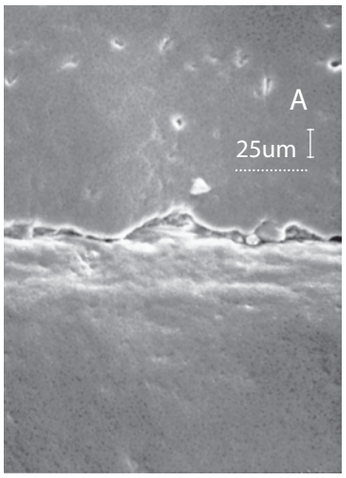
Iatrogenic Factors Favoring Plaque Retention

Restorative dentistry—from a simple restoration to a full-mouth reconstruction—can do more harm than good to the patient's oral health if performed improperly! Placing only optimum restorations is synonymous with preventive periodontics (tertiary prevention, p. 198).

Fillings and crowns that appear to be perfect clinically and macroscopically almost always exhibit deficiencies at the margins when viewed microscopically. When margins are located subgingivally, they always present an irritation for the marginal periodontal tissues.

Overhanging margins of restorations and crowns accumulate additional plaque. Gingivitis ensues. The composition of the plaque changes. The number of gram-negative anaerobes (e. g., *Porphyromonas gingivalis*), the organisms responsible for initiation and progression of periodontitis, increases rapidly (Lang et al. 1983).

Gross iatrogenic irritants such as poorly designed *clasps* and *prosthesis saddles* may exert a direct traumatic influence upon periodontal tissues.





51 Amalgam Restoration—Clinical View and SEM

Left: Viewed in the scanning electron microscope, a clearly visible margin defect is observed. Such a defect is a perfect niche for the accumulation of plaque. The amalgam restoration (**A**) is at the top of this figure, the adjacent enamel below. The white dot under the 25 µm legend are representative of the size of coccoid microorganisms (ca. 1 µm).

Courtesy *F. Lutz*



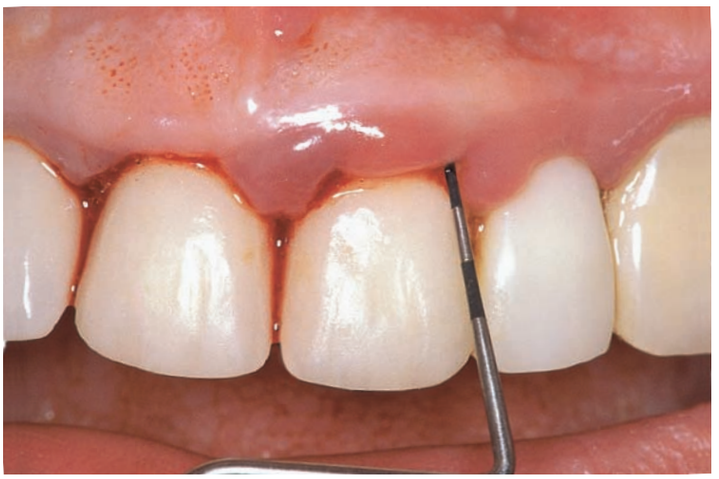


52 Amalgam—Proximal Overhang

Gross overhangs such as this, located subgingivally, invariably lead to plaque accumulation and to gingivitis (note hemorrhage). Pathogenic, gram-negative anaerobes are frequently observed. In contrast to amalgam and especially gold restorations, composite resin restorations are particularly retentive of bacteria.

Left: Radiograph of the same case.





53 Crown Margin Overhang and Open Margins

The cement that was used to cement this crown has begun to extrude from the open margin. The massive retention of plaque between the crown and the prepared tooth leads to severe gingivitis and establishment of a pathogenic bacterial flora.

Left: Section through a porcelain-fused-to-metal crown with a margin that is both overhanging (arrows) and open. Calculus (**C**) and plaque have accumulated apically.

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Subgingival Plaque

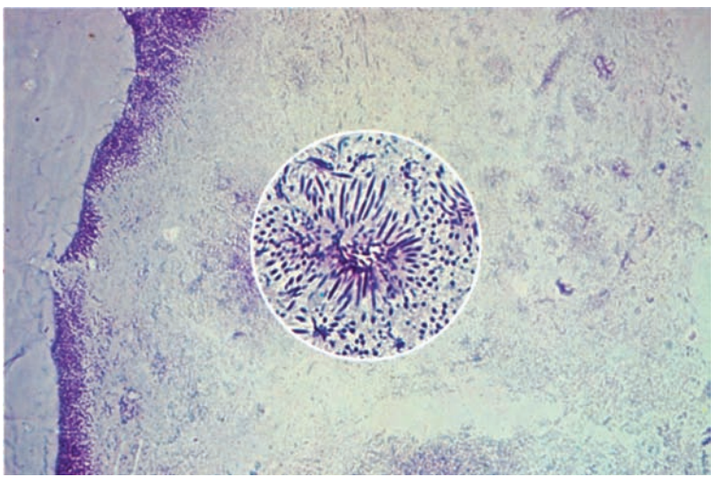
Extending apically from the supragingival region, a subgingival plaque biofilm will often form within the existing gingival sulcus/pocket; this was previously called the "adherent" plaque. In addition to gram-positive bacteria such as streptococci, actinomyces, etc., as the probing depth increases so does the number of anaerobic *gram-negative* bacteria (p. 36).

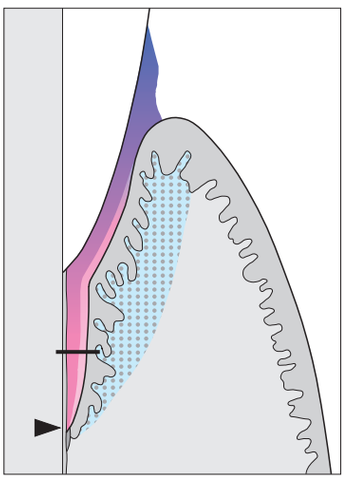
This subgingival biofilm can also calcify. A dark, hard and difficult to remove calculus ("serum calculus") accumulates. In addition, the gingival pocket also contains loose agglomerates of non-adherent, often mobile bacteria (with a high concentration of gram-negative anaerobes and spirochetes). In acute phases, *periodontopathic* bacteria often increase dramatically. These include *Actinobacillus actinomycetemcomitans, P. gingivalis, T. forsythensis,* spirochetes etc. (pp. 30, 33, 38). Despite these alterations in the subgingival plaque, periodontitis, even in the acute stage, cannot be characterized as a "highly specific" infection because large differences have been reported in the bacterial composition between patients and even within different pocket locations in the same patient (Dzink et al. 1988, Slots & Taubmann 1992, Lindhe 1997).

54 Subgingival Pocket Flora
This is a relatively thin adherent biofilm (blue-violet). One observes loose accumulations of gram-negative, anaerobic, and also motile bacteria. Formations resembling test tube brushes, consisting of filamentous bacteria are also observed (inset).

Histology courtesy *M. Listgarten*

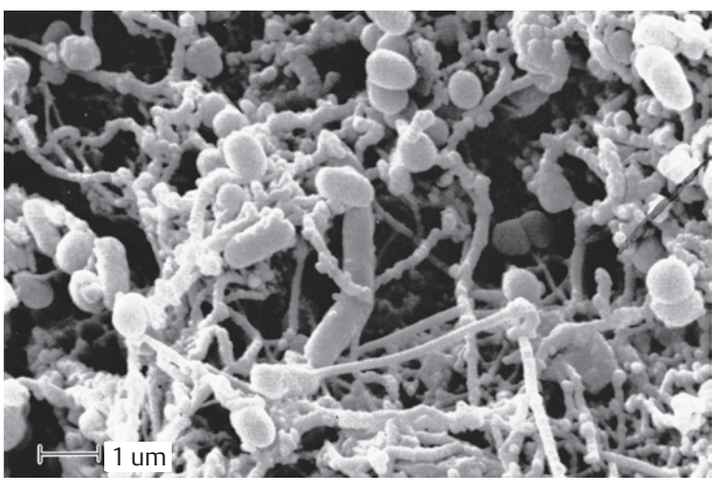
Right: As pocket depth increases (arrow), the resident flora becomes increasingly gram-negative and anaerobic.





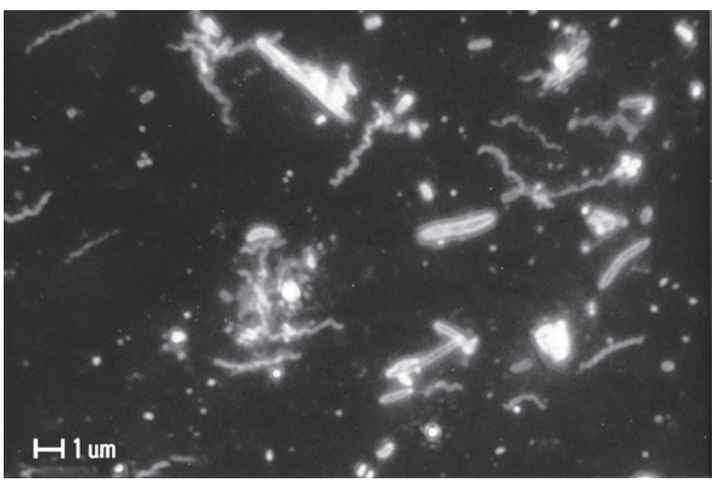
55 Surface of the Biofilm on the Root
Within a pocket, the root surface of a tooth manifesting periodontitis is covered with a densely intertwined bacterial colonization composed of many different bacterial morphotypes (scanning electron photomicrograph).

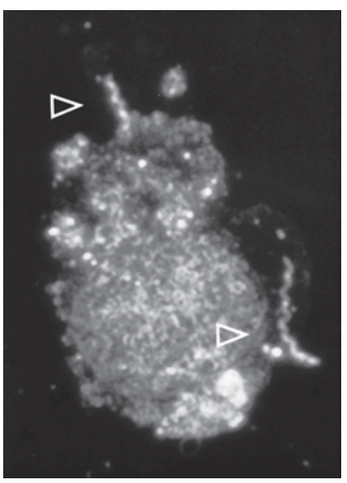
The *morphology* of the bacteria permits neither a determination of the species nor any clues concerning pathogenicity.



56 Microorganisms of the Non-Adherent Plaque—"Planktonic" Phase
In a dark-field preparation, motile rods and small to larger spirochetes predominate, while cocci and filaments are rare: Typical signs of an active pocket (exacerbation; cf. p. 63).
Right: Intact phagocytes (PMN) in the pocket exudate do not lose their capacity for phagocytosis. The arrow depicts a spirochete being engulfed.

Courtesy *B. Guggenheim*

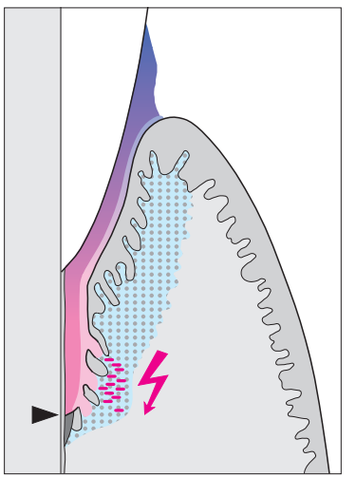


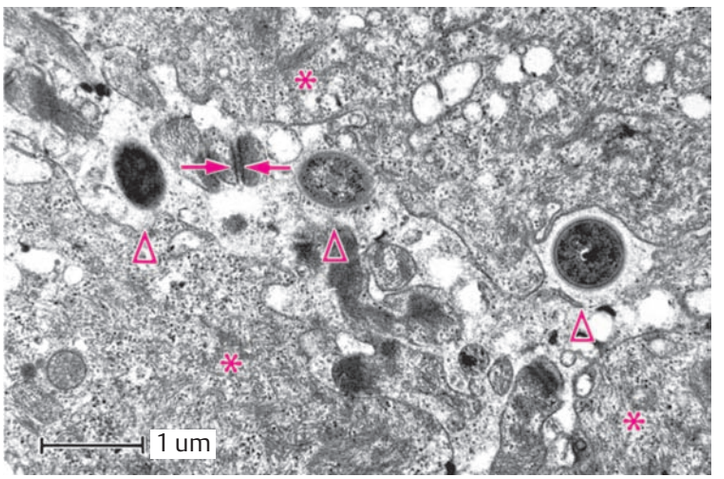


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Bacterial Invasion Into Tissue?

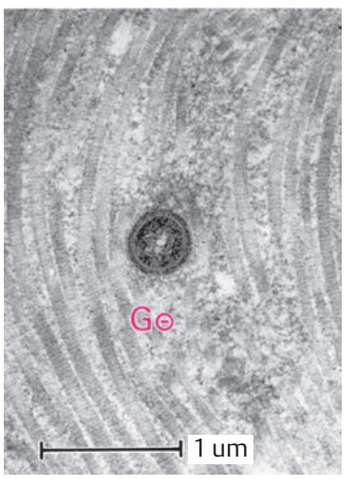
In patients with a compromised immune response, for example in cases of early onset, aggressive periodontitis (p. 96), bacteria with pathogenic potential have the ability to invade the cells of the pocket epithelium and the subepithelial connective tissue, and to remain viable there for varying periods of time. This usually occurs only in the depth of the pockets where the bacteria can avoid the infiltrate (defense), which is usually located close to the gingival margin. The periodontopathic bacteria (p. 33) produce virulence factors (leukotoxins from *Actinobacillus actinomycetemcomitans,* lipopolysaccharides and enzymes), which can initially shut down the chemotactic guidance of defense cells (e. g., PMN) or even kill PMNs. With time, the invading microorganisms are recognized by the activated immune system and killed. If tissue invasion is mild, small areas of necrotic tissue resorption may result, but if the bacterial invasion is massive, acute suppurative abscesses can result (Allenspach-Petrzilka & Guggenheim 1983, Christensson et al. 1987, Frank 1988, Saglie et al. 1988, Slots 1999, Van Winkelhoff & Slots 1999). It is unclear whether oral microorganisms that invade tissue cells actually *colonize*, or whether the individual microorganisms simply invade.

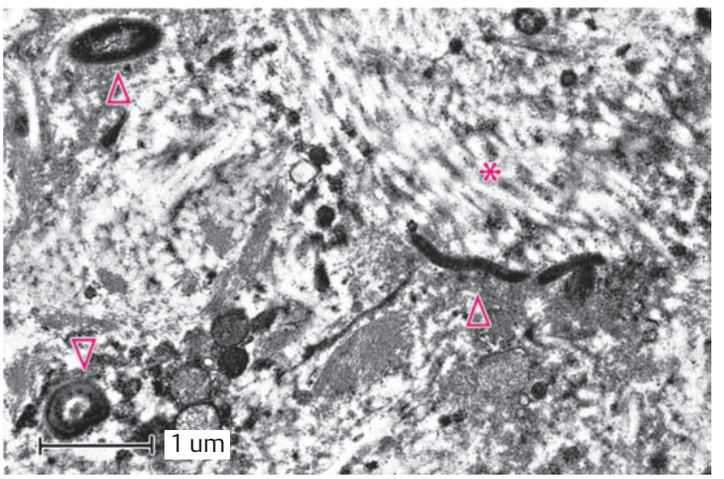




57 Bacteria within the Pocket Epithelium
Bacteria (red triangles) in the widened intercellular spaces of the pocket epithelium. Three epithelial cells (✻) and one desmosome (double arrow) are observed.
The exudate and PMNs have significantly widened the intercellular spaces between the JE cells.

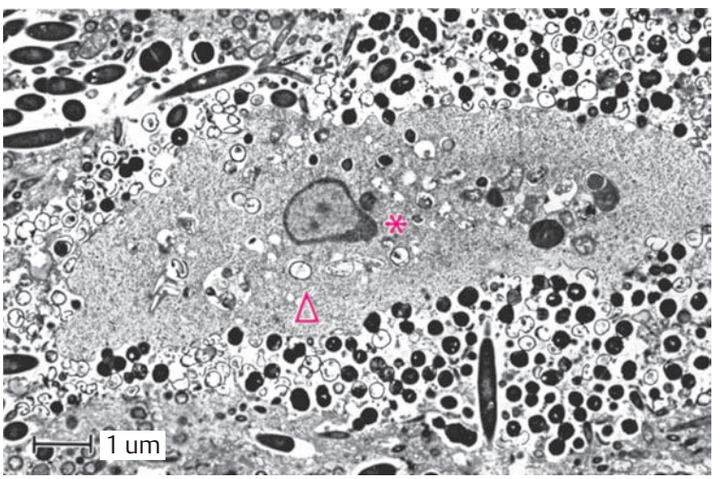
Left: In the depth of the active pocket, one observes ulcerated epithelium, through which bacteria may invade the connective tissue (red bars).





58 Bacterial Invasion—Infection
Bacteria of various species are observed within the connective tissue (arrows) adjacent to a deep periodontal pocket. Tissue damage (✻ = degraded collagen) may result, or tissue may remain completely healthy in appearance.

Left: A gram-negative bacterium (**G**⊖) is observed in the midst of otherwise essentially intact collagen fibrils



59 Necrosis—Suppuration
Almost the entire photomicrograph is filled by a dead phagocytic cell (PMN, ✻). The cell contains phagolysosomes, some of which exhibit digested material (arrow).
The dead phagocyte is surrounded by dead bacteria and bacterial cell walls. This pus must either be resorbed by the host tissue, or expelled (abscess, fistula).

TEMs courtesy *B. Guggenheim*

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Classification of Oral Microorganisms

Thanks to new laboratory techniques (e.g., 16S rRNA analyses), over 530 species and subspecies have been isolated and classified from subgingival and supragingival bacterial samples (Slots & Taubman 1992, Moore & Moore 1994, Socransky et al. 1999). Some of the cultivable species are listed below.

Today, only about a dozen microorganisms are classified as *periodontal pathogens*. Foremost among these are gram-negative organisms, including *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Tannerella forsythensis* (formerly *Bacteroides forsythus)* and *Prevotella intermedia* (p. 33).

Some of these bacteria possess significant biochemical capacities for the pathogenesis of inflammatory periodontal diseases. For example, they are capable of colonizing root surfaces and cell surfaces, and therefore maintain a secure position in the micro-ecological cosmos of the pocket flora. The microorganisms are usually capable of co-aggregation, i.e., they aggregate with one or more other types of bacteria to form a so-called complex or "cluster" (Socransky et al. 1998, 1999). Such complexes have been characterized as highly pathogenic or only slightly pathogenic.

60 Microorganisms in the Plaque Biofilm and in the Non-adherent Planktonic Phase

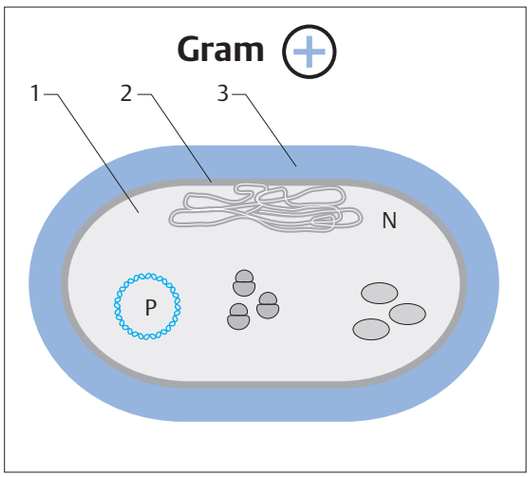
	Gram ⊕ positive		Gram ⊖ negative	
Prokaryotes	**Facultative anaerobes**	**Obligate anaerobes**	**Facultative anaerobes**	**Obligate anaerobes**
Cocci ●	**Streptococcus** – *S. anginosus (S. milleri)* – *S. mutans* – *S. sanguis* • **Ss** – *S. oralis* – *S. mitis* – *S. intermedius*	**Peptostreptococcus** – *P. micros* • **Pm** **Peptococcus**	**Neisseria** **Branhamella**	**Veillonella** – *V. parvula*
Rods ▬	**Actinomyces** – *A.naeslundii* • **An** – *A. viscosus* • **Av** – *A. odontolyticus* – *A. israelii* **Propionibacterium** **Rothia** – *R. dentocariosa* **Lactobacillus** – *L. oris* – *L. acidophilus* – *L. salivarius* – *L. buccalis*	**Eubacterium** – *E. nodatum* • **En** – *E. saburreum* – *E. timidum* – *E. brachy* – *E. alactolyticum* **Bifidobacterium** – *B. dentium*	**Actinobacillus** – *A. actinomycetemcomitans* • **Aa** **Capnocytophaga** – *C. ochracea* – *C. gingivalis* – *C. sputigena* **Campylobacter** – *C. rectus* • **Cr** – *C. curvus* – *C. showae* **Eikenella** – *E. corrodens* • **Ec** **Haemophilus** – *H. aphrophilus* – *H. segnis*	**Porphyromonas** – *P. gingivalis* • **Pg** – *P. endodontalis* **Prevotella** – *P. intermedia* • **Pi** – *P. nigrescens* – *P. melaninogenica* – *P. denticola* – *P. loescheii* – *P. oris* – *P. oralis* **Bacteroides** – *T. forsythensis* • **Tf** – *B. gracilis* **Fusobacterium** – *F. nucleatum* • **Fn** – *F. periodonticum* **Selenomonas** – *S. sputigena* – *S. noxia*
Spirochetes and mycoplasms ≈	**Mycoplasm** – *M. orale* – *M. salivarium* – *M. hominis*		**Spirochetes of ANUG** **Treponema sp.** – *T. denticola* • **Td** – *T. socranskii* – *T. pectinovorum* – *T. vincentii*	
Eukaryotes	**Candida** – *C. albicans*	**Entamoeba**		**Trichomonas**

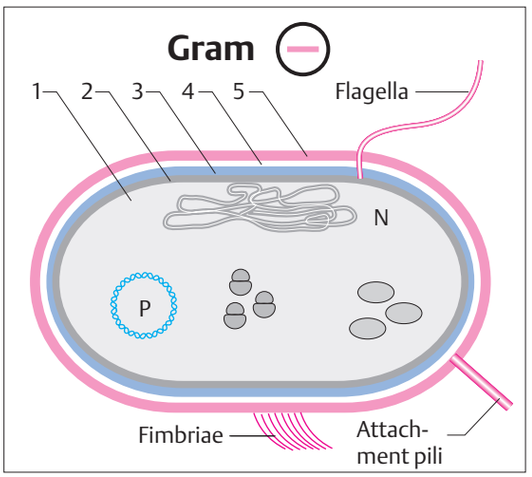
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Cell Walls of Gram-Positive and Gram-Negative Bacteria

The technique of *Gram staining* renders differences in the composition of bacterial cell walls visible in the microscope:

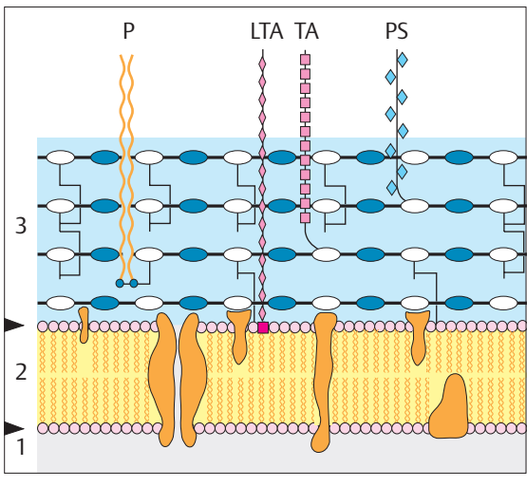
- The plasma membrane (phospholipid double membrane; osmotic barrier) which surrounds the cytoplasm of both gram-positive and gram-negative bacteria, as well as the cellular skeleton that provides integrity consists of murein (a peptidoglycan), which represents only a thin layer in gram-negative microorganisms.
- *Gram-positive* bacteria possess only this single, thick cell wall, in the form of a giant netlike molecule ("sacculum"). Antigens such as teichoic acids and proteins emanate from the murein layer. Penicillin can inhibit or kill gram-positive microorganisms by preventing cell wall synthesis by means of blocking the molecular binding of the polysaccharide chains.
- In *gram-negative* bacteria, especially the external membranes, and particularly the external layer, are highly complex; contained therein is the endotoxin lipopolysaccharide (LPS), which has a two-fold effect upon the host organism: *toxic* via the contained lipid A, and *antigenic* via the O-specific polysaccharide chain (O-antigen; cf. p. 38).

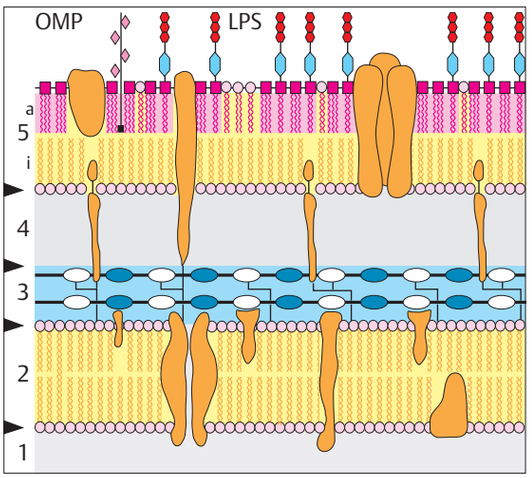




61 Anatomy/Morphology of Gram-Positive *(left)* **and Gram-Negative** *(right)* **Bacteria**

1 Cytoplasm with organelles: Genome (**N**), plasmid (**P**), ribosomes
2 Cytoplasmic membrane: This phospholipid bilayer functions as an osmotic barrier
3 Peptidoglycan: This large molecule provides protection
4 Periplasmatic space: This is gram-negative specific
5 Outer membrane: Found only in gram-negative organisms, with inner and outer layers

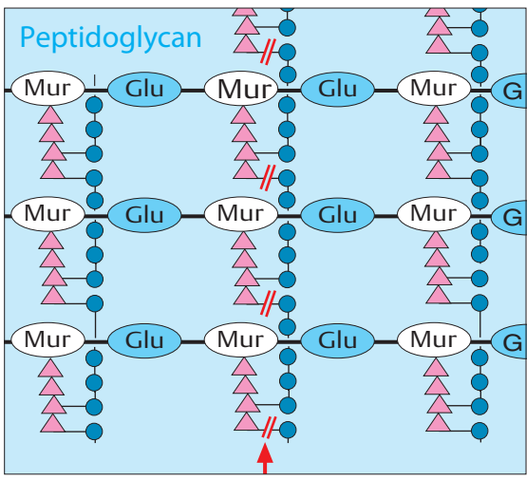


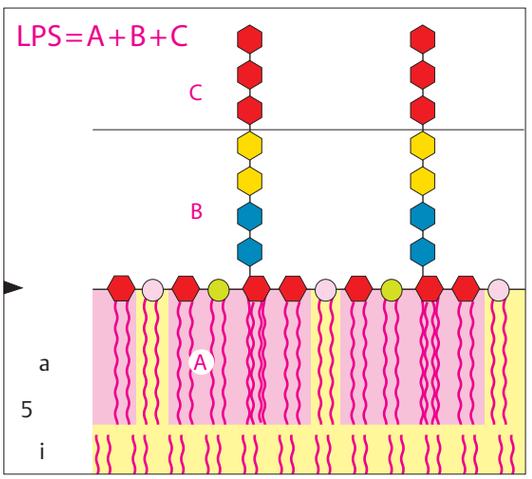


62 Cell Wall Differences

Left: Gram-positive cell walls
P Cell wall proteins
LTA Lipoteichoic acid
TA Teichoic acids
PS Specific polysaccharides

Right: Gram-negative cell walls
4 Periplasmatic space
5 Inner (**i**) and outer (**a**) layer of the outer membrane with proteins (orange, e. g., **OMP** = outer membrane proteins), lipopolysaccharides (**LPS** in layer **5a**).
1, 2, 3 See legend, Fig. 61.





63 Cell Wall—Details

Left: Gram-positive cell walls. Peptidoglycan consists of alternating units of murein (**Mur**; N-acetyl-muraminic acid) and **Glu** (N-acetyl-glucosamine) and is cross-linked via peptide (blue circles, red triangles). Penicillin blocks this (arrow).
Right: Gram-negative cell wall
LPS = A+B+C, "endotoxin" (p. 38)
A Lipid A (red, toxic effect)
B Core polysaccharide
C O-specific antigen
Modified from *F. Kayser et al.; L. Stryer*

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Periodontitis—Classical or Opportunistic Infection?

Classical Infection

In classical infection, upon which the postulates of Henle and Koch are based (see below), the defense capabilities of the host are breached by a *specific, highly virulent* bacterium. The bacterium proliferates within the tissues and elicits typical symptoms of disease. Examples of classical infections include diphtheria, scarlet fever and tuberculosis.

Periodontitis is *not* a classical infection. However, those types of periodontitis associated with *Actinobacillus actinomycetemcomitans may* be classified as classical infections.

Opportunistic Infection

Opportunistic microorganisms are only pathogenic in a compromised host. They are regularly found in the natural flora. They normally do not cause damage to the host. However, in patients with reduced resistance, the existence of risk factors or immunosupression, a selective increase in bacteria with *weak virulence factors* may occur, leading to an opportunistic infection. This situation does not fulfill Koch's postulates, but rather those proposed by Socransky for periodontitis (see below, Socransky & Haffajee 1992).

64 Classical Infection

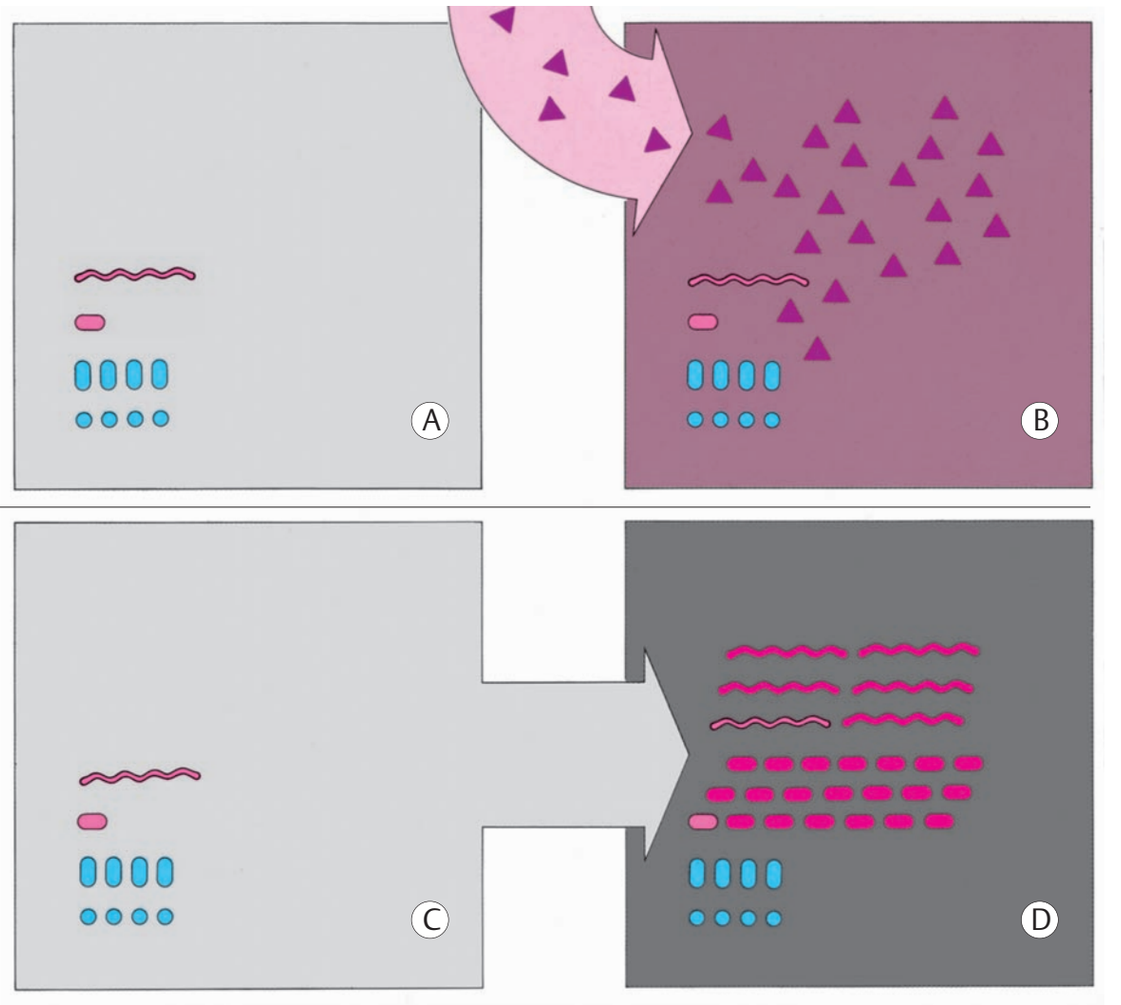
A *Initial situation*: Non-specific, "natural" colonization with gram-positive (blue) and a few gram-negative (red) microorganisms ("resident" flora). The host is healthy (ecological balance).

B *Infection:* An increased number of exogenous specific pathogenic microorganisms (infective dose, violet triangles) penetrates the defense mechanism of the host and begins to proliferate selectively.

65 Opportunistic Infection

C *Initial situation*: the same non-pathogenic flora is present, as in figure 64A.

D *"Shift"/infection*: Alterations in the micro-ecology and the general resistance of the host leads to a destabilization of the ecological balance. One or more species react to this new situation with selective proliferation as well as altered activity leading to tissue damage (disease).



Classical vs. Opportunistic Infection—"Postulates"

In addition to the classical infections with a single pathogen, there is a large group of opportunistic infections in which entire groups of microorganisms can be involved in the initiation and progression of diseases.

Postulates of Henle and Koch	Postulates of Socransky
The **causative agent** must always be found in the clinical lesion, and not in healthy subjects.	**Association**: The causative agent must be found in active "sites" in higher numbers than in non-active sites.
The putative pathogen must be **cultivable** in pure culture.	**Elimination**: The elimination of the causative agent must stop the progression of the disease.
The pathogenic characteristics of the causative agent must elicit in **animal models** a disease identical to that in humans, and the agent must be identified in the animal.	**Host response**: The cellular or humoral immune response must validate the specific role of the causative agent in the disease.
	Virulence factors: The causative agent must possess virulence factors that are relevant for the initiation and progression of the disease.
In addition: Beyond the postulates of Henle and Koch, there must be clear evidence of the immunological "**pathogen-host relationship**."	**Animal models**: The pathogenicity of the causative agent in an animal model must provide conclusive evidence that it can cause periodontitis in humans.

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Putative Periodontopathic Bacteria

For almost 100 years, a bacterial etiology for the existence of periodontitis has been sought. Early on, the *non-specific plaque hypothesis* prevailed, but since the 1970's research has indicated specific bacteria as the etiologic agents in periodontal diseases. Nevertheless, it is certain that not all possible periodontopathic microorganisms have yet been identified. With this in mind, Figure 66 (Socransky & Haffajee 1992, Tonetti 1994) presents the most likely periodontopathic microorganisms, but this table makes no claim to completeness; it will certainly be modified and enhanced by future research.

According to Socransky's postulates, the pathogenic potential of a microorganism is determined not only by its association with the disease, but also by:

- Improvement of the disease condition after elimination of the pathogen by therapy
- Activation of the host immune response to the specific infection
- Detection of putative virulence factors (pp. 34–35)
- The elicitation of similar periodontal disease symptoms in animal experiments.

Species \ Studies	① Association	② Elimination	③ Host Response	④ Virulence Factors	⑤ Animal Studies
Aa *Actinobacillus actinomycetemcomitans*	+ + +	+ + +	+ + +	+ + +	+ + +
Pg *Porphyromonas gingivalis*	+ + +	+ + +	+ + +	+ + +	+ + +
Pi *Prevotella intermedia*	+ + +	+ +	+ +	+ + +	+ + +
Fn *Fusobacterium nucleatum*	+ + +	+	+ + +	+ +	+
Tf *Tannerella forsythensis**	+ + +	+ +	+	+ + +	+
Cr *Campylobacter rectus*	+ + +	+ +			
Ec *Eikenella corrodens*	+ + +	+		+	+ +
Pm *Peptostreptococcus micros*	+ + +	+	+		
Ss *Selenomonas sputigena*	+ + +				
Eubacterium sp.	+ +		+ +		
Spirochetes	+ + +	+ + +	+ + +	+ + +	+

66 Frequency of Research Studies That Have Demonstrated the Degree of Pathogenicity of Various Microorganisms According to Socransky's Postulates.
The *number of "plus signs"* indicates the frequency of positive results in the studies.
For all of the microorganisms listed, the direct association with the disease (**1**) was investigated, and in most cases also the other listed criteria (**2–5**).

The intensity of the *background color* (red = gram-negative; blue = gram-positive) depicts the association of the bacteria with the various criteria of periodontitis; and thus, is a measure of their relative pathogenicity.

Association with Periodontitis

+ + + Highly pathogenic
+ + Moderately pathogenic
+ Slightly pathogenic
no research studies

+ + + Highly pathogenic	+ + Moderately pathogenic	+ Slightly pathogenic
• Aa *Actinobacillus actinomycetemcomitans*	*P. intermedia*	*S. intermedius*
• Pg *Porphyromonas gingivalis*	*C. rectus*	*P. nigrescens*
• Tf *Tannerella forsythensis**	*E. nodatum*	*P. micros*
• Td *Treponema denticola* (Spirochetes of NUG)	*Treponema sp.*	*F. nucleatum*
		Eubacterium sp.
		E. corrodens

67 Rank Ordering of the Pathogenicity of Individual Bacteria
(Haffajee & Socransky 1994)

A. actinomycetemcomitans
P. gingivalis
Tannerella forsythensis* *(2003; formerly **B. forsythus**)*
Certain spirochetes ...
appear to be the most virulent periodontal pathogenic microorganisms.

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Virulence Factors

The destructive potential of bacteria is related to their relative concentration (percentage composition of the total bacterial flora), but also to their so-called *virulence factors*. Bacteria (usually gram-negative) possessing virulence factors are usually found in periodontal pockets when periodontal destruction is actively progressing. Some of these virulence factors have already been mentioned: endotoxin, exotoxin, enzymes, chemotactic substances and antigens. These and other virulence factors that have thus far been demonstrated are listed below.

Virulence Transfer

Virulence factors are transferred to daughter cells following cell division, but such factors can also be transferred from one bacterium to another or to other species of bacteria by means of plasmid transfer.

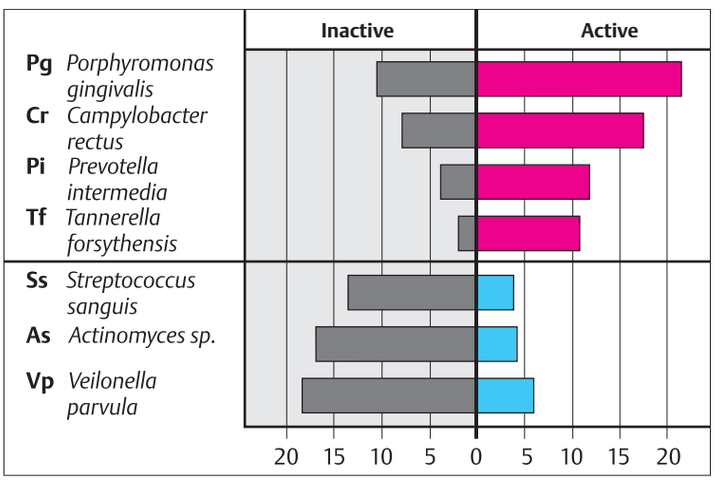
Plasmids determine various characteristics of bacteria, e.g., the production of toxins (virulence plasmids) and resistance factors (resistance plasmids) against antibiotics.

Bacteriophages are viruses that proliferate within bacterial cells and have the ability to transfer DNA fragments or plasmids between bacteria (Fig. 70; Preus et al. 1987, Haubek et al. 1997, Willi & Meyer 1998).

68 Number (in percent) of Microorganisms in Active and Inactive Pockets (Dzink et al. 1988)

The course of periodontitis is tooth surface specific and episodic.

The absolute number and the percentage composition of periodontopathic gram-negative bacteria (red) are significantly increased in *active* pockets. In *inactive* lesions, one finds primarily gram-positive "resident" microorganisms (blue), which are less damaging to host tissue and which to a certain degree actually inhibit the pathogens.



69 Bacterial Virulence Factors

Virulence is multifactorial. It is influenced by the inherent pathogenic potential of a bacterium, its environment, and its interaction with the host. Virulent bacteria require a competitor! In order to elicit periodontitis, microorganisms must

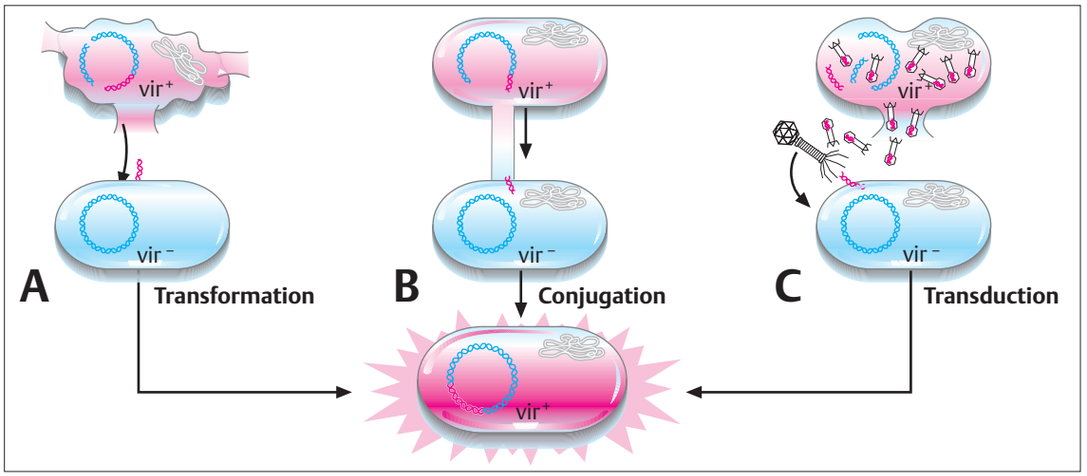
- Establish themselves near the host tissue
- Avoid being eliminated by saliva or exudates
- Find appropriate nutrition
- Avoid the defense mechanisms of the host and other microorganisms
- Be capable of destroying periodontal tissues.

Even though virulent, pathogenic bacteria in the pocket possess a *significant potential for damage*; this potential is small in comparison to that of the host: The clinically detectable destruction of soft and hard tissues is caused mainly by the immune defense mechanisms of the host.

Fact: In order to induce periodontitis, even virulent bacterial strains require partners (complexes: p. 37).

Goals	Bacterial Factors	
Adhesion to host tissues – Surface structures	– Fimbriae, pili – other adhesins	Microorganisms
Colonization, proliferation	– Nutrition chain development – Proteases for foodstuff metabolism (Host proteins, Fe^{2+}) – Inhibition of inhibitors	Microorganisms
Host response **– Deceiving** **– Inhibiting** **– Eliminating**	– Capsule, mucous – PMN receptor blocker – Leukotoxins (Aa) – Immunoglobulin-destroying proteases (Pg) – Complement-degrading proteases	Microorganisms
Penetration into host tissues, host cells	– Invasins	Microorganisms
Tissue damage – direct		Microorganisms
Enzymes	– Collagenases – Hyaluronidase, Chondroitin sulfatase – Trypsin-like proteases (Pg, Tf, Td)	Microorganisms
Bone resorption	– LPS/lipolysaccharide – LTA/lipoteichoic acids – Capsule-, membrane substances	Microorganisms
Cellular toxins, poisons	– butyric acid, propionic acid, indoles, amines – ammonia, hydrogen sulfide and other volatile sulfur compounds	Microorganisms
Tissue damage – indirect	– Host inflammatory response to microbial plaque antigens – Sensitive regulation of pro-inflammatory mediators such as TNF, IL-1, IL-6, and thus sensitive regulation of the synthesis of prostaglandin E2 (PGE2), matrix metalloproteinases (MMP) etc.	Host

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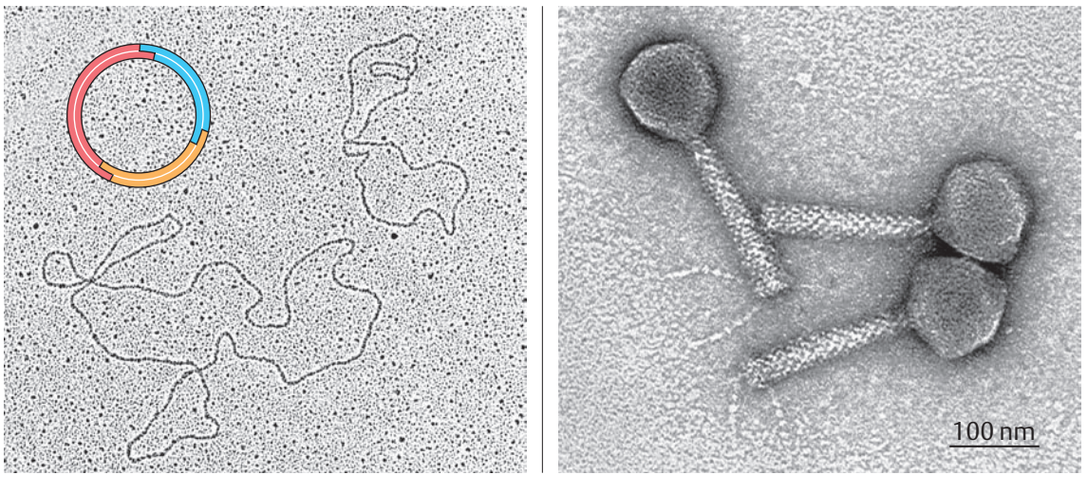


70 Transmission Pathway of Virulence Factors
Circular DNA molecules (plasmids) contain the gene for virulence factors (vir^+). Three pathways for such DNA transfer are understood:

A *Transformation*: A non-virulent bacterium (vir^-) takes the free DNA from a dead virulent bacterium.

B *Conjugation:* Two vital bacteria exchange DNA "sexually" by direct cell contact.

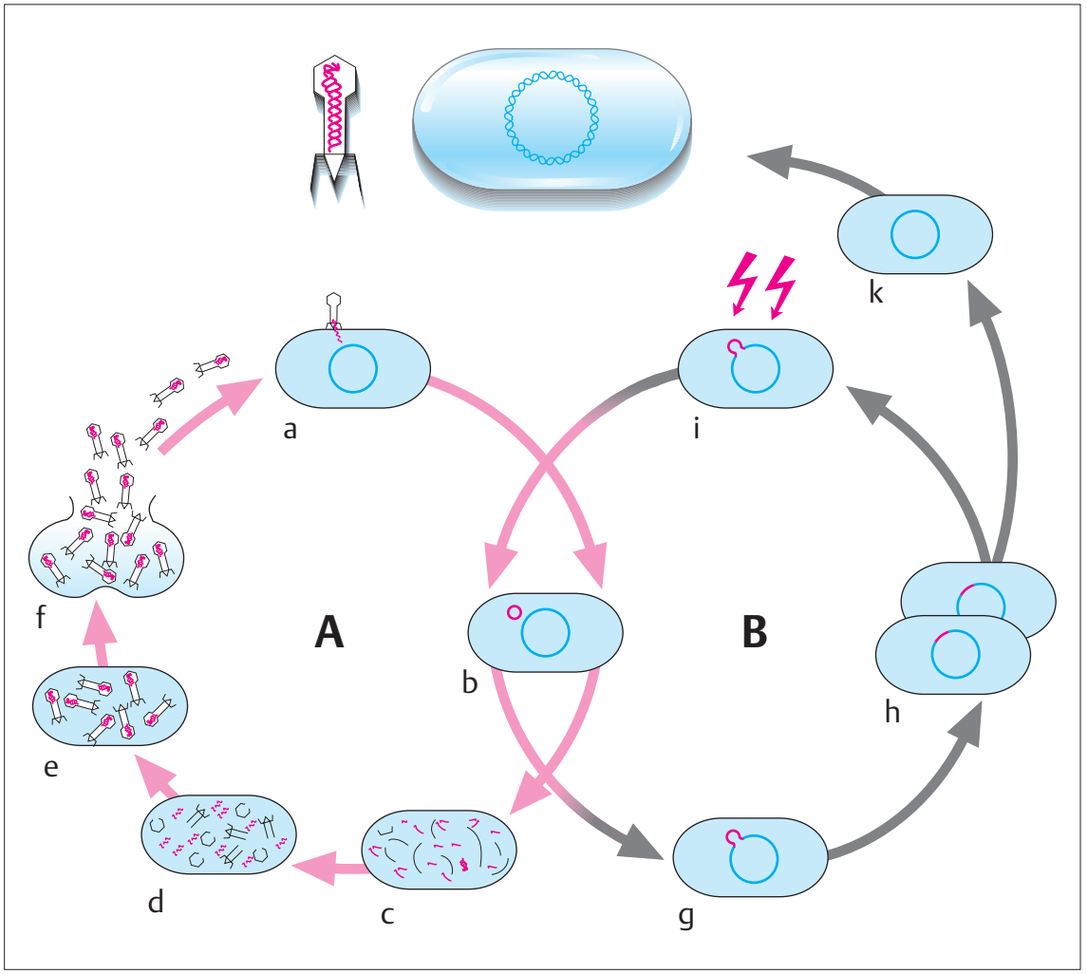
C *Transduction:* Phages (viruses) transfer the DNA (Fig. 72).



71 Plasmids and Bacterial Phages
Left: Plasmids are circular DNA molecules consisting of 1,000–450,000 base pairs (2–500 genes). They contain genes for virulence (toxins) as well as resistance factors. In the color schematic (left) three genes are represented.

Right: Bacteriophages are viruses that infect bacteria and propagate within bacteria (transduction).

TEM courtesy *J. Meyer.*



72 Transduction: DNA Transfer via Bacteriophages
Phages proliferate within bacterial cells by two mechanisms (cycles).

A **Lytic cycle**:
The bacterium dies.

a The phage injects its genome.

b The injected DNA adopts a ring configuration.

c/d Viral DNA—regulated production of phage components: capsule, genome virulence factor.

e New phages are created (viral morphogenesis).

f Bacteriolysis: Release of innumerable new phages.

B **Lysogenic cycle**:
The bacterium survives.

b Separate DNA (see above).

g The viral genome is incorporated into the bacterial genome.

h The bacterium is latently infected and divides. The new bacteria are "virulent" and vital (e. g., diphtheria toxin gene).

i Continuous irritation renders the phage genome self-supporting (→ cycle **A**).

k Special cases: the bacterium "loses" the phage genome and remains vital.

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Marker Bacteria in Periodontitis

About a dozen bacteria in the oral cavity—so-called "marker" bacteria—have been variously associated with periodontitis. Of these, the best documented "agents of periodontal disease" are:

- *Porphyromonas gingivalis (Pg)*
- *Actinobacillus actinomycetemcomitans (Aa)*
- *Tannerella forsythensis (Tf)*

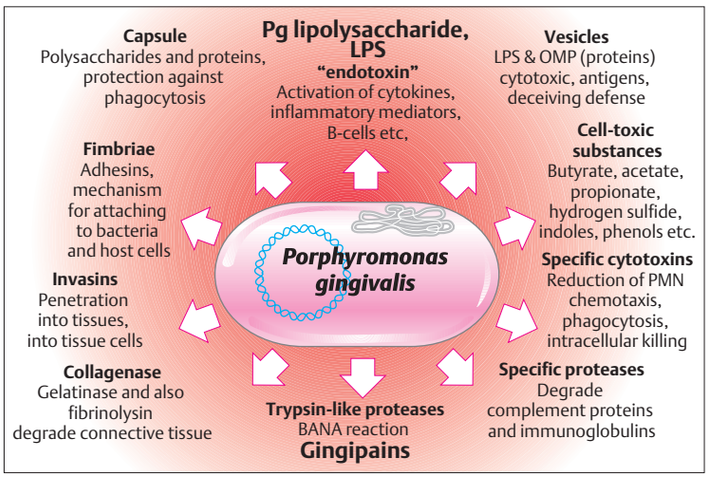
All of the other bacteria within the ecosystem of the oral cavity produce far fewer virulence factors.

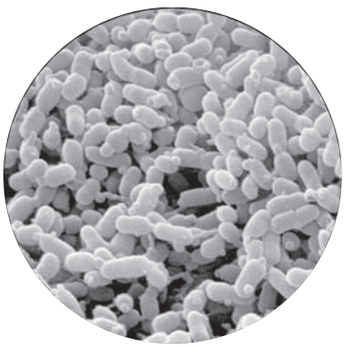
Virulence Factors of the Marker Bacteria

- *Toxins*: Best characterized are the leukotoxin from various *Aa* clones, and the special lipopolysaccharide from *Pg* (LPS; p. 38).
- *Ability to invade*: *Pg* and *Aa* can penetrate host *cells* and thus have the ability to avoid the non-specific immune response, the "first line of defense" (p. 42).
- Enzymes and proteases: Upon contact of *Pg* with epithelial cells, numerous enzymes (e. g., extracellular proteases) as well as "gingipains" that reduce host immune response are set free.

73 Virulence Factors of *Porphyromonas gingivalis (Pg)*

Pg requires many of these factors to exist, i. e., to acquire its nutrition, and to maintain itself within the ecology of the periodontal pocket. Its most significant "weapon" against the host organism is its toxic and antigenically effective LPS (p. 38).





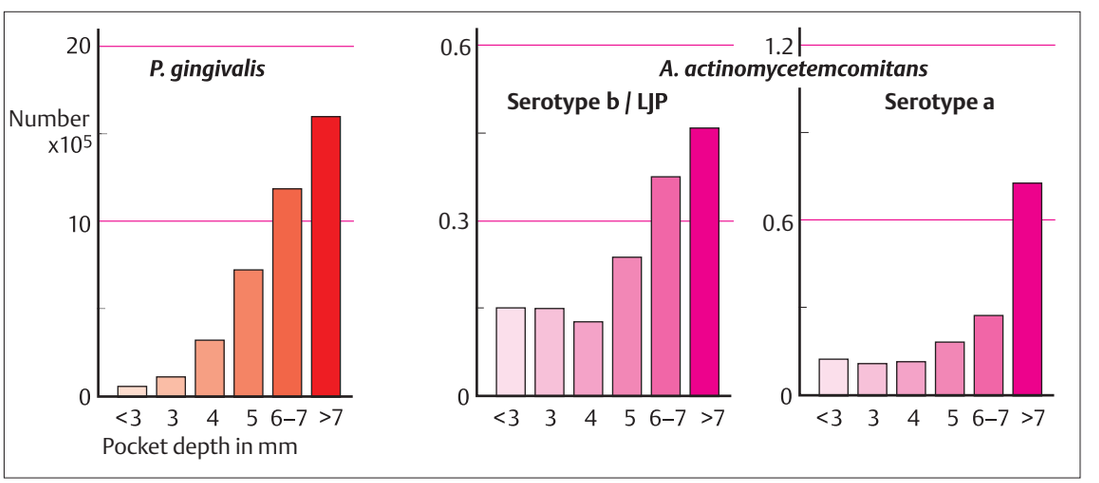
Right: Pg smear. Note the numerous vesicles.

TEM Figs. 73 & 75 courtesy *B. Guggenheim*

74 The Pocket as "Reservoir"

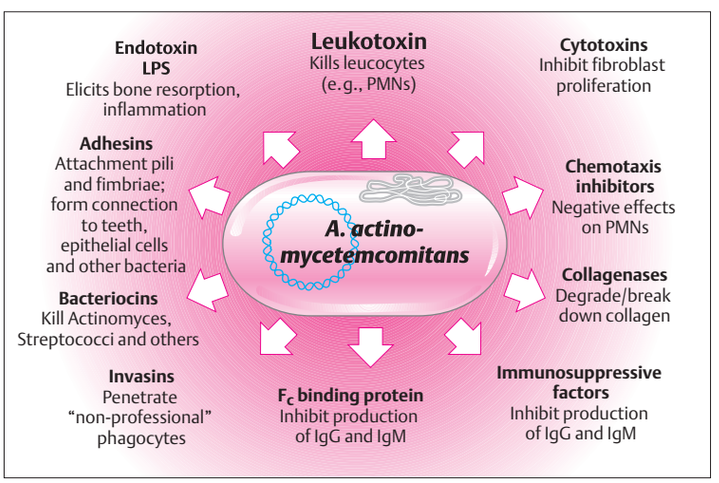
The deeper the *(anaerobic)* pocket, the larger the average number of pathogenic bacteria (*Pg* and *Aa*/serotype b).

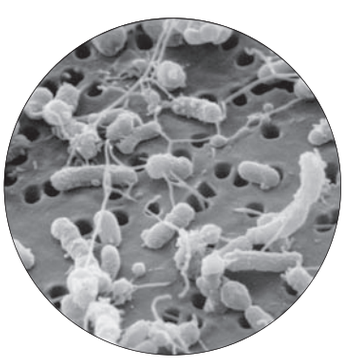
In order to initiate disease, a critical number of pathogenic microorganisms is required. For the three microorganisms depicted here, this number varies considerably. The critical number is, for example, for *Aa*/serotype a lower than for *Pg*.



75 Virulence Factors of *A. actinomycetemcomitans (Aa)*

The *Aa* leukotoxin is one of the most potent toxins. It has the capability to directly inhibit some of the most important components of the human immune system, e. g., PMNs, immunoglobulins and complement activation. Something over 40 *Aa*-subtypes do not express leukotoxin. One subtype does this in extremely high measure, and it is associated with aggressive periodontitis (LJP). Modified from *J. Lindhe et al.*





Right: Smear of *Aa*.

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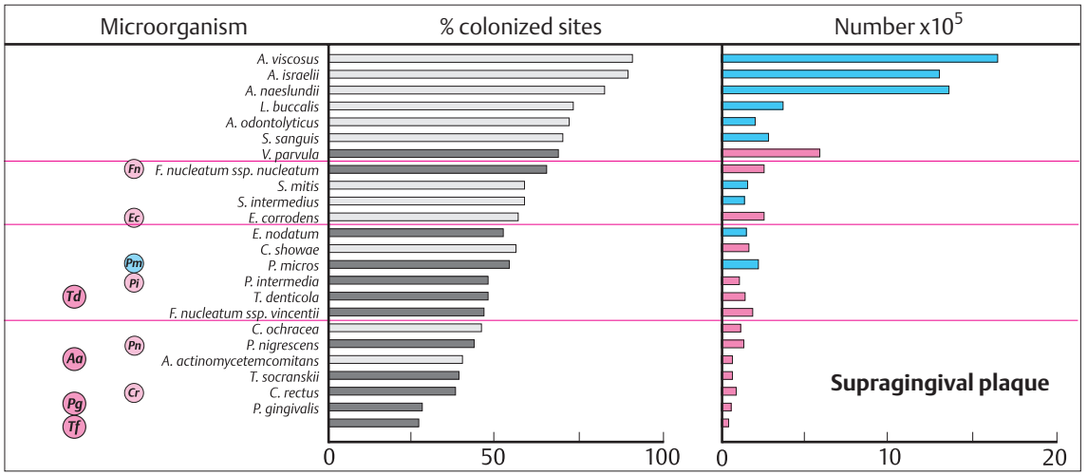
Pathogenic "Single Fighters" vs. Pathogenic Complexes?

Just as many microorganisms in the oral cavity profit from the activities of *Aa* and *Pg*, these two organisms are also dependent upon the others: In most cases, plaque formation begins with the "early colonizers" (*Actinomyces naeslundi/viscosus*, streptococcus sp. etc.; p. 24); *Aa* and *Pg* enter the biofilm only later. They are capable of colonizing epithelia, but less so hard tooth structure. Pg is a strict anaerobe, and is therefore present in increased numbers in deep pockets (which must be taken into consideration during treatment planning).

As the biofilm "matures," the composition of plaque changes considerably, e.g., from primarily gram-positive to gram-negative bacteria. Socransky and co-workers (1998, 1999) described the so-called "red-complex" of *P. gingivalis, T. forsythensis* and *T. denticola* as the "end stage" of the confrontation between pathogenicity and resistance vs. host response.

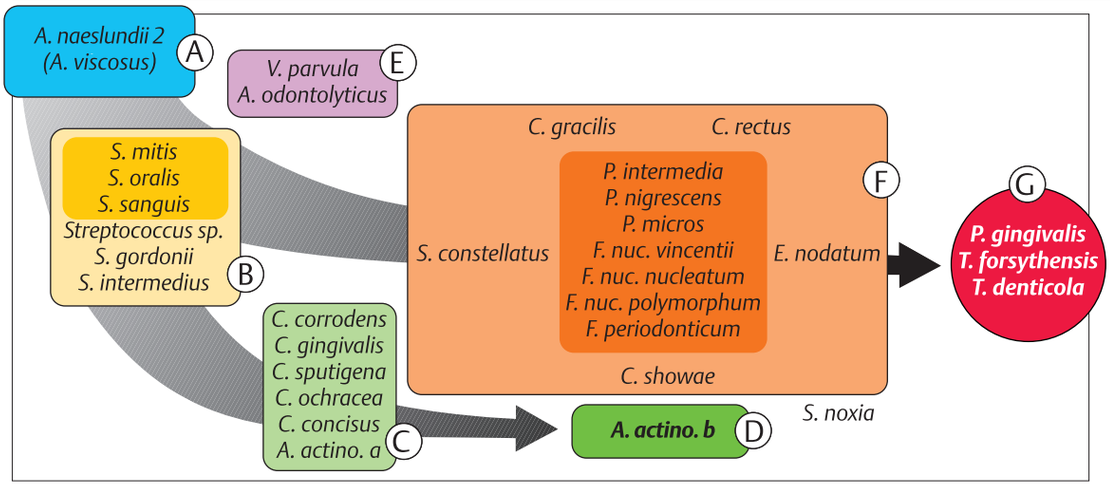
Conclusion

Actinomyces actinomycetemcomitans, P. gingivalis and the "red-complex" (G; Fig. 77) represent the bacterial risk factors for periodontitis in *susceptible individuals* because of their pathogenic virulence factors.

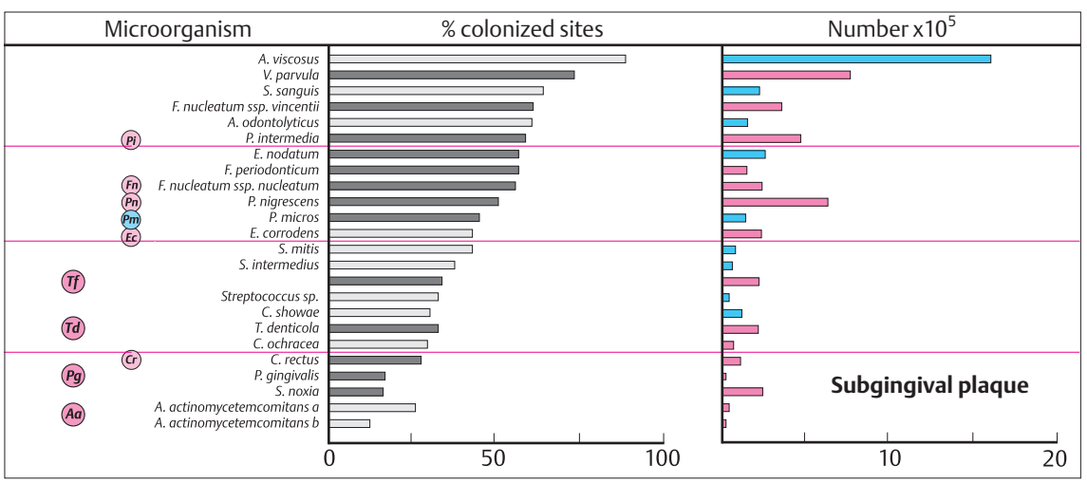


76 Bacteria of the Supragingival Plaque
Percentage of colonized sites (middle) and absolute number (right) of 24 out of 40 tested microorganisms (over 16,000 samples from 213 adults; *Socransky et al.* 1999). The "marker bacteria" are circled on the left.

Bright	aerobe
Dark	anaerobe
Blue	gram-positive
Red	gram-negative



77 Development of Complexes
Dependent upon host response, availability of nutritive substances, and interbacterial competition, the variable "consortium" develops. Bacteria no longer work as single entities: The biofilm acts and reacts as a stable "organism." One pathway (**A** → **G**) leads by way of several intermediate stages to the "red complex," while another pathway (**A** → **D**) leads in the direction of *Aa*.
DNA tests permit the identification of such groupings (p. 185)



78 Bacteria of the Subgingival Plaque
The data and sources for this information derive from Fig. 76. In the subgingival milieu, the type and number of gram-positive organisms decrease, with an ever-increasing number of various gram-negative organisms. Most of the gram-negative marker bacteria are increased (left).
Relatively seldom, and then in only small numbers, one observes *P. gingivalis* and *A. actinomycetemcomitans*: "quality before quantity"?

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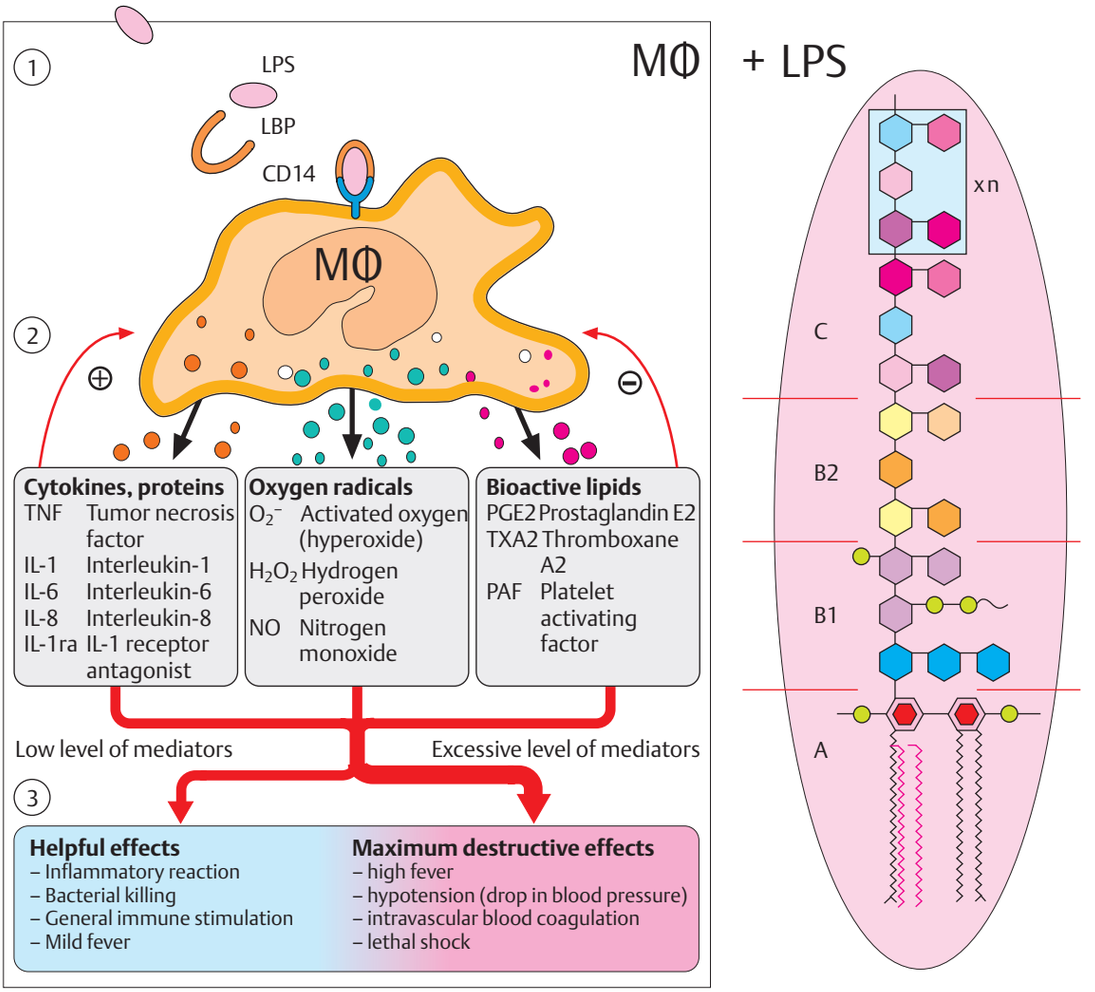
Endotoxins—Lipopolysaccharide (LPS)

Bacterial endotoxins are some of the most fascinating biological molecules. They are heat-stable (pyrogens!), extremely toxic and therefore responsible for inflammatory processes, fever and shock. Chemically, all bacterial endotoxins are lypopolysaccarides (LPS; Rietschel & Brade 1992).

Lipopolysaccarides are one component of the outer membrane of the cell wall of *gram-negative* bacteria (lipid bilayer, p. 31), and render the membrane relatively impermeable (e. g., to antibiotics).

Every bacterial species and subspecies can be differentiated by their typical LPS. The O-specific chain (C) is the antigenic portion of the molecule (*surface antigen*). Lipid A is exclusively responsible for the toxicity and the activation of the host-immune reaction via macrophages (MΦ); lipid A is found in the LPS of the outer membrane. Only *free* LPS molecules are toxic for the host, not those embedded within the bacterial cell wall. Free LPS occurs:

- Through vesicle formation
- During cell division (proliferation)
- After destruction of the cell wall of dead bacteria.



79 LPS—Structure and Function
The stimulation of macrophages (MΦ) by LPS initiates the immune defense reaction and the inflammatory reaction.

1 LPS, coupled with the LPS-binding protein LBP, activates macrophages via the specific LPS-receptor **CD14**.

2 This produces the following mediators: cytokines, oxygen radicals, lipids (e. g., prostaglandins), which may work singularly or in combination. Cytokines enhance (+) while lipids inhibit (-) their individual synthesis via "autocrine" mechanisms.

3 Dependent upon the concentration of mediators, a broad spectrum of host reactions is initiated.

Right: Configuration of LPS.

A **Lipid A**: Up to six variable fatty acids, diglucosamine (red), phosphate (green)

B **Nuclear zones 1 and 2**: diverse sugars

C **O-specific chains**: repeating units; type-specific antigens.

Modified from *E. Reitschel & H. Brade (1992)*

Interaction Between LPS and the Host

Even though metabolism by plaque bacteria results in many biologically active substances (endotoxins, enzymes, chemokines, phlogogens) that may cause *direct* damage, LPS from gram-negative bacteria plays the most important role in the pathogenesis of periodontal diseases: It stimulates macrophages to produce cytokines, activate the *alternative* complement pathway, is antigenically effective and also cytotoxic. With slight plaque accumulation, LPS is present in only low concentrations, but this may "awaken" and activate a dormant host immune system.

The action of LPS is primarily *indirect*: It activates not only macrophages but also endothelial cells and fibroblasts to produce cytokines. LPS molecules from various periodontopathic bacteria induce quite varying amounts of cytokines. LPS from *Pg*, for example, induces much higher levels of cytokines than other microorganisms associated with periodontitis (Takada et al. 1991).

The host may respond to LPS with varying intensity, but the pathogenesis of periodontal disease is primarily determined by the host's immune reaction. This can vary considerably from person to person (Pathogenesis, p. 39).

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Pathogenesis—
Reactions and Defense Capabilities of the Host

Periodontal diseases represent a family of related, usually chronic, and very often aggressive bacterial infectious diseases.
The etiologic agents, the *primary pathogens*, are several virulent bacteria found in dental plaque and in the oral cavity. The most important ones are *Aa, Pg* and *Tf*, although other species may play enhancing roles (cf. p. 33). Bacteria *must* be present if periodontitis is to be initiated and propagated, but bacteria are not solely responsible for this disease.
Host factors in combination with additional risk factors (smoking, stress etc.) have been shown by recent research to significantly influence the susceptibility, expression (e. g., type and severity) and progression of periodontitis. For this reason it is absolutely necessary to achieve an understanding of the possible reactions of the host organism to the bacterial challenge.

This chapter is therefore structured as follows:

- New concepts concerning pathogenesis—a paradigm shift
- Host defense: mechanisms and participants—cells and molecules
- Non-specific, congenital and specific, acquired immunity—interactive relationships
- Surface molecules, markers (CD), receptors
- Regulating molecules, mediators: cytokines—eikosanoides—matrix metalloproteinases
- Risk factors: genetics—environment—life style
- Pathogenesis I—initial inflammatory reactions, PMN diapedesis
- Pathogenesis II—histologic stages
- Pathogenesis III—molecular niveau
- Attachment loss: breakdown of connective tissue and bone
- The transition from gingivitis to periodontitis—cyclic course of destruction
- Periodontal infections and systemic diseases
- Etiology and pathogenesis—summary and conclusions

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New Concepts of Pathogenesis

Research on the etiology and pathogenesis of periodontitis has provided so much new knowledge in recent years that it is reasonable to speak of a *paradigm shift*. This is based primarily on new knowledge concerning biofilms (microorganisms), molecular biology, host susceptibility, risk factors and genetics.

Biofilm

The adherent bacterial flora—dental plaque—is a highly organized biofilm. Bacteria within the biofilm are well protected from the host response as well as from antimicrobial agents. The only effective therapy is purely physical destruction and elimination of the biofilm by means of scaling (tooth and root surface cleaning, supra- and subgingivally).

Molecular Biology

New knowledge about molecular and cellular mechanisms has led to better understanding of the processes by which bacteria in the biofilm elicit immune and inflammatory reactions in the host, leading to connective tissue destruction and resorption of the alveolar bone.

Host Sensitivity and Risk Factors

In order for the aforementioned mechanisms to occur, leading to the initiation and establishment of periodontitis, a susceptible host must be present. The microorganisms, by themselves, *cannot* cause the disease process. Environmental factors and risk factors such as smoking or inherited (unfavorable) host defense mechanisms modify the host reaction and are primarily responsible for the destruction, progression, severity and clinical picture of periodontitis.

Genetics

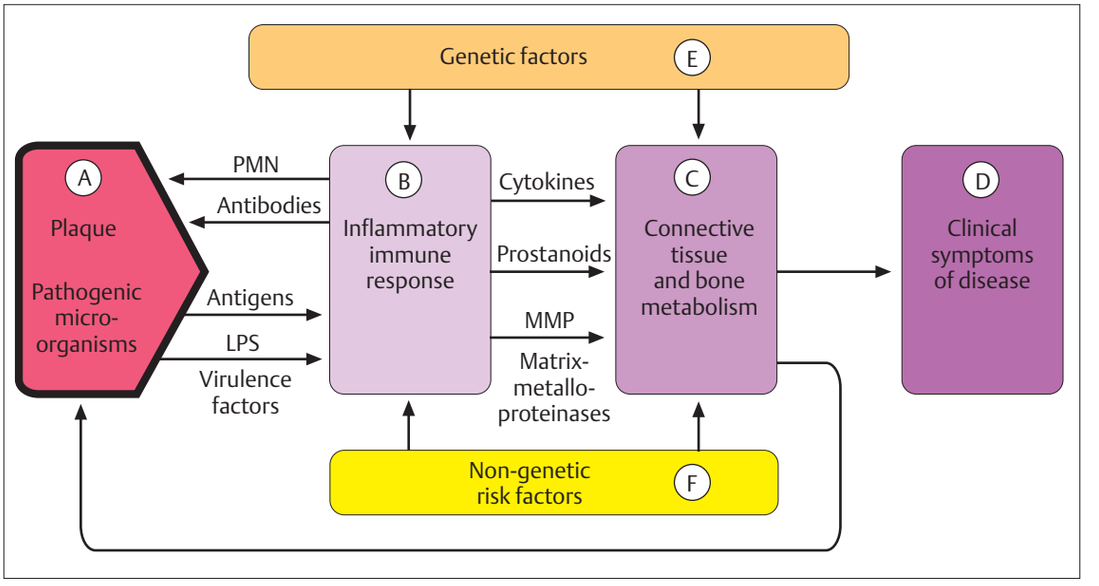
The various molecular biological mechanisms, the host susceptibility to inflammatory damage, and congenital risk factors are determined for the most part by genetics (p. 51). Therefore heredity assumes a much larger significance today than was previously assumed; humans are born with their predisposition towards periodontitis!

80 Pathogenesis of Human Periodontitis
The disease cannot be phenotypically homogeneous because of the many influences upon the host reactions.

A	Microbiology	pp. 23–40
B	Host reactions	pp. 41–45
C	Metabolism	pp. 60/61
D	Clinical Symptoms	pp. 62/63
E	Genetics	pp. 51–53
F	Risk Factors	pp. 51–54

PMN Polymorphoneuclear Granulocytes (p. 42)
LPS Lipopolysaccharide (p. 38)

Modified from *R. Page & K. Kornman, 1997*



Therapeutic and Diagnostic Consequences

Since the 1960's, prevention and therapy have consisted primarily in the removal or reduction of the infectious bacteria. In the future, attempts will be made to utilize the host response diagnostically and to influence it in therapy; for example, progressive periodontitis is characterized not only by elevated levels of bacterial substances, above all lipopolysaccharides (LPS), but also by *pro-inflammatory* mediators. These include the cytokines TNFα, IL-1 and IL-6, as well a prostaglandins (especially PGE2) and matrix metalloproteinases (MMP).

In contrast, in periodontal health or in a case of a stable lesion, the level of bacterial substances (LPS) is low and the *inflammation-reducing* cytokines such as IL-10, TGFβ as well as MMP- inhibitors (TIMP) are higher.

In the future, therapeutic measures will include attempts to reduce disease-eliciting factors, and to stimulate resistance factors. In addition, it will be necessary to maintain and even increase our efforts to eliminate or reduce other risk factors such as diabetes, smoking, stress and environmental factors.

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Host Response—Mechanisms and "Participants"

Various mechanisms within the host organism inhibit bacterial infection. In addition to physical and chemical barriers (skin/keratinization; mucosa/mucous; saliva with mucins, lysozmes, histozines, etc.) the human immune system plays the most important protective role.

One can differentiate between:

- Non-specific, naturally present, "congenital" immunity (p. 42)
- Specific, acquired immunity (p. 43)

First Axis of the Defense System
This consists of cells of non-specific immunity (phagocytes, natural killer cells) and also diverse molecular effectors (complement, C-reactive protein, etc.)

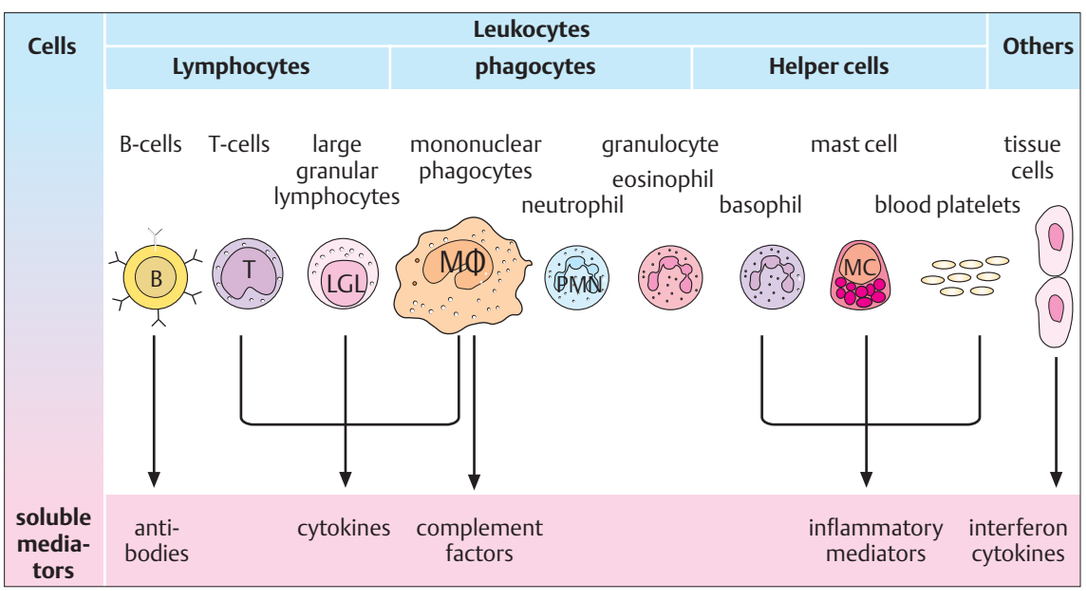
Second Axis of the Immune System
This consists of specific immunity, above all the lymphocytes (T- and B-cells, as well as antigen-presenting cells, e. g., macrophages/MΦ) as well as the various immunoglobulins.

Cellular components of the host response

- **Inflammatory cells**
  - PMN polymorphonuclear granulocyte (p. 42)
    Neutrophils
    Eosinophilic granulocytes
    Basophilic granulocytes, mast cells
  - Thrombocytes
- **Resident cells**
  - Fibroblasts, endothelial cells, epithelial cells
- **Antigen-presenting cells (APC)** (p. 42)
  - Monocytes/macrophages
  - Langerhans cells, dendritic cells
- **Lymphocytes** (p. 43)
  - T-cells
    T-helper cells T_H1 and T_H2 = T4-cells
    T-cytotoxic cells Tc = T8-cells
  - B-cells, plasma cells (PC)
  - NK = "natural killer cells"

81 Cellular Components of the Immune System
The important cell types for immune response include inflammatory cells, resident cells, antigen-presenting cells and lymphocytes. These are described in detail in the following pages.

Modified from *R. Sanderink 1999*



82 Components of the Immune System—Cells and Soluble Mediators
A large percentage of the soluble mediators such as complement factors, acute phase proteins (C-reactive protein) etc. are produced in the liver.
Each and every cell that is involved in the maintenance of the structure of the periodontium also synthesizes a large spectrum of cytokines (p. 47) and other mediators. It is important to recognize that even the resident (non-mobile) cells are active in the immunologic process.

Modified from *M. Roitt et al. 1995*

Effectors and regulatory cellular surface molecules

- **Ig Immunoglobulins** (p. 43)
  - 5 classes: IgA, IgM, IgG, IgD, IgE
    Subclasses: IgA1 and A2, IgG1 through G4
- **C Complement** (p. 42)
  - C 1-9 cascade
  - MAC/major attack complex / C9
- **Cytokines** (p. 47/48)
  - Interleukins (IL)
  - Cytotoxic factors (TNF)
  - Interferon (IFN)
  - Colony-stimulating factors (CSF)
  - Growth factors (GF)
- **Chemokines** (p. 48)
  - "classic," α- and β-Chemokines
- **Eicosanoids** (p. 49)
  - Prostaglandins (e. g., PGE2)
    Leukotrienes (e. g., LTB4)
- **Other mediators** (p. 50)
  - Matrix metalloproteinases
- **Receptors, surface antigens** (p. 46)
  - MHC classes I and II (HLA)
  - CD antigens (classification)
  - Receptor molecules
  - Adhesins

83 Humoral Components of the Immune System
Included in this table are also receptors and surface antigens (markers), without which the cell-cell relationships would not be possible (signal formation, antigen presentation etc.).

Modified from *R. Sanderink 1999*

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Non-specific, Congenital Immunity—the First Line of Defense

Non-specific immunity represents the quite potent first line of defense. It is phylogenetically ancient, quick to respond, and involves the mechanisms of *phagocytosis* (killing and digesting of microorganisms) and *acute* inflammation.

The *cellular components* of the non-specific immune system consist of the phagocytic cells (neutrophilic granulocytes [PMN] and monocytes/macrophges [MΦ]) as well as the natural killer cells [NK]).

Soluble effector molecules include complement (C) and acute phase proteins (C-reactive proteins), which are synthesized for the most part in the liver.

The inflammatory reaction is highly regulated by *mediator molecules*. Among these is bradykinin, but above all the pro-inflammatory cytokines (pp. 47–48), prostaglandins (p. 49), and many other components of the primary defense axis:

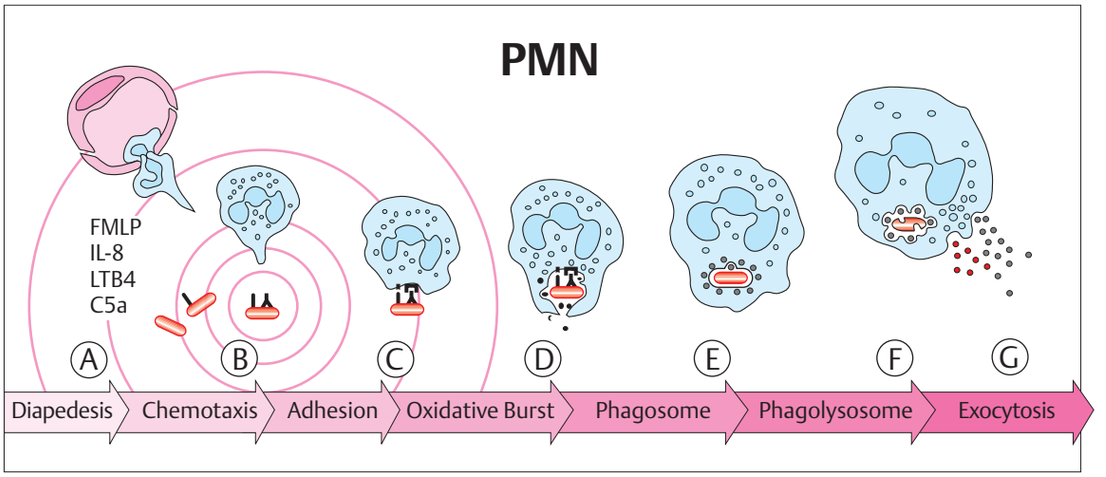
- Polymorphonuclear granulocytes (PMN)
- Monocytes/macrophages (MΦ)
- Natural killer cells (NK)
- Complement (C)
- Additional inflammatory mediators

Non-specific immunity possesses no "memory."

84 Polymorphonuclear Granulocytes (PMN)

PMNs represent the first line of defense. They can be constantly recovered from the gingival sulcus.

A Adhesion to the blood vascular walls and penetration into the tissues
B Chemotactic movement
C Capture of opsonized microorganism (MO)
D Phagocytosis of the MO
E Formation of phagosomes
F Phagolysosomal killing
G Exocytosis (release of killing mediators)

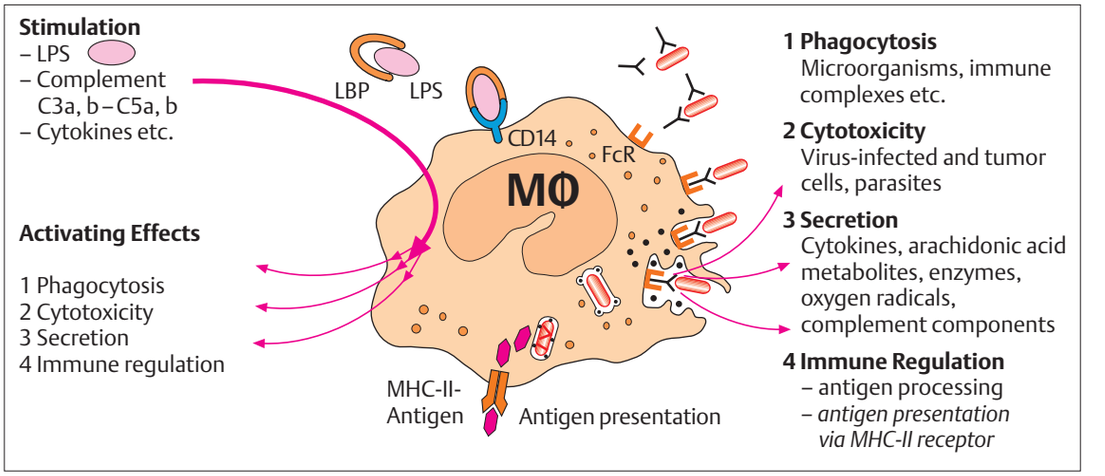


85 Macrophages (MΦ)

The most important component of specific host defense system: They present to the T-lymphocytes the antigens of the phagocytized microorganisms (APC).

LPS Lipopolysaccharide
LBP LPS-binding proteins
CD14 Receptors for the LPS-LBP complex
FcR Immunoglobulin receptor
MHC II Major histocompatibility complex II, p. 46

Modified from *Gemsa et al. 1997*

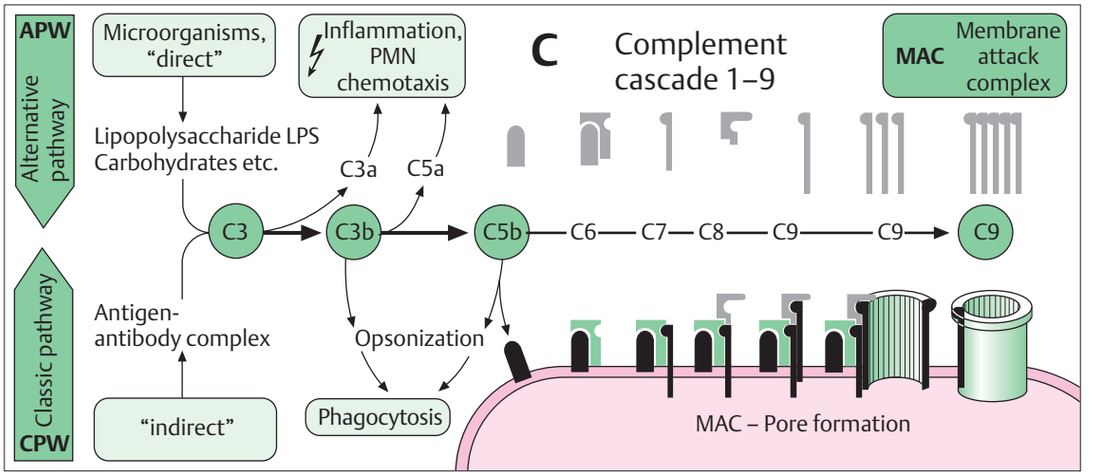


86 Complement (C)

Soluble complement factors C-1 through C-9 circulate in the blood. Most of the C-components are proteins possessing enzyme functions and can be activated through two different pathways.

- Chemotactic activity: **C5a** stronger than **C3a**.
- C3b opsonizes initially, followed by **C5b** etc.
- Pore formation is bacteriocidal through the **MAC**

Modified from *Koolman & Röhm 1998*



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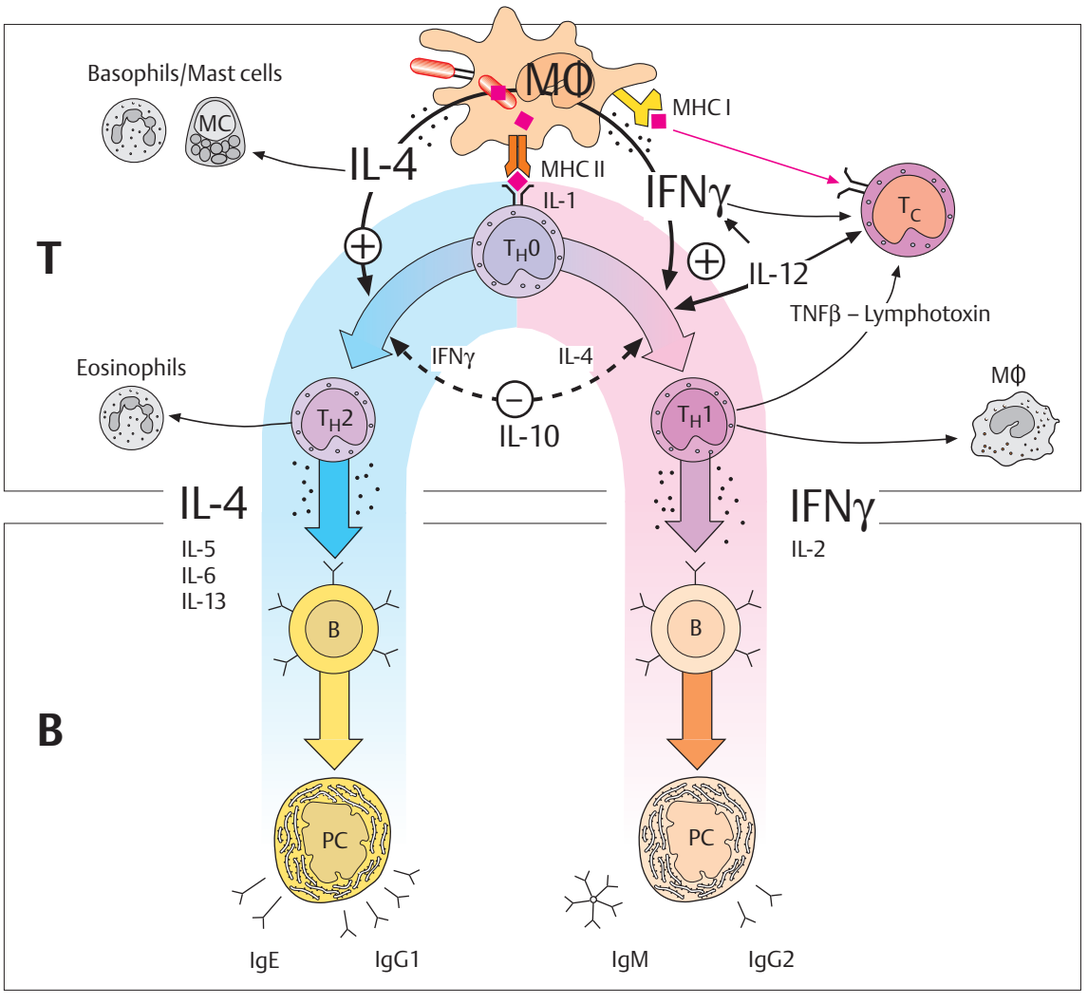
Specific, Acquired Immunity—the Second Line of Defense

- T-Lymphocytes
- B-Lymphocytes/plasma cells
- Immunoglobulins

The phylogenetically younger, specific immunity is responsible for the fine regulation of host defense. Lymphocytes are the key cells of the immune system: They monitor the various immune responses. Two main cell groups can be distinguished: T-cells and B-cells.

It requires more time to set the second line of defense into motion, but specific immunity possesses a "memory" function (immunization!)

- *T-lymphocytes* fulfill numerous functions:
  - Cytotoxic T-cells (T_c-, T8-, CD8 cells) eliminate foreign cells as well as damaged host cells (pore-forming "lymphotoxin" = TNFβ);
  - T-helper cells (T_H-, T4-, CD4 cells)—the subclasses T_H1 and T_H2 secrete various groups of cytokines and thereby program various pathways of the immune reaction (T_H1 → MΦ or T_H2 → Ig).
- *B-cells* as well as immunoglobulin-forming *plasma cells* are responsible for the opsonization of foreign bodies (pp. 44–45), which is regulated mainly by the T_H2 pathway.



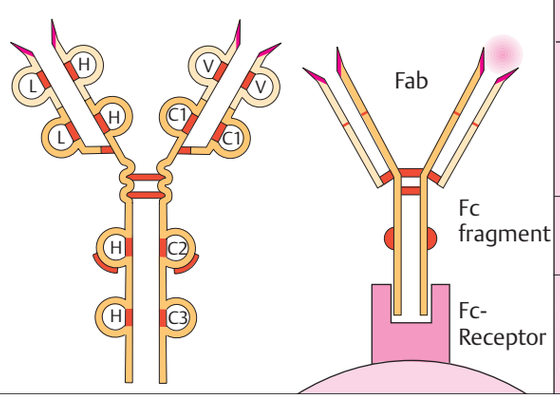
87 T-Cells/T-Lymphocytes
These cells are activated following direct cell contact of antigen-presenting MHC-I and -II molecules (upon MΦ and B-cells), with the T-cell receptor complex (p. 46). T-helper cells (**T_HO**) differentiate via IL-4 and other cytokines (p. 47) to **T_H2** cells, via IFNγ to **T_H1** cells; this T-cell subpopulation enhances the differentiation of B-cells in addition to the other defense cells (gray). T-cells (right) synthesize cytotoxins such as lymphotoxin (= tumor necrosis factor, TNFβ).

88 B-Cells, Plasma Cells (PC)
B-cells are activated by cytokines from the T-helper cells (IL-4 and IFNγ) as well as antigens.
Mature B-cells express numerous surface-fixed **Ig** (= B-cell receptor) and under the influence of cytokines evolve into plasma cells (**PC**), which release massive amounts of Ig molecules. Cytokines from the T_H1 and T_H2 cells enhance the "switch" from IgE and IgM to the IgG subclasses of immunoglobulins.

Modified from *Zinkernagel 1998*

Ig Immunoglobulins

Classes	Subclasses
IgG	IgG1 IgG2 IgG3 IgG4
IgM	
IgA	IgA1 IgA2
IgD	
IgE	



Fc receptors of defense cells			
MΦ	Fcγ Fcγ Fcγ Fcμ Fcε	RI RIIa RIII R RII	CD64 CD32 CD16
PMN	Fcγ Fcγ Fcα	RIIa RIII R	CD32
Mast-cell/ Basophilic granulocyte	Fcε	RI	

89 Immunoglobulins (Ig)
Left: Ig classes and subclasses.

Middle: Ig molecules are Y-shaped glycoproteins with heavy (**H**) and light (**L**) chains, as well as constant (**C**) and variable (**V**) domains.

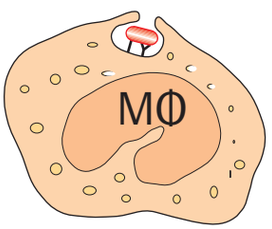
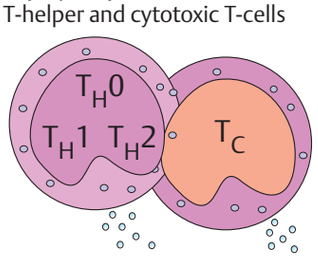
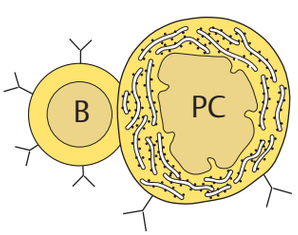
Right: The **FaB** fragment contains the antigen-binding site. **Fc** fragment binds on Fc receptors of various defense cells and on complement (CPW, Fig. 90).

Modified from *Roitt et al. 1995*

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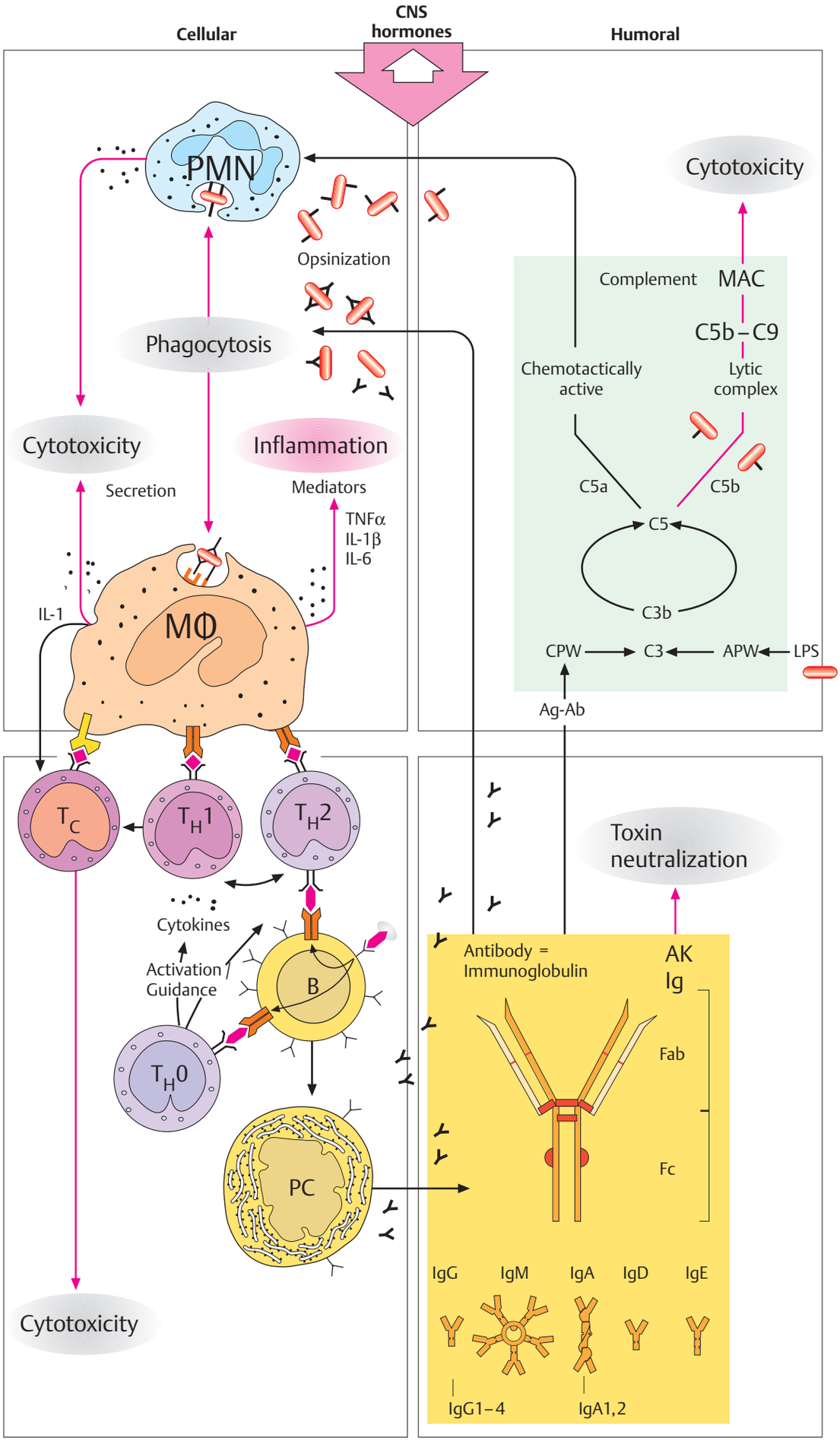
90 Cellular and Humoral Components

Components of the Immune System—Summary

Cell Type /Molecules	Characteristics	Functions, Effects
Polymorphonuclear granulocyte (PMN, Microphage) 	• Differentiation, maturation and clonal expansion in bone marrow • Lifespan: 2-3 days • 10-20 µm diameter • **Fc, C3- C5-receptors** • Enzyme-containing granules	• Diapedesis, chemotaxis • Phagocytosis: Adherence, phagolysosome formation, digestion, "oxidative burst" • Release of lysosomal enzymes • Release of inflammatory mediators: prostaglandins (e. g., **PGE_2**), leukotrienes (e. g., **LTB4**), cytokines
Complement system; cascade C1–C9	*CPW—classical pathway* • Classical activation pathway, elicited by antibody/immunoglobulins (Ab/Ig), which aggregate with antigens (Ag): **Ag/Ab** complex *APW—alternative pathway* • Alternative activation — independent of antibodies — via bacterial mucopolysaccharide (e. g., **LPS**); leads to separation of **C3** into **C3b** (and **C3a**) and to activation of **C5**	• Immune adherence: binds to the **Fc** fragment of antibodies • Elevated capillary permeability • Anaphylatoxin • PMN chemotaxis (see also **C5a**) • Opsinization of bacteria • Irreversible, structure-dependent and functional membrane damage via formation of the **MAC** ("membrane attack complex"), which leads to pore formation and ultimate cell lysis
Monocyte / Macrophage (MΦ) 	• Differentiation, maturation and clonal expansion within bone marrow • Lifespan: months • 12—25 µm diameter • Diverse receptors: – **Fc** for immunoglobulins – **CR** for complement (**C3**) – **CD** 14 for lipopolysaccharide (p. 40)	• Phagocytosis, antigen processing • Antigen presentation (for **T**- and **B**-cells) • Production and secretion of bioactive substances: – Cytokines (p. 47-48): **IFNα** (anti-viral), **TNFα, IL-1, IL-6, IL-8** – Components of the complement system – Lysosomal enzymes – Arachidonic acid metabolites (p. 49) – Oxygen and nitrogen radicals (p. 42)
T-lymphocytes T-helper and cytotoxic T-cells 	• Stem cells from bone marrow • Maturation: Thymus-dependent (**T**) • Lifespan: Months • 6-7 µm diameter; activated: 10 µm • Antigen recognition: T-cell receptor (**TCR**) • Surface antigens / Marker molecules: – **CD4** on helper cells (**T_H0; T_H1, T_H2**) – **CD8** on cytotoxic cells (**T_c**) • **T**-memory cells	*"Cell-mediated immunity"* • **T**-helper cells: Assist with antibody production and the cytotoxic T-cell response (**T_c**-cells); activation of macrophages • Suppression of an immune response • Release of cytokines of various subclasses (**T_H0, T_H1, T_H2**): **IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-10, IL-12, IL-13; IFNγ, TNFβ** • **Memory function of memory cells**
B-lymphocyte/plasma cell 	• Stem cells from bone marrow (**B**) • Maturation: fetal liver, bone marrow, Peyer's patches • Lifespan: Months • 6-7 µm diameter • Activated (plasma cell, **PC**): 10-15 µm diameter • Surface **Ig** as antigen receptor • **B**-Memory cell	*"Humoral Immunity"* • Immunoglobulin synthesis • **B**-lymphocyte: **Ig** antigen-specific, variable class • Plasma cell: **Ig** antigen- and class-specific • Clonal expansion following activation • Memory function of memory cells
Antibody (Ab) = Immunoglobulin (Ig) Molecular weight / percentage **IgG** 150 000 / 80% **IgM** 900 000 / 13% **IgA** 300 000 / 6% **IgD** 185 000 / 1% **IgE** 280 000 / 0.02%	• 5 classes: **IgA, IgD, IgE, IgG, IgM** • Subclasses: **IgA1, IgA2, IgG1, IgG2, IgG3, IgG4** • Basic molecule: – Heavy (H) and light (L) polypeptide chains – Constant (C) and variable (V) domains: Millions of antigen-specific variations are possible	• Opsinization of microorganisms • Antigen binding: Antigen-antibody complex • Complement activation (**CPW**) • Toxin neutralization • Neutralization of viruses • Hypersensitivity reaction (**Types 1-3**)

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Interactions Between Non-specific and Specific Immunity



91 Interactions

Non-Specific Immunity—First Line of Defense
Modified from *Roitt et al. 1985*

- *PMN granulocytes*: The cells represent the first and most important defense reactions within the junctional epithelium and gingival sulcus (phagocytosis). As PMNs are destroyed, lysozomal enzymes and toxic radicals are set free, which leads to cytotoxicity.
- *Complement*: Activated initially independent of antibodies by way of the initial, alternative pathway (APW); later it is dependent on antibodies via the classical pathway (CPW): Opsonization, chemotaxis, membrane destructive (MAC, Fig. 86) → cytotoxicity.
- *Macrophages (MΦ):*
  - phagocytosis
  - release of cytokines and inflammatory metabolites and enzymes
  - antigen presentation: MΦ regulates T- and B-cells

Specific Immunity—Second Line of Defense

- *T-lymphocytes*: These are responsible for cell-mediated immunity
  - T-helper cells (T_H) work to regulate and/or activate via their cytokines
  - T_C-cells are cytotoxic
  - Memory cells of both types provide the immunologic "memory" of the T-cells
- *B-lymphocytes/plasma cells*: These are responsible for humoral immunity. Upon contact with antigens and activation via T_H cells, B-lymphocytes differentiate into antibody-producing plasma cells; "memory" cells of the B-lymphocytes
- *Antibodies (Immunoglobulins):*
  - Classes: IgG, IgM, IgA, IgD, IgE
  - Subclasses: IgG1–4 and IgA1–2 are immunoglobulins that are serum proteins, which bind specific antigens and are induced by these antigens. They have opsonization functions, neutralize toxins and activate complement (CPW).

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Regulatory Cell Surface Molecules: Markers, Receptors

- MHC—major histocompatibility complex
- CD—cluster of differentiation
- Receptor molecules
- Adhesins and ligands

MHC molecule groups (classes I and II) make possible the differentiation between "self" and "non-self" (rejection reaction after organ transplantation).

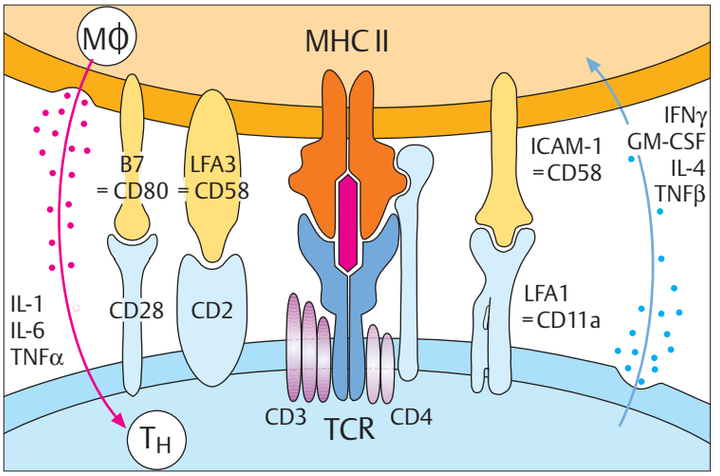
CD4-marker: Lymphocytes and leukocytes express marker molecules ("antigens") on their surface, which permits a classification of the cell populations.

A systematic and individualized nomenclature, the CD-system, was developed for these markers.

Receptor molecules: Receptors are found on the surfaces of all cells, and most of these can receive bioactive molecules that either enhance or inhibit cell functions, e. g., cytokines, chemokines, complement factors, antigens and antibodies.

Adhesins serve as co-receptors, stabilize the primary receptor binding, and are responsible for the important "second signal" (general activation).

92 Antigen Presentation
By means of its MHC-II receptors, the macrophage presents foreign substrates, e. g., bacterial antigens (red) to a T-helper cell via the T-cell receptor complex (TCR+ CD3+ CD4).
MHC-II complexes (orange in the figure) are found on the antigen-presenting cells (MΦ, dendritic cells, as well as B-cells).
Support for this primary binding is provided by co-receptors, which also are important for the so-called "second signal" (definitive activation of the target cells).



Receptor Binding, MΦ—T_H
Supportive and signal-building co-receptors with their CD numbers. The old names indicate their functions, e. g.:

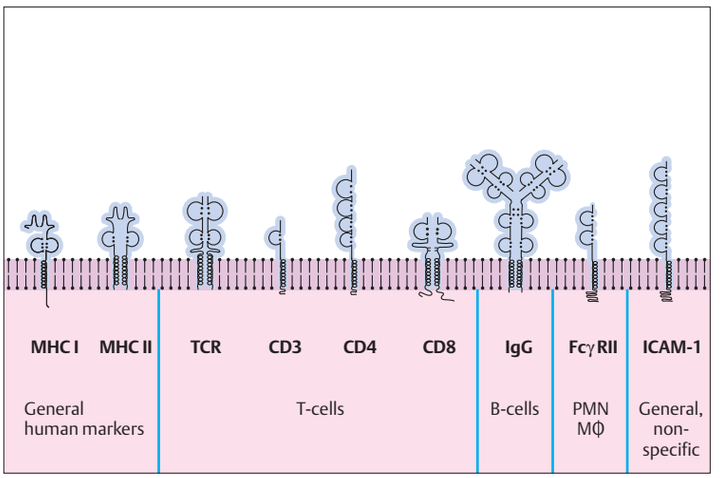
LFA leukocyte function-associated antigens
ICAM Intercellular adhesion molecule

Release of cytokines following activation by. . .

- *MΦ:* TNFα, IL-1, IL-6
- *T-cells*:INFγ, GM-CFS, IL-4, TGFβ etc.

93 Immunoglobuin Gene Super Family
This family of surface molecules (receptors) contains numerous important members:

- MHC-I and –II molecules
- T-cell receptors (TCR) with their co-receptors; these are necessary for T-cell activation (see above)
- Immunoglobulins (pictured: membrane-bound IgG = "B-cell receptor")
- Fc receptors (Fig. 89)
- Diverse ICAM (adhesins)

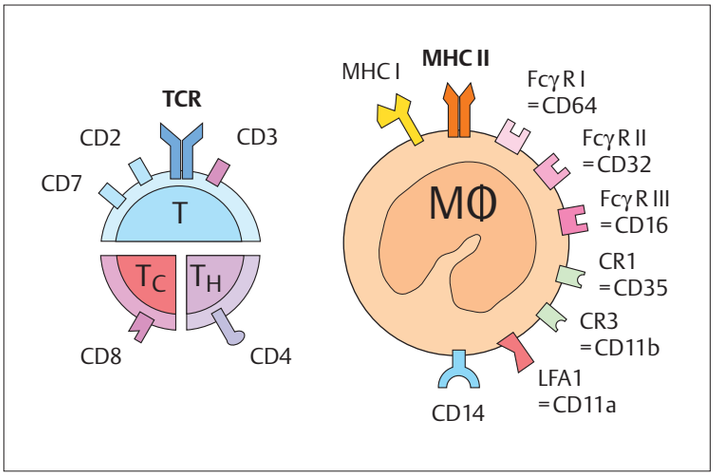


Family of Adhesins
Contained within this family of accessory surface molecules are:

- Integrins
- Lectins
- Selectins
- ICAM of the Ig-gene superfamily.

Adhesins increase the avidity of cell-cell binding. This is especially important for the "ordered" diapedesis of inflammatory cells out of the blood vessels (p. 55).

94 Most Important Surface Markers of T-cells and MΦ
Macrophages interact with numerous biologically active molecules such as immunoglobulins (via Fc receptors), complement (via C-receptors, CR) as well as bacterial LPS (via CD14).
T-cells require fewer receptors: Following activation (antigen presentation by MΦ), they are effective through their repertoire of cytokines (p. 47). The subpopulations of cytotoxic T-cells (T_C) carry the CD8-receptor; T-helper cells (T_H) carry the CD4-receptor.



TCR T-cell receptor
MHC Major histocompatibility complex (classes I and II)
FcR Receptors of the Fc fragments of immunoglobulins (Fig. 89)
CR Complement receptor
CD14 Receptor for lipopolysaccharide (LPS)

CD Classification
So far, over 130 surface molecules have been classified and their functional significance described.

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Cytokines

Cytokines are hormone-like, small molecular weight peptides or glycopeptides. They regulate all of the important biological processes, such as *cell proliferation*, *cell growth, cell activation, inflammation, immunity* and *repair*. Some cytokines (e. g., IL-8, MCP-1) have a chemotactic effect on immune cells. Members of the cytokine family include:

- Interleukins (formerly "lymphokines")
- Cytotoxic factors (tumor necrosis factor α and β)
- Interferons (anti-viral IFNα and β, "immune-IFNγ")
- Colony stimulating factors (CSF)
- Growth factors (GF)

In combination with other mediators, the cytokines build a large network, which is responsible not only for normal *tissue homeostasis* but also for every type of immune response. Most of the cytokines are local, a small group is also effective systemically (TNF, IL-1, IL-6). Specific receptors for these molecules are present on the target cells (for IL-1 through IL-10, e. g., CD121 through CD130; p. 46).

During the inflammatory reaction, which is a component of natural immunity (p. 42), the pro-inflammatory cytokines (IL-1β, IL-6, IL-8, TNFα, IFNγ) do battle with the inhibitory "immune-regulating" molecules (IL-1ra, IL-10, TGFβ; p. 48).

Cytokines

P = pro-inflammatory
A = anti-inflammatory
C = chemotactic

Interleukins IL

P	**IL-1α**	interleukin 1α
P	**IL-1β**	interleukin 1β
	IL-2	interleukin 2
	IL-3	interleukin 3
	IL-4	interleukin 4
	IL-5	interleukin 5
P	**IL-6**	interleukin 6
	IL-7	interleukin 7
C	**IL-8**	interleukin 8
	IL-9	interleukin 9
A	**IL-10**	interleukin 10
	IL-11	interleukin 11
P	**IL-12**	interleukin 12
A	**IL-13**	interleukin 13

... and others

Cytotoxic Factors

P	**TNFα**	tumor necrosis factor α
	TNFβ	tumor necrosis factor β = "lymphotoxin"

Interferon IFN

	IFNα	interferon α
	IFNβ	interferon β
P	**IFNγ**	interferon γ

Colony Stimulating Factors CSF

G-	**CSF**	granulocyte-CSF
M-	**CSF**	macrophage-CSF
GM-	**CSF**	granulocyte/MΦ-CSF
Multi-CSF = IL-3		

Growth Factors GF

A	**TGFα**	tissue growth factor α
A	**TGFβ**	tissue growth factor β
	EGF	epithelial GF
	FGF	fibroblast GF
	PDGF	platelet-derived GF
	IGF	insulin-like GF
	BMPs	bone morphogenetic protein
	PTHrP	parahormone-related protein

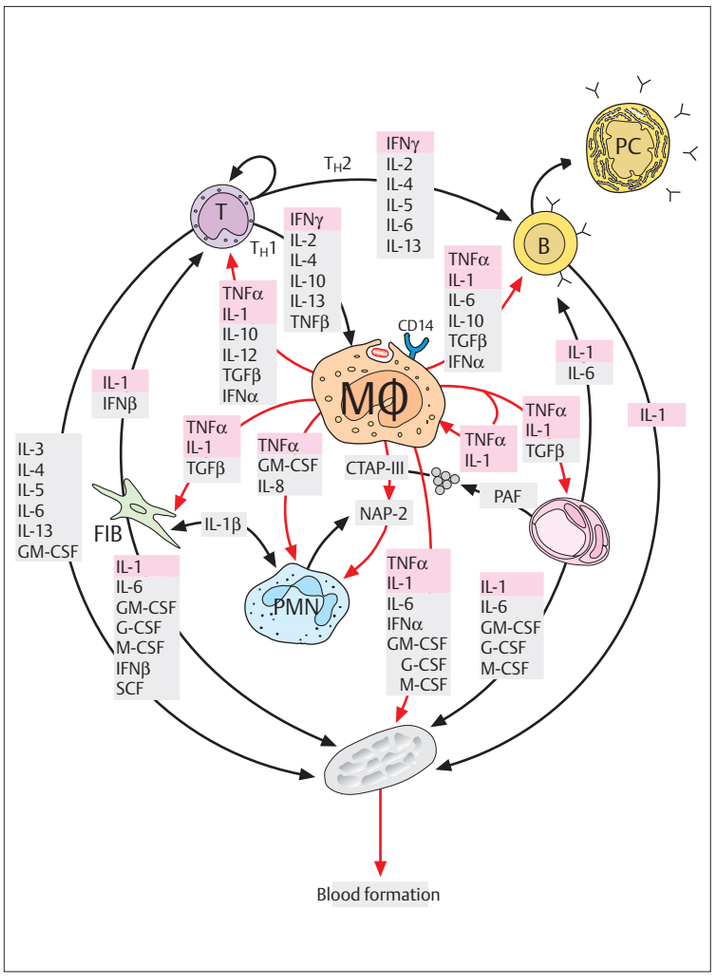
Chemotactically Active Cytokines

α-chemokines

	IL-8	interleuken 8

β-chemokines

	RANTES	regulated on activation, normal T-cell expressed and secreted
	MCP-1	monocyte-chemotactic protein
	MIP	macrophage-inflammatory protein



95 Cytokine Network

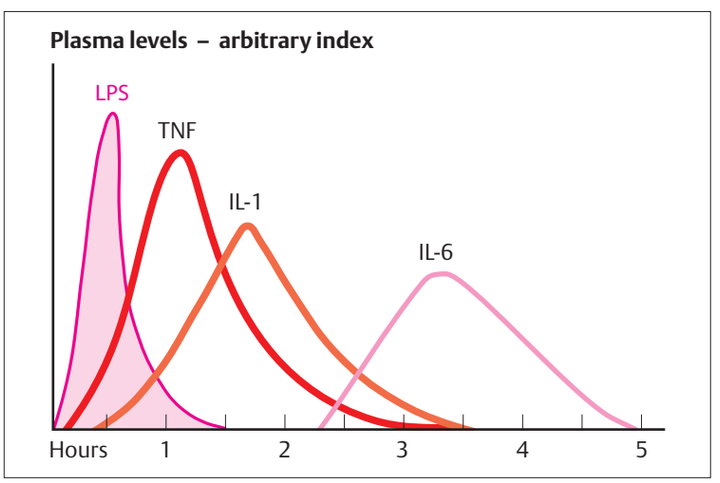
The cytokine-producing cells and their target cells exchange signals continuously. This supports primarily tissue homeostasis ("cellular internet").

A high degree of regulation can be achieved over certain physiologic or exceptional functions, for example immune defense, inflammation, healing, growth and cellular proliferation. TNF and IL-1 are important *cytokine inductors* that exert local as well as systemic effects (e. g., effects on the neuroendocrine system, pituitary hormones, liver).

In addition to the *pro-inflammatory* cytokines, one also observes their antagonists: *anti-inflammatory* cytokines (IL-1 receptor antagonist IL-1ra, IL-10, TGFβ).

CTAP-III	connective tissue activating protein III, precursor of NAP-2
NAP-2	neutrophil activating peptide 2
PAF	blood platelet activating factor
SCF	stromal cell factor

Modified from *Gemsa et al. 1997*



96 Cytokine Cascade

Following injection of lipopolysaccharide (filled curved) the blood plasma always reflects the sequence TNF, IL-1 and IL-6: "without TNF, no IL-1; without IL-1, no IL-6" (cascade-like production of these cytokines of the inflammatory process; cf. Fig. 95, pink-highlighted areas)!

Cytokines are not stored, rather they are constantly produced.

Modified from *Abbas et al. 1996*

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Cytokines and Their Effects

97 Inflammatory Cytokines

Periodontitis is characterized by an increased secretion of pro-inflammatory and catabolic cytokines, mainly the major activators IL-1 and TNFα.
These activate the release of additional cytokines such as IL-6, inflammatory mediators such as the prostaglandins (PGE2, p. 49) and of tissue destructive enzymes such as matrix metalloproteinases (MMP, p. 50).
Especially IL-1 and TNF enhance the net loss of bone because they inhibit synthesis while supporting resorption (disturbed homeostasis; p. 61).

Mediators / Cytokines of Non-specific Immunity – The Acute Inflammatory Reaction

Cytokine	Origin	Target cells	Effects on target cells
TNF(α) *Gene polymorphism*	Monocytes / MΦ T-lymphocytes	MΦ PMN Osteoclasts Hypothalamus Liver	Phagocytosis, IL-1 synthesis, general activation, inflammation enhances bone resorption Fever, cachexia, APP (acute phase proteins)
IL-1 **IL-1α** **IL-1β** *Gene Polymorphism*	Monocytes / MΦ others	T-cells, $CD4^+$ B-cells Osteoblasts Osteoclasts Endothelial cells Hypothalamus Liver	Stimulates secretion of IL-2 Enhances proliferation Inhibits bone formation Stimulates bone resorption Activation, inflammation Fever APP (acute phase proteins)
IL-6	Monocytes / MΦ Endothelial cells, T-cells	Thymocytes Mature B-cells Liver	Co-stimulation Proliferation APP, fibrinogen etc.
IL-8 *Chemokines*	Monocytes / MΦ, endothelial cells, fibroblasts, T-cells	PMN Leukocytes	Chemotaxis, activation Chemotaxis, activation

98 Chemokines

The α- and the β- chemokines of the host are differentiated in their sequence of amino acids: between the two cystein residues (-C-C- in the β-chemokines) there is an additional amino acid (X in -C-X-C-) in the α-chemokines.
The receptors of these chemokines upon MΦ and T-cells are "misused" by the HI-virus (p. 148)!

Chemokine = „Chemoattractant Cytokines" — Helix Receptors

Chemokine	Type	Target cells	Effect
α-Chemokine **– IL-8**	– -C-X-C- MΦ, Tissue cells	PMN	Low concentration: Chemotaxis High concentration: Activation
β-Chemokine **– MCP-1** **– MIP-1α** **– RANTES**	– -C-C- T-cells • MΦ chemoattr. protein 1 • MΦ inflamm. protein 1α • regulated on activation, normal T-cell expressed and secreted	 MΦ MΦ MΦ, CD4-Memory cells	Recruiting and activation Recruiting and activation Recruiting and activation

99 Immunoregulatory Cytokines

Control mechanisms including activation, inhibition and selective mechanisms attempt to combat foreign substances and organisms such that the least possible tissue destruction occurs.
Not listed in this table is IL-1ra (anti-inflammatory), the receptor antagonist of IL-1.

Mediators /Cytokines of Immunologically-elicited Inflammation (Chronic)

Cytokine	Origin	Target cells	Effects on target cells
IFNγ *Immune-interferon*	T-cells, NK-cells	Mono / MΦ, NK all cells	Activation Enhanced expression of MHC molecules of classes I & II
Lymphotoxin **TNFβ**	T-cells	PMN, NK endothelial cells	Activation Activation
IL-10	T-cells	Mono / MΦ B-cells	Inhibition Activation
IL-5	T-cells	Eosinophils, B-cells	Activation Proliferation and activation
IL-12	Macrophages	NK-cells, T-cells	Activation Proliferation, activation Differentiation of CD4-/T_H0-cells into T_H1-cells

100 Cytokines of the Second Line of Defense

Listed here are only three of a long list of cytokines that are responsible for the recruitment, differentiation and activation of target cells (colony stimulating factor CSF and others, including those for mediation of wound healing).

Modified from *Abbas et al.* 1996

Mediators / Cytokines of Lymphocyte Activation, Proliferation and Differentiation

Cytokine	Origin	Target cells	Effects on target cells
IL-2	T-cells	T-cells NK-cells B-cells	Proliferation, cytokine production Proliferation, activation Proliferation, Ig formation
IL-4	$CD4^+$-T-cells Mast cells	B-cells Mono / MΦ T-cells	Isotype change to IgE Inhibition of activation Proliferation
TGFβ	T-cells Monocytes / MΦ	T-cells Mono / MΦ other cells	Inhibition of proliferation Inhibition of activation Inhibition of proliferation

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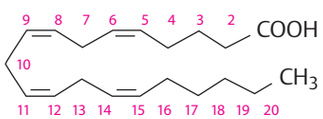
Eicosanoids – Prostaglandins and Leukotrienes

Eicosanoids (C20 molecules) comprise a large group of mediators with a broad spectrum of efficacy. They consist of the 4x unsaturated fatty acid *arachidonic acid* (ARA). ARA is a constituent of the phospholipid plasma membrane of all human cells. ARA is not freely available; rather, it is released from the inner membrane layer by the action of *phospholipase A2* and is subsequently processed further enzymatically (*ARA cascade*), specifically by *lipoxygenases* to leukotrienes (LT), and through *prostaglandin synthases* (= cyclooxygenases 1 and 2; COX-1 and COX-2) to prostaglandins (PG), prostacyclins and thromboxanes.

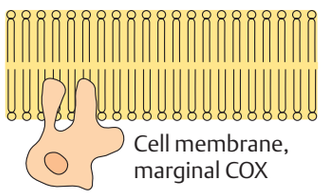
Especially important in periodontology are:

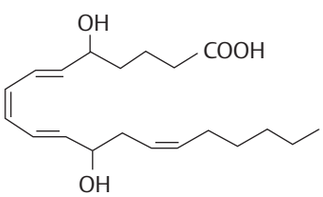
- The chemotactically effective leukotriene (LTB4)
- Prostaglandin PGE2 (synthesized locally by macrophages), which, in elevated concentration, is one of the most potent inflammatory mediators.
  PGE2 is produced via COX-1 (gene on chromosome 9; *MΦ-positive and MΦ-negative = normal phenotype;* see below) as well as through COX-2 (gene on chromosome 1; p. 53).

Arachidonic Acid Cascade

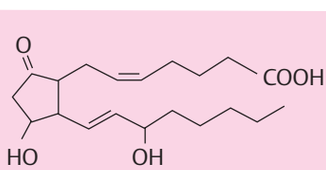


Arachidonic acid – ARA

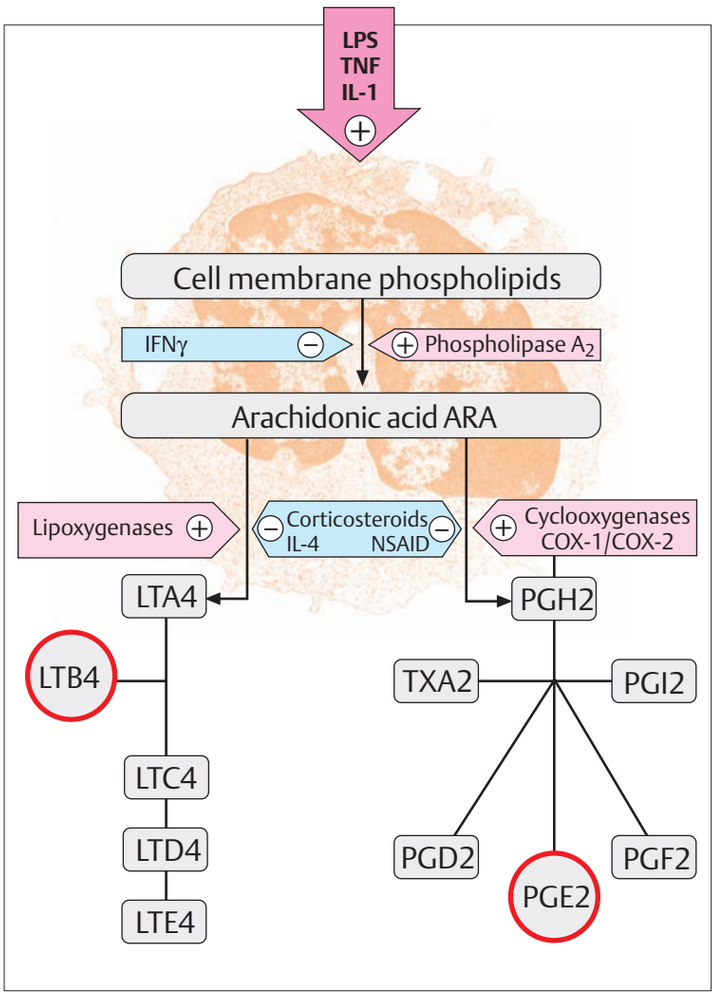




Leukotriene B4 – LTB4



Prostaglandin E2 – PGE2



101 Arachidonic Acid Cascade
Macrophages secrete diverse eicosanoids in response to bacterial metabolites such as LPS and/or via cytokines (TNF, IL-1):

- Prostaglandins e. g., **PGD2, PGE2, PGF2**
- Thromboxane, e. g., **TXA2**
- Prostacyclins, e. g., **PGI2**
- Leukotrienes, e. g., **LTA4-LTE4**

These bioactive molecules are not stored in the cells, but are constantly produced.

Prostaglandins exert *physiologic effects* upon stomach mucosa, blood clotting, smooth musculature (vessels, intestines), uterus (contractions, at high dosage even abortion!).

An "overproduction" (inflammation, fever, etc.) is theoretically preventable at various sites by means of inhibitory substances (blue field) of the individual enzymes (red field).
Available today for use in practice are corticosteroids and non-steroidal inflammatory inhibitors (NSAID, p. 294).

COX-1 versus COX-2

COX-1 is responsible for maintaining PGE2 production at a *constant, physiologic level*, essential for the protection of the gastrointestinal mucosa (mucin) and the function of blood platelets (clotting). Through the actions of pro-inflammatory cytokines (IL-1, TNF) COX-1 is *not* up-regulated, in contrast to COX-2, which is largely responsible for the high levels of PGE2 during inflammation.
A different PGE2 secretion by macrophages in response to bacterial stimuli (*MΦ-positive phenotype* to COX-1; Offenbacher et al. 1993) is responsible for a generally elevated ability to mount the inflammatory response (cf. p. 52).

Medicinal Therapeutic Possibilities

The most important inhibitors of PGE2 synthesis are the non-steroidal anti-inflammatory drugs (NSAIDs) such as acetyl salicylic acid (aspirin). These agents block COX-2 as well as COX-1. The threatening side effects of COX-1 inhibition (stomach ulcer; hemorrhage) most often prohibit a high dosage or long-term regimen.
With the development of so-called super aspirins, which are pure COX-2 inhibitors (p. 294), it appears that in the near future it will be possible to suppress the high level of regulation of PGE2 by means of COX-2 molecules.

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Enzymatic Mechanisms—Matrix Metalloproteinases

Periodontopathic bacteria in the subgingival plaque, which initiate and propagate the inflammatory process, cause periodontal destruction *directly* (via *bacterial proteolytic enzymes*, e.g., "gingipains"), but especially also *indirectly* by means of the complex stimulation of a large group of proteolytic *host enzymes*, which have the capability to destroy the extracellular matrix of connective tissue and bone.

Most important in this regard is the family of zinc-dependent enzymes, the more than 14 matrix metalloproteinases (MMP; Birkedal-Hansen 1993, Deschner 1998).

The major MMP elements include gelatinases, collagenases, stromelysins, matrilysins and others.

Stimulation and Expression of MMPs

Bacterial products (e.g., LPS) can directly stimulate macrophages to produce the precurser molecules (pro-MMP). The macrophages then synthesize and secrete both cytokines and prostaglandins, which then can stimulate fibroblasts and other tissue cells to increase MMP synthesis and secretion. At the same time, other factors become active (growth factors, hormones), which play an indirect role in the synthesis of MMPs and their *inhibitors* (e.g., TIMP, tissue inhibitors of MMP).

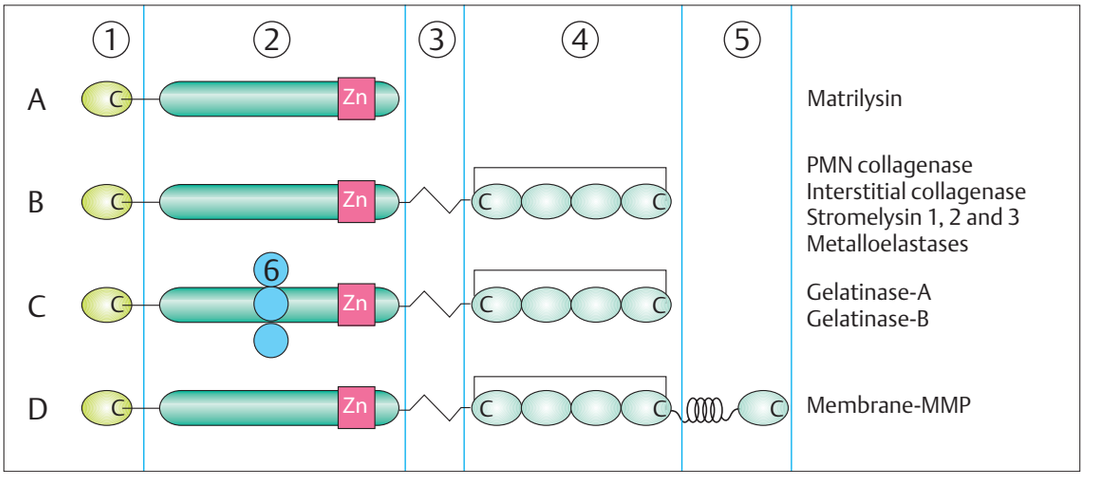
102 Matrix Metalloproteinases—Classes
Activated inflammatory and structural cells synthesize a broad array of zinc-containing proteolytic enzymes (and simultaneously their inhibitors, even TIMP). Matrix metalloproteinases destroy the extracellular matrix of connective tissue; they exhibit overlapping substrate specificity.

Figs. 102 and 103: Modified from *J. Reynolds and M. Meikle 1997*

Enzyme	MMP Number	Substrate
Gelatinases		Collage degradation
– Gelatinase A	**MMP-2**	Native collagen IV, V, VII, X
– Gelatinase B	**MMP-9**	Elastin and fibronectin
Collagenases		
– Fibroblast type	**MMP-1**	Collagen types I, II, III, VII, VIII, X
– PMN Type	**MMP-8**	
Stromelysins		
– Stromelysin-1	**MMP-3**	Proteoglycan – nuclear protein
– Stromelysin-2	**MMP-10**	Fibronectin, laminin
– Stromelysin-3	**MMP-11**	Collagen IV, V, IX, X and elastin
Matrilysin	**MMP-7**	Fibronectin, laminin, collagen IV
Metalloelastase	**MMP-12**	Elastin
Membrane type	**MMP-14**	Pro-gelatinase A

103 Structure of MMP
MMP molecules A-C are free enzymes; the fourth (D) is a membrane MMP of the cell surface. Non-activated MMP exhibit six primary domains:
1 Propeptide (latent enzymes)
2 N-terminal end, *catalytic portion*, with Zn^{2+}!
3 "Hinge" between 2 and 4
4 C-terminal end
5 Transmembrane domain
6 Gelatin-binding site

MMP is activated by plasmin and other substances.



Natural inhibitors of MMP expression, and therefore also of destruction, are regulated both *locally* (TIMP, IL-10, TGFβ) and *systemically* (steroidal inhibitors).

Inhibition and Inactivation of MMP

The healthy extracellular matrix of connective tissue and bone undergoes constant turnover. Regulatory mechanisms are targeted toward an equal balance between synthesis and breakdown, i.e., for tissue homeostasis. In the case of inflammation, particularly in periodontitis, this balance is altered in favor of the catabolic enzymes.

Non-steroidal, synthetic inhibitors are of great interest for periodontal therapy. Most important here are the *chemically modified tetracyclines* (CMT 1–10; Ryan et al. 1996).

One of these agents, an MMP-blocking modified doxycycline (DOX), has recently reached the commercial market as a long-acting LDD (low-dose DOX: *Periostat*™; p. 294).

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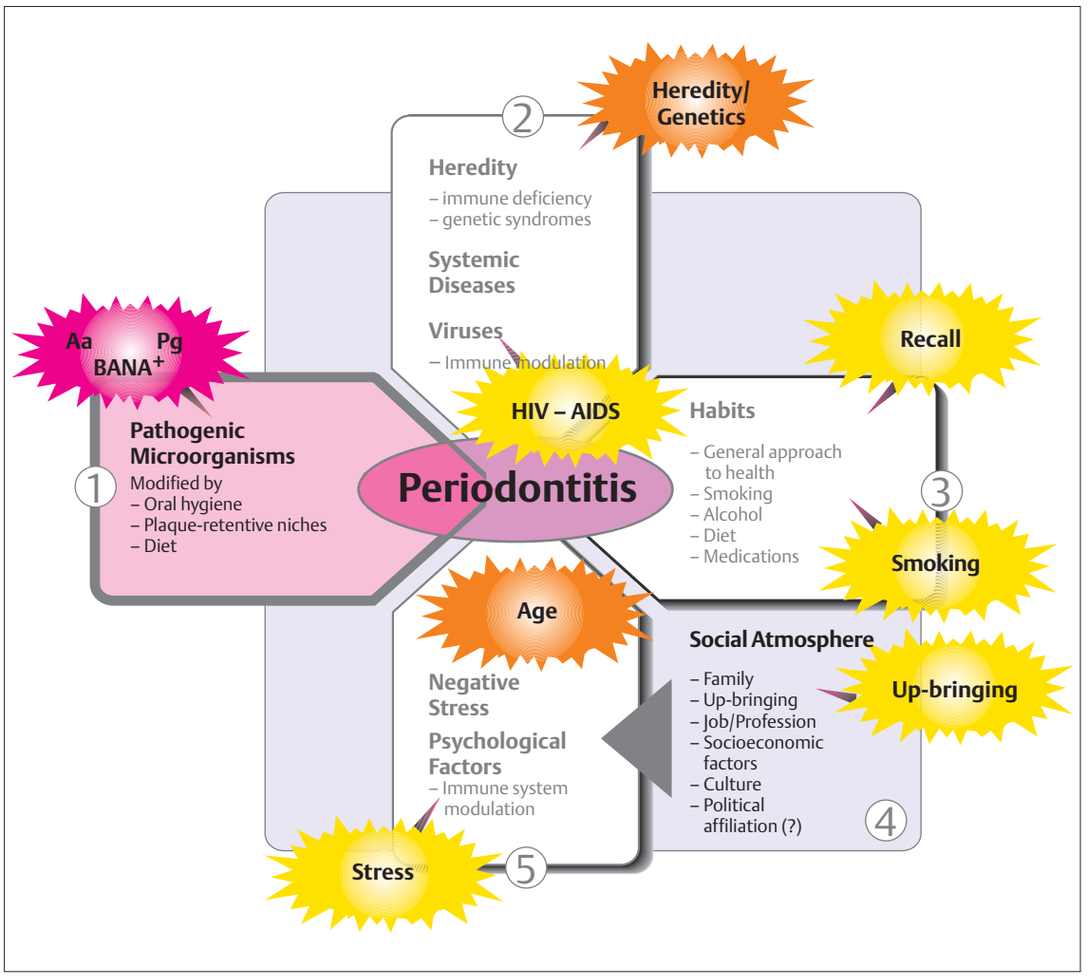
Risk for Periodontitis—the Susceptible Host

Microorganisms as the Agents of Disease

The role of bacterial plaque as the primary etiologic factor for gingivitis and periodontal diseases is unquestioned. For the progression of gingivitis to periodontitis, the "marker bacteria" such as *A. actinomycetemcomitans* (*Aa*) and the "red complex" of the BANA-hydrolyzing bacteria *P. gingivalis*, *T. forsythensis*, and *T. denticola* (Fig. 77) are always present.

The Host and Its Environment

In addition, a long list of other factors, so-called secondary etiologic factors or "risk factors" co-determine the initiation, the progression and the clinical picture. These risk factors negatively influence the tissue as well as the defense reactions (immunity) of the host; they render the host more susceptible to the disease process. These risk factors may be just as important as the bacteria in the pathogenesis of periodontitis.



104 Risk Factors and Their Odds Ratios

Primary "risk factors"
– Specific pathogens from plaque
- *A. actinomycetemcomitans:* ×2
- *BANA + complex*: (Pg-Tf-Td)* ×3.6
- *P. gingivalis:* ×2.7

Secondary "risk factors"
– Non-alterable risk factors
- Genetic defects ?
- IL-1 gene polymorphism: ×2.7
- Ethnic origin: ?
- Gender: ?
- Age: ?

– Alterable risk factors
- Smoking: ×2.8–6.7
- Stress: ×3–5
- Up-bringing: ×3
- Lack of recall: ×3.2
- Diabetes mellitus: ×2–3
- HIV / AIDS: ?

* BANA-positive bacteria hydrolyze N-α-benzoyl-DL-arginine-2-naphthalamide (a synthetic trypsin substrate)

Adapted from *N. Clarke* & *R. Hirsch 1995* (p. 22)

Classification of Risk Factors ("Odds Ratios")

In terms of their importance for prognosis and choice of therapy, the recognition and weighing of risk markers or risk factors for the patient, each tooth and each aspect of each tooth are of critical importance during examination and collection of clinical data.

The term *"odds ratio"* refers to a statistically determined multiplier. It expresses the increased risk vis-à-vis normal susceptibility, but it is more of a relative figure than a definitive value.

Risk factors can be classified in many ways (see also p. 54):

- Microorganisms—host
- Host systemic—local
- Genetic or non-genetic (inherited or acquired)
- Avoidable or non-avoidable, etc.

One impressive and practical classification simply discriminates between:

- Risk factors that can be altered (see also p. 54)
- Risk factors that cannot be altered

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Genetic Risk Factors—Diseases, Defects, Variations

In the case of periodontitis—a "multifactorial" disease—*genetic* and *non-genetic* factors influence each other, and their effects cannot always be sharply distinguished from each other. Both usually increase the pathogenesis as well as the clinical symptoms of the disease. One example is a low IgG2-blood level (caused by genetics and smoking).

Genetic Diseases—Genetic Defects

A genetic defect (e. g., in Papillon-Lefèvre Syndrome with its mutated cathepsin-C receptors) can be powerful enough to elicit disease by itself. This pertains primarily to single-gene diseases or chromosomal anomalies (Hart & Kornmann 1997). In such genetic diseases, periodontitis often occurs in adolescence, and sometimes even during eruption of the primary teeth. Many patients with pre-pubertal, juvenile or aggressive periodontitis exhibit granulocyte defects (PMN; see below).

Genetic Risk Factors

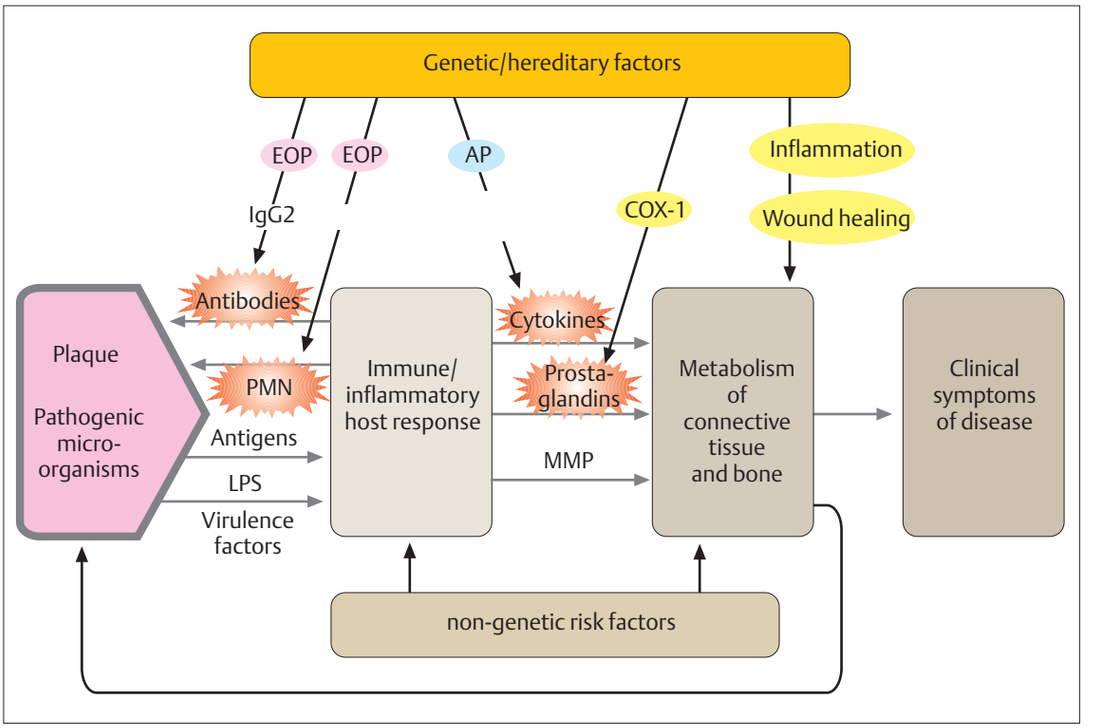
The majority of diseases are *multifactorial*, with a genetic component as the basis (gene variations: polymorphisms, e. g., the IL-1 gene). Such genetic risk factors may be associated with various loci (multi-gene diseases). The genetic insufficiency, in itself, does not in such cases lead to clinical manifestations of disease; only over the course of time, in adulthood, the clinician may note that a patient has become more susceptible, for example, to chronic periodontitis. Defects, polymorphisms with risk for periodontitis include:

- Fc receptors (FcγRII on PMN granulocytes)
- IgG2 level
- IL-1 gene (+) polymorphisms (p. 189)
- COX-1 (+) gene: elevated PGE2 production
- MΦ (+) phenotype: inflammation/wound healing
- Others: polymorphisms of IL-4, IL-10, TNFα, FMLP, Vit.-D3, cathepsin-C receptors, among many others.

105 Genetic Risk Factors—Influence on Pathogenesis
Demonstrated (red) and probable (yellow) genetically elicited defective immune functions. Their gene loci are portrayed in Fig. 106.

- *Antibodies*: reduced IgG2-level
- *PMN defective function*: LAD type 1 (leukocyte adhesion deficiency); adherence deficiency, FcγRII deficiency.
- *Cytokines*: positive IL-1 genotype
- *Prostaglandins COX-1*: with positive COX-1 genotype, MΦ produce excessive PGE2
- *Inflammation, wound healing*: negative effects with a positive (MΦ+) phenotype

Modified from *T. Hart & K. Kornman 1997*



PMN Defects

From family studies, research with twins, as well as DNA analysis, the role of inherited PMN defects in aggressive periodontitis has long been acknowledged (Michalowicz et al. 1991, Michalowicz 1994, Hart et al. 1994). All of these research studies clearly show the important role of the PMN during host defense against infection. Numerous and diverse PMN functions may be combined or altered: Chemotaxis, peroxide production, phagocytosis, bacteriocidal activity/"killing," LTB-4 production (see also van Dyke 1995).

The following systemic diseases resulting from granulocyte defects are also associated with periodontal diseases (for additional examples: see Hart et al. 1994):

- Leukocyte adhesion deficiency (LAD) type 1
- Chediak-Higashi Syndrome
- Down Syndrome
- Papillon-Lefèvre Syndrome
- Diabetes mellitus
- Chronic granulomatosis
- "Lazy Leukocyte Syndrome"
- Crohn's Disease, others

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Genetic Susceptibility to Periodontitis

Leukocyte Adhesion Deficiency, Type 1 (LAD Type 1)

Chemotactically-regulated PMN diapedesis cannot occur because of lacking adhesins on the PMN surface and lacking corresponding ligands on the endothelial cells; despite numerous PMNs in the vessels, one observes only few of these cells in the surrounding tissues. Early-onset, aggressive periodontitis is the result.

Low IgG2 Level

A low IgG2 level, resulting from genetic predisposition or tobacco use is associated with aggressive periodontitis. IgG2 binds to polysaccharide-like antigens, and is therefore important for defense against gram-negative bacteria.

PMN Receptors for IgG—FcγRII

Affinity and avidity for bacteria (the initiation of phagocytosis) are high when these bacteria are *opsonized*, for example, via immunoglobulins. The Fc-domain of IgG (Fig. 89) binds to the FcγRII receptors of the PMN. Defective or lacking receptors leads to aggressive periodontitis.

Gene Polymorphisms—IL-1-positive Genotype

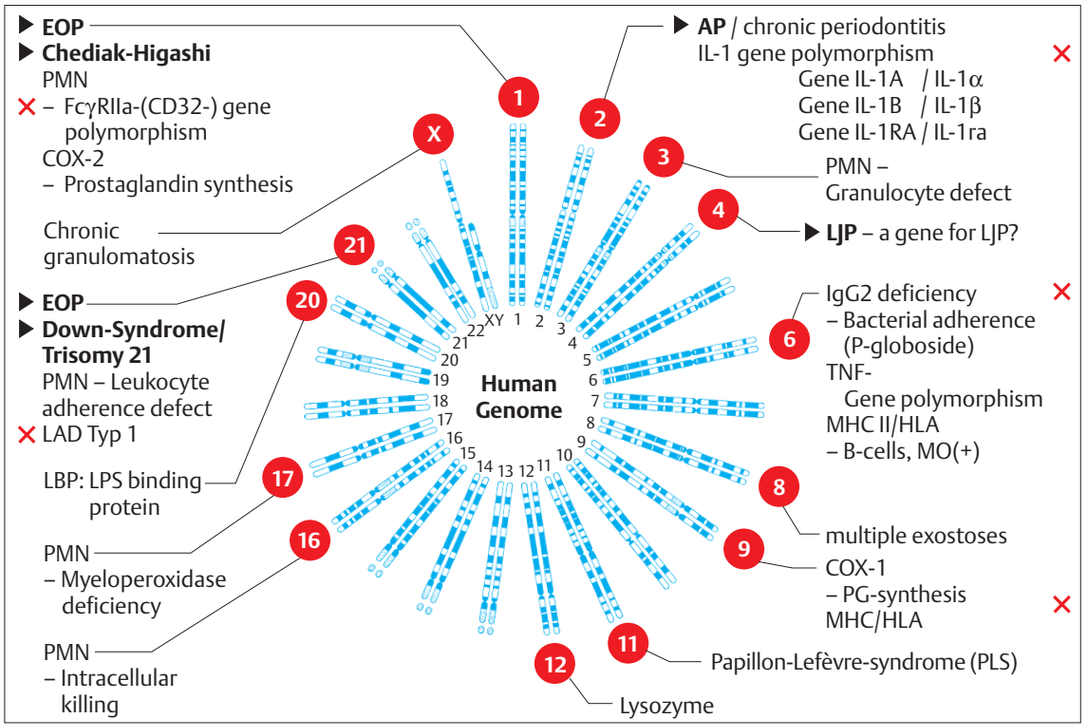
The positive genotype (p. 189) sets the stage for stimulation of macrophages, with four-times the IL-1 content; this is associated with chronic adult periodontitis.

Gene for Cyclooxygenase 1 (COX-1)

The macrophage gene COX-1, which is responsible for the physiologically *constant* prostaglandin production, produces excessive amounts of PGE2, one of the most powerful inflammatory mediators (p. 49), when appropriately stimulated (TNFα, IL-1).

MΦ (+) Phenotype

Not all MΦ react equally to the same level of stimulation (e. g., LPS). The positive phenotype synthesizes and secretes the cytokine-inductors TNF and IL-1 in elevated levels, which have major effects on *inflammation* and *wound healing*.



106 Human Chromosomes with Genes That May be Associated With the Periodontium or With Periodontitis (as of 1998)
To date it is primarily single-gene defects and gene polymorphisms by which the gene loci and the corresponding functional disturbances are understood (gene defects, gene variants; gene alleles).

Note: nomenclature change, 1999

AP → chronic periodontitis, type II/CP

EOP } → Aggressive periodontitis,
LJP } type III/AP

The Human Genome

The ambitious goal of the Human Genome Project was to decipher the entire DNA sequence (ca. 30 billion base pairs) of the human genome by the year 2003. Because of the intensive competition from private researchers, the goal was accomplished in the year 2000, although several gaps remain. It will require much more time until the function of the individual genes (ca. 30,000—40,000?), and their coded proteins and their functions and structures of the domain (estimated at 1,000—5,000 3D structures) will be clarified.

For this project (Structural Genomics Project), several scientific consortia in the USA and Germany have combined to found, for example, the "Protein Structure Factory" in Berlin. With precise knowledge of the structure and function of important human proteins, it will eventually be simpler to understand the complicated interplay of normal or defective genes, and to utilize such knowledge for diagnosis and therapy (e. g., for targeted medicines).

The likelihood is high that such new and practical knowledge can also be applied to the periodontal diseases.

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Alterable Risk Factors, Modifying Co-Factors

Periodontopathic microorganisms are non-alterable risk factors that initiate periodontal diseases (pp. 51–53); however, there is also a large number of alterable risk factors—earlier called co-factors—which influence the course of periodontitis to greater or lesser degree depending on their importance or intensity. It is possible to a certain degree within this group to differentiate between general (systemic) and local risk factors:

Systemic:
- Systemic diseases (diabetes, HIV infection, etc.;p. 119)
- Smoking
- Stress
- Medicines
- Education, social circumstances
- Lifestyle
- Environment
- Nutrition

Local:
- Saliva quality and quantity
- Mouth breathing
- Exogenous, mechanical, chemical, thermal, corrosive and actinic irritations
- Allergic reactions
- Function: occlusal trauma
- Orofacial clenching/bruxing phenomena
- Work-related parafunctions.

Serious systemic diseases such as untreated diabetes, blood disorders, hormonal imbalance etc., can be partially responsible for initiation of and accelerating progression of gingivitis or periodontitis. This will be covered in the chapters "Types of Disease" (p. 77) and "Oral Pathologic Alterations" (p. 119).

Smoking is acknowledged today as an important risk factor. Tar products cause local irritation of the gingiva, nicotine is a sympathomimetic which leads to reduced metabolism in the periodontal tissues, and combustion products influence the chemotactic reactions of PMNs. An important risk is the lowering of the IgG2 level (p. 53).

Stress may be elicited by various causes such as work overload, social circumstances, environmental effects etc., and can lead to a negative influence upon the immune system or to increases in the pro-inflammatory mediators; the latter are associated with elevated susceptibility to periodontitis.

Side effects of medicaments can play a role in the initiation or progression of gingivitis or periodontitis, and are described in the chapter "Oral Pathologic Alterations" (p. 119).

Poor up-bringing and a *compromised socioeconomic status* may lead to insufficient appreciation for systemic health as well as oral hygiene, and this can have a negative effect on the periodontal structures.

Negative environmental factors can reduce the efficacy of the immune system and therefore also host defense against infections.

Nutrition can influence the speed of plaque formation as well as its composition. Extreme diets and inadequate nutrition can weaken the immune system and therefore the host's ability to respond to marginal infections.

The *saliva* has protective functions. Salivary mucins (glycoproteins) cover all mucosal surfaces as a protective film. Depending upon its flow rate and viscosity, saliva has a more or less powerful cleansing effect. Salivary content of bicarbonate, phosphate, calcium and fluoride determines its buffering capacity and its remineralizing potential. The antimicrobial activity of saliva is determined by its content of secretory immunoglobulins (sIgA) as well as lysozyme, catalase, lactoperoxidase and other enzymes.

Mouth *breathing* leads to drying out of the mucosa, and the protective effect of saliva is lost.

Exogenous irritants can injure the mucosa, gingiva and periodontium to varying degrees:
- *Mechanical* injuries, e. g. improper use of the toothbrush and other hygiene aids, can lead to injury and acute inflammation.
- *Chemical* irritants such as highly concentrated topical medicaments and acids can lead to lesions of the gingiva and mucosa. The same is true of *thermal* irritants (burns). These lesions are usually reversible, but in serious cases necrosis may result.
- Non-noble materials (e. g., root canal pins) may *corrode* following root fracture and cause damage to the periodontal ligament through corrosion products (Wirz et al. 1997 b)
- *Actinic* irritation: Mucositis and xerostomia may occur following radiation therapy for tumors in the head and neck region.

Allergic reactions may appear as mild erythema all the way to painful blister formation.

The chapter "Function—Functional Therapy" (p. 459) details *occlusal trauma, orofacial clenching phenomena and work-related parafunctions.*

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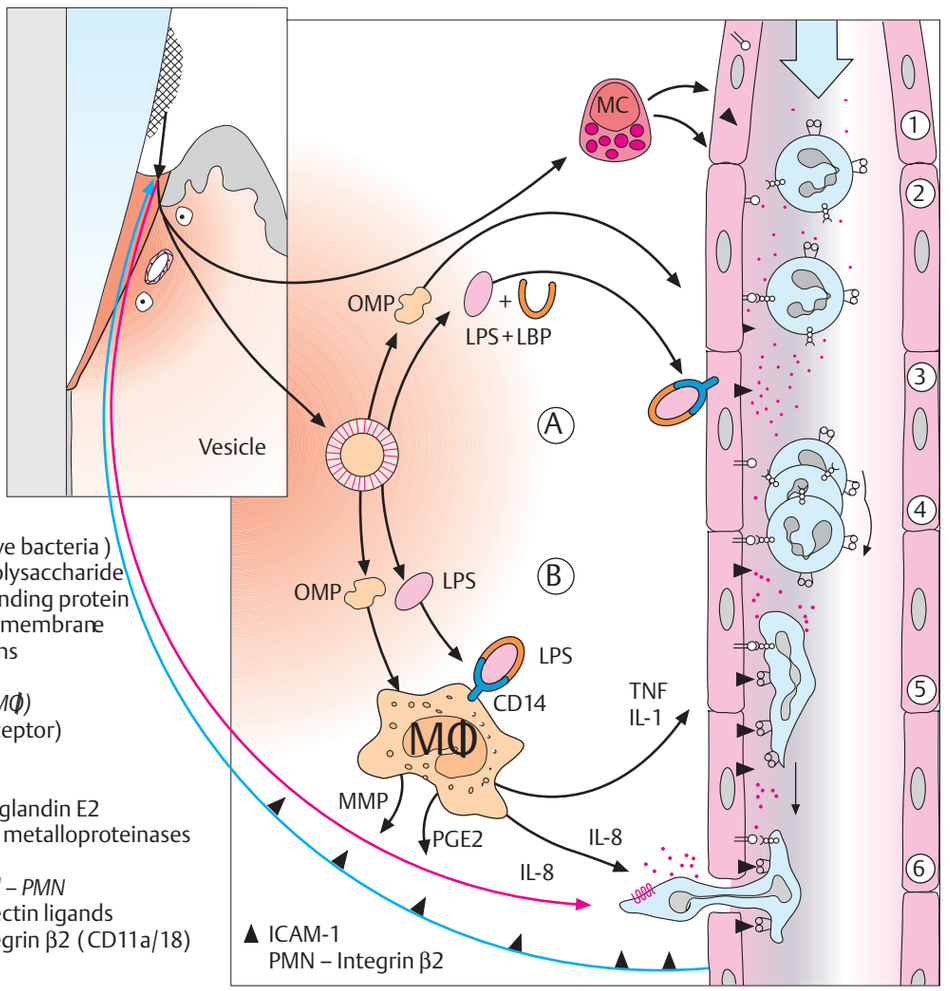
Pathogenesis I—Initial Inflammatory Reactions

Reactions in Healthy Tissues

Bacterial plaque metabolites "attract" PMNs. Proteins from bacterial vesicles and LPS, which react with LBP, as well as chemotactically active substances such as formyl peptide (FMPL, p. 58) stimulate tissues and vessels *directly* (A), supported by the mast cells (MC) near the vessels, or *indirectly* (B) by way of the macrophages (MΦ). These cells also produce pro-inflammatory cytokines (IL-1, TNF), MMP, PGE2, and IL-8, a chemokine that is also produced by the junctional epithelial cells near the sulcus. A chemotactically effective concentration gradient is created upon which the defense cells (PMNs) become oriented as they exit the vessels and proceed toward the biofilm ("blue arch").

Vascular Reactions

Post-capillary venules expand in reaction to signal substances (e.g., histamine from mast cells, prostacyclins, NO etc.), and the flow of blood slows. Endothelial cells and host defense cells in the blood stream (primarily PMNs) express adhesins which enhance the adherence of the PMNs to the vessel wall, and subsequently make possible their diapedesis into the irritated tissue.



Vesicle
(Gram-negative bacteria)
LPS Lipopolysaccharide
LBP LPS-binding protein
OMP Outer membrane proteins

Macrophage (MΦ)
CD14 (LPS Receptor)
TNF und IL-1
IL-8
PGE2 Prostaglandin E2
MMP Matrix metalloproteinases

Endothelial cell – PMN
Selectin – Selectin ligands
ICAM-1 – Integrin β2 (CD11a/18)

107 Leukocyte Recruiting, Vessel-PMN Interactions

1 *Recruiting*: In the slowed blood stream of the widened venules, PMNs approach the vessel walls.

2 *Contact—selectins*: Molecules of the selectin family upon endothelial cells and PMNs "brake" this movement.

3 *"Rolling":* The PMNs roll toward the vessel wall guided by the selectins (ELAM-1).

4 *Activation of integrins*: Chemokines from endothelium and tissue activate integrin β2 on PMNs.

5 *Fixation/Adherence*: ICAM-1 on the endothelial cells and integrins (PMN) retain the PMN. The PMN then wanders, by means of chemotactic guidance, toward the expanded intercellular spaces.

6 *Transmigration/Diapedesis*: The PMN leave the venuole. It wanders apically toward the sulcus bottom, i. e., toward the plaque, guided by its chemoreceptors (7-helix receptor, cf. HIV, p. 148)

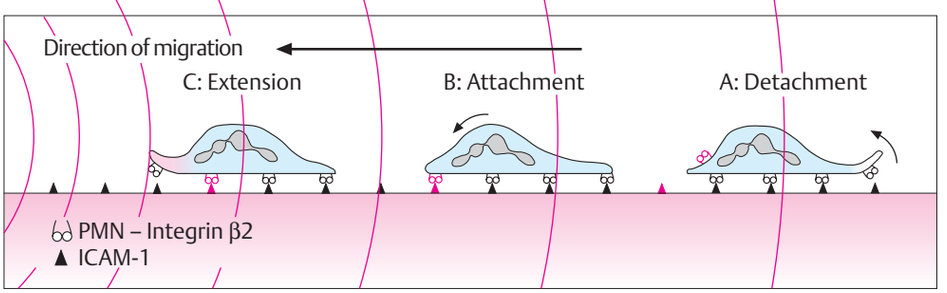
Chemotaxis

- Classical chemotactic substances
- α-Chemokine
- β-Chemokine

Bacteria
Host
FMLP (Formyl peptide, p. 58)
C5a (Complement)
LTB4 (Leukotriene, p. 49)
PAF (Platelet activating factor)

Interleukin 8 (IL-8)

MCP-1
MIP-1α und 1β
RANTES



108 Migration of a PMN (from right to left): Detachment—Attachment—Extention

The PMN migrates upon cells and matrix substances along the gradient of the diverse chemokines in contact with their integrin molecules with ICAMs.
ICAMs are adhesins that belong to the superfamily of immunoglobulin genes (p. 46); they can be expressed on all tissue components.

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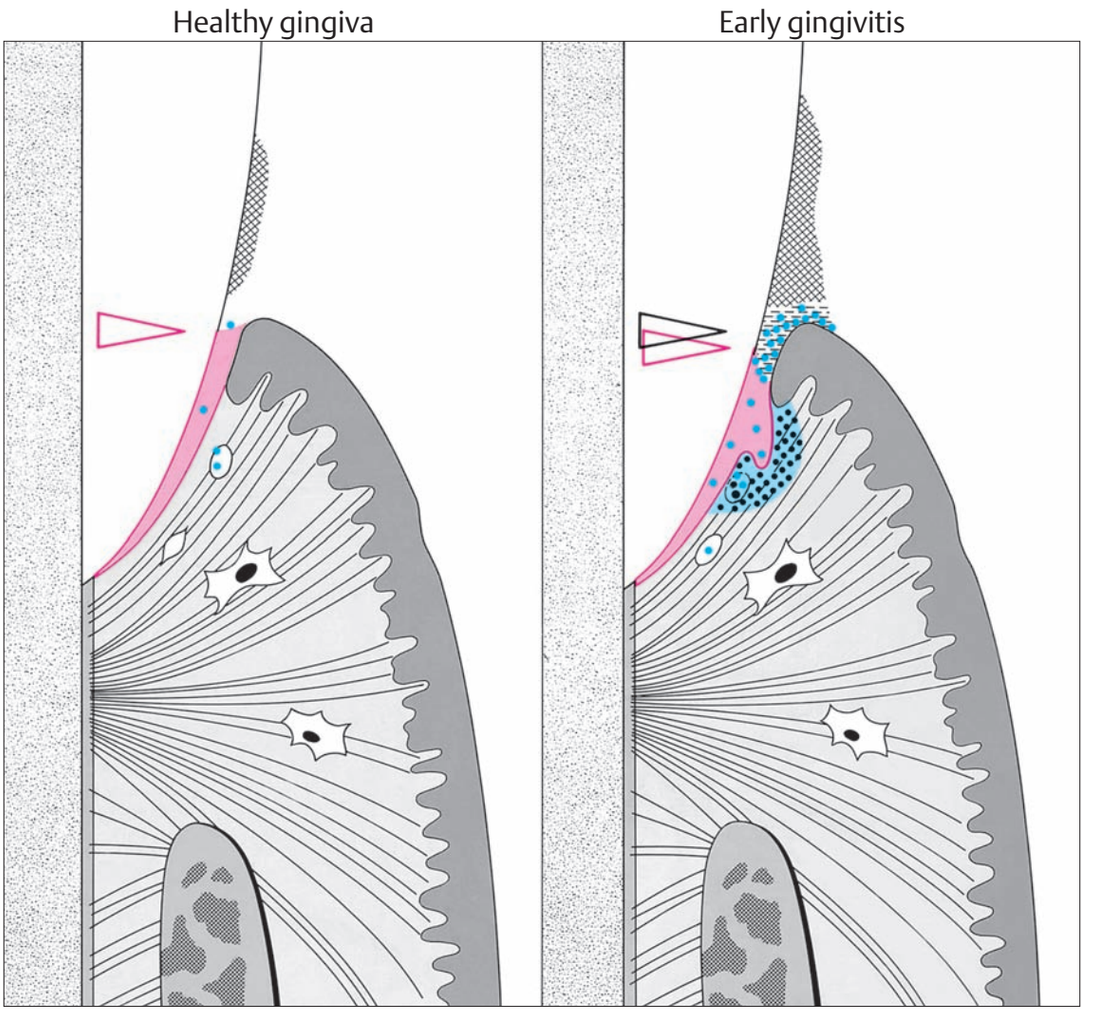
Pathogenesis II—Histology

As early as 1976, Page and Schroeder, on the basis of a literature review and their own experiments, described the histologic development of gingivitis and periodontitis. Their now-classic publication differentiated an initial, an early and an established gingivitis, and demarcated these from periodontitis. With today's knowledge "initial gingivitis" is no longer considered to be an early stage of disease, rather as the physiologic response of tissues and the immune system to dental plaque, even when plaque is present only in minute quantities (Schroeder 2000)

Early Gingivitis

Even in clinically healthy gingiva, some polymorphonuclear granulocytes (PMNs) transmigrate the junctional epithelium (p. 55). If this PMN migration becomes accompanied by a gingival sulcus infiltrate containing subepithelial T-cells, the condition is referred to as *early gingivitis*. Only in children can this stage of the disease process be maintained over longer periods of time.

In most adults, this "early lesion" develops quickly into an *established gingivitis*, which can vary considerably.



109 Healthy Gingiva
Very little plaque accumulation (hatched area), normal junctional epithelium (red), minimal sulcus depth (red arrow).
A few polymorphonuclear leukocytes (PMNs, blue dots) transmigrate the junctional epithelium in the direction of the sulcus bottom. Dense collagenous fiber apparatus, intact fibroblasts. This "condition" was earlier described as "initial gingivitis."

110 Early Gingivitis
Heavier plaque accumulation. In this early lesion, PMNs (blue dots) transmigrate the junctional epithelium and create a wall against plaque bacteria in the slightly deepened sulcus (red arrow apical to the plaque border).
In the subepithelial area, an early infiltration of lymphocytes is observed (black dots).

Modified from *R. Page & H. Schroeder 1982*

Plaque	Few, mainly Gram-positive aerobes	Mainly Gram-positive aerobes
Junctional epithelium / Pocket epithelium	Normal junction epithelium, exhibiting no rete	Initial alteration and lateral proliferation of the junctional epithelium coronally
Vasculature Inflammatory cells, infiltrate Exudate	Few PMNs from the subepithelial vascular plexus transmigrate the junctional epithelium Very little exudate ("sulcus fluid") No subepithelial round-cell infiltrate	Vasculitis, out-flow of serum proteins, PMN migration, accumulation of lymphoid cells, T-cell dominance, few plasma cells; appearance of immunoglobulins and complement
Connective tissue Fibroblasts, collagen	Normal	Cytopathic alterations of fibroblasts; collagen loss in the infiltrated connective tissues
Alveolar bone	Normal	Normal
Disease course	–	Early lesion: 8–14 days after uninhibited plaque accumulation

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Established Gingivitis

This can persist over many years without developing into periodontitis. It appears not to be caused by any specific microorganisms, but is influenced by their quantity and by the metabolic products from the biofilm.

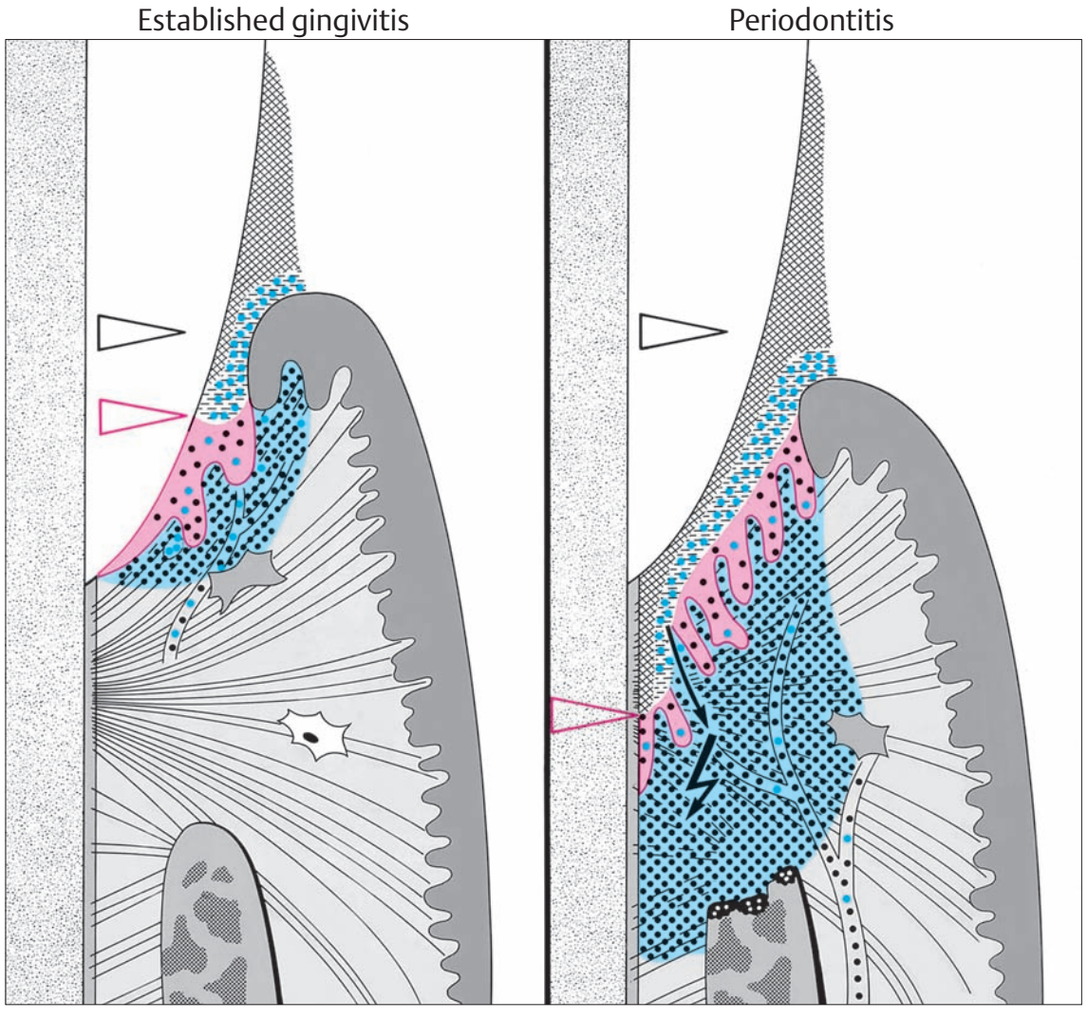
Periodontitis

The transition from gingivitis into *periodontitis* (*progressive lesion*) is caused on the one hand by changes in the pathogenic potential of the plaque, and on the other hand by an inappropriate or inadequate host response to the infection, as well as the existence of risk factors (p. 55).

It is possible to identify periods of stagnation and exacerbation, which progress slowly (chronic) or rapidly (aggressive) depending on the type of disease (p. 112).

The histopathologic characteristics of the gingival and periodontal lesions cannot explain the myriad of individually varying and therefore difficult to classify forms of the disease (breakdown, progression etc.).

Only the most recent knowledge from molecular biology makes it possible to understand the immunologic processes within the periodontal tissues. These processes will be described in the following two pages.



111 Established Gingivitis
Increased plaque accumulation leads to massive influences on the constituents of the gingiva. All of the characteristics of gingivitis remain, but these can now also be identified histologically, and may vary in severity. The junctional epithelium—"epithelial attachment"—becomes apically displaced by the advancing plaque front (gingival pocket; arrow), but there is no loss of connective tissue attachment. The differentiated inflammatory infiltrate protects the deeper structures.

112 Periodontitis
The most important histologic differences between gingivitis and periodontitis include progressive connective tissue attachment loss and bone resorption, as well as apical proliferation and partial ulceration of the junctional epithelium (pocket epithelium; the base of the pocket is indicated by the red arrow).
In acute phases there is bacterial invasion of the tissue with resultant micro- or macro-abscesses.

Gram-positive and Gram-negative	Sub-epithelial, mainly anaerobic and Gram-negative	**Plaque**
Lateral proliferation of JE; apical migration; pseudopocket formation	Apical proliferation of the pocket epithelium, ulceration of the pocket epithelium, true pocket formation	**Junctional epithelium / Pocket epithelium**
Acute inflammatory alterations; plasma cells; immunoglobulins in connective tissue and sulcus; elevated GCF flow; leukocyte "wall" at plaque front.	Acute inflammatory manifestations as with gingivitis; massive infiltration; plasma cell dominance; copious and partially suppurative exudation; expansion of the inflammation and immunopathology.	**Vasculature Inflammatory cells, infiltrate Exudate**
Severe fibroblast damage, further collagen loss, stabilization of the exudates.	Further collagen loss in the infiltrated tissues, simultaneous fibrosis in peripheral gingival areas	**Connective tissue Fibroblasts, collagen**
Normal	Resorption of alveolar bone (attachment loss)	**Alveolar bone**
Manifest 3-4 weeks after plaque accumulation, but can persist for many years without further progress	Periods of stagnation and exacerbation, slowly or rapidly depending upon the type of disease	**Disease course**

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Pathogenesis III—Molecular Biology

More than 20 years after the histologic description of the structural biology of periodontal diseases (Page & Schroeder 1976, pp. 56–57); Kornman, Page and Tonetti (1997) attempted to describe the pathogenesis of gingivitis and periodontitis with consideration for the actual molecular biology and newest genetic knowledge.

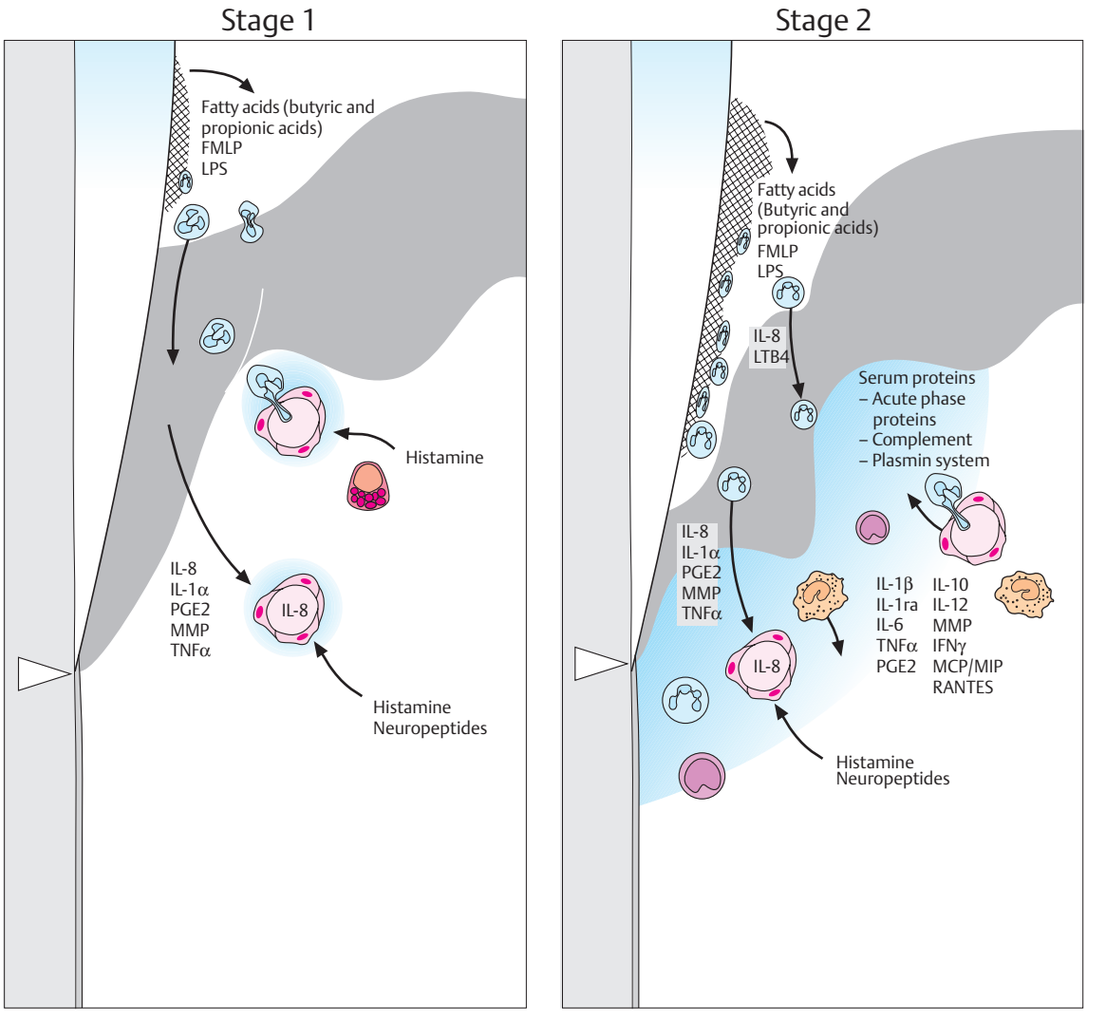
They divided their "expanded" pathogenesis into four stages or "momentary pictures," which were related in every case to a significant change in the activity of the immune system:

1 Reactions of the healthy periodontium to plaque
2 Initial acute, local inflammatory reactions
3 High regulation of the inflammation and infiltrate
4 Chronic immune reactions; attachment loss

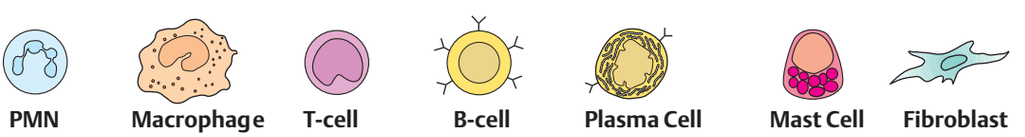
The primary activities of cells and molecules that participate in the local changes in the marginal periodontium are described briefly in the figures below.

113 Stage 1:
Initial Reactions to the Plaque
Plaque bacteria produce metabolites, e. g., fatty acids (butyric acid, propionic acid), and the peptides FMLP and LPS, which stimulate the cells of the junctional epithelium to synthesize inflammatory mediators (IL-8, TNFα, IL-1α, PGE2, MMP). Free nerve endings produce neuropeptides and histamine, which highly regulate the *local vascular reaction*. Perivascular mast cells release histamine, which causes the endothelium to release IL-8 in the vessels. IL-8 attracts PMNs.

114 Stage 2:
Activation of Macrophages and the Serum Protein System
This vascular reaction (p. 55) causes serum proteins (e. g., complement) to spill into the connective tissues and activate the *local inflammatory reaction*.
Later, leukocytes and monocytes are recruited. Activated macrophages produce inflammatory mediators, e. g., IL-1β, IL-1ra, IL-6, -10 and –12, TNFα, PGE2, MMP, IFNγ as well as chemotaxins such as MCP, MIP and RANTES.



Host Defense Cells



Signal and Effector Molecules
Host:
- Cytokines
  * proinflammatory
- Eicosanoids
- Proteases etc.
  ** chemotactically effective

Bacterial:
- Antigens, toxins and chemotaxins**

Abbreviations for molecules A-L
for all stages

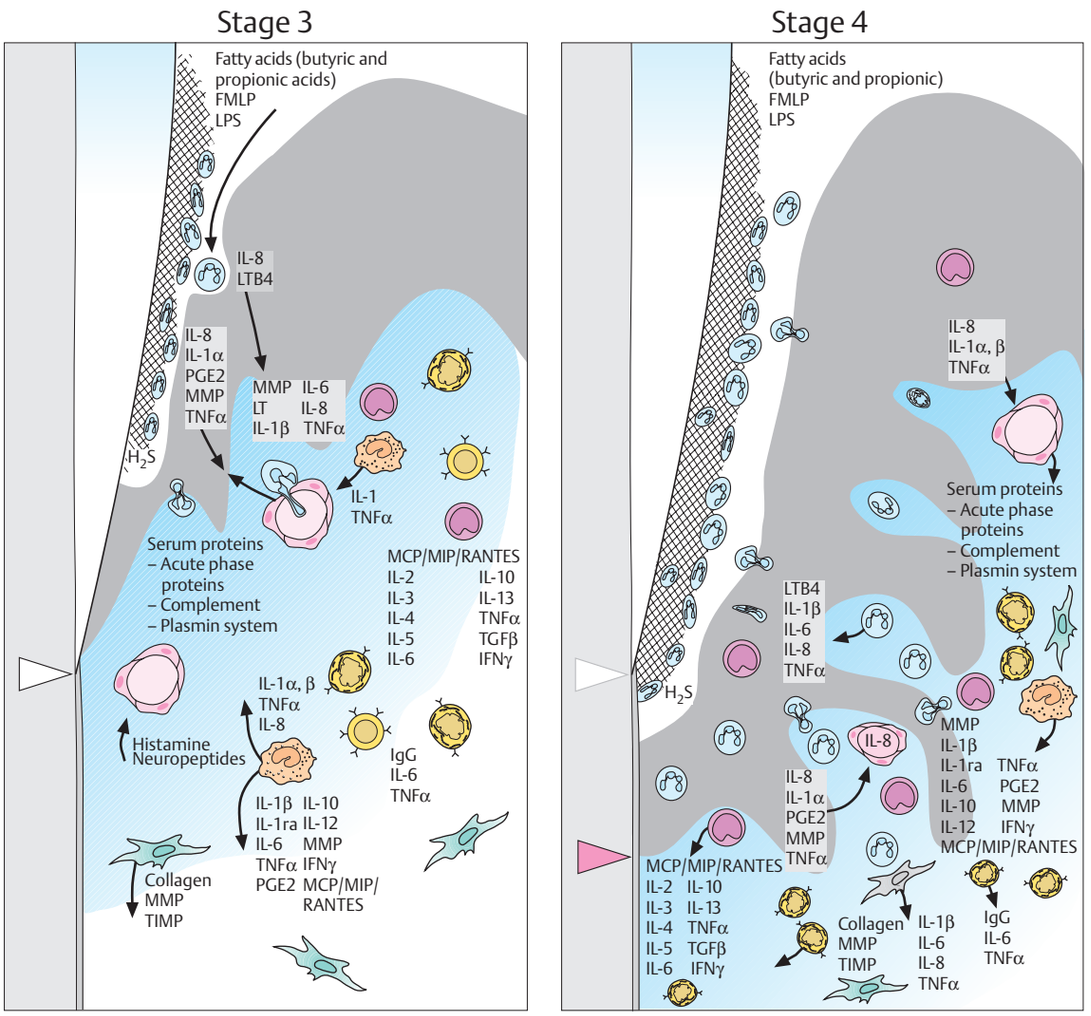
FMLP**	N-Formyl-Methionyl-Leucyl-Phenylalanine	**IL-1ra**	Interleukin-1-receptor-antagonist	**IL-10**	Interleukin 10
		IL-2	Interleukin 2	**IL-12***	Interleukin 12
IgG	Immunglobulin G	**IL-3**	Interleukin 3	**IL-13**	Interleukin 13
		IL-4	Interleukin 4	**IFNγ***	Interferon γ
IL-1α*	Interleukin 1α	**IL-5**	Interleukin 5		
IL-1β*	Interleukin 1β	**IL-6***	Interleukin 6	**LPS**	Lipopolysaccharide
		IL-8*	Interleukin 8		

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In a healthy individual, the term *local* defense means that plaque-irritated tissues recruit and activate only those cells and substances that are necessary for an effective defense. A network of mediators (cytokines, prostanoids, enzymes) deriving from immigrated immune cells and resident host cells serve to coordinate the actual situation and attempt also to maintain tissue homeostasis as long as possible, without tissue loss.

If the pathogenic microorganisms continue to exert "pressure" over a longer period of time (chronic inflammation), and if the immune response is not sufficiently competent ("susceptible host"), the tissue balance is tipped into a stage of increased breakdown that is enhanced by pro-inflammatory mediators and destructive enzymes.

These molecular and cellular reactions to bacterial metabolites and virulence factors vary considerably from person to person, but nevertheless permit an understanding of the numerous possible clinically relevant forms of gingivitis and periodontitis.



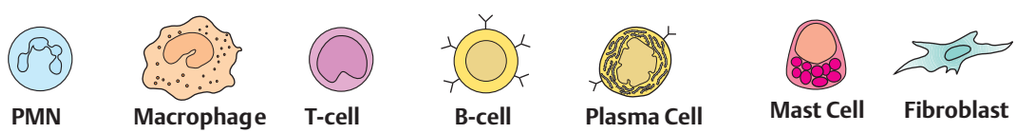
115 Stage 3: Up-regulation of the Activity of Inflammatory Cells—Detachment of the Junctional Epithelium Leads to a Gingival Pocket

The *inflammatory infiltrate* is dominated by lymphocytes. Activated T-cells coordinate the response via cytokines (IL-2 through –6, IL-10 and –13, TNFα, TGFβ, IFNγ). Plasma cells produce Igs and cytokines. Activated PMNs synthesize diverse cytokines, leukotrienes and MMP. Activated fibroblasts produce MMP and TIMP instead of collagen. The infiltrate (blue) expands.

116 Stage 4: Initial Attachment Loss

In the infiltrated connective tissue, there is elevated activity of macrophages, with highly regulated mediators and host reactions. Immunocompetent cells produce numerous cytokines (IL-1β, IL-6, IL-8, TNFα) as well as PGE2, MMP and TIMP. Plasma cells dominate the infiltrate. The disturbed tissue homeostasis leads to the destruction of collagen, connective tissue matrix and bone. *Periodontitis* is the consequence.

Modified from *K. Kornman et al. 1997* [pinxit *T. Cockerham*]



Host Defense Cells

Abbreviations for molecules L – Z
for all stages

LTB4**	Leukotriene B4	**MMP**	Matrix metalloproteinases	**TIMP**	Tissue inhibitors of MMP
MCP**	Monocyte chemoattractive protein	**PGE2**	Prostaglandin E2	**TGF**β	Transforming growth factor β
MIP	Macrophage inflammatory protein	**RANTES****	Regulated on activation, normal T-cell expressed and secreted	**TNF**α*	Tumor necrosis factor α

Signal and Effector Molecules

Host:
- Cytokines
  * proinflammatory
- Eicosanoids
- Proteases etc.
  ** chemotactically effective

Bacterial:
- Antigens, toxins and chemotaxins**

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Attachment Loss I—Destruction of Connective Tissue

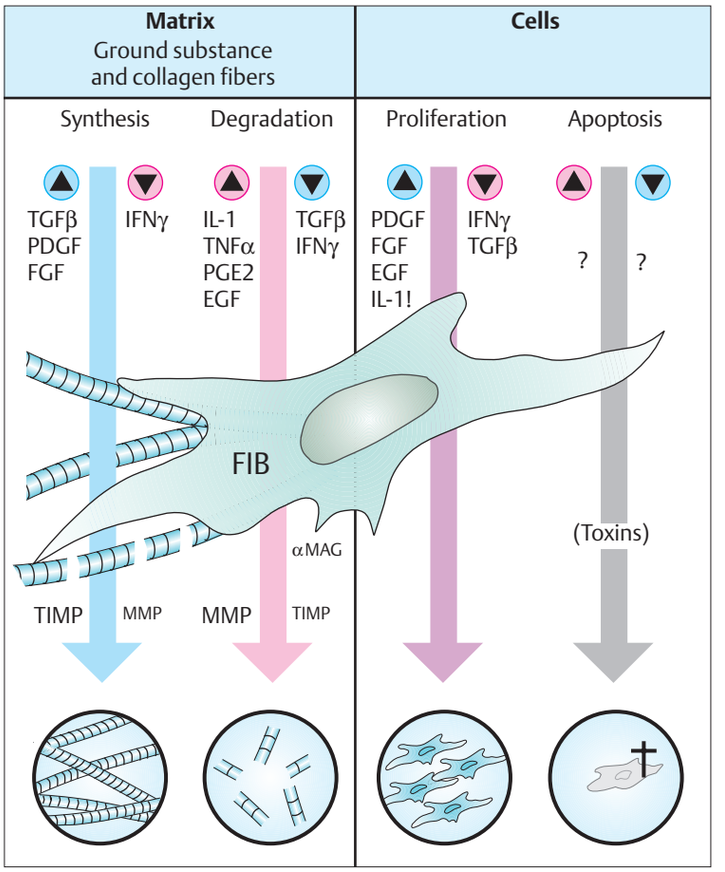
Attachment loss (AL) is one of the primary symptoms in active phases of periodontitis: Extracellular matrix and collagen, e.g., periodontal ligament fibers, are destroyed. The critical milestone in this process is the major change in the activity of resident fibroblasts: Tissue homeostasis is lost, the balance between synthesis and resorption is forced toward more active destruction. This stimulation of tissue resorption may have several different causes:

- In *chronic periodontitis*, it is above all the macrophages (MΦ), activated by bacterial metabolites (e.g., LPS) that stimulate the resident fibroblasts toward secretion of destructive (secondary) mediators such as PGE2 and enzymes (e.g., MMP). On-going efforts in research today are targeted toward reducing such macrophage hyperactivity through use of various medicaments.
- In *acute inflammation and abscesses*, PMNs within the connective tissues are activated by the extremely high chemokine concentrations. During the "respiratory burst" and afterwards, a massive amount of lytic enzymes is set free (acid hydrolases, elastase, neutral proteases etc.), and these have the capacity to destroy host tissue.

117 Connective Tissue—Homeostasis of Apposition and Resorption

Influence of messenger substances (cytokines, primarily growth factors) and secondary mediators such as PGE2, MMP and TIMP on fibroblasts (FIB). Synthesis and resorption of extracellular matrix (collagen fibers and ground substance) remain in balance.
Also the number of cells is regulated by proliferative and apoptotic signals. Apoptosis (genetically determined cell depth) is initiated by many mechanisms, for the most part yet to be understood.

▲ Enhanced
▼ Reduced



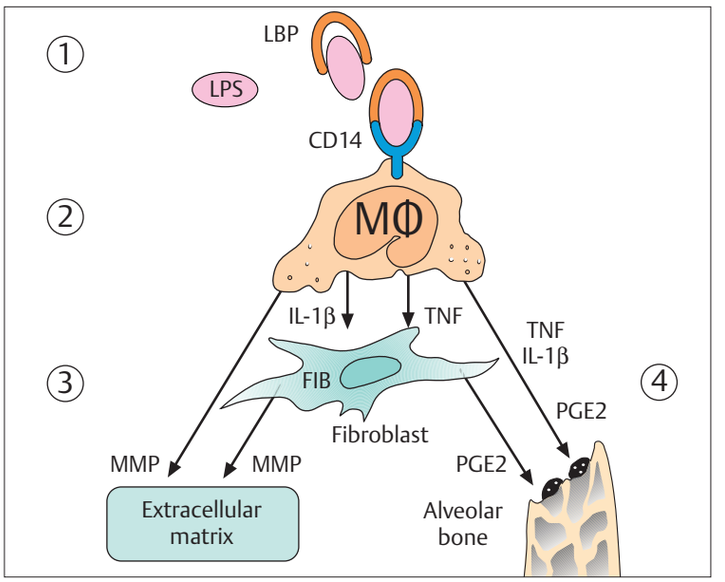
Alphabetical Listing of the Mediators

EGF Epidermal Growth Factor
FGF Fibroblast Growth Factor
IFNγ Interferon γ
IL-1 Interleukin 1
αMAG α-Macroglobulin
MMP Matrix Metalloproteinases
PDGF Platelet-Derived Growth Factor
PGE2 Prostaglandin E2
TGFβ Transforming Growth Factor β
TIMP Tissue Inhibitor of MMP
TNFα Tumor Necrosis Factor α

118 Inflammation-provoked Destruction of Connective Tissue Matrix and Bone

1 Lipopolysaccharide (LPS) activated macrophages (MΦ)
2 Up-regulated inflammatory mediators (IL-1β, TNF, PGE2) and enzymes (MMP) activate fibroblasts, and degrade connective tissue and bone matrix *directly*.
3 Fibroblasts also destroy collagen by means of MMP.
4 Bone resorption results directly (via MΦ) or indirectly through stimulation of osteoclasts.

Modified from *R. Page et al. 1997*



CD14 LPS receptor
IL-1β Interleukin 1β
LBP LPS binding protein
LPS Lipopolysaccharide
MΦ Macrophage
MMP Matrix Metalloproteinases
PGE2 Prostaglandin E2
TNFα Tumor Necrosis Factor α

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Attachment Loss II—Bone Resorption

The numerous mechanisms that serve homeostasis but also effect increased local synthesis and resorption of the alveolar bone are well-understood: The instigating agents of tissue loss in periodontitis are bacterial substances such as lipopolysaccharides, lipoteichoic acids (LTA) etc. These lead to an increased release of cytokines and mediators such as IL-1, TNFα, IFNγ, growth factors (e.g., bone morphogenetic protein, BMP) and local factors such as PGE2, MMP and others (abbreviations in the figure legend, left).

These factors stimulate the activity of osteoclasts directly, or may work indirectly, first on pre-osteoclasts, and thus increase the "pool" of bone resorbing cells. The mentioned bacterial substances and the host-mediators also demonstrate a direct repression or modulation of the bone-forming osteoblasts (Schwartz et al. 1997).

During acute phases of periodontitis, it appears that a direct initiation of bone resorption by bacterial products such as LPS, LTA and peptidoglycans is possible.

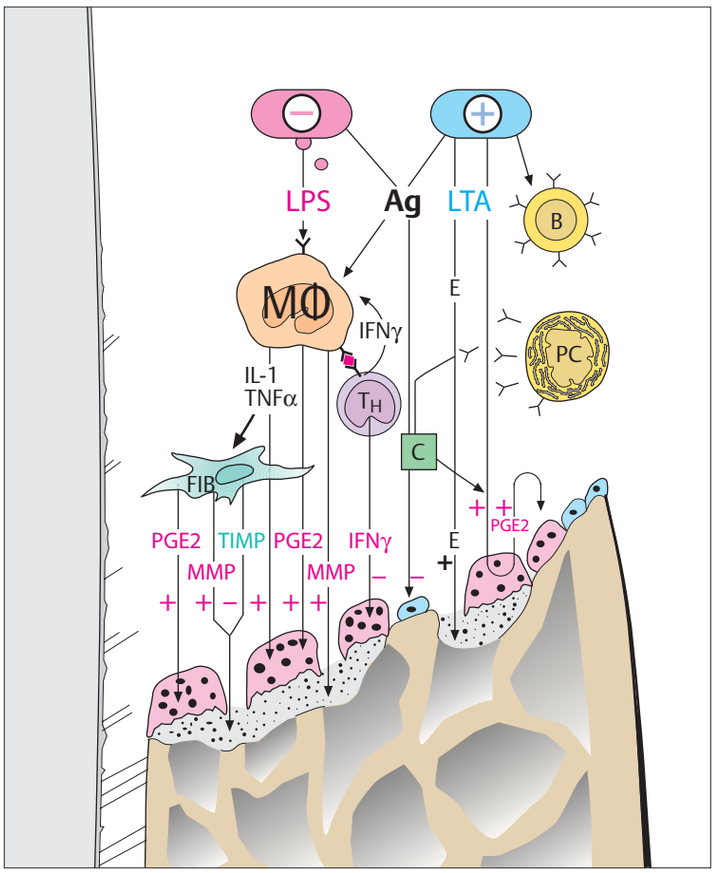
Stimulatory (+) and Inhibitory (-) Components of Bone Resorption

Gram-negative bacteria:
Ag Antigens
LPS Lipopolysaccharides

Gram-positive bacteria:
Ag Antigens
LTA Lipoteichoic Acid
E Enzymes

Host cells:
B B-cells
PC Plasma Cells
T_H T-Helper Cells
MΦ Macrophages
FIB Fibroblasts

Host molecules:
C Complement
IL-1 Interleukin 1
IFNγ Interferon γ
MMP Matrix Metalloproteinases
PGE2 Prostaglandin E2
TIMP MMP-Inhibitors
TNFα Tumor Necrosis Factor α

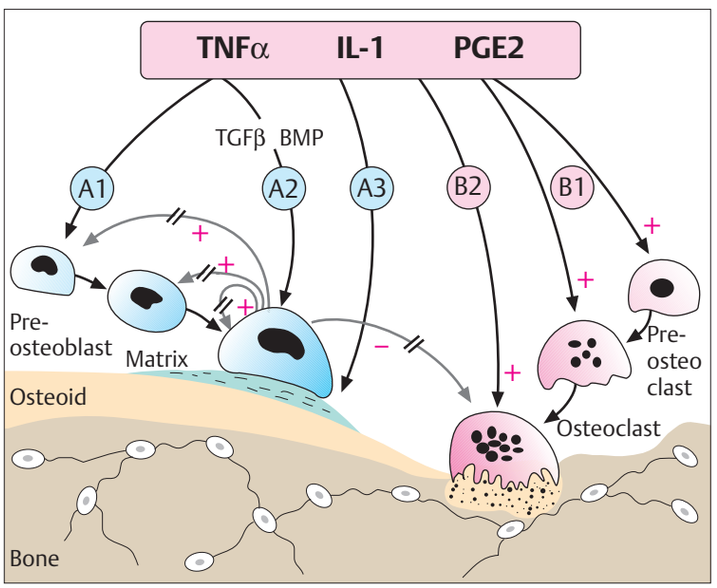


119 Mechanisms of Local Periodontal Bone Destruction
The arrows eminating from the macrophages (MΦ) and from stimulated fibroblasts (FIB) indicate the enzymatic destruction of the *organic* bone matrix (MMP/TIMP).
Activated, multinuclear osteoclasts (red) resorb *inorganic/mineral portions* of the alveolar bone (acid-released calcium phosphate).
Osteoclasts exhibit on their outer surface a "sucker," and within a brush-like border (secretion of acids, resorption of released minerals).

Additional Local (Growth) Factors:
BMP Bone Morphogenetic Protein
TGFβ Transforming Growth Factor β

Inhibition of osteoblasts:
A1 Pre-osteoblasts are inhibited in differentiation to osteoblasts
A2 Production of TGFβ, BMP is inhibited
A3 Matrix production is inhibited

Stimulation of osteoclasts:
B1 Osteoclast differentiation is enhanced
B2 Osteoclast activity is enhanced



120 Bone Remodeling During Progressive Periodontitis—Local Factors for Inhibition and Stimulation!
The local factors released from the inflammatory cells, osteoblasts and osteoclasts alter normal tissue homeostasis: Osteoblasts are inhibited, osteoclasts are stimulated. The differentiation, proliferation and the capability of osteoblasts to synthesize matrix substances and cytokines (e.g., for the auto- and paracrine stimulation) are thereby influenced.

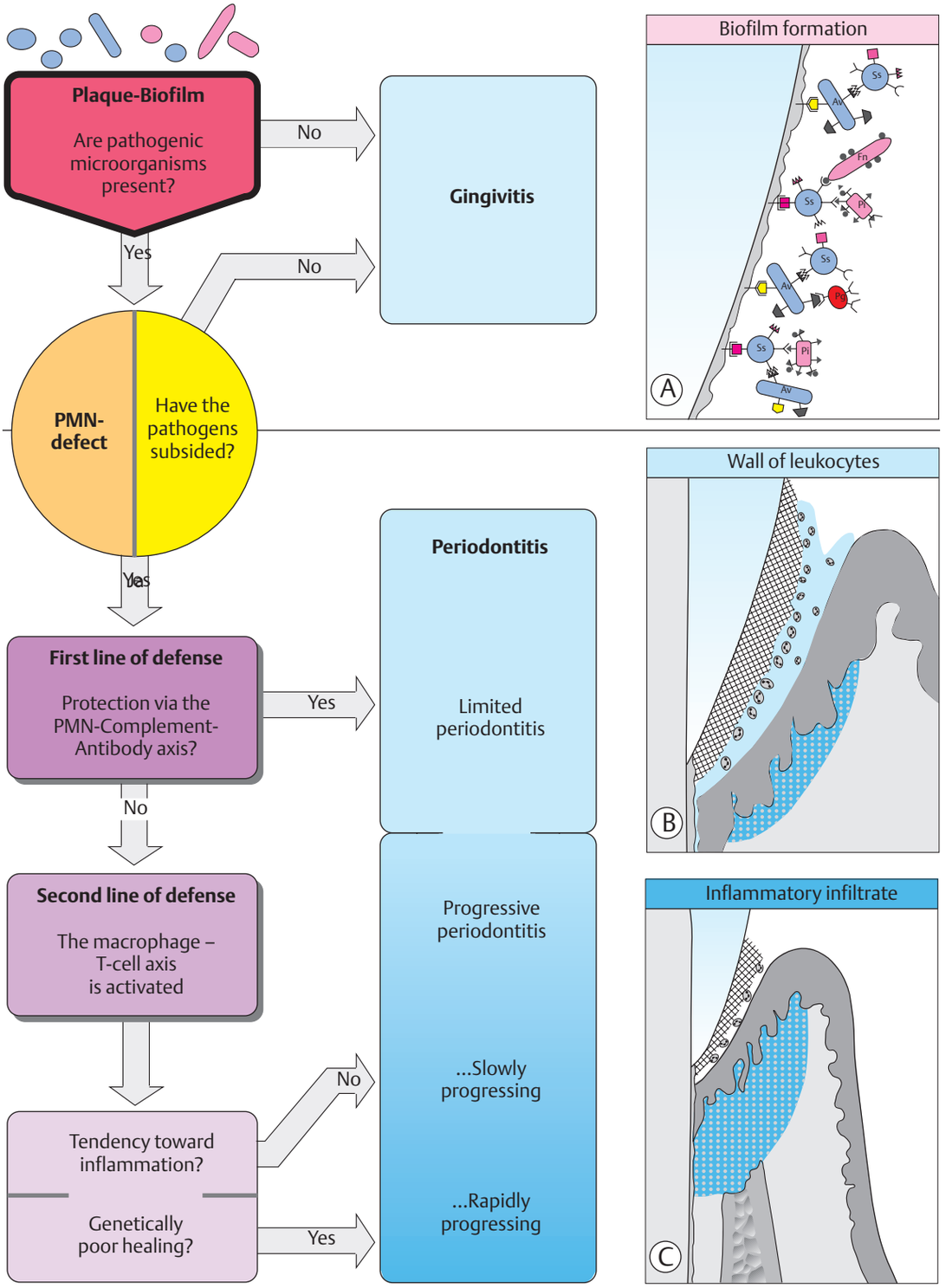
Modified from *Z. Schwartz et al. 1997*

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Pathogenesis—Clinical Features: From Gingivitis to Periodontitis

The stages in the pathogenesis of gingivitis and periodontitis have already been described histopathologically (p. 56) and at the molecular level (p. 58).

In 1993, Offenbacher and his coworkers presented a promising concept to explain why the presence of so-called pathogenic bacteria (p. 33) is associated in some patients with gingivitis and in others with periodontitis. A most important role is played by the neutrophilic granulocytes (PMN): If PMNs exhibit a defect in diapedesis, a failure to respond to chemotaxis, inadequate mobility, inability to phagocytose and "digest" the bacteria, the PMNs are incapable of preventing bacterial invasion or the establishment of the subgingival biofilm. Also, when too few PMNs are present or if the bacteria are capable of avoiding them selectively, a clinically obvious and established gingivitis will develop (Schroeder 1994). The progress of chronic or aggressive periodontitis then depends upon the additional immunologic defense mechanisms and the genetically determined inflammatory response and healing capabilities of the tissue (varying host suceptibility).



121 Clinical Flow Diagram: from Gingivitis to Periodontitis
Biofilm metabolites activate host defense. Host response varies individually and will be more or less severe depending on the inflammatory reaction → gingivitis.

A *Early colonizers* (*Ss* and *Av*) make possible colonization by further bacterial species (*Fn, Pi, Pg*) into the developing biofilm (cf. Fig. 44).
Polymorphonuclear granulocytes (PMNs) are the first defense cells in the sulcus (Miyasaki 1991). If the PMN response is defective or if the bacteria are able to avoid the PMN response, pathogenic bacteria become established in the subgingival area, and elicit a *"limited" periodontitis.*

B A so-called leukocyte wall, formed primarily by PMNs, covers the plaque.
The initial defense axis (*congenital immunity*) consists of phagocytic cells (PMNs), complement and antibodies.
If the first axis fails, the secondary defense axis (MΦ and T-cells) must be activated: *acquired immunity*. The inflammatory process becomes chronic, and periodontitis progresses.
The MΦ/ T-cell reaction varies in intensity: Tendencies toward inflammation and the genetics of healing are different, and the progression of the periodontitis will also vary.

C Host defense mechanisms of the inflammatory infiltrate create this second defense axis.

Modified from *K. Miyasaki 1991, S. Offenbacher et al. 1993*

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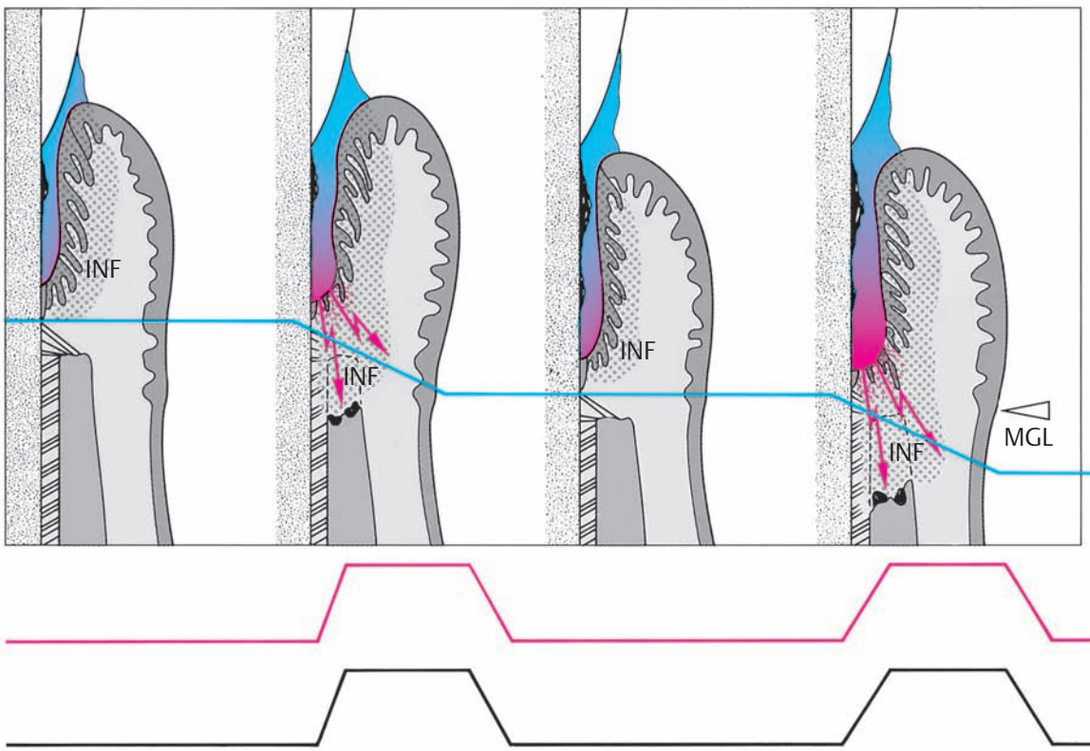
Cyclic Course of Periodontitis

Stable Lesion—Active Progressing Lesion

Periodontitis seldom progresses continuously. Most often, as demonstrated by Goodson et al. (1982) and Socransky et al. (1984), the attachment loss occurs cyclically, in "bursts" on individual teeth or on individual surfaces of teeth. During acute phases, the gram-negative, anaerobic and motile bacteria increase in numbers. Within a very short period of time, direct *microbial invasion* of the tissues can occur. This invasion reacts to acute host defense mechanisms, with the formation of *micronecrosis* or suppurating *abscesses*. Attachment loss occurs via collagen destruction.

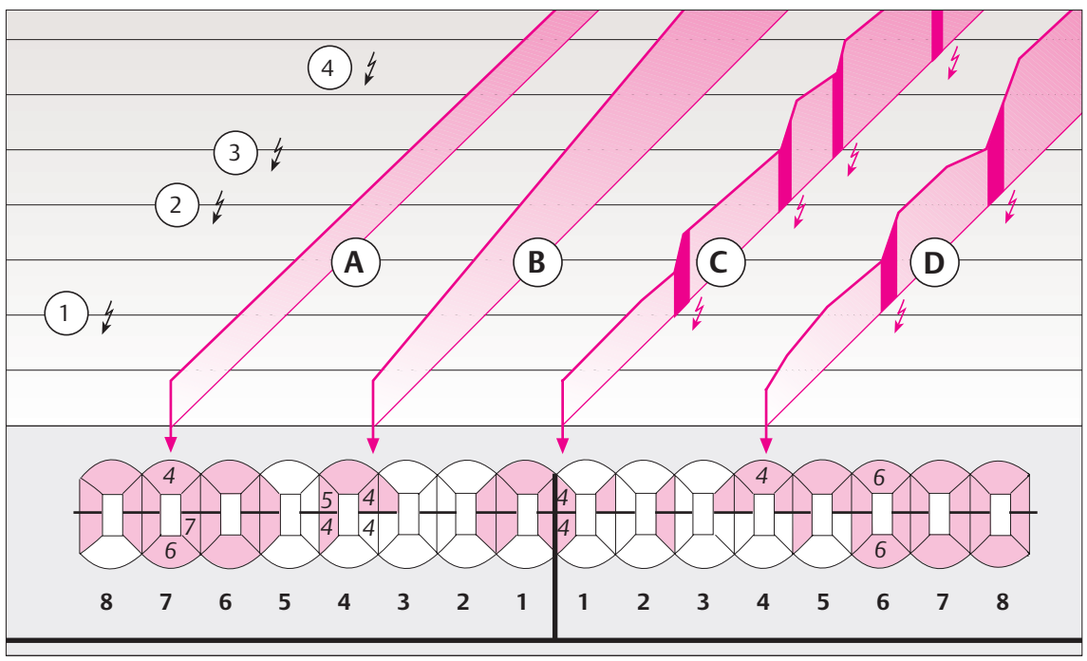
Additional mechanisms remain under discussion (Page et al. 1997):

- Changes in the biofilm with high levels of LPS; consequence: Very high levels of IL-1, TNF, PGE2 and MMP, with the already discussed consequences.
- Significant disturbance of the normal chemotactic gradients (IL-8, FMPL): The PMNs "explode" within the connective tissue, which is thereby damaged.
- PMN diapedesis is inhibited by LPS from *P. gingivalis* and various polyamines: PMNs are effectively removed from the acute defense reaction.



122 Cyclic Nature
Alterations in the subgingival biofilm, the increase in pathogenic bacteria and their invasion into tissues (see text) elicit the *acute inflammatory tissue reaction* (red arrows), *attachment loss* and *bone resorption.*
An enhanced host response can eliminate the bacterial insult and lead to *stagnation* of the acute exacerbation. A certain degree of tissue regeneration may even occur during such stages.

Modified from *M. Newman 1979*



123 Regular and Irregular, "Site-Specific" Attachment Loss
The chart (below) reveals probing depths to 6 mm. Such pockets can remain inactive for years (**A**). It is only seldom that pocket depth grows deeper *continuously* (**B**). On individual surfaces of teeth, it is much more common to see symptoms of activity (phases **1–4**):

- **A** Probing depth remains constant for years
- **B** Continuous process: 1 mm attachment loss
- **C** Four acute phases: With attachment loss
- **D** Two acute phases: With loss and remission

Modified from *S. Socransy et al. 1984*

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Periodontal Infections and Systemic Diseases

Does Periodontitis Make Us Sick?

The strong influence of host factors in the pathogenesis and progression of periodontitis has been previously described. New knowledge from research has shown that the chronic infectious disease "periodontits" may also lead to serious systemic diseases in particularly susceptible individuals. Periodontitis, at the very least, must be viewed as a weighty risk factor for these systemic, and also multifactorial, diseases (Mealey 1999). Interactions have been demonstrated, or are suspected, with the following systemic diseases:

- Cardiovascular diseases:
  angina pectoris, myocardial infarction, endocarditis
- Difficulties during pregnancy: premature birth,
  low birth weight, increased infant mortality
- Stroke, brain abscess
- Pulmonary infections
- Diabetes Mellitus (DM)

Cardiovascular Diseases: Angina Pectoris, Myocardial Infarction, Endocarditis

We have known for a long time the "classic" risk factors for this group of diseases: high triglycerides and low density cholesterol, stress, smoking and simply being a male. Beck and coworkers (1996) demonstrated in a large, longitudinal study of over 1100 men that periodontitis with deep probing depths increased the risk of coronary heart diseases, independent of the other risk factors.
In very general terms, *any infection* is a risk factor for atherosclerosis, embolism, and endocarditis. Infections with gram-negative microorganisms are associated with the flowing of inflammatory mediators into the vascular system, including also the systemically active cytokines (TNF, IL-1, IL-6), growth factors and prostaglandins.
Some gram-positive organisms may also be causative for severe cardiovascular diseases: Streptococci from the oral cavity, especially *S. sanguis*, can elicit the feared endocarditis or support it (De Bowes 1998, Herzberg & Meyer 1998, Meyer & Fives-Taylor 1998, Chiu 1999).

Problems in Pregnancy: Premature Birth, Low Birth Weight, Elevated Infant Mortality

Premature births with a birth weight of under 2500 g are the direct consequence of premature contractions and rupture of the membrane. Risk factors include smoking, drug abuse, alcohol, diabetes, high and low age of the mother, and bacterial infection of the urogenital tract.
However, in one out of four cases, other reasons for premature birth and low birthweight were identified (Offenbacher et al. 1996, 1998; De Bowes 1998). The initiation of contractions and subsequent birth are influenced significantly by prostaglandins (PGE2, PGF2α—components of the "morning after pill" RU 486!); these are mediators, of course, that have long been known to be increased in patients with periodontitis.

Stroke, Brain Abscess

Microorganisms from many different infected organs can reach the brain. Organisms from the oral cavity are rare there, and therefore appropriate research is lacking. Ziegler and coworkers (1998) described possible relationships between oral infections (including severe periodontitis) and stroke. Brain abscesses are usually a result of anaerobic infections. Except for individual case reports, no comprehensive research studies are available to definitively conclude that infections of the brain could be caused by oral microorganisms (Saal et al. 1988, Andersen & Horton 1990).

Pulmonary Infections

Oral, nasal and pharyngeal microorganisms frequently contaminate the upper airways (De Bowes 1998, Scannapieco et al. 1998, Scannapieco 1999). Clinic patients and the homebound, especially those in need of constant care, often exhibit poor oral hygiene and a high level of plaque accumulation. Such plaque represents a significant reservoir for potential pathogenic invasion of the respiratory tract (Terpenning et al. 1993).

Diabetes Mellitus (DM)—Type 1, Type 2

Intensive research has demonstrated that diabetes significantly increases the risk for periodontitis and its progression (p. 215). Other studies have asked the question whether periodontitis influences metabolic control in diabetes patients (Yki-Järvinen 1989, Grossi & Genco 1998, Lalla et al. 2000). Rayfield and coworkers (1982) found a direct correlation between the number of acute infections and complications in the control of the blood sugar level. There is a good correlation between acute infections and reduced response to insulin, a condition that can inhibit clinical recovery after prolonged periods of time (Sammalkorpi 1989).

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Etiology and Pathogenesis—Summary

Healthy Gingiva

The tissues of healthy gingiva (pp. 8–13) contain within their epithelial and connective tissue structures a certain defense potential against the microorganisms of dental plaque. Even in healthy tissues, one observes the ever-present immunologic defense reactions (interactions between host and bacteria: the human being as "biotope").

Plaque

Dental plaque is a biofilm. Within only hours after thorough cleaning, it is re-formed upon the pellicle of the tooth structure. Plaque also occurs as a bacterial accumulation upon soft tissues (e.g., mucosal surfaces) and is therefore not always removed periodically by common oral hygiene procedures! Initially gram-positive bacteria proliferate and form an organized biofilm. These bacteria do not invade the host tissues and therefore effect such tissues only via their metabolites.

Connective Tissue

The bacterial metabolites recruit mainly polymorphonuclear granulocytes (PMN) from the activated subepithelial venule plexus. The PMNs exit in small numbers from the vessels. In this *clinically healthy stage*, very few if any other inflammatory cells are observed. Immunoregulatory mediators far exceed the pro-inflammatory mediators. There is no damage to fibroblasts or collagen.

Junctional Epithelium/Gingival Sulcus

In addition to blood plasma constituents (sulcus fluid), PMNs wander in small numbers, following the chemotactic gradient, through the intercellular spaces of the junctional epithelium, and enter the gingival sulcus. Here they build a defensive wall against the plaque bacteria, but are not capable of eliminating an organized biofilm via phagocytosis.

With good oral hygiene, the balance between the bacterial presence and the initial, non-specific defense (PMN mediators of inflammation) can be maintained over years. This "condition" was earlier referred to as "initial gingivitis," however with regard to today's knowledge, it cannot be defined as a true "disease."

The "old" periodontologists of the first half of the twentieth century would perhaps define this histologic situation as "physiologic inflammation" of the gingiva.

Established Gingivitis

If, in a patient with healthy gingiva, the biofilm is permitted to grow and mature by cessation of oral hygiene measures, gram-negative bacteria quickly establish a foothold, and their metabolites (e.g., LPS) traverse the junctional epithelium and enter the connective tissue. This leads to the establishment of "*early gingivitis*" (Page & Schroeder 1976). This condition is characterized by elevation in the sulcus fluid flow and PMNs. In the subepithelial area, one observes primarily T-cells. Also observed is initial collagen loss, cytopathic alterations of the resident fibroblasts and early lateral proliferation of the juctional epithelium. This "early gingival lesion" is only a shortly-lived (4–14 days) pre-stage of *established gingivitis*. Only in children can the early gingivitis lesions persist over long periods of time. In adults, one observes almost exclusively the *established gingivitis* lesions, with very different degrees of manifestation.

Plaque

The biofilm persists. The host defense can attack it only superficially, and if oral hygiene is inadequate, it will expand broadly. With time, the gram-negative bacteria increase in percentage.

Connective Tissue

As a result of the continuous diffusion of antigens and toxins (e.g., lipopolysaccharides), more and more macrophages are activated which, via the pro-inflammatory cytokines (TNF, IL-1, IL-6, IL-8) and other secondary mediators of inflammation (above all, PGE2; p. 49) the endothelial cells of the vessels are signaled to now permit other cellular blood-borne substances to exit the vasculature in addition to PMNs and plasma proteins (Expression of specific adhesins). In the rapidly expanding inflammatory infiltrate, both humoral (immunoglobulins from B-cells/plasma cells) and cellular (T-cells) immune reactions occur. The host defense now becomes more targeted via antigen processing and targeted immunoglobulin production: The mounting of the specific, adaptive immunity, the second line of defense (pp. 43 and 62).

Junctional Epithelium/Sulcus/Gingiva

The junctional epithelium proliferates laterally, but not apically.

Inflammatory swelling (edema, hyperplasia) of the gingiva can place segments of the plaque subgingivally, resulting in a situation in which primarily gram-negative, anaerobic bacteria come to reside within the deepened suclus (gingival pocket, pseudopocket). *Established gingivitis* is the result. At this point, there is no attachment loss. With targeted and adequate treatment, this gingivitis is completely reversible.

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Chronic and Aggressive Periodontitis

The etiology and the pathogenesis of all forms of periodontitis are essentially similar. However, the various clinical courses of disease (chronic, aggressive, etc.) can be explained by the varying intensity and quality of the bacterial insult on the one hand, and the host response to the infection, as well as the number and type of collateral risk factors on the other hand.

Plaque

Gingivitis develops into periodontitis with attachment loss and formation of true periodontal pockets only if the plaque contains virulent periodontopathic bacteria at a critical level (p. 33), and in response to an inadequate local immune response in a susceptible host individual.

As pocket depth increases, the plaque becomes progressively more gram-negative and anaerobic. Protected within the biofilm, especially in the subgingival region, the bacteria can no longer be eliminated by the host defense mechanisms. Because the biofilm has become established between the tooth root and vital pocket soft tissue, natural restitution of the health of the tissues has been rendered virtually impossible.

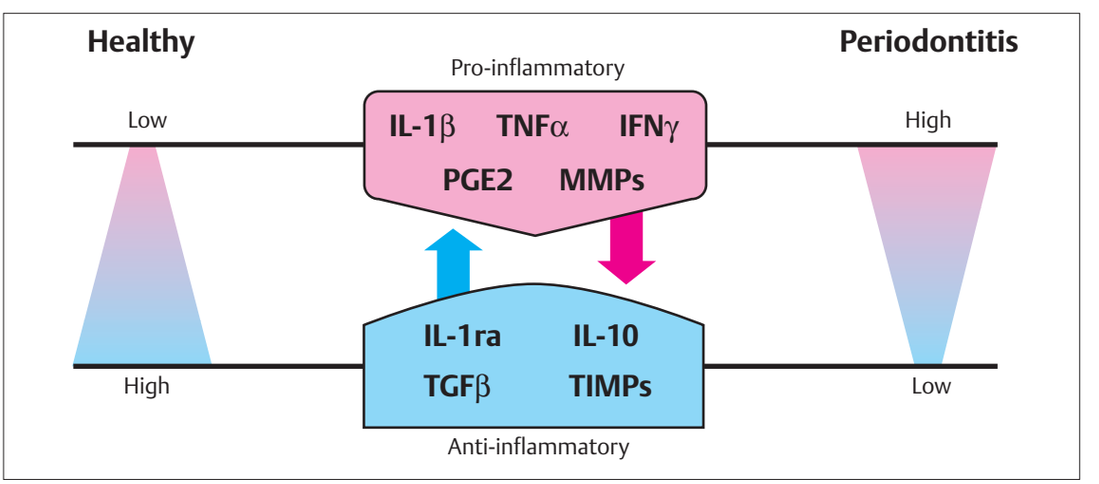
The virulent pathogenic bacteria have the capacity to destroy at ever-increasing rates the defense response and the tissue homeostasis, as was described for gingivitis.

High LPS concentrations and the presence of numerous mediators maintain strict control over the both targeted and peripheral host reactions.

Gingiva/Periodontal Ligament/Alveolar Bone

In patients with periodontitis, it is quite common that gingivitis also persists, but its degree of severity can vary widely. In the case of juvenile, aggressive periodontitis (formerly LJP), gingival inflammation may remain quite mild even in the face of pronounced attachment loss (p. 116).

In the presence of non-alterable (e. g., genetic) and/or alterable (e. g., smoking) risk factors, there may occur an increase in pro-inflammatory mediators (inadequate host response), a condition which could force periodontopathic microorganisms apically, and therefore involve deeper regions of the periodontium (*periodontal ligament and alveolar bone*) in the destructive process. The result is destruction of the tooth-supporting apparatus and attachment loss (progressive lesion; periodontitis).



124 The "Bottom Line": Mediators and the Origins of Periodontitis

Periodontal *health* is characterized by few pathogenic bacteria, a low level of pro-inflammatory cytokines, prostaglandin E2 and matrix metalloproteinases MMP and a high level of tissue inhibitors of MMP (TIMP) and cytokines that suppress the immune-inflammatory reaction (IL-1ra, IL-10, TGFβ). In *periodontitis* one observes exactly the opposite!

Modified from *R. Page et al. 1997*

Fact

At the end of the day, all of the previously described clinical, histologic and molecular biologic reactions are steered by signal molecules of the cytokine network ("internet of the tissues"), and by anabolic/anti-inflammatory or catabolic/pro-inflammatory mediators.

Knowledge of the interactions of these guiding factors permits recognition of the individual forms of periodontitis (primarily the aggressive form) and permits diagnosis and ultimately also appropriate treatment. In addition to the classical diagnostic techniques (probing depth, attachment loss, attachment level, radiographic appearance, plaque indices, bleeding indices etc.), specific diagnostic tests are already on the market which modify and enhance the new knowledge: Bacteriological tests (e. g. DNA tests, p. 183) and tests of the host's reaction (e. g., gene tests: IL-1 gene polymorphism, p. 189). Today, one has hope that in the near future the very bases of the described mechanisms in the complex disease of periodontitis can be stabilized to prevent destruction of periodontal tissues pharmacologically, biochemically or by genetic technology.

There is hope that, in the near future, periodontitis will not simply be treated by means of mechanical therapy, but rather that the clinical diagnosis of still *healthy* individuals at a young age can provide the practitioner with means for primary prophylactic measures to keep the disease process under control or prevent it entirely.

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Indices

The inflammatory diseases of gingiva and periodontium, as well as their symptoms and the etiologic agents of these conditions (microbial plaque/biofilm) can be assessed clinically using qualitative and/or quantitative indices. In most instances, indices are used in epidemiologic studies, but they can also be useful during clinical examination of individual patients.

Indices are expressed numerically to describe defined diagnostic criteria: A disease process or its severity is described or classified using numbers (1, 2, 3 etc.). Simple indices record only the presence or absence of a symptom or an etiologic agent with "yes or no" entries, e.g., after sulcus probing: (+) = "bleeding," (-) = "no bleeding."

An appropriate index permits quantitative and qualitative statements about the criteria under investigation (disease, disease etiology), and is simple, objective, reproducible, rapid and practical. It should also be amenable for use by auxiliary personnel. Indices must provide data that are amenable to *statistical evaluation*.

Although indices for the most part were developed for *epidemiologic studies*, international standardization among various research groups has proven to be impossible: Many investigators use various indices or employ no indices whatever in periodontitis studies, rather they collect data on pocket probing depth and/or attachment loss in millimeters, which are then attributed to certain degrees of severity. Severity grades I–III: for probing depths up to 3 mm (I), between 4 and 6 mm (II) and those of 7 mm and greater (III). Other epidemiologists may assess these three severity grades using different millimeter measurements. Therefore it is scarcely possible to precisely compare the results of various studies to each other. Nevertheless, approximate conclusions can be drawn, e.g., concerning the worldwide incidence of periodontitis (p. 75).

Indices are also used in the *private practice with individual patients*: Especially plaque and gingivitis can be readily assessed in numerical fashion.

Repeated determination of an index during the course of preventive or active therapy can help ascertain the degree of patient motivation/compliance and treatment success or failure.

In this chapter, only a few of the many indices will be briefly described, primarily those which have been used for international epidemiologic studies, as well as some that are indicated for use on individual patients in private practice.

Plaque Indices
- Plaque index (PI)—O'Leary et al. 1972
- Approximal plaque index (API)—Lange 1986
- Plaque index (PI)—Silness & Löe 1964

Gingivitis Indices
- Bleeding on probing (BOP)—Ainamo & Bay 1975
- Papilla bleeding index (PBI)—Saxer & Mühlemann 1975
- Gingival index (GI)—Löe & Silness 1963

Periodontal Indices
- Periodontal disease index (PDI)—Ramfjord 1959
- Community periodontal index of treatment needs (CPITN)—WHO 1978
- Periodontal screening and recording (PSR)—ADA/AAP 1992

"Gingival Recession Index"
- Recession is measured in mm from the cementoenamel junction to the gingival margin (Jahnke et al. 1993, p. 162) or classified according to Miller (1985, pp. 162–163).

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Plaque Indices

125 Plaque Index simplified/PI—Plaque Control Record, PCR (PI; PCR—O'Leary et al. 1972)

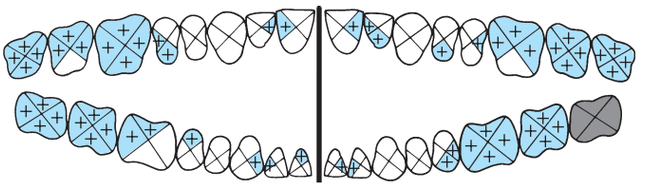
This precise index records the presence of supragingival plaque on all four tooth surfaces. For this test, the plaque is disclosed. The presence (+) or absence (–) of plaque is recorded in a simple chart, and the plaque incidence in the oral cavity is expressed as an exact percentage.

- PI is an index for the practice.

PI Plaque Index simplified

– No plaque at the gingival margin (not scored)

\+ Plaque at the gingival margin



Calculation

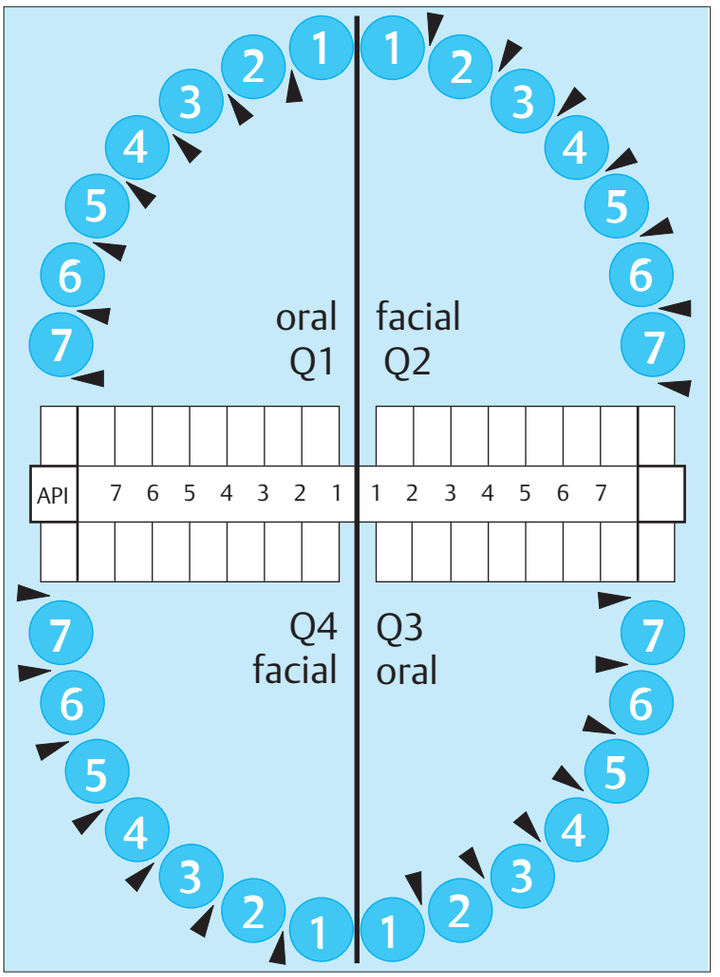
$$PI = \frac{\text{Number of sites with plaque}}{\text{Number of sites evaluated}} \times 100 \quad \text{Example: } \frac{57}{124} \times 100 = 46\,\%$$

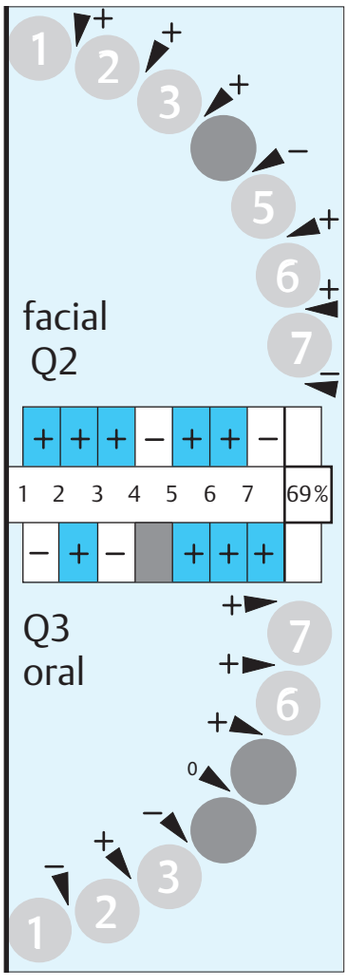
Abbreviation	Grade
PI=PCR	⊕ & ⊖

126 Approximal Plaque Index—API (Lange 1986)

Following application of disclosing solution, a simple yes/no decision is made concerning whether the examined *interproximal surfaces* are covered by plaque (+) or not (–). The proportion of plaque-covered interproximal spaces is expressed as a percentage. Usually, analogous to the papilla bleeding index (PBI, Fig. 129), in a given quadrant the interproximal spaces are scored from only one aspect, i. e., from the facial (Q2 and Q4) or from the oral aspect (Q1 and Q3). The API is indicated for individual patient data collection and for motivation. It correlates with the PBI and is calculated as shown in the following formula:

$$\textbf{API} = \frac{\text{No. of plaque (+) sites}}{\text{No. of sites examined}} \times 100$$



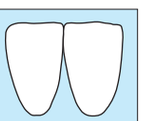
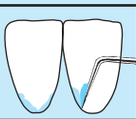
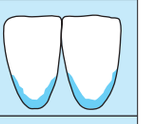
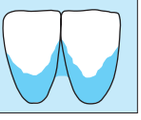


Right: The problem of missing teeth (dark): If only one tooth is missing (above), the measurement site remains intact; if two adjacent teeth are missing (below) one measurement site is lost. In the depicted quadrants (2 and 3) the API is 69 %.

127 Plaque Index (PI; Silness & Löe 1964)

This index ascertains the *thickness* of plaque along the gingival margin; only this plaque plays any role in the etiology of gingivitis. To visualize plaque, teeth are dried with air. Plaque is not stained.

The PI is indicated for epidemiologic studies in which the gingival index (GI) is recorded simultaneously. It is less useful for routine dental office charting.

Grade		
0	**No** Plaque	
1	**Thin** plaque layer at the gingival margin, only detectable by scraping with a probe	
2	**Moderate** layer of plaque along the gingival margin; interdental spaces free, but plaque is visible to the naked eye	
3	**Abundant** plaque along the gingival margin; interdental spaces filled with plaque	

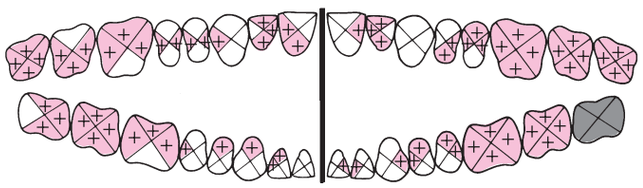


Abbreviation	Grade
PI	0–3

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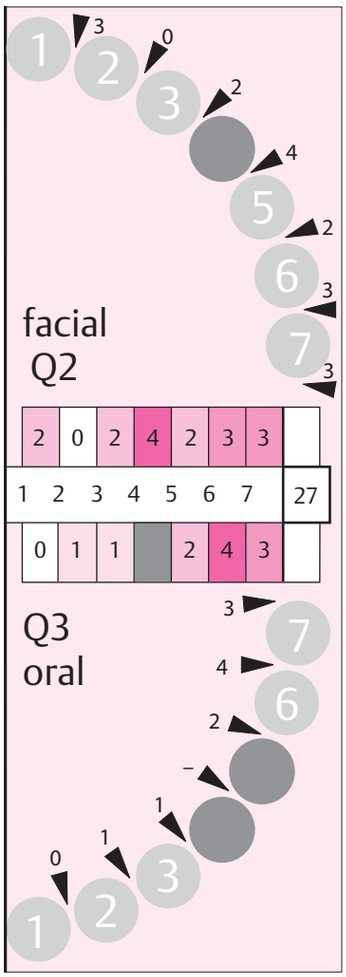
Grade	Abbreviation
⊕ & ⊖	BOP

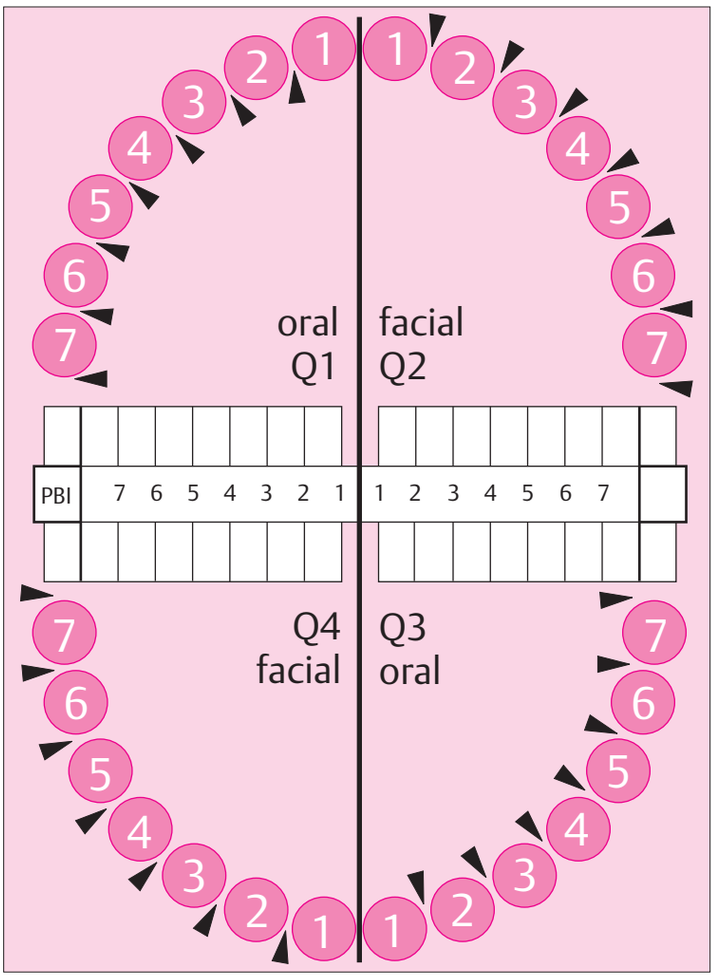
BOP bleeding on probing
– no bleeding upon probing (not recorded)
+ bleeding upon probing



Calculation

$$BOP = \frac{\text{Number of bleeding sites}}{\text{Number of sites evaluated}} \times 100 \qquad \text{Example } \frac{71}{124} \times 100 = 57\,\%$$





Grade	Abbreviation
0–3	GI

Grade	
0	Normal gingiva; no inflammation; no discoloration (erythema); no bleeding
1	Mild inflammation; slight erythema; minimal superficial alterations. **No bleeding**
2	Moderate inflammation; erythema; **bleeding on probing**
3	Severe inflammation; severe erythema and swelling; **tendency to spontaneous bleeding;** possible ulceration.

Gingival Indices

128 Bleeding on Probing – BOP (Ainamo & Bay 1975)

As in the PI (Fig. 125), all four surfaces of all teeth are assessed with regard to whether probing elicits bleeding (+) or not (–). The severity of gingivitis is expressed as a percentage.
Because more than 100 sites must be measured, the BOP is indicated only for individual patient examinations (e. g., data collection, recall).

129 Papilla Bleeding Index—PBI (Saxer & Mühlemann 1975)

The PBI discriminates four *different degrees (intensities) of bleeding* subsequent to careful probing of the gingival sulcus in the papillary region (see p. 70). Probing is performed in all four quadrants. To simplify the recording of the PBI, quadrant 1 is probed only from the oral aspect, quadrant 2 from the facial, 3 again from oral, and from the facial in quadrant 4 (see black arrows in diagram). Bleeding scores are entered into the chart (middle).
The PBI can be reported as the *bleeding number* (= sum of all values) or as an index (average severity, as in this formula:

$$\textbf{PBI} = \frac{\text{Bleeding Number}}{\text{Number of sites measured}}$$

- The PBI is valuable for the practitioner, who can compare values over time (patient motivation, risk factors).

Left: In this example (quadrants 2 and 3), the PBI is 2.1; the bleeding number is 27. Missing teeth? Compare Fig. 126, right.

130 Gingival Index—GI (Löe & Silness 1963)

The GI records gingival inflammation in three grades. It is measured on six selected teeth (16, 12; 24 and 36, 32; 44; cf. Fig. 127) on facial, oral, mesial and distal sites. The symptom of *bleeding* comprises a score of 2.

- The GI was developed for epidemiologic studies. It is less applicable for individual patients because the differences between the scoring levels are too gross.

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Papilla Bleeding Index—PBI

The PBI was developed for use in the private practice and not for epidemiologic studies. It is a *sensitive indicator* of the severity of gingival inflammation in individual patients. The PBI does not require a great amount of time, since only 28 measurement sites in the complete dentition are evaluated (Saxer & Mühlemann 1975).

The PBI has proven to be particularly useful for assessing inflammation in the interdental papillae by recording bleeding on probing in the interdental areas during the course of treatment. The index therefore offers an excellent means for patient *motivation* (p. 222). While the patient watches in a mirror, the practitioner can score the intensity of papillary inflammation. The patient can see when the gingival tissue bleeds, which helps him to realize where the diseased sites in the mouth are located.

The patient will realize and experience the reduction in inflammation during the course of therapy as the index is repeated at each visit, and this is a good motivator for thorough and continuing patient compliance.



131 Grade 1—Point
20–30 seconds after probing the mesial and distal sulci with a periodontal probe, a single bleeding point is observed.

132 Grade 2—Line/Points
A fine line of blood or several bleeding points become visible at the gingival margin.

133 Grade 3—Triangle
The interdental triangle becomes more or less filled with blood.

134 Grade 4—Drops
Profuse bleeding. Immediately after probing, blood flows into the interdental area to cover portions of the tooth and/or gingiva.

Recording the PBI

Bleeding is provoked by sweeping the sulcus using a blunt periodontal probe under light finger pressure from the base of the papilla to its tip along the tooth's distal and mesial aspects. After *20–30 seconds*, when a quadrant has been completely probed, the intensity of bleeding is scored in four grades and recorded on the chart.

The sum of the recorded scores gives the "*bleeding number*." The PBI is calculated by dividing the bleeding number by the total number of papilla examined.

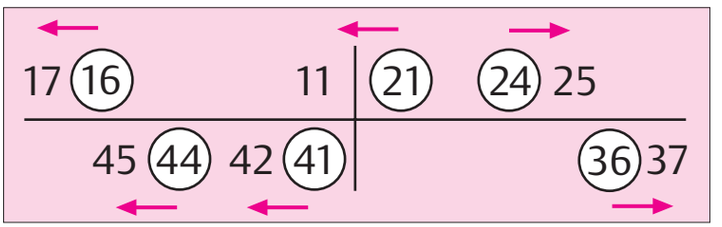
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Periodontal Indices

The determination of the *severity* of periodontitis through use of an index is really impossible. In contrast to a gingivitis index, which must only record the intensity of inflammation, any periodontitis index must, above all, measure the pocket probing depths and the degree of loss of tooth-supporting tissues (attachment loss). Periodontal indices find their most important use in epidemiologic studies. In private practice, a periodontal index may provide a quick overview of a patient's condition.

Many years ago numerous epidemiologic studies were performed using the *Periodontal Disease Index* (PDI) of Ramfjord (1959). More recently the CPITN has been recommended by the World Health Organization (WHO; p. 72). The CPITN was modified by the American Dental Association (ADA 1992) and the American Academy of Periodontology (AAP 1992) for a triage-type rapid diagnosis in general dental practice (PSR, p. 73).

Periodontal Disease Index (PDI)



Grade	
0	Inflammation-free. No gingival alterations
	Gingiva
1	Mild to moderate gingivitis at isolated sites on the gingiva surrounding the tooth
2	Mild to moderate gingivitis surrounding the tooth
3	Severe gingivitis, visible erythema, hemorrhage, gingival ulcerations
	Periodontium
4	Attachment loss to 3 mm, measured from the CEJ
5	3–6 mm attachment loss
6	Attachment loss greater than 6 mm

135 Measurement of the PDI Using Ramfjord Teeth—Replacement Teeth
Ramfjord demonstrated that for epidemiologic purposes six teeth (circled) could be taken as representative of the entire dentition. If any of the "Ramfjord teeth" are missing, the replacement teeth (not circled) are substituted.

136 Periodontal Disease Index (PDI)—Degree of Severity
Within the PDI, grades 1, 2 and 3 represent a *gingivitis index*, while grades 4, 5 and 6 represent an index for the extent of periodontal destruction (*attachment loss*) independent of gingivitis and probing depths.

The PDI is not indicated for private practice but for epidemiologic studies.

Periodontal Disease Index (PDI)

The PDI does not include examination of all 28 teeth (third molars are excluded in almost *all* indices), but rather a sample of six teeth that are representative of the entire dentition. These teeth were chosen so that each tooth type, both jaws and each quadrant are taken into account: teeth 16; 21, 24; 36; 41 and 44. These are the so-called "Ramfjord teeth." If one of these teeth is missing, its distal neighbor (17, 11; 25; 37; 42 or 45, respectively) may be substituted (Marthaler et al. 1971).
The PDI evaluates these selected teeth for both gingivitis and attachment loss, with three gradations for each. For periodontitis grades 4, 5 and 6, probing depth ("pocket depth") is *not* measured, rather the distance from the cementoenamel junction to the bottom of the pocket is recorded (attachment loss).

An average PDI score (e.g., 2.8) *cannot* be taken to indicate that only gingivitis (to grade 3) is present, or whether attachment loss has already occurred on individual teeth. For example, mild gingivitis and slight attachment loss on individual teeth could lead to an average value of < 3. Grades 1–3 and grades 4–6 must there be evaluated separately.

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Community Periodontal Index of Treatment Needs—CPITN

The CPITN was developed in 1978 by the World Health Organization (WHO, 1978; Ainamo et al. 1982). It is used primarily for epidemiologic studies (p. 76). The major difference between the CPITN and other indices is that it determines not only the severity of gingivitis (bleeding) and periodontitis (pocket probing depth), but also provides information concerning the type of disease process and therefore also the extent of therapy that is necessary. Thus the CPITN provides not only conclusions about the incidence of gingivitis and periodontitis in a population, but also about the necessary expense, in both time and money, that will be necessary for treatment of a population group.

The CPITN does not consider the attachment loss on individual teeth, rather only the clinical situations requiring treatment:

- Gingival inflammation
- Bleeding
- Calculus
- Pocket probing depth

The CPITN is measured and ascertained using a *special probe* on all teeth, and the most severe areas in each *sextant* is noted in the chart.

137 Special Probes and the Codes 0–4 for Recording the CPITN and PSR

These probes are characterized by a small sphere of 0.5 mm diameter and a black band between 3.5 and 5.5 mm.

For use in the practice, additional grooves at 8.5 and 11.5 mm may be included. Probing is performed with a ca. 0.25 N force. The highest code in each sextant is entered into a simple form (see below).

Special probes



Code 0



Code 1



138 CPITN and PSR Codes—Definitions

Codes 0–4 define health (0) or disease in gingiva and periodontium (1–4). In principle they are identical for both indices. Because it was developed for private practice, the PSR is somewhat more detailed than the CPITN, because in addition to the actual codes, an asterisk (*) can be added as an indication that the case is more complicated, e. g., by ...

- Furcation involvement
- Elevated tooth mobility
- Mucogingival problems (e. g., lack of attached gingiva)
- Recession > 3 mm

Table for recording the PSR:

4*	1	3
3	2*	3

Comments:

- Pocket-free sextant in the anterior region, but severe recession in the mandibular anterior sextant (*)!
- Pockets up to 5 mm in sextants 3, 4, 6
- Sextant 1: Furcation involvement and pockets of 7 mm.

	CPITN-Codes	PSR-Codes	
0	– Healthy	– No bleeding – No calculus – Band 100% visible	0
1	– **Bleeding** on probing — BOP	– **Bleeding** – No calculus or defective restorations – Band 100 % visible	1
2	– **Calculus** Supra- and subgingival – Iatrogenic marginal irritations	– Bleeding – **Calculus** – Band 100 % visible	2
3	– **Pockets** Shallow, up to 5 mm	– **Band only partially visible** Probing depths 3.5–5.5 mm	3
4	– **Pockets** Deeper, ≥ 6 mm	– **Band no longer visible** Probing depths greater than 6 mm	4

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Periodontal Screening and Recording—PSR

The PSR is a modified CPITN, developed by the American Academy of Periodontology (AAP 1992) and the American Dental Association (ADA 1992). As with the CPITN, taking the PSR is a relatively quick procedure, not requiring extensive forms to be filled out (no "paper war"!). This index serves for early detection of periodontitis.

The index reveals for the practitioner the current state of the gingiva (bleeding) as well as previously occurring pathologic processes in the form of pocket depth and the associated attachment loss. Further, the PSR provides indication as to whether additional, more-detailed examinations are necessary: If a code 3 or 4 is diagnosed on any tooth surface, a complete periodontal examination must be performed and a panoramic radiograph or a full-mouth radiographic series taken.

The PSR assists the general practitioner in the decision concerning whether a patient should be referred to a specialist for more complex periodontal therapy.







139 Clinical Definition of Codes 0–4

0 Probing elicits no bleeding; healthy
1 BOP (plaque, but no calculus)
2 The probe encounters supra- and subgingival calculus; bleeding
3 Probing depths between 3.5 and 5.5 mm, i. e., the black band remains at least partially visible
4 Probing depths 6 mm or more, the black band disappears subgingivally (bleeding, plaque, calculus)

CPITN – Treatment need	
0	– Home care
I	– OHI (oral hygiene instruction)
II	I + calculus removal and scaling
III	I + II + complex therapy

PSR – Treatment need	
– Preventive treatment only	0
– OHI – Plaque and debris removal	1
– OHI – Subgingival plaque and calculus removal	2
– As in 2 + Complete periodontal exam and radiographs ⇨ Possible referral to specialist	3
– As in 2 and 3 + More extensive treatment, possibly surgery ⇨ Refer to specialist	4

140 CPITN and PSR—Treatment Needs

In contrast to the "codes," the treatment needs deriving from them diverge somewhat:

- In contrast to the CPITN, in the PSR even with healthy gingiva (**Code 0**) preventive measures are advised. (Oral hygiene instruction = primary prophylaxis)
- With **Code 1**, professional plaque removal (PSR) should be performed in addition to the patient's own oral hygiene (CPITN).
- At **Code 2**, both indices recommend supra- and subgingival plaque and calculus removal.
- At **Code 3**, both the diagnosis and the therapy are somewhat extended: complete periodontal charting, panoramic radiography or full-mouth series.
- The same holds for **Code 4**. The suggestion "refer to a specialist," is used in the PSR mostly as an aid in the general practice.

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Epidemiology

Descriptive epidemiology deals with the occurrence, the severity and the distribution of diseases, as well as physical and/or mental incapacity, or mortality, in any selected population.
Analytical epidemiology seeks furthermore to discern the cause(s) of a disease. During the comprehensive examination of patients with periodontitis, in addition to the primary etiologic agent—plaque biofilm—other important factors such as heredity, socioeconomic status, behavior patterns, systemic diseases, risk factors as well as ethnic origin should also be ascertained as closely as possible. From this data, one can derive the likely consequences of prophylaxis and treatment, also from a public health standpoint (Albandar & Rams 2002).
In the field of periodontology, epidemiologists deal primarily with the dissemination of and the etiologic factors for gingivitis and periodontitis.
Not all results of the classic epidemiologic studies of the previous decades enjoy unquestioned acceptance today, however. The early studies did not consider all etiologic factors (see above), nor the varying forms of the disease, symptoms of activity and localization of the disease process. Furthermore, many studies did not draw conclusions concerning the treatment needs of the populations under study (AAP 1996).

Epidemiology of Gingivitis

Numerous epidemiologic studies have been performed worldwide, especially in children and adolescents. Their results reveal enormous differences. The morbidity rates (percent affected in the studied population) range from about 50 to almost 100 % (Stamm 1986, Schürch et al. 1991, Oliver et al. 1998).
Further, the reported degrees of severity of gingivitis vary greatly among the studies. These differences can be explained primarily by the use of non-standardized examination methods (various indices) and the constantly changing classifications of the diseases themselves. Other etiologic considerations that could explain the large differences include the quite varied status of prevention (plaque control) in the population groups studied, as well as geographic, social and ethnologic factors.
The incidence and severity of gingivitis may even vary in the same patient group with short-term repeated examinations (Suomi et al. 1971, Page 1986). In addition, the degree of severity of gingivitis over the course of one's life can vary enormously: It achieves its maximum in adolescents reaching puberty, then recedes somewhat, exhibiting a slight tendency to increase in adults as age increases (Stamm 1986, Fig. 178 right).
The existence of gingivitis cannot be taken as evidence that periodontitis will eventually develop (Listgarten et al. 1985, Schürch et al. 1991). The *public health significance* of gingivitis epidemiology may therefore be called into question.
In studies in which both gingivitis and plaque were considered, a clear positive correlation between oral hygiene and severity of gingivitis emerged (Silness & Löe 1964, Koivuniemi et al. 1980, Hefti et al. 1981).

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Epidemiology of Periodontitis

Many new epidemiologic studies of periodontitis have been published from many countries (Ahrens & Bublitz 1987, Fig. 142; Miller at al. 1987; Miyazaki et al. 1991a, b, Fig. 143; Brown & Löe 1993, Fig. 141; Papapanou 1994, 1996; AAP 1996; Oliver et al. 1998). As was the case with gingivitis studies, the results must be carefully interpreted. It is very difficult to compare the results of various studies when different parameters and different measurement techniques were employed without calibration. Up until now, epidemiologic studies, especially of elderly patients, have not even considered the causes of tooth loss all the way to edentulousness (caused by periodontitis?).

Furthermore very little attention has been given to fact that the measured parameters are applicable only to individual sites around individual teeth, and cannot be taken as a generalization, for example as an indication of the loss of tooth-supporting tissues in the entire dentition.

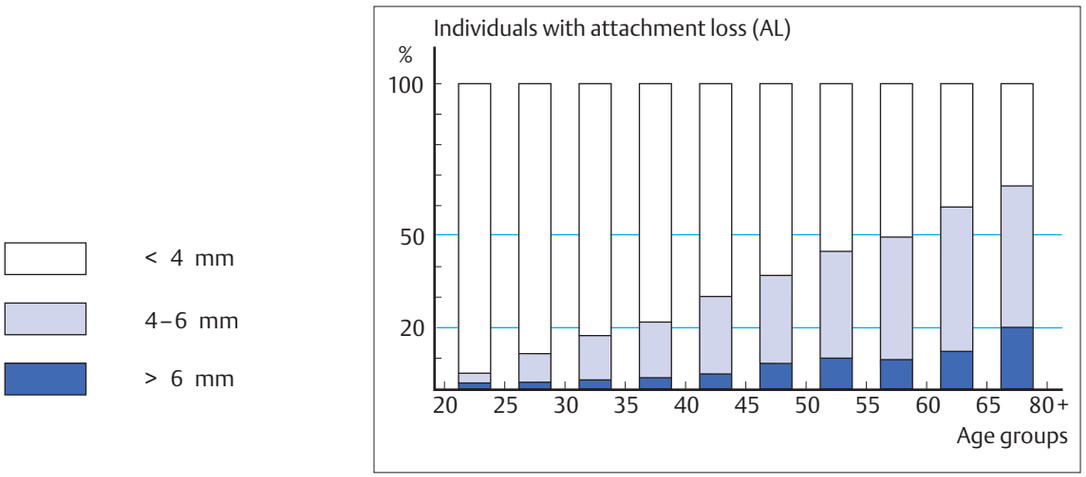
Most epidemiologic studies are really only "momentary" studies of the extent of disease (mean values).

Only Löe et al. (1986) studied the course of attachment loss over many years *longitudinally*, in a group of Norwegian students and academics on the one hand and tea plantation workers in Sri Lanka on the other hand. They also compared the ethnic and socioeconomic differences between these two very different population groups. The results demonstrated that in the Norwegian group the mean attachment loss in the entire dentition was 0.1 mm per year, while this figure was 0.2–0.3 mm in those subjects examined in Sri Lanka! The molars were most often affected in both groups.

Forms of Periodontitis

Epidemiologic studies rarely differentiate between the rare, early forms of the disease which can progress very rapidly even in young adults (aggressive periodontitis), and the more widespread, usually slowly-progressing chronic periodontitis. True aggressive forms are probably quite rare (2–5 % of all cases) in Europe and the USA.

More precise figures are available concerning aggressive, localized periodontitis (formerly: LJP, p. 118): In Europe, about 0.1 % of young people are affected, while in Asia and Africa high morbidity rates up to 5 % have been reported (Saxen 1980; Saxby 1984, 1987; Kronauer et al. 1986).



141 Attachment Loss in 15,000 Employed Individuals in Various Age Groups in the USA
After recording attachment loss (distance from the cementoenamel junction to the bottom of the pocket) approximately 30 % of all subjects exhibited 4–6 mm of attachment loss, while only 7.5 % had more than 6 mm of attachment loss.
In the same study, pocket probing depths were measured, and were distinctly less than the attachment loss figures, because probing does not evaluate recession.

Attachment Loss—Studies in the USA

Miller et al. (1987) and Brown & Löe (1993) examined over 15,000 employed persons in the USA, ranging in age from 18 to 80 years. In addition to other parameters, most important in the studies was the measurement of attachment loss. Approximately 76 % of all subjects exhibited attachment loss 2 mm or more, but only 7.6 % had attachment loss of more than 6 mm. Both studies showed that loss of tooth-supporting tissues increased with age, but contended that periodontitis (and recession) cannot be termed a "disease of the elderly."

CPITN Studies

In recent years, the CPITN was used most frequently the world over for epidemiologic studies. During the examination of 11,305 subjects in Hamburg (Fig. 142), the use of this index revealed that only 2.8 % were totally periodontally healthy (Code 0) and required no treatment. Nine percent exhibited bleeding on probing (Code 1) and 44 % had pocket probing depths up to 5.5 mm (Code 3). These patients required supra- and more importantly subgingival scaling, which could be performed by qualified auxiliary personnel (dental hygienist). Only in 16 % of the subjects were probing depths of 6 mm and greater detected (Code 4). These patients

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required additional complex therapy beyond simple scaling (root planing, surgical procedures) by the dentist. Severe periodontitis (Code 4) increased with age; mild periodontitis was correspondingly less often recorded in elderly patients.

WHO Studies

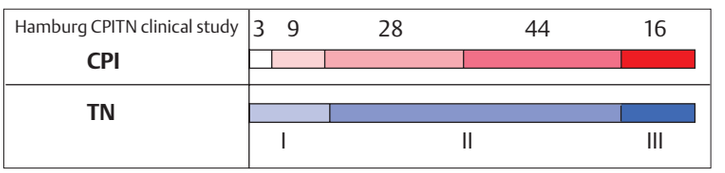
In a literature review of numerous investigations from Europe, the USA and Latin America, Miyazaki et al. (1991 a, b) encountered inconsistent results. Despite significant differences among the various countries, severe forms of periodontitis (CPITN Code 4) were observed at a level of only 10–15 %.

One overall conclusion can be drawn: In Europe, the USA and Latin America, gingivitis and mild periodontitis are quite common. Profound manifestations of attachment loss are only observed in ca. 10–15 % of these populations.

It is important to note, however, that scored codes or millimeter measurements do not mean that periodontitis is generalized throughout the mouth: A patient is classified as "affected" even if only a single tooth surface of the entire dentition has a single pocket depth measurement of 6 mm, corresponding to Code 4! This fact relegates the published "10–15 %" affected rate to a somewhat lower level.

Periodontitis is likely more widely distributed in Asia and Africa compared to the populations described here.

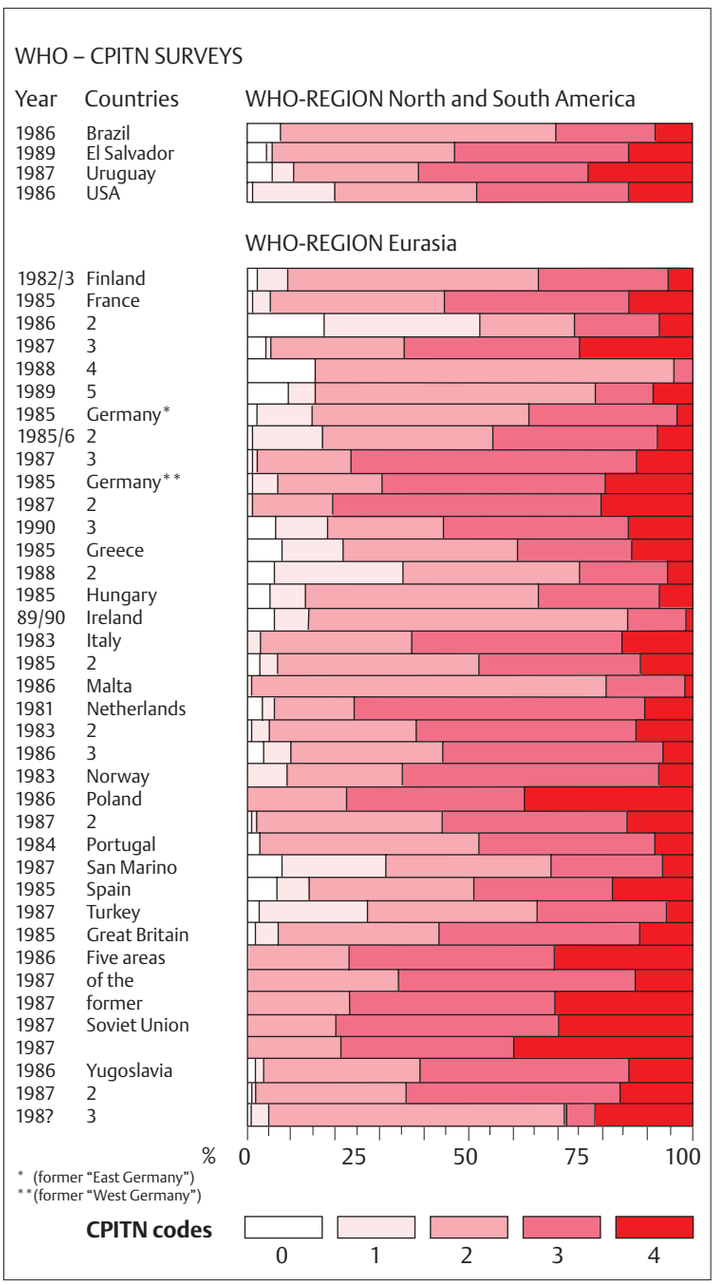
142 CPITN Study of 11,305 Subjects in Hamburg, Germany
Percentage distribution of the degrees of severity (Codes 0–4) for periodontal diseases (above) and the treatment needs and types of therapy that derive from the data (TN I-III, below).



143 Forty-two (!) CPITN Studies from America and Europe
The large differences between the clinical studies in various countries are clearly obvious. But even sequential epidemiologic studies in a single country, e. g., in France or in Germany, exhibit enormous differences that can hardly be explained by true differences in the severity of the disease. One must therefore assume that the individual degrees of severity as recorded by the CPITN were interpreted quite differently by the various examiners.

Despite these limitations, this summary of numerous studies can be viewed positively, because severe periodontitis (Code 4) and "treatment need" III (= complex therapy) were diagnosed in "only" ca. 10–15 % of all subjects.

One must consider, however, that a single patient, e. g., with a "Code 4," that may be present only in a single quadrant or on a single tooth or a single site on a tooth, does not indicate that complex therapy (TN III) should be performed on a single tooth nor in the entire dentition. Such information does indicate that such a patient is likely more susceptible to periodontitis.



Types of Plaque-associated Periodontal Diseases

Gingivitis—Periodontitis

The general term "periodontal diseases" encompasses inflammatory as well as recessive alterations within the gingiva and the periodontium (Page & Schroeder 1982; AAP 1989, 1996; Ranney 1992, 1993; Lindhe et al. 1997; Armitage 1999).

While gingival recession may have various etiologies, including morphologic, mechanical (improper oral hygiene) and even functional (p. 155), gingivitis and periodontitis are plaque-associated diseases. Today it is becoming more and more clear that bacteria alone, including even the so-called periodontopathic bacteria, can always elicit gingivitis, but *not* periodontitis in every case. The initiation of periodontitis, its speed of progress and the expression of its clinical picture also involve the responsibility of negative host factors and additional so-called risk factors (Clark & Hirsch 1995). One of the most important are defects of the acute host response resulting from functional disturbances of polymorphonuclear granulocytes (PMN), other insufficient immunologic reactions, and the predominance of pro-inflammatory mediators, many of which are genetically determined. The now well established alterable risk factors include, particularly, "unhealthy" habits such as smoking, alcohol consumption and a less than well-rounded diet (p. 22).

Furthermore, systemic disorders and syndromes are usually *un*alterable, obligate or facultative risk factors for periodontitis. In this regard, especially Diabetes mellitus is viewed as prejudicial (p. 132). Finally, the entire social environment of an individual can play an important role for the initiation and propagation of periodontitis (pp. 22, 51).

All of the factors discussed in the chapter "Etiology and Pathogenesis" (p. 21) enhance the occurrence of all diseases, including periodontitis. Thus, periodontitis is considered to be of multifactorial etiology. It remains difficult in the face of a manifest disease process to differentiate the "etiologic weight" of bacteria on the one hand and the host response and risk factors on the other hand, or to differentiate between and among such etiologic factors. The contemporary, on-going search for responsible "risk markers" is therefore very important.

Today, clinicians can use additional clinical parameters such as bleeding on probing, the severity of the disease in relation to patient age, as well as microbiologic and genetic tests in order to establish some semblance of a proper diagnosis and prognosis (p. 165).

In the future, more precise research into the reduced host defense occasioned by immunologic defects, as well as the type and quantity of participating cytokines and inflammatory mediators (e. g., in sulcus fluid, blood or saliva) will simplify diagnosis and permit more definitive predictions about the probable course of the disease, as well as indicating more targeted therapy.

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Classification of Periodontal Diseases—Nomenclature

Most recent research results and clinical knowledge have led the nomenclature of the periodontal diseases into a state of constant change. Indeed, because of varying interpretations of scientific results, it has often led to conflict between authors and scientific societies.

Relatively recently, the new nomenclature from the *American Academy of Periodontology* (AAP, 1989) was generally accepted. This classification distinguished between gingivitis (G) and adult periodontitis (AP) of the following types: Early-onset periodontitis (EOP) with further subclassifications (PP, JP, RPP), periodontitis associated with systemic diseases (PSD), the acute necrotizing, ulcerative gingivoperiodontitis (ANUG/P), and the therapy-resistant, refractory periodontitis (RP).

After only a few years, however, this "new" classification was no longer satisfactory! The *European Federation of Periodontology* (EFP) proposed in 1993 a new classification, and even *this* was modified in 1999/2000 during an international workshop in collaboration with the AAP (Armitage 1999).

The "old" classification from 1989 was criticized because it placed too much weight upon the age of the patient vis-à-vis the initiation of the disease process. For example "adult periodontitis" (AP) as a chronic disease could also be observed in adolescents; rapidly-progressing periodontitis (RPP) was observed not only in young individuals (EOP) but also could appear "suddenly" in older patients; the localized, juvenile form (LJP) was not observed *only* in young patients. Furthermore, "refractory periodontitis" (RP) cannot be viewed as a specific disease entity, because following treatment for periodontitis the disease might recur or the disease might simply not respond to the therapy.

Classification 1999/2000

This third edition of the *Color Atlas of Periodontology* consistently utilizes the most recent nomenclature (1999/2000), but several "old" and well-known terms are used for occasional clarification, e. g., LJP, which is now classified as "Type III, aggressive periodontitis A, localized."

The table below is a brief summary of the new nomenclature to assist the dentist. The complete and extremely comprehensive "Classification of Periodontal Diseases and Conditions" can be found in the appendix ("Classifications," p. 519). Some view the new classification as too expansive and comprehensive, while others criticize it as incomplete!

Classification of Periodontal Diseases (International Workshop of the AAP/EFP, 1999)

Type I	**Gingival Diseases**
	A Plaque-Induced Gingival Diseases
	B Non-Plaque-Induced Gingival Lesions
Type II	**Chronic Periodontitis**
	A Localized
	B Generalized
Type III	**Aggressive Periodontitis**
	A Localized
	B Generalized
Type IV	**Periodontitis as a Manifestation of Systemic Disease**
	A Associated with Hematological Disorders
	B Associated with Genetic Disorders
	C Not Otherwise Specified (NOS)
Type V	**Necrotizing Periodontal Disease**
	A Necrotizing Ulcerative Gingivitis (NUG)
	B Necrotizing Ulcerative Periodontitis (NUP)

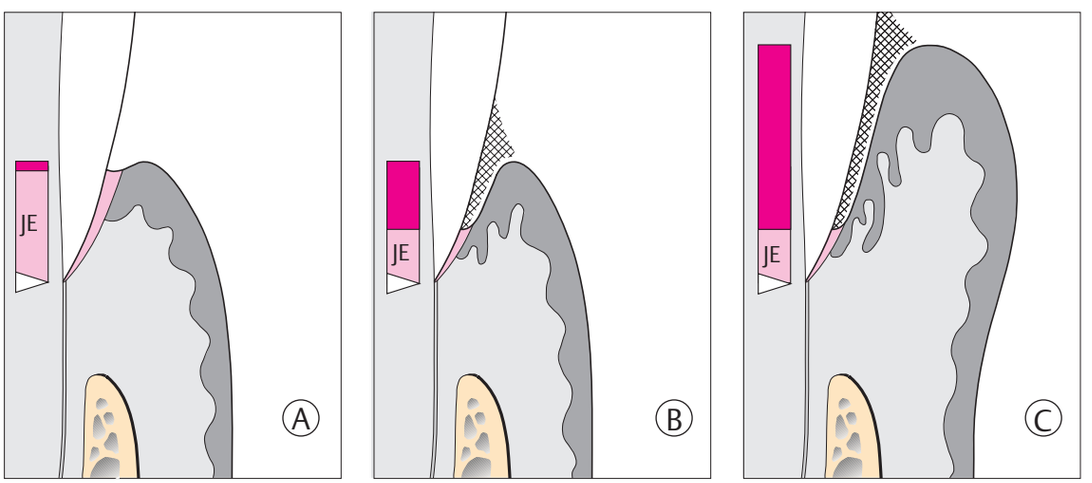
Types VI-VIII Further forms as well as transitions and "status pictures" of the diseases described, as well as the strengths and weaknesses of the new classifications are described in details (pp. 519–522) and in the original publications.

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Gingivitis

Plaque-induced Gingivitis, Gingivitis Simplex, Type I A 1

Gingivitis is ubiquitous. It is a bacterially-elicited inflammation (non-specific mixed infection) of the marginal gingiva (Löe et al. 1965).
In the chapter "Etiology and Pathogenesis," the development and progression of gingivitis from healthy tissue to an early lesion and on to established gingivitis was described (pp. 56–59; Page & Schroeder 1976, Kornman et al. 1997). In children, the early lesion (gingivitis) with T-cell dominance can persist for many years; on the other hand, in adults, the persistent lesion is almost exclusively *established* gingivitis (with plasma cell dominance), which can assume quite variable clinical severity. It is possible, clinically and pathomorphologically, to differentiate gingivitis into mild, moderate and severe, although this classification is relatively subjective.
For a more precise diagnosis of gingival inflammation, it is prudent to employ accepted indices that quantitate bleeding upon probing of the gingival sulcus.



144 Sulcus and Gingival Pocket

A **Sulcus:** Histologically, in healthy gingiva the sulcus is maximally 0.5 mm deep. However, upon probing, the probe may penetrate the junctional epithelium up to 2 mm.
B **Gingival Pocket:** In gingivitis, the coronal portion of the junctional epithelium detaches from the tooth. There is no true attachment loss.
C **Pseudopocket:** With swelling of the gingiva, a pseudopocket may develop.

The borderline between healthy gingiva and gingivitis is difficult to ascertain. Gingiva that appears to be clinically healthy will nevertheless almost always exhibit *histologically* a mild inflammatory infiltrate. If there is an increase in clinical and histologic inflammation, one observes lateral proliferation of the junctional epithelium. The JE becomes detached from the tooth at its marginal aspect, as the bacterial front progresses between the tooth surface and the epithelium. The result is a *gingival pocke*t.

In cases of severe gingivitis with edematous swelling and hyperplasia of the tissues, a clinical pseudopocket often forms.

Gingival pockets and pseudopockets are not *true* periodontal pockets because no connective tissue attachment loss has occurred, nor has there been *apical* proliferation of the junctional epithelium. However, because the milieu of pseudopockets is an oxygen-poor environment, periodontopathic anaerobic microorganisms can thrive.

It is true that gingivitis may develop into periodontitis. However, even without treatment, gingivitis can persist over many years in a stationary manner, exhibiting only minor variations in intensity (Listgarten et al. 1985). With treatment, gingivitis is fully reversible.

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Histopathology

The clinical and histopathologic pictures of established gingivitis are well correlated (Engelberger et al. 1983).

The mild infiltration that occurs even in clinically healthy gingiva may be explained as a host response to the small amount of plaque that is present even in a clean dentition. Such plaque is composed of non-pathogenic or mildly pathogenic microorganisms, primarily gram-positive cocci and rods.

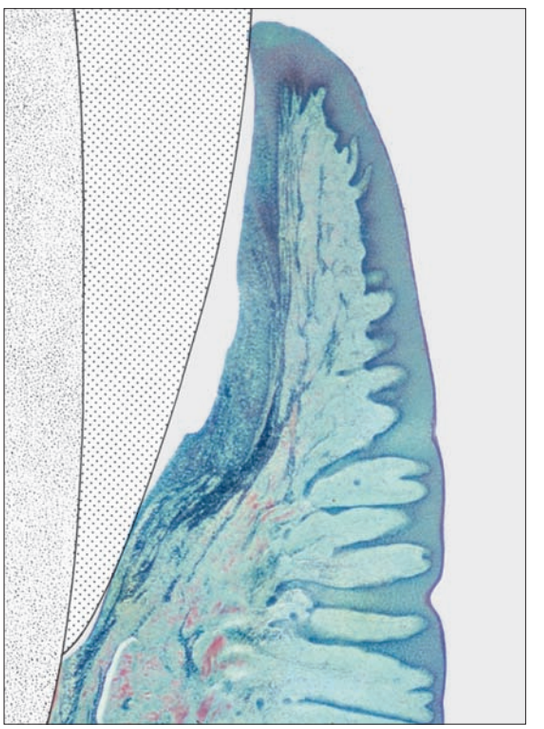
As plaque accumulation increases, so does the severity of *clinically* detectable inflammation, as evidenced by the quantity and expanse of inflammatory infiltrate. The subepithelial infiltrate consists primarily of differentiated B-lymphocytes (plasma cells), with a smaller number of other types of leukocytes.

With increasing inflammation, more and more PMN granulocytes transmigrate the junctional epithelium. As a consequence, the junctional epithelium takes on the characteristics of a pocket epithelium (p. 104, Fig. 209; Müller-Glauser & Schroeder 1982), but without any significant apical proliferation.

145 Healthy Gingiva (left)
Even in clinically healthy gingiva (GI = 0, PBI = 0), one can observe a very discrete sub-epithelial inflammatory infiltrate. Scattered PMNs transmigrate the junctional epithelium, which remains for the most part intact (HE, x10).

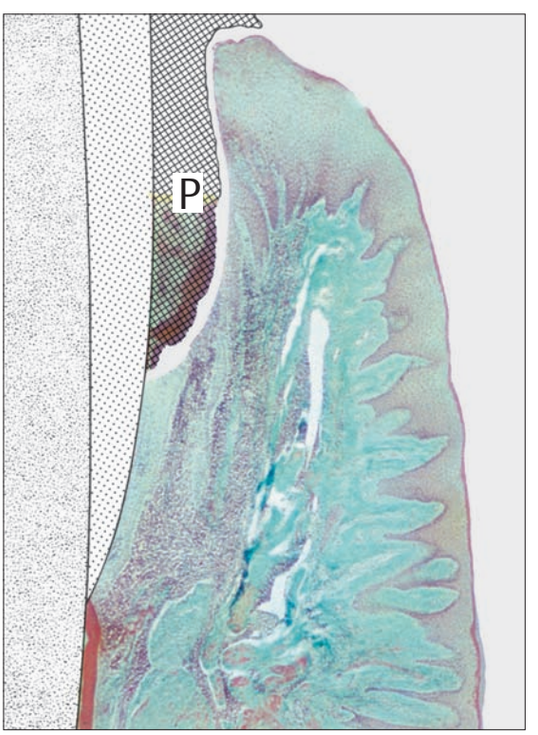
146 Mild Gingivitis (right)
As clinical inflammation increases (GI = 1, PBI = 1) the amount of inflammatory infiltrate increases and collagen is lost (Masson stain, x 10).

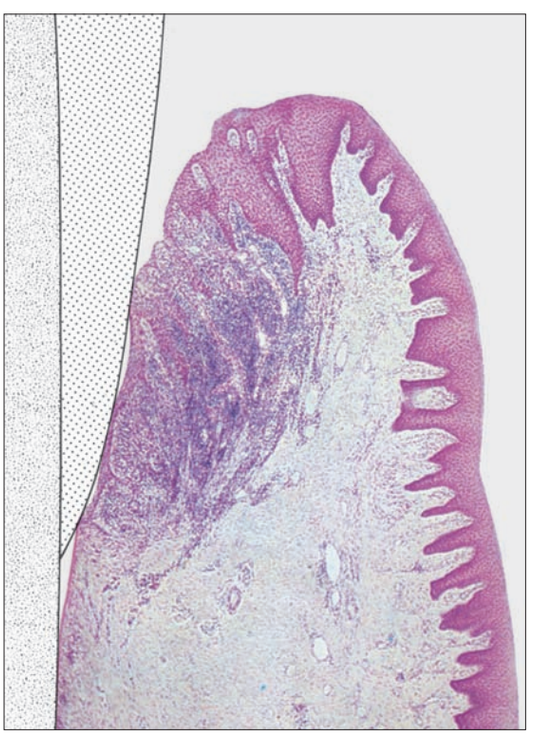




147 Moderate Gingivitis (left)
When gingivitis is clinically apparent (GI = 2, PBI = 2), the infiltrate becomes more dense and expansive. Collagen loss continues. The junctional epithelium proliferates laterally; a gingival pocket develops. **P** = subgingival plaque. (Masson stain, x 10)

148 Severe Gingivitis (right)
Pronounced edematous swelling (GI = 3, PBI = 3–4). The inflammatory infiltrate is expansive; collagen loss is pronounced. The junctional epithelium is transformed into a pocket epithelium (gingival pocket). Only in the apicalmost area are any remnants of intact junctional epithelium observed. Apical to the JE, the connective tissue attachment is intact (no loss of attachment; HE, x10).





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Clinical Symptoms

- Bleeding
- Erythema
- Edematous and hyperplastic swelling
- Ulceration

The earliest clinical symptom of an established lesion is *bleeding* subsequent to careful sulcus probing. This hemorrhage is elicited by the penetration of the probe tip through the disintegrating junctional epithelium and into the highly vascular sub-epithelial connective tissue. At this stage of the inflammatory process (PBI = 1), *no gingival erythema* may be visible clinically. Clinical symptoms of advanced (established) gingivitis include profuse bleeding after sulcus probing, obvious *erythema* and simultaneous *edematous swelling*. In the most severe cases, spontaneous bleeding and eventually *ulceration* may occur. The *chronic* types (and severities) do not elicit pain; pain occurs only in *acute* gingivitis (e.g., NUG, p. 85).

Even severe gingivitis may *never* progress to periodontitis. With proper treatment, gingivitis is reversible.





149 Healthy Gingiva (left)
The gingiva is pale salmon-pink in color, and stippled. The narrow free gingival margin is distinguishable from the attached gingival. After gentle probing with a blunt periodontal probe, no bleeding occurs.

150 Mild Gingivitis (right)
The localized erythema is scarcely visible, and one observes slight edematous swelling. Some of the former stippling is lost, and there is minimal bleeding upon probing.





151 Moderate Gingivitis (left)
Obvious erythema and edematous swelling. No stippling is apparent, and there is hemorrhage following probing of the sulcus.

152 Severe Gingivitis (right)
Fiery redness, edematous and hyperplastic swelling; complete absence of any stippling; interdental ulceration, copious bleeding on probing, and spontaneous hemorrhage.

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Mild Gingivitis

A 23-year-old female came to the dentist for a routine check-up. She had no complaints and was not aware of any gingival problems, although in her medical history she indicated that her gingiva bled occasionally during tooth brushing. Her oral hygiene was relatively good. She had received tooth brushing instructions from a dentist once, but no subsequent OHI. The patient was not on a regular recall schedule. Calculus removal had been performed sporadically in the past during routine dental check-ups, and several restorations placed.

Findings:

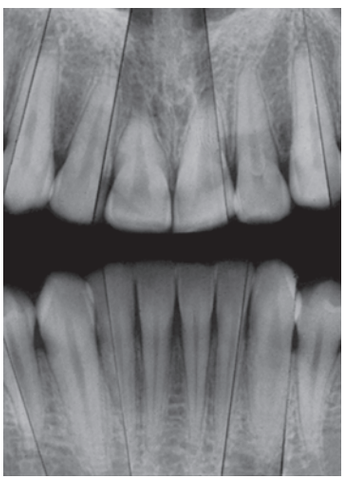
API	(Approximal Plaque Index): 30 %
PBI	(Papilla Bleeding Index): 1.5
PD	(Probing depths): ca. 1.5 mm maxilla, ca. 3 mm mandible
TM	(tooth mobility): 0

Diagnosis: Gingivitis in initial stage
Therapy: Motivation, OHI, plaque and calculus removal
Recall: Prophylaxis at 6-month intervals
Prognosis: Very good

153 Mild Gingivitis in the Anterior Area
In the maxilla, one observes no overt signs of gingivitis, except for a mild erythema.
In the mandible, especially in the papillary areas, slight edematous swelling and erythema can be detected (arrows).

Right: Radiographically there is no evidence of loss of interdental bone height. The maxillary central incisors exhibit short roots.





154 Papilla Bleeding Index (PBI)
After gentle probing of the sulci with a blunt periodontal probe, hemorrhage of grades 1 and 2 occurs. This is a cardinal sign of gingivitis.

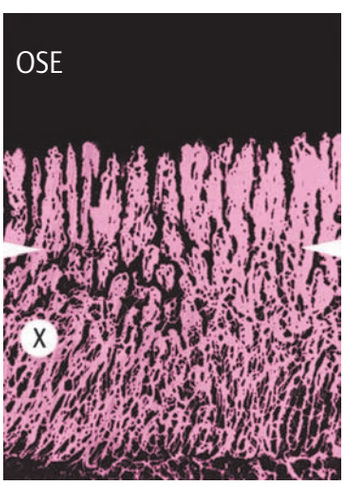


155 Stained Plaque
Around the necks of the teeth and in interdental areas, small plaque accumulations are visible.

Right: Gingival vascular plexus (**X**) in the region of the junctional epithelium in a case of mild gingivitis. Above the white arrows, one observes the most marginal vascular loops in the area of the adjacent oral sulcular epithelium (**OSE**; canine preparation).

Courtesy *J. Egelberg*



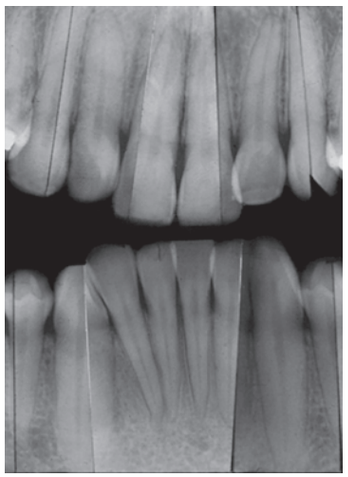


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Moderate Gingivitis

A 28-year-old female presented with a chief complaint of gingival bleeding. She "brushes her teeth," but had never received any oral hygiene instruction from a dentist or hygienist. Calculus had been removed only infrequently, and a professional debridement had never been systematically performed. Generalized crowding of the teeth in both arches is evident, combined with an anterior open bite. These anomalies reduced any self-cleansing effects and made oral hygiene difficult. This also likely increased the severity of gingivitis.

Findings:
API: 50 % PD: ca. 3 mm maxilla, ca. 4 mm mandible
PBI: maxilla 2.6, mandible 3.4. TM: 0 maxilla, 1 mandible
Diagnosis: Maxilla, moderate gingivitis; mandibular anterior region, severe gingivitis with pseudopockets.
Therapy: Motivation, oral hygiene, plaque and calculus removal; after re-evaluation, possible gingivoplasty.
Recall: Every six months initially.
Prognosis: With patient cooperation and compliance, very good.





156 Moderate Gingivitis in Anterior Segments
Erythema and swelling of the gingiva. The symptoms are more pronounced in the mandible than in the maxilla.

Left: Radiographically there is no evidence of destruction (demineralization) of the interdental bony septa.



157 Papilla Bleeding Index (PBI)
The pronounced gingivitis that is particularly obvious in the mandibular anterior area is corroborated by the PBI. Bleeding scores of 2 and 3 are recorded after "sweeping" the sulcus with a periodontal probe in the papillary regions.





158 Stained Plaque
Moderate plaque accumulation in the maxilla. In the mandible, heavier plaque, especially at the gingival margins.

Left: Vascular plexus of the gingiva near the junctional epithelium in a case of severe gingivitis (Fig. 155, right)

Courtesy *J. Egelberg*

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Severe Gingivitis

A 15-year-old male was referred for evaluation and treatment of suspected juvenile periodontitis (LJP/Type III A). The extremely pronounced gingivitis was, however, inconsistent with this diagnosis. Sulcus probing and radiographic examination revealed no attachment loss on anterior teeth or molars.

The patient practiced virtually no oral hygiene, stating that it was impossible to brush his teeth because the gingiva bled at the slightest touch. He had never received adequate motivation, nor any oral hygiene instruction, nor any treatment for his gingivitis.

Findings:

API: 88 %
PBI: 3.5
PD: pseudopockets to 5 mm
TM: 0

Diagnosis: Severe gingivitis with edematous hyperplastic enlargement of the facial aspect of the anterior area; mouth breathing as possible etiologic co-factor (?).

Therapy: Motivation, oral hygiene instruction, definitive debridement. After re-evaluation, possible gingivoplasty.

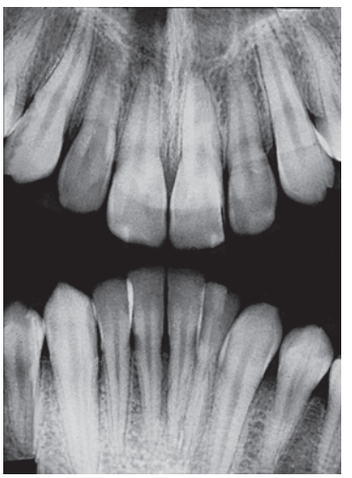
Recall: Initially every three months.

Prognosis: With patient cooperation and compliance, good.

159 Severe Gingivitis
The clinical symptoms of severe gingivitis including erythema, edema and hyperplastic enlargement, are observed. The anterior region is more severely affected (slight crowding, mouth breathing?). Probing reveals no attachment loss; the base of the pseudopockets are not apical to the cementoenamel junction.

Right: Radiographically, one observes no evidence of bone loss on the interdental septa.





160 Papilla Bleeding Index (PBI)
Copious bleeding (PBI grade 4) occurs after sweeping the anterior sextant pseudopockets with a blunt periodontal probe. The inflammation is less pronounced in the premolar and molar regions.

If gingival hyperplasia is severe, the clinician must exclude other possible etiologic factors such as medicament-induced lesions and systemic disorders.



161 Stained Plaque
Moderately heavy accumulation of supragingival plaque. Not visible is the expanse of the subgingival plaque within pseudopockets. The pronounced inflammation, especially in the anterior region, anticipates significant plaque accumulation subgingivally.

Right: The papilla between 21 and 22 is grossly enlarged, erythematous and devoid of any stippling.





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Ulcerative Gingivitis/Periodontitis

NUG/P = Necrotizing Ulcerative Gingivitis/Periodontitis, Type V A (NUG) and V B (NUP)

Ulcerative gingivitis is usually an acute, painful, rapidly progressing inflammation of the gingiva, which may enter a sub-acute or chronic stage. Without treatment, this disease usually develops quickly into localized ulcerative periodontitis. It seldom occurs as a generalized process, nor is its severity always identical. It may be quite advanced in anterior segments, while the premolars or molars are not affected at all, or only mildly so. The reasons for this remain unknown (oral hygiene? ischemia? locally predominating pathogenic bacteria? plaque-retentive areas? tooth type?). Probing depths are usually shallow because gingival tissue is lost to necrosis as attachment loss proceeds. Secondary ulceration of other oral mucosal surfaces is rarely observed, and only in severe cases (AAP 1996e). Caution: Ulceration can represent an early oral symptom in HIV-positive patients and AIDS victims (p. 151).

Ulcerative gingivitis/periodontitis has become less common in recent decades than earlier (exception: HIV-positive and AIDS patients). In the younger population, the *morbidity* has been variously reported between 0.1–1 %.

The *etiology* of NUG is not completely understood. In addition to plaque and a previously existing gingivitis, the following local and systemic predisposing factors are suspected:

Local Factors
- Poor oral hygiene
- Predominance of spirochetes, fusiforms and *P. intermedia*, and occasionally *Selenomonas* and *Porphyromonas* in the plaque
- Smoking (local irritation by tar products)

Systemic Factors
- Poor general health, psychic stress, alcohol
- Smoking: nicotine as a sympatheticomimetic, and carbon monoxide (CO) as a chemotaxin (p. 216)
- Age (15–30 years)
- Season of the year (September/October and December/January; Skâch et al. 1970)

NUG/P patients usually exhibit similar *life styles and habits*: The teeth do not occupy a high position in the patient's consciousness. They are usually young adults, heavy smokers (tobacco: high content of tar and nicotine), exercise poor oral hygiene, and become interested in treatment only during acute, painful exacerbations.

The *clinical course* is acute, but fever occurs only seldom. Within only a few days, interdental papillae may be lost to ulceration. The acute phase may gravitate into a chronic interval stage if host resistance improves (see pre-disposing factors) or through self-treatment (rinsing with a disinfectant mouthwash). Untreated ulcerative gingivitis exhibits a high recurrence rate, and may develop rapidly into ulcerative periodontitis (attachment loss with shallow pockets!).

Therapy: In addition to local debridement, the early stages of treatment should be supported with medicaments. Topical application of ointments containing cortisone or antibiotics, or metronidazol may be effective. In severe cases, systemic metronidazol (e. g., Flagyl) may be prescribed (see Medicaments, p. 287). After reduction of the acute symptoms in advanced cases, surgery to correct gingival contours may be indicated.

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Histopathology

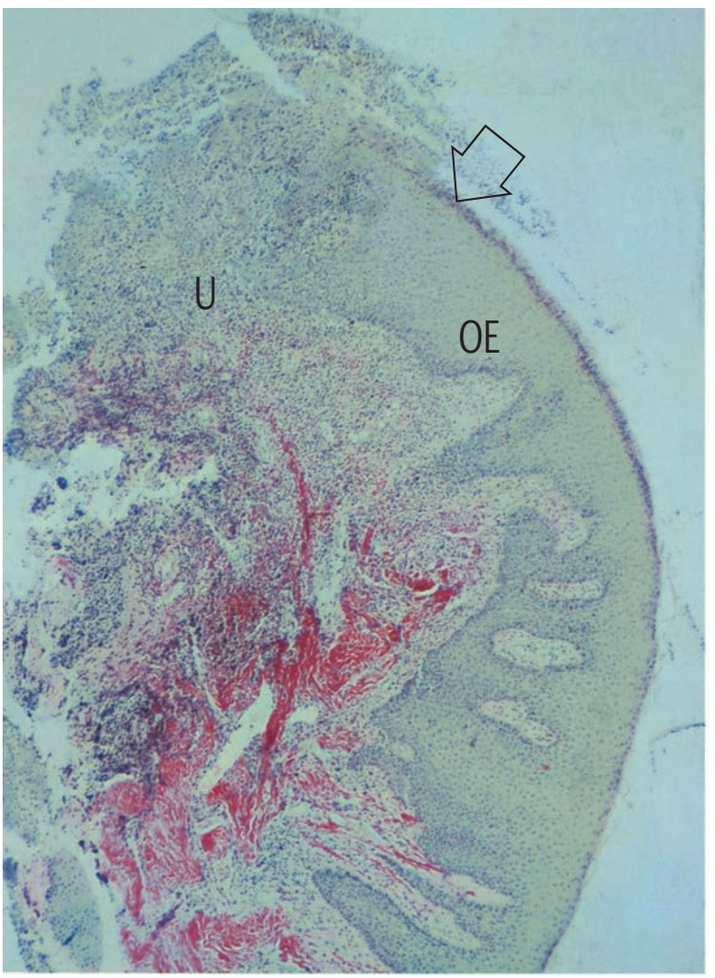
The clinical and histopathologic pictures in NUG are correlated. The histopathology of NUG is, however, significantly different from that of simple gingivitis.

As a consequence of the acute reaction, an enormous number of PMNs transmigrate the junctional epithelium in the direction of the sulcus and the col. In contrast to the situation in simple gingivitis, PMNs also migrate toward the oral epithelium and the tips of papillae, which undergo necrotic destruction. The ulcerated wound is covered by a clinically visible, whitish pseudomembrane that consists of bacteria, dead leukocytes and epithelial cells as well as fibrin. The tissue subjacent to the ulcerated areas is edematous, hyperemic and heavily infiltrated by PMNs. In long-standing disease, the deeper tissue regions will also contain lymphocytes and plasma cells. Within the infiltrated area, collagen destruction progress rapidly.

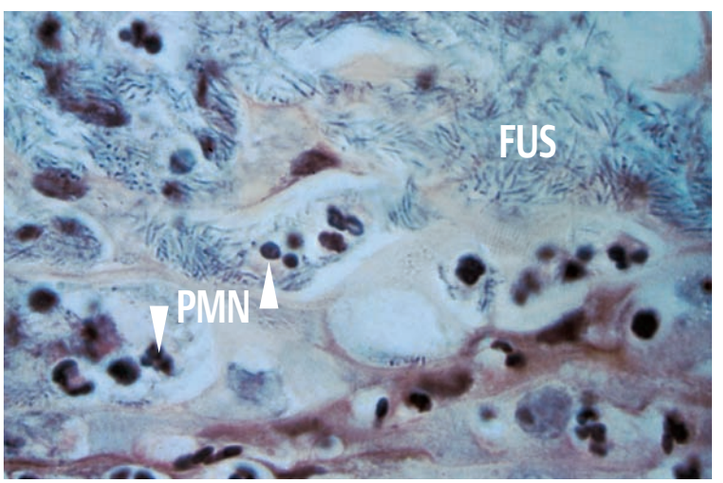
Spirochetes and other bacteria often penetrate into the damaged tissues (Listgarten 1965, Listgarten & Lewis 1967).

162 Papilla Biopsy
Affected papilla excised from a patient with mild ulcerative gingivitis, resembling the clinical situation in Fig. 164. The tip of the papilla and the tissue approaching the col have been destroyed by ulceration (**U**).
The oral epithelium (**OE**, stained yellow) remains essentially intact. In the deeper layers of the biopsy, one observes the red-stained intact collagen, while beneath the decimated papilla tip the collagen is largely obliterated (van Gieson, x10).

The arrow indicates the section that is enlarged in Fig. 163.



163 Surface of the Disintegrated Tissue
The upper portion of the figure exhibits thickly packed fusiform bacteria (**FUS**). Spirochetes present in the section are not visible with this staining technique, but the numerous **PMNs** are obvious. The brownish structures with weakly staining nuclei are dying epithelial cells (van Gieson, x1000).



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Clinical Symptoms—Bacteriology

- Necrotic destruction of the gingiva, ulceration
- Pain
- Halitosis
- Specific bacteria

Beginning in the tissues of the interdental col, there is *necrotic destruction* of papilla tips, followed by destruction of entire papillae and even portions of the marginal gingiva. It is not clear whether gingival destruction is caused primarily by vascular infarction or by invasion of bacteria into the tissues. In rare instances, depressed ulcerations may also be seen on the cheeks, lips or tongue. In the absence of treatment, the osseous portion of the periodontal supporting apparatus can also become involved. The earliest clinical symptom of ulcerative gingivitis is localized pain.

The patient with NUG is characterized by a typical, insipid, sweetish *halitosis*.

Generalized ulcerative gingivitis must be differentiated from acute *herpetic gingivostomatitis* (p. 131), which is generally accompanied by fever.





164 Earliest Symptom of NUG (left)
The most coronal portion of the papilla tip is destroyed by necrosis from the col outward. The defects are covered by a typical whitish pseudomembrane. The first pain may be experienced even before any ulcerations become visible clinically. NUG should be diagnosed and treated in this early, *reversible* stage!

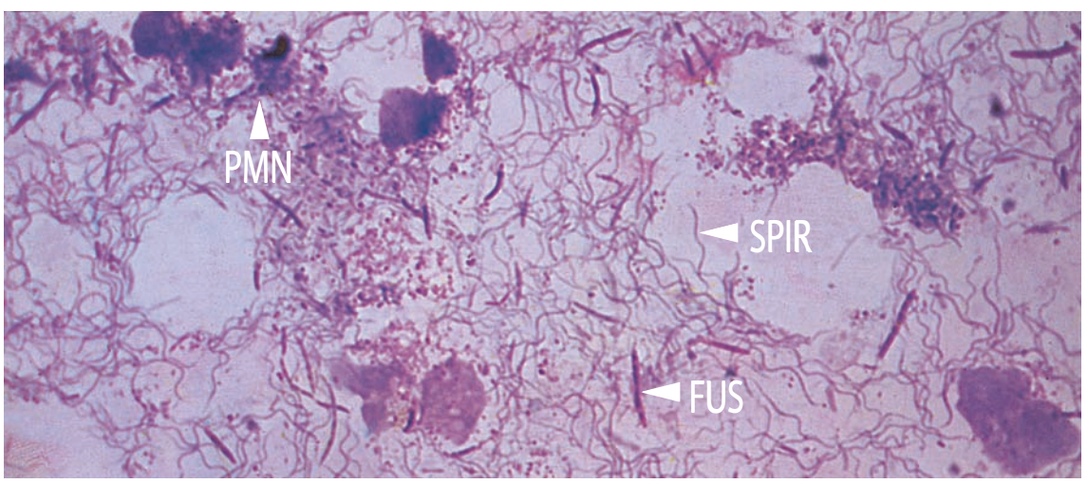
165 Advanced Stage of Ulceration (right)





166 Complete Destruction of the Papilla (left)
Between the premolars there is no visible lesion, but areas of early necrosis can be seen on the mesial and marginal aspects of the canine. Note the complete destruction of the papilla between canine and first premolar.

167 Acute Recurrence (right)
The papillae have been completely destroyed. The previous acute stage has led to reverse architecture of the gingival margin. Beginning ulcerative periodontitis.



168 Bacteriology—Smear of the Pseudomembrane
In addition to dead cells, granulocytes (**PMN**) and fusiform bacteria (**FUS**), enormous numbers of spirochetes (**SPIR**) are visible (van Gieson, x1000)

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Ulcerative Gingivitis (NUG)

A 19-year-old female experienced gingival pain and hemorrhage for three days.

Findings:

API:	70 %
PBI:	Anterior segments, 3.2
	Premolar and molar regions, 2.6
PD:	2–3 mm proximal
TM:	0–1

Diagnosis: Acute ulcerative gingivitis, early stage; NUG, Type V A.

Therapy: At the first appointment, careful mechanical debridement; topical medicaments: antibiotic or cortisone-containing ointment, metronidazol gel (e.g., Elyzol, p. 292). The patient may be instructed to rinse at home with mild hydrogen perborate solution.

Subsequent appointments: Motivation, repeated oral hygiene instruction, plaque and calculus removal.

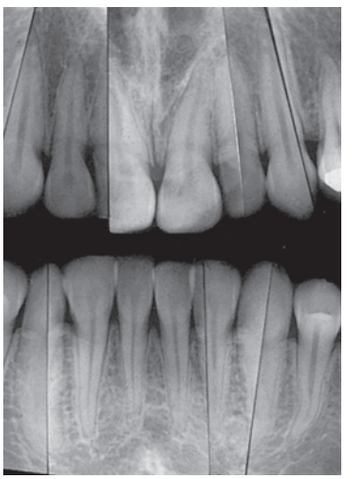
Recall: Short-interval (information! plaque control).

Prognosis: With treatment and *patient compliance*, good.

169 Initial Stage
Initial acute ulcerative destruction of several papilla tips (arrows). Other papillae exhibit signs of mild inflammation, but no destruction.

Right: Radiographically one observes no evidence of resorption of interdental septal bone.





170 Initial Destruction of Papilla Tips in the Maxilla
Necrosis of the papilla tips between central and lateral incisors is apparent, with erythema and swelling. Note the initial necrosis of the papilla tips between the lateral incisor and canine. Between the central incisors, one notes erythema of the papilla, but not yet any signs of necrosis. If the patient experiences pain when this area is probed gently, it is evidence that the necrotic process has already begun in the col area.



171 Destruction of Papilla Tips in the Mandible
Each papilla exhibits signs of incipient ulceration, and each is already covered by a pseudomembrane consisting of fibrin, dead tissue cells, leukocytes and bacteria.

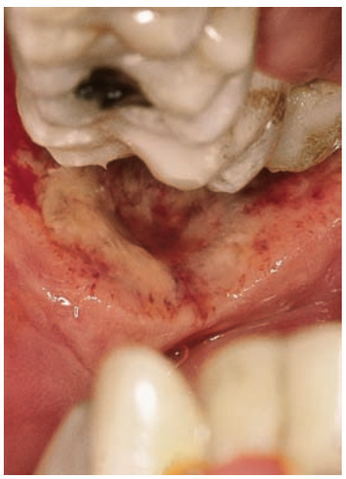


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Ulcerative Periodontitis (NUP)

Ulcerative periodontitis always develops—often very rapidly—from a preexisting ulcerative gingivitis. Probing depths may be shallow because the destruction of tissue occurs simultaneously with the attachment loss. While the clinical signs associated with NUG can be eliminated by proper treatment, ulcerative periodontitis (NUP) always leads to irreversible damage (attachment loss, bone loss). Acute and subacute phases occur cyclically. Acute phases are always accompanied by pain, in contrast to simple gingivitis. Ulcerative periodontitis is seldom generalized.

Therapy: Similar to the case with ulcerative gingivitis, in the acute phase of NUP careful and complete plaque and calculus removal should be performed. This mechanical therapy can be supported by topical medicaments. After the pain subsides, systematic instrumentation (debridement) follows. In advanced cases, minor surgical procedures may be necessary after all acute symptoms subside: In mild cases, gingivoplasty; in advanced cases, contouring flap surgery. A strict recall program is absolutely necessary, because NUP has a tendency toward recurrence.





Acute Stage

172 Ulcerative Gingivoperiodontitis—Second Acute Episode
This 26-year-old male experienced a second, pronounced acute episode. In addition to gingivitis, in the maxillary anterior and molar segments attachment loss has occurred. (Treatment: p. 90)

Left: Expansive, painful mucosal ulcer adjacent to tooth 18. Extraction of tooth 18 is indicated following the acute episode.





Interval Stage

173 Localized Ulcerative Periodontitis
This 22-year-old male had experienced two acute episodes, which were not treated professionally. In this *interval stage*, the patient is free of pain.
Left: Advanced but very localized attachment loss in the anterior segment of the mandible. The interdental crater presents a niche for plaque accumulation, which can lead to exacerbation if host response is compromised.





174 Expansive, Generalized Ulcerative Periodontitis
This 30-year-old male complained of gingival recession. He had experienced pain in recent years, but did not seek treatment.
The now pain-free lesions have progressed to various degrees; *interval stage (HIV-patient)*.

Left: In the anterior region of the mandible, especially between teeth 41 and 42, the attachment loss has progressed significantly.

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Ulcerative Gingivoperiodontitis—Therapy

A 26-year-old male (see also Fig. 172) who was systemically healthy, presented complaining of severe pain and "inflamed gingiva." A similar inflammation had occurred one year previously, but it resolved after the use of mouthwashes, without treatment by a dentist. The patient smoked ca. 40 cigarettes per day.

Findings:

API: 80% PBI: 3.2
PD: 3–5 mm TM: 0–1
Second acute phase (one year later)

Diagnosis: Generalized, acute ulcerative gingivoperiodontitis.

Therapy: In acute stages, careful supragingival cleaning, chlorhexidine rinses; "slow release" medicaments (e.g., metronidazol: Elyzol gel) may be used if pockets are present. After the acute symptoms subside, systematic supra- and subgingival plaque and calculus removal. Surgical intervention (gingivoplasty) was indicated in this case, but was not performed.

Recall: Not desired by the patient.

Prognosis: Questionable; patient non-compliance.





175 Ulcerative Gingivoperiodontitis in a 26-year-old patient
Note the gingival destruction, especially in the papillary regions of the anterior teeth, gingival swelling, bleeding on probing and spontaneous hemorrhage, with heavy plaque accumulation. One year previously, at about the same time of the year (December), identical symptoms had occurred.

Right: Detail of the gingival condition around tooth 22.





176 Clinical View Six Days Later
During two appointments (on day 1 and day 4), the teeth were carefully debrided (the supportive medicinal treatment is described in the text). The acute symptoms subsided to such a degree that a comprehensive scaling and root planing could be performed.

Right: Detail tooth 22.





177 Final Clinical Result
After repeated and systematic supra- and subgingival scaling (no surgical procedures) the NUG/NUP "healed" completely. The gingiva remains somewhat "puffy"; a gingivoplasty would be indicated. The patient "felt good" again, and refused any additional therapy, a reaction that is typical for such patients.

Right: Final clinical appearance; detail of tooth 22.

Hormonally Modulated Gingivitis

Types I A 2 a

Changes in the body's hormonal balance generally do not cause gingival inflammation, but can increase the severity of an already existing plaque-induced gingivitis. In addition to insulin deficiency (Diabetes mellitus; pp. 132, 215), it is primarily the female sex hormones that often are associated with the progression of plaque-elicited gingivitis:

- Puberty gingivitis
- Pregnancy gingivitis
- Gingivitis from the "Pill" (rare)
- Gingivitis menstrualis and intermenstrualis
- Gingivitis climacterica

Puberty Gingivitis

Epidemiologic studies have demonstrated that gingival inflammation during puberty is somewhat more pronounced when compared to the years preceding and following puberty (Curilović et al. 1997, Koivuniemi et al. 1980, Stamm 1986). If oral hygiene is poor and/or if the adolescent is a mouth breather, a typical gingival hyperplasia may ensue, especially in the maxillary anterior area (Figs. 178 and 179).
Therapy: Oral hygiene instruction, plaque and calculus removal; gingivoplasty if the hyperplasia is severe. Mouth breathing may require consultation with an appropriate medical specialist (ENT).

Pregnancy Gingivitis

This condition is not observed in every pregnant woman. Even if oral hygiene is good, however, the gingivae will exhibit an elevated tendency to bleed (Silness & Löe 1964).
Therapy: Oral hygiene; recall every one to two months until breast-feeding is discontinued.

"Pill" Gingivitis

Gingival reaction to oral contraceptives is rare today (Pankhurst et al. 1981). Symptoms: Slight bleeding, rarely erythema or swelling.
Therapy: Oral hygiene.

Gingivitis menstrualis/Intermenstrualis

This gingival condition is exceedingly rare. Desquamation of gingival epithelium occurs during the twenty-eight day menstrual cycle, similar to vaginal epithelium. In exceptional cases, the desquamation can be so pronounced that a diagnosis of "discreet" gingivitis may be made, even less frequently gingivitis menstrualis or intermenstrualis (Mühlemann 1952).
Therapy: Good oral hygiene to prevent secondary plaque-associated gingivitis.

Gingivitis climacterica

This alteration of the mucosa is also rare. The pathologic alterations are observed less on the marginal gingiva than on the attached gingiva and oral mucosa, which may appear dry and smooth, with salmon-pink spots. Stippling disappears and keratinization is lost. Patients complain of xerostomia and a burning sensation.
Therapy: Careful oral hygiene (pain!), and topical vitamin A-containing ointments and dentifrices. In severe cases, the gynecologist may elect to intervene by means of systemic estrogen supplements.

The following pages depict puberty gingivitis and pregnancy gingivitis.

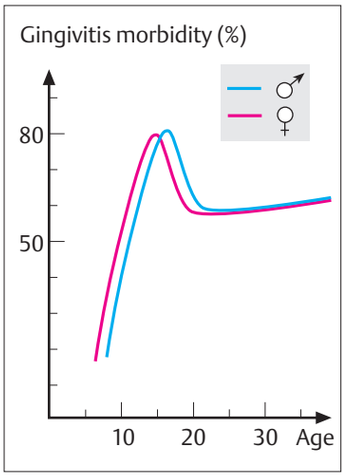
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Puberty

178 Puberty Gingivitis
In this 13-year-old female, more or less severe hemorrhage occurred after gentle sulcus probing. Plaque and mouth breathing were causes of the gingival inflammation. The pubertal hormonal surge may have been a cofactor.

Right: Morbidity of gingivitis in 10,000 persons. A peak is observed during puberty (Stamm 1986).





179 Puberty Gingivitis, Orthodontic Treatment
This 13-year-old male lost his maxillary central incisors due to an accident. Gingivitis, possibly puberty-related, was present before the accident.
Orthodontic means were used to move the lateral incisors mesially. In the absence of adequate plaque control, a severe inflammatory hyperplasia occurred between the maxillary lateral incisors.



Pregnancy

180 Mild Pregnancy Gingivitis
In this 28-year-old female, who was in her seventh month of pregnancy, a cursory inspection did not reveal symptoms of gingivitis in the anterior region. In the premolar and molar areas, however, a moderate gingivitis was detected.

Right: Copious hemorrhage occurred immediately after probing in the area of a defective restoration (plaque niche).





181 Severe Pregnancy Gingivitis
This 30-year-old patient exhibited moderate gingivitis even before her pregnancy. This photograph, taken during the eighth month, reveals severe inflammation and pronounced hyperplastic, epulis-like gingival alterations in the anterior segment.



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Severe Pregnancy Gingivitis—Gravid Epulis

This 24-year-old woman was eight months pregnant. She presented complaining that she "bites the swollen gums" on the left side of her mouth (gravid epulis). A severe, generalized gingivitis was also in evidence.

Findings:

API: 70 %

PD: 7 mm in the region of teeth 34 and 35, otherwise up to 4 mm (pseudopockets)

PBI: 3.2

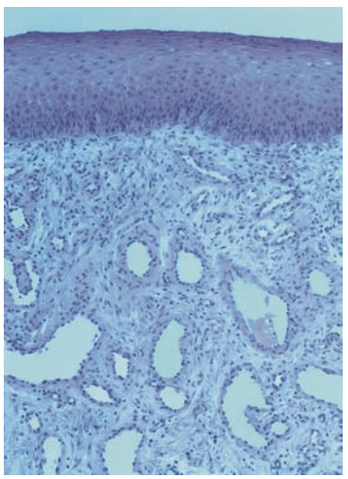
TM: 0–1

Diagnosis: Severe generalized pregnancy gingivitis with a large pyogenic granuloma (epulis) near teeth 34–35.

Therapy: During the pregnancy, repeated oral hygiene instruction, motivation, plaque and calculus removal; gingivoplasty (electrosurgery, laser) around teeth 34 and 35. After breast-feeding is terminated, re-evaluation and further treatment planning.

Recall: Frequency depends on patient compliance.

Prognosis: With treatment, good.

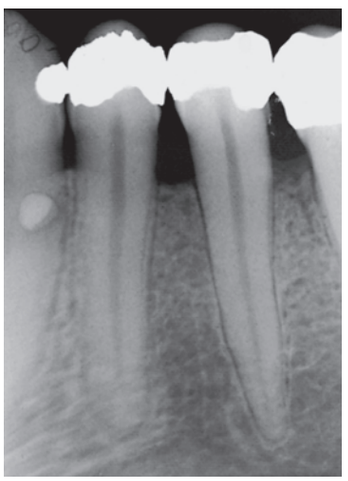


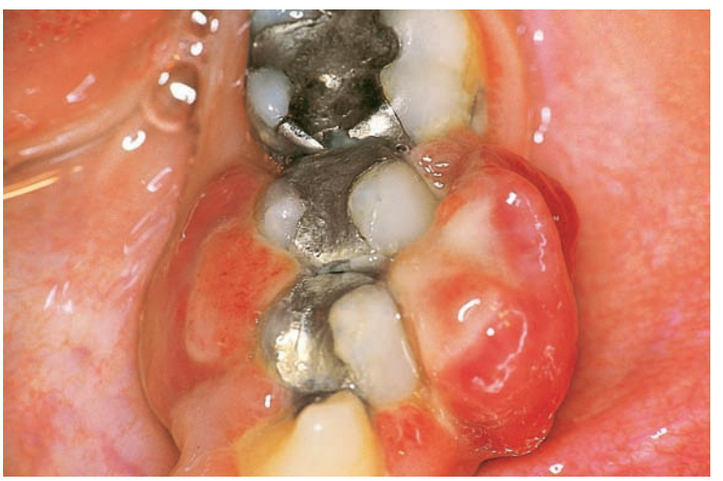


182 Severe Pregnancy Gingivitis

Due to poor oral hygiene, a pronounced gingivitis developed during the last half of the pregnancy. A large epulis is observed buccally and lingually around the mandibular premolars.

Left: The histologic section (of gingiva, not the epulis; see black line) exhibits normal oral epithelium, a relatively mild inflammatory infiltrate and widely dilated vessels (HE, x40)





183 Gravid Epulis

The surface of the epulis is ulcerated because the patient's maxillary teeth bit into the tissue during mastication. For this reason, the redundant tissue had to be removed while the patient was still pregnant. Considerable hemorrhage may be expected during such surgery (laser, electrosurgery).

Left: The radiograph depicts some horizontal loss of the crestal compact bone of the interdental septa (demineralization).



184 Three Months After Gingivoplasty; Two Months Post-Partum

Definitive periodontal therapy and treatment planning for restorative work that is sorely needed should begin at this time (e. g., replacement of the old amalgam restorations with inlays or composites).

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Pregnancy Gingivitis and Phenytoin

Some systemic medications, in combination with microbial plaque, can elicit gingival overgrowth. This adverse effect is best known for phenytoin (diphenylhydantoin, p. 121), which has been used for decades in the treatment of most types of *seizure disorders*.

Phenytoin is also often prescribed following skull trauma and neurosurgical procedures, so that relatively many persons (up to 1 % of the population?) take the drug at some time during life.

It is also well known that phenytoin can cause *teratogenic damage*, and therefore must *never* be taken during pregnancy. In the case depicted here, neither the patient nor her treating physician were aware of this potential side effect. The 22-year-old epileptic had taken phenytoin for many years, including during the first seven months of her pregnancy. She developed prominent gingival overgrowth, which was particularly pronounced in the maxillary right quadrant. Probing depths up to 7 mm (pseudopockets) were associated with copious hemorrhage (BOP). Fortunately, the child was born with no birth defects.

185 Phenytoin-Induced Gingival Overgrowth and Pregnancy Gingivitis
The epileptic patient is seven months pregnant. Up until the time of her dental visit, she was taking phenytoin. The gingivae exhibit generalized, pronounced inflammatory hyperplasia and overgrowth.

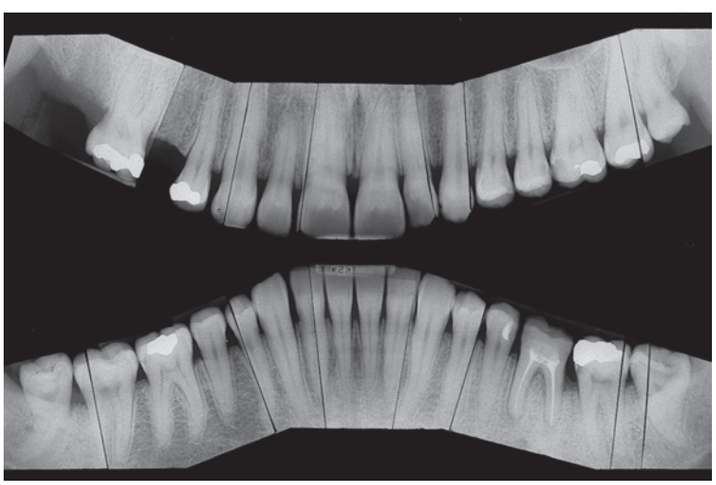
Right: Extreme gingival overgrowth around tooth 12.





186 Radiographic Appearance
Despite the severe hyperplastic gingivitis, no bone loss is observed. Teeth 15 and 17 had already been extracted.

Right: Note normal interdental osseous septa even in the region around tooth 12, which exhibited pronounced, severe overgrowth.





187 After Periodontal Treatment
After the delivery, the patient underwent scaling, gingivoplasty and extraction of teeth 17, 15 and 28; this led to the reinstatement of healthy gingival relationships, although the phenytoin regimen had to be resumed.

Right: Even in the region where overgrowth was most severe (tooth 12), virtually healthy gingival relationships are observed.





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Periodontitis

Periodontitis maintains its position as one of the most widespread diseases of mankind, but fortunately only ca. 5–10 % of all cases are aggressive, rapidly-progressing forms (Ahrens & Bublitz 1987, Miller et al. 1987, Miyazaki et al. 1991, Brown & Löe 1993, Papapanou 1996).
Periodontitis is a multifactorial disease of the tooth-supporting structures, elicited by a microbial biofilm (dental plaque). It usually develops from a pre-existing gingivitis; however, not every case of gingivitis develops into periodontitis. The quantity and virulence of microorganisms on the one hand, and host resistance factors (immune status, genetics and therefore heredity, as well as the presence of risk factors) on the other hand are the primary determinants for the initiation and progression of periodontal destruction (p. 21).
The classification of periodontitis evolves from dynamic, pathobiologic criteria, as recommended by the AAP (Armitage 1999; more extensive listing on p. 78; complete classification on p. 519):

- Chronic Periodontitis (Type II, formerly AP)
- Aggressive Periodontitis (Type III, formerly EOP/RPP)
- Necrotizing Periodontitis (Type V B/NUP, formerly ANUP)

The two main forms (Types II and III) are further subclassified according to their expansion into *localized* (A: ≤ 30 % of all sites involved) or *generalized* (B: > 30 % of all sites involved).
Similarly, the degree of severity—as determined by clinical attachment loss—is divided into *mild* (1–2 mm), *moderate* (3–4 mm) and *severe (≥ 5* mm).
An entire group of periodontal diseases comprise the aggressive forms (Type III), which were previously referred to as PP (prepubertal periodontitis), LJP (localized juvenile periodontitis), and RPP (rapidly progressive periodontitis).
The *pathobiologic* nomenclature, i. e., the description of the clinical course of periodontitis, is presented on pages 98 and 99. One must, of course, keep in mind that a “practical” diagnosis for every *patient*, for every *tooth*, and for every *site* must be performed clinically and radiographically to assess the severity, i. e., the *pathomorphology* of the disease process.

In this chapter, the following aspects of periodontitis will be presented:

- Severity of periodontitis
- Types of pockets
- Morphology of bone loss
- Furcation invasion
- Histopathology
- Clinical and radiographic symptoms
- Clinical cases depicting the various forms of periodontitis

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Pathobiology—The Most Important Forms of Periodontitis

The pathobiological nomenclature for the various forms of periodontitis is not a rigid, definitive classification. Today, one differentiates between chronic and aggressive forms of the disease, which may be localized or generalized (pp. 519–522). The chronic form of the disease may transform into an aggressive form, e.g., in the elderly, where the immune system is less effective. Most types of periodontitis progress in a step-wise fashion (random burst theory). Stages of exacerbation alternate with stages of remission.

As science continually provides new knowledge concerning microbiology as well as pathogenesis—especially the host response to the infection—the diagnosis and the nomenclature can no longer simply be defined according to the clinical course of the disease; it is now possible to characterize periodontitis as an *Aa*- or a *Pg*-associated disease (etc.). In the future, it may become even more important to characterize the diverse parameters of the host immune response, the mediators and the risk factors; these are ultimately responsible for the existence of the disease and the speed of its progression.

Type II (Chronic Periodontitis; formerly AP)

This most common form of periodontitis begins between the ages of 30 and 40 years, generally from a pre-existing gingivitis. The entire dentition may be equally affected (*generalized*, Type II B). More often, however, the distribution of the disease is irregular, with more severe destruction primarily in molar areas but secondarily also in anterior segments (*localized*; Type II A). The gingivae exhibit varying degrees of inflammation, with "shrinkage" in some areas and fibrotic manifestations elsewhere.
Exacerbations occur at rather lengthy intervals. Risk factors (e.g., heavy smoking, IL-1-positive genotype) can accentuate the clinical course. In the elderly, the disease can lead to tooth loss, which may also be due to a decrease in host immune response and more frequent acute stages.

Therapy: Chronic periodontitis can be successfully treated by means of purely mechanical therapy, even if the patient's cooperation/compliance is not optimum.

188 Characteristics of Type II—formerly AP

Clinical Symptoms	
• Morbidity	ca. 85–95 % of all adults (?) ca. 95 % of all periodontal patients
• Initiation—Course	ca. 30th year of life slow, "chronic" course
• Periodontal Findings	– all teeth affected, localization to molars and incisors – gingival inflammation, gingival swelling, some recession – alveolar bone: uneven destruction
• Systemic Diseases	none
Blood Cell Defects	*neutrophilic granulocytes*, *monocytes*
	– –
Bacterial Infection	mixed flora; in *active pockets*, frequently *P. gingivalis*, *P. intermedia*, *Fusobacterium nucleatum*, *A. actinomycetemcomitans*
Heredity	none (possible polymorphisms, e.g., IL-1)

Type III B (Aggressive Periodontitis; formerly EOP/RPP)

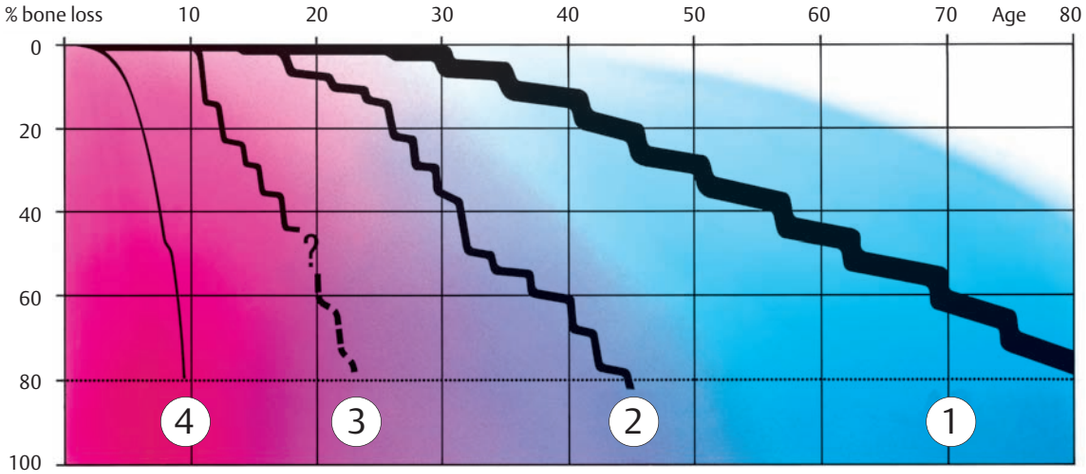
Aggressive forms of periodontitis are relatively rare (Page et al. 1983a, Miyazaki et al. 1993, Lindhe et al. 1997, Armitage 1999). They are usually diagnosed between the ages of 20 and 30 years. Females appear to be more frequently affected than males. The severity and distribution of attachment loss vary considerably. One observes infrequent acute stages, which may transition into chronic disease. The cause of the active stages is specific microorganisms (*Aa*, *Pg* etc.), which may actually invade the ulcerated tissues. Risk factors (smoking, systemic disease such as diabetes, conditions of psychic tension and stress) and pro-inflammatory mediators that reduce the immune response can amplify the disease picture.

Therapy: The majority of aggressive cases can be successfully treated by means of purely mechanical therapy. In severe cases, a supportive systemic antibiotic regimen may be indicated.

189 Characteristics of Type III B—formerly EOP/RPP

Clinical Syptoms	
• Morbidity	ca. 5–15 % of all periodontitis patients
• Initiation—Course	possible in all ages, young individuals predominate rapid cyclic course
• Periodontal Findings	– many to all teeth affected – gingiva more or less inflamed – rapid bone loss – frequent symptoms of activity
• Systemic Diseases	?, possible genetic determination
Blood Cell Defects	*neutrophilic granulocytes*, *monocytes*
Chemotaxis reduced	++ ++
Increased migration	++ ++
Bacterial Infection	mixed flora and specific *P. gingivalis*, *T. forsythensis*, *F. nucleatum*, *A. actinomycetemcomitans* (invasion?), *P. intermedia*, spirochetes
Heredity	sex-linked dominant (?)

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190 Forms of Periodontitis

1. Chronic, slowly progressive periodontitis in adults—Type II
2. Aggressive, rapidly progressing periodontitis—Type III B
3. Aggressive, localized (juvenile) periodontitis—Type III A
4. Aggressive, generalized, prepubertal, rapidly progressing periodontitis—Type IV B

Type III A (Aggressive Periodontitis, formerly EOP/LJP)

This rare disorder occurs early and attacks the *permanent* dentition. It begins in puberty, but is usually not diagnosed until several years later, often when lesions are discovered serendipitously (e.g., on bitewing radiographs taken for caries assessment). In the initial stages, incisors and/or first molars are affected in both maxilla and mandible; later, other teeth may also be affected. Hereditary factors (genetics, ethnic origin) have been demonstrated. Girls are affected more frequently than boys. In the early stages of aggressive periodontitis, one seldom observes pronounced gingivitis. The gingival pockets almost always (90 %) harbor *Aa*. The patient's serum contains immunoglobulins against *Aa* leukotoxins, which damage PMNs.

Therapy: With early diagnosis and therapy consisting of vigorous mechanical debridement and supportive systemic administration of medicaments, the destructive process can be halted rather easily. Osseous defects may eventually regenerate.

Type IV B (Aggressive Periodontitis; formerly EOP/PP)

This extraordinarily rare form of periodontitis may be detected even upon eruption of the deciduous teeth and is usually associated with genetic aberrations and systemic disorders (Page et al. 1983b, Tonetti & Mombelli 1999, Armitage 1999; cf. p. 118). The disease progresses rapidly and is usually generalized:

- A localized form begins at ca. age 4 and exhibits only mild gingival inflammation with relatively little plaque.
- The generalized form (Type IV B) begins immediately after eruption of the deciduous teeth. It is associated with severe gingivitis and gingival shrinkage. The microbiology remains unclear.

Therapy: The localized form can be halted by a combination of mechanical therapy and systemic antibiotics. The generalized form appears to be refractory to therapy.

191 Characteristics of Type III A (formerly EOP/LJP)

Clinical Syptoms			
• Morbidity	0.1 % in young Caucasians, > 1 % in young Blacks		
• Initiation—Course	ca. age 13, at the beginning of puberty often rapid "burst-like" course		
• Periodontal Findings	– primarily only in first molar and/or incisor teeth – gingiva often appear normal – crater-like bone loss		
• Systemic Diseases	none, genetically determined		
Blood Cell Defects	*neutrophilic granulocytes*, *monocytes*		
Chemotaxis reduced	++	+	
Phagocytosis reduced	+	-	
Receptor defect	+	?	
Bacterial Infection	mixed flora and specifically *A. actinomycetemcomitans* (serotypes a, b), *Capnocytophaga* sp. (?)		
Heredity	autosomal recessive (sex-linked dominant?)		

192 Characteristics of Type IV B (formerly EOP/PP)

Clinical Syptoms			
• Morbidity	very rare (few cases reported)		
• Initiation—Course	immediately after eruption of deciduous teeth clinical course is almost continuous and destructive		
• Periodontal Symptoms			
– generalized form	– all teeth affected – gingiva inflamed and hyperplastic		
– localized form	– individual teeth affected, very rare		
• Systemic Diseases	Hypophosphatasia, susceptibility to respiratory tract infections, otitis media, skin infections		
Blood Cell Defects	*neutrophilic granulocytes*, *monocytes*		
Adherence (vessel wall) disturbed	++	+	
Chemotaxis reduced	++	++	
Receptor deficiency	+	+	
Bacterial Infection	mixed flora, specific bacteria are unknown		
Heredity	autosomal recessive		

(Figs. 188–192 modified from *Page et al. 1983a/b, 1986; Schroeder 1987b)*

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Pathomorphology—Clinical Degree of Severity

Periodontitis is a general term. As already described, it subsumes *pathobiologically dynamic forms of disease progressing at varying rates*, and exhibits differing microbial etiologies and varying influences by the host immune response (p. 21). It may sound trite, but one must understand that all forms of periodontitis *begin* at some point in time. They develop at various times during a patient's life, usually from a pre-existing plaque-elicited gingivitis. In the absence of treatment, the disease progresses, albeit at varying rates. It is therefore clear that during clinical data collection, which leads to the diagnosis for an individual case, not only must the type of disease be ascertained, it is also necessary to determine in what pathomorphologic stage the disease process currently exists or, in other words, how far atta–chment loss has progressed. From these diagnostic criteria, clinical course, expanse (*localized*, e. g., less than 30 % of all sites, or *generalized*) and clinical degree of severity, the practitioner can derive a prognosis and estimate the degree of difficulty of the necessary treatment, which will naturally be higher for aggressive periodontitis (Type III) than for a similarly advanced chronic periodontitis (Type II).

Case Diagnosis—Single-Tooth Diagnosis—"Sites"

While it is usually possible to diagnosis the type of periodontitis for the entire dentition, it is rather a more difficult matter to precisely define the clinical degree of severity. Periodontitis almost always progresses at different rates in different areas of the mouth, on different teeth, and even at different sites on individual teeth. Therefore, any statement about "average" disease severity is usually meaningless.

The following pathomorphologic classification (degree of severity) cannot be understood as pertinent to the individual case (patient); it relates more to *single tooth diagnosis* as well as *single tooth prognosis*. The reasons for the often quite irregular localized destruction are not always clear (oral hygiene, plaque-retentive niches, localized specific bacteria, tooth type, function?). In addition to describing gingivitis, practitioners will continue to use the terms mild, moderate and severe to differentiate the stages of periodontitis. It is perhaps important to remember that different authors, "schools," and periodontologic societies use various synonyms for these different clinical manifestations of disease.

Clinical Degrees of Severity

• mild/slight	… *levis*	… *superficialis*
• moderate	… *media*	… *media*
• severe/advanced	… *gravis*	… *profunda*

Most recently, the AAP (1996c) has defined the degree of severity of periodontitis not exclusively according to probing depths and terms such as mild, moderate or severe; rather more consideration is given also to gingival inflammation, bone loss, attachment loss, furcation invasion and tooth mobility.

Clinical Degrees of Severity Class	Form	Gingival Inflammation, Bleeding (BOP)	Probing Depths (PD)	Clinical Attachment Loss (AL)	Bone Loss	Furcation Invasion	Tooth Mobility (TM)
Class 1	**Gingivitis**	+ to +++	1–3 mm	–	–	–	– ?
Class 2	**Slight Periodontitis**	+ to +++	4–5 mm	1–2 mm	+	–	– ?
Class 3	**Moderate Periodontitis**	+ to +++	6–7 mm	3–4 mm	horizontal ++ individual vertical	~ F1	+
Class 4	**Severe Periodontitis**	+ to +++	>7 mm	≥5 mm	multiple vertical ++	F2, F3	++

Conclusions

All of the pathomorphologic classifications of periodontitis presented on this page (cf. AAP Glossary 2001) attempt to describe the severity of the disease *as it exists immediately after clinical examination*; these descriptors say very little about the pathobiology, the *dynamics* or the speed of progression of periodontitis (prognosis).

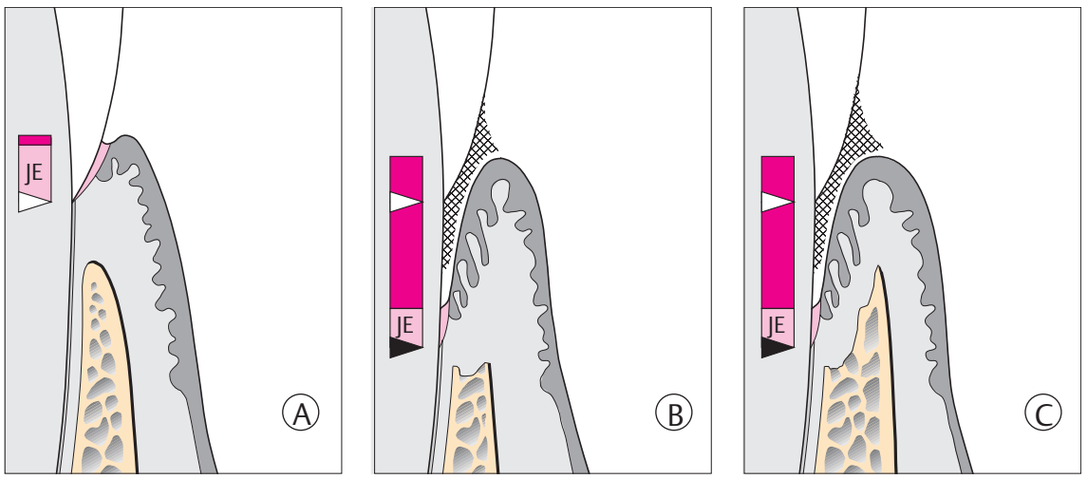
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Pockets and Loss of Attachment

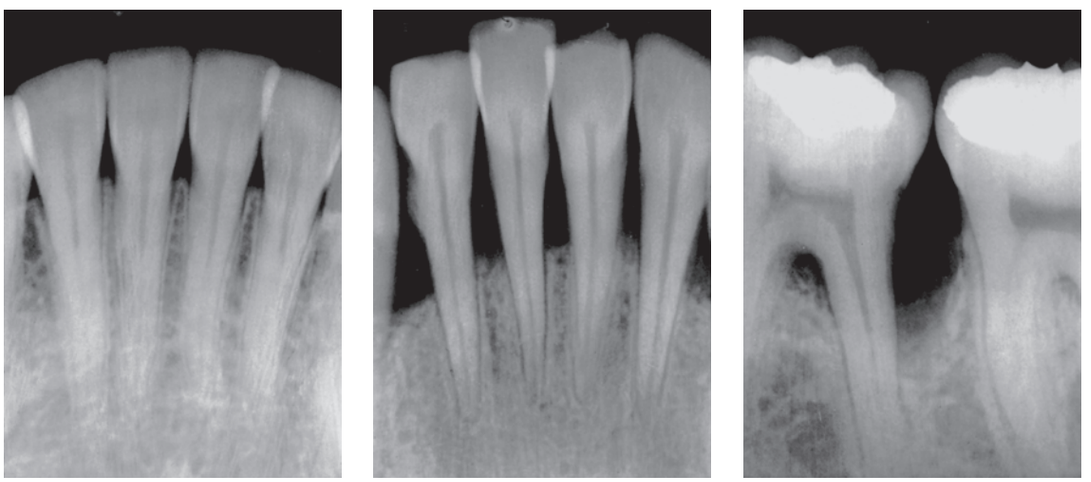
Pocket formation without any loss of connective tissue attachment is seen in gingivitis in the form of the *gingival pocket* and the *pseudopocket* (p. 79). A *true* periodontal pocket will exhibit attachment loss, apical migration of the junctional epithelium, and transformation of the junctional epithelium into a pocket epithelium (Müller-Glauser & Schroeder 1982). The *true* periodontal pocket may assume two forms (Papapanou & Tonetti 2000):

- *Suprabony* pockets, resulting from horizontal loss of bone
- *Infrabony* pockets, resulting from vertical, angular bone loss. In such cases the deepest portion of the pocket is located apical to the alveolar crest.

Whether pocket development is horizontal or vertical appears to be due to the thickness of the interdental septum or the facial and oral bony plates.
True loss of attachment results from microbial plaque and the metabolic products of plaque microorganisms. The range and effective radius of destruction is ca. 1.5–2.5 mm (Tal 1984; Fig. 195).



193 Types of Periodontal Pockets
A Normal Sulcus
The apical termination of the junctional epithelium (JE) is at the cementoenamel junction (open arrow).
B Suprabony Pocket (red)
Attachment loss; proliferation of pocket epithelium. A remnant of junctional epithelium (pink) persists at the base of the pocket.
C Infrabony Pocket
Bony pocket

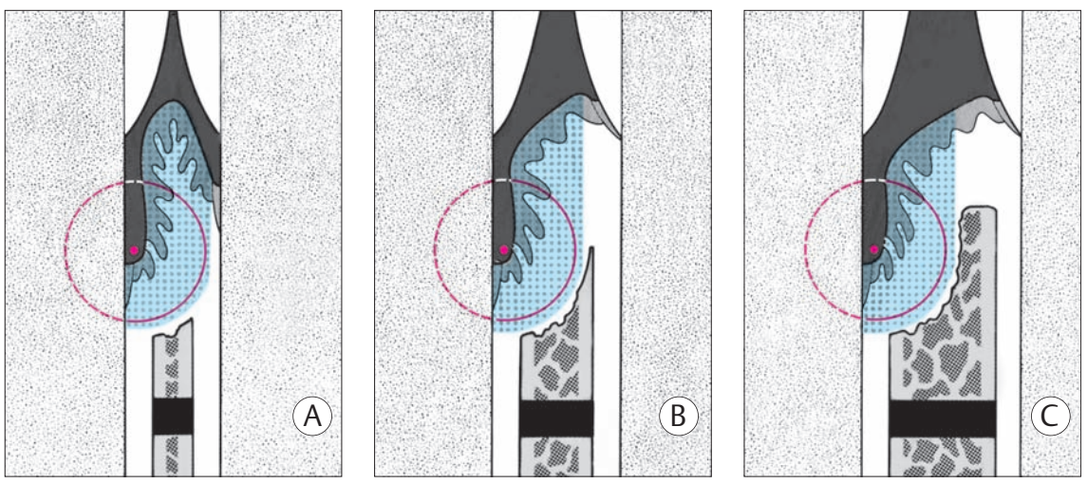


194 Types of Bone Loss

No attachment loss (left)
Normal alveolar septa. Lamina dura and alveolar crest remain intact.

Horizontal Bone Loss (middle)
Up to 50 % loss of interdental septal bone.

Vertical Bone Loss, Furcation Involvement (right)
Severe bone loss distal to the first molar. The furcation of this tooth is also involved.



195 "Range" of Destruction = Contour of Bone Resorption
The destructive process radiating from the plaque measures ca. 1.5–2.5 mm (red circle). The *expanse (width) of the interdental septa* therefore determines for the most part the type (morphology) of bone loss.

A Narrow: horizontal resorption
B Average: horizontal resorption, incipient vertical resorption
C Wide: vertical resorption, bony pocket

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Intra-alveolar Defects, Infrabony Pockets

The infrabony pocket (infra-alveolar vertical bone loss) may exhibit various forms in relation to the affected teeth (Goldman & Cohen1980, Papapanou & Tonetti 2000).

Classification of Bony Pockets

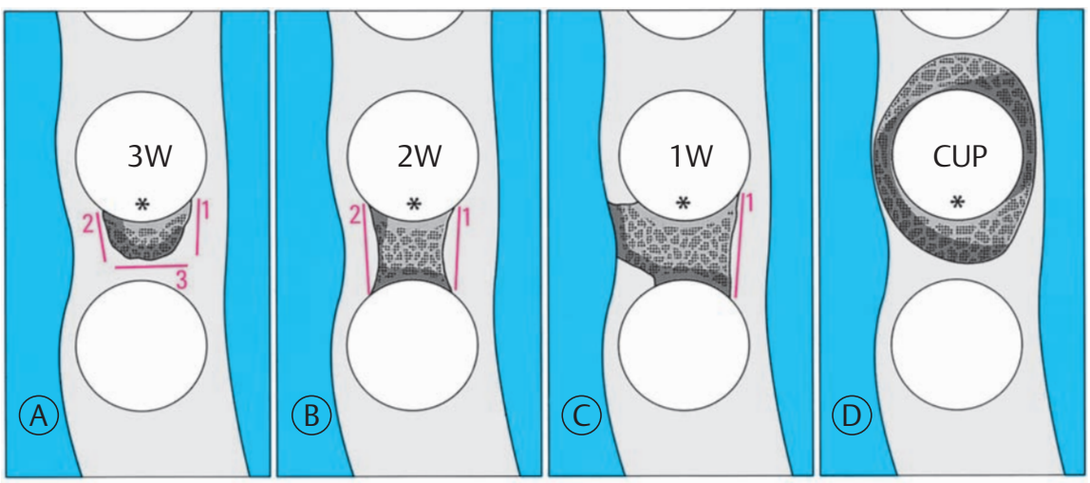
- *Three-wall bony pockets* are bordered by one tooth surface and three osseous surfaces
- *Two-wall bony pockets* (*interdental craters*) are bordered by two tooth surfaces and two osseous surfaces (one facial and one oral)
- *One-wall bony pockets* are bordered by two tooth surfaces, one osseous surface (facial or oral) and soft tissue
- *Combined bony pockets (cup-shaped defects)* may be bordered by several surfaces of a tooth and several of bone. The defect surrounds the tooth.

The causes for this wide variation in pocket morphology and resorption of bone are myriad and cannot always be wholly elucidated in each individual case.

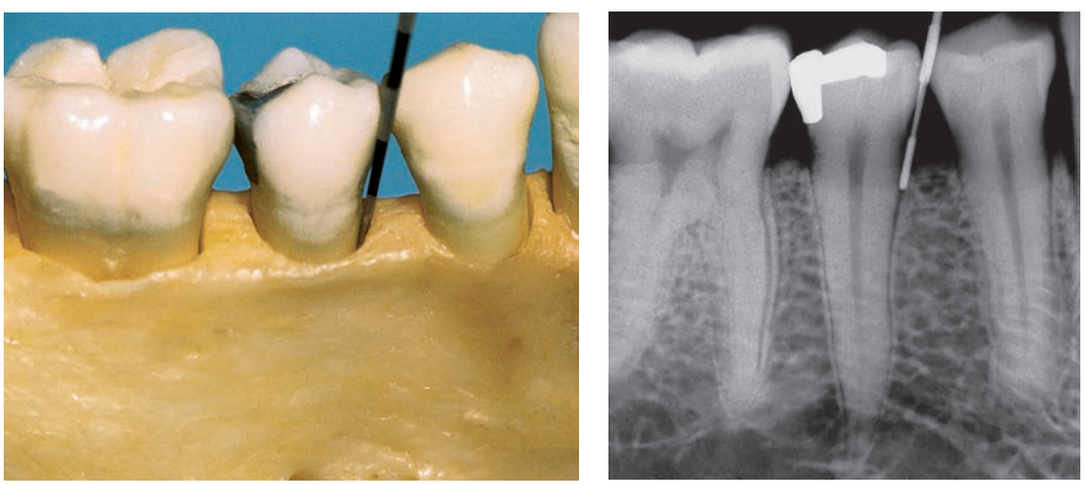
196 Schematic Representation of Pocket Morphology

A **Three-wall** bony defect
B **Two-wall** bony defect
C **One-wall** bony defect
D **Combined** bony defect, crater-like resorption ("cup")

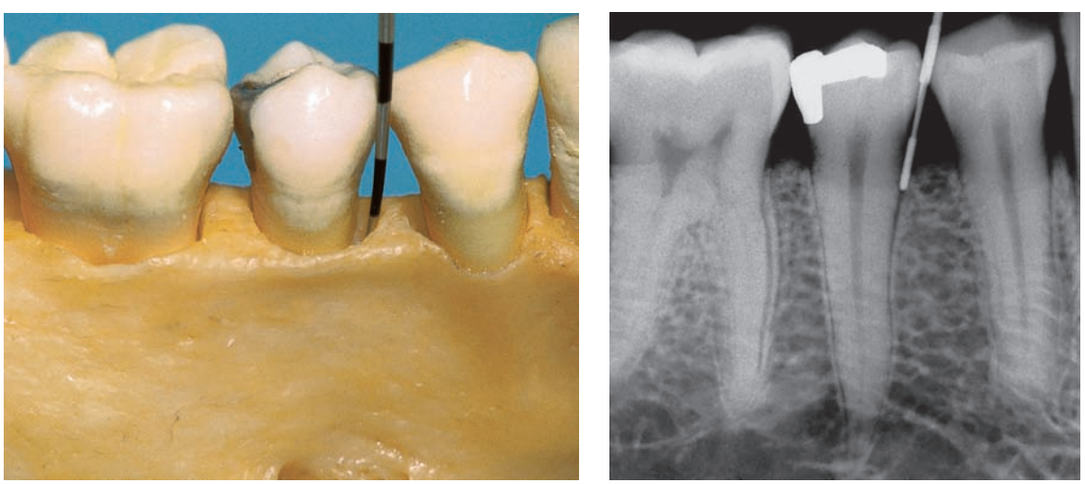
The walls of each pocket are shown in red (1–3).



197 Small Three-Wall Defect
Early pocket formation on the mesial aspect of the second premolar. The color-coded probe (CP12) measures a depth of ca. 3 mm. If gingiva were present, the total probing depth would be ca. 5 mm.



198 Deep Three-Wall Bony Pocket
The periodontal probe descends almost 6 mm (measured from the alveolar crest) to the base of this three-wall defect.



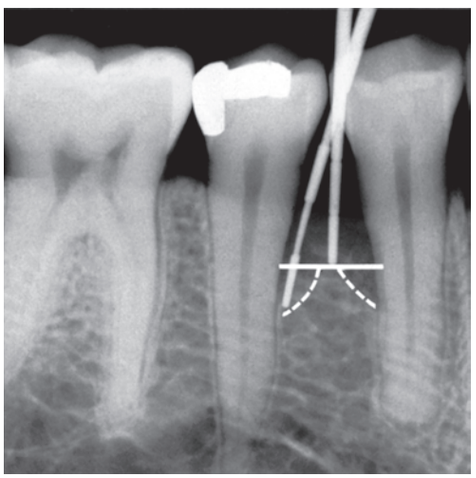
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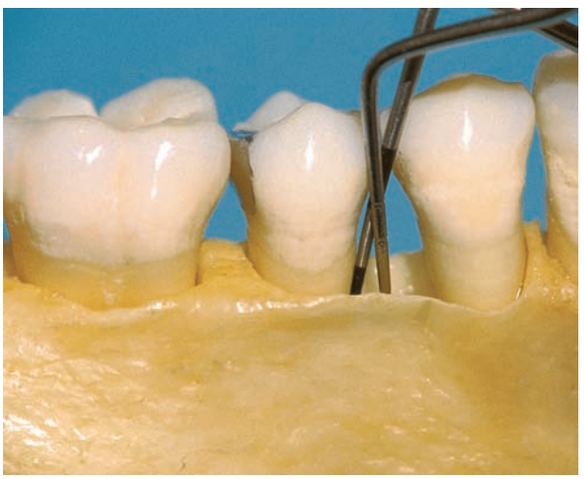
Mention was already made of the significance of bone thickness (Fig. 195). Since the bony septa between the roots become thinner coronally, the initial stage of periodontitis generally presents as horizontal resorption. The greater the distance between the roots of two teeth, the thicker will be the intervening septum, and the development of a vertical defect is more likely.

In addition to such simple osseous morphology, other factors almost certainly play some role in the type of resorption:

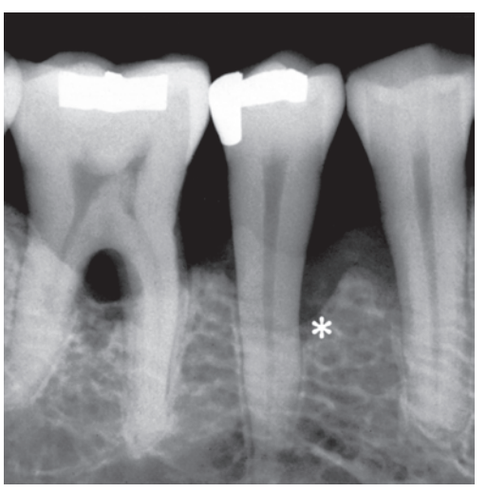
- local acute exacerbation elicited by specific bacteria in the pocket
- local inadequate oral hygiene (plaque)
- crowding and tipping of teeth (plaque-retentive areas)
- tooth morphology (root irregularities, furcations)
- improper loading due to functional disturbances (?).

The morphology of the bony pocket is of importance in both prognosis and treatment planning (defect selection). The amount of bone remaining will affect the chances of osseous regeneration after treatment.



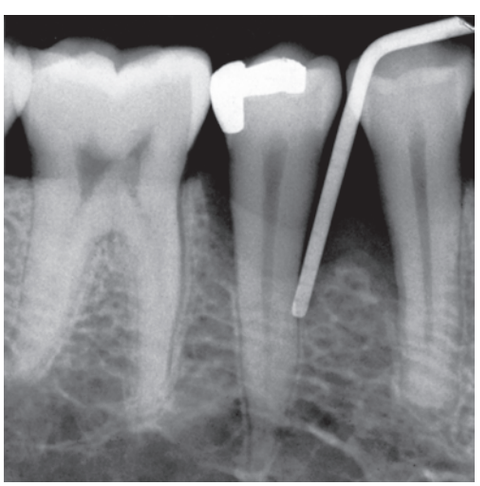


199 Two-Wall Bony Pocket, Interdental Crater
The coronal portion of this defect is bordered by only two bony walls (and two tooth surfaces). In the apical areas the two-wall defect becomes a three-wall defect (see left probe tip in the radiograph, left).





200 One-Wall Bony Pocket on the Mesial of Tooth 45
Advanced bone loss in premolar/molar area. On tooth 45, the facial wall of bone is reduced almost to the level of the mesial pocket (*). A portion of the lingual plate of bone remains intact. The facial root surface and the interdental spaces could be covered with soft tissue to the cementoenamel junction, masking the defect clinically.





201 Combined Pocket, Cup-Shaped Defect
In the region of tooth 45, the apical portion of the osseous defect courses around the tooth, creating a “moat” or “cup” (Goldman probe in situ). The bony pocket is therefore bordered by several osseous and several tooth surface walls.

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Furcation Involvement

Periodontal bone loss around multirooted teeth presents a special problem when bi- or trifurcations are involved. Partially or completely open furcations tend to accumulate plaque (Schroeder & Scherle 1987). Exacerbations, abscesses, progressive loss of attachment and rapid deepening of periodontal pockets occur frequently, especially with through-and-through furcation involvement. In addition, open furcations are particularly susceptible to the development of dental caries.

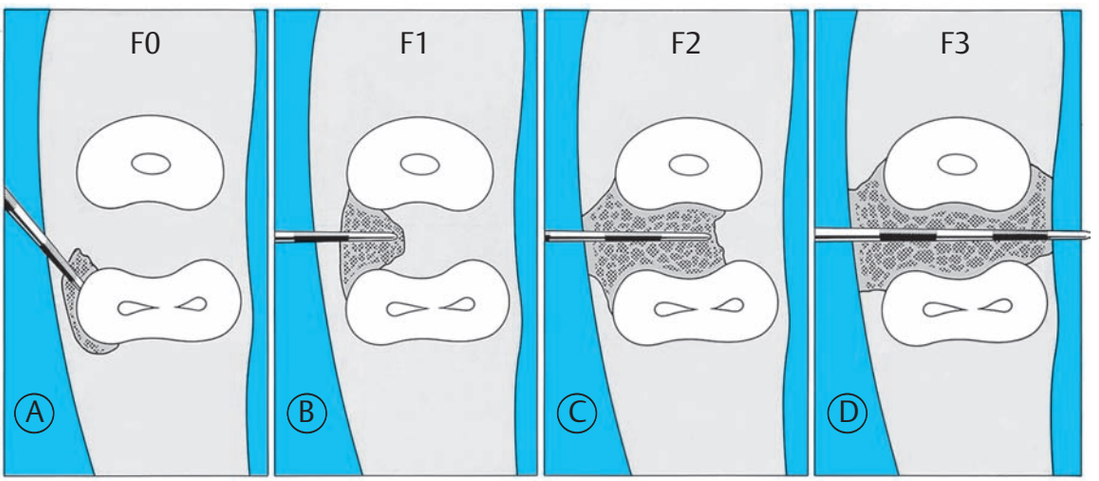
Modified from Hamp et al. (1975), three classes of *horizontally* measured furcation invasion are acknowledged:

Class—Horizontal

Class F1: The furcation can be probed in the horizontal direction up to 3 mm.

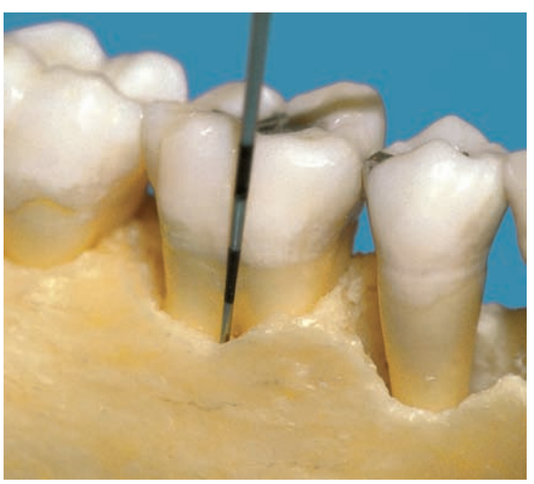
Class F2: The furcation can be probed to a depth of more than 3 mm, but is not through-and-through.

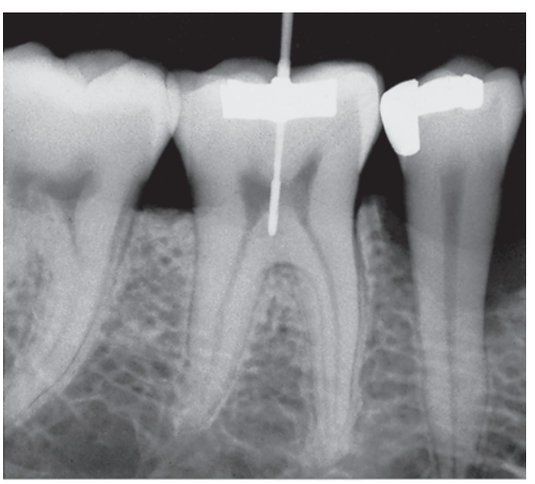
Class F3: The furcation is through-and-through and can be probed completely.



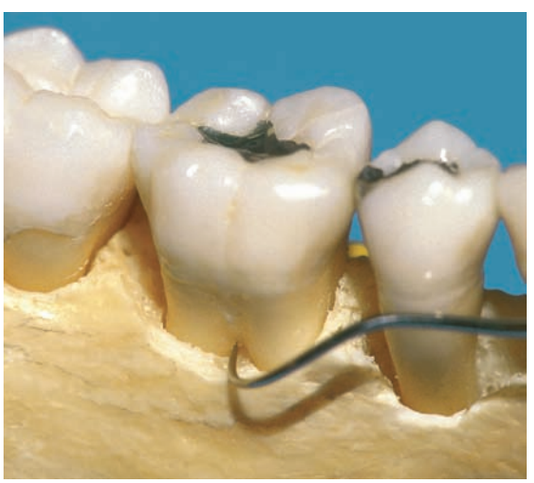
202 Classification of Furcation Involvement—Horizontal Measurement
Furcation involvement may be combined with infrabony pockets.

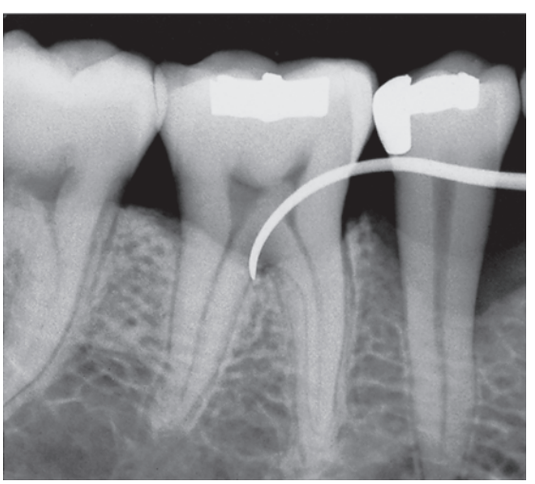
A F0: Pocket on the mesial root, but without furcation involvement.
B F1: Furcation can be probed 3 mm horizontally.
C F2: Furcation can be probed deeper than 3 mm.
D F3: Through-and-through furcation involvement.





203 No Furcation Involvement—F0
In a clinical situation, in the region of the buccal furcation entrance, a ca. 5 mm deep *suprabony pocket* would be detectable.





204 Furcation Involvement—F1
Using a curved, pointed explorer (CH3; Hu-Friedy) the buccal furcation can be probed to a depth of less than 3 mm. Probing is performed from both buccal and lingual aspects. Special furcation probes are available on the market today. They are rounded and have millimeter indications; e. g. Nabers-2 (Hu-Freidy).

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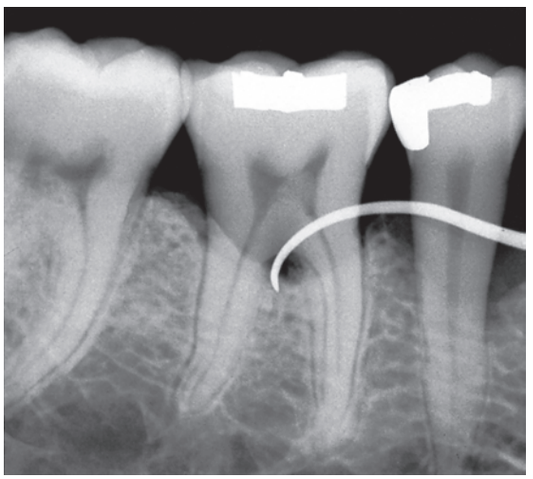
This classification of bifurcation involvement in the mandible is also applicable to the maxilla, where trifurcation involvement is virtually impossible to diagnosis on the radiograph. In the case of maxillary trifurcations, it is imperative to differentiate between which roots the invasion exists and the extent of the horizontal bone loss. For a complete diagnosis, probing most be performed not only from the buccal, but also from distopalatal and mesiopalatal aspects.

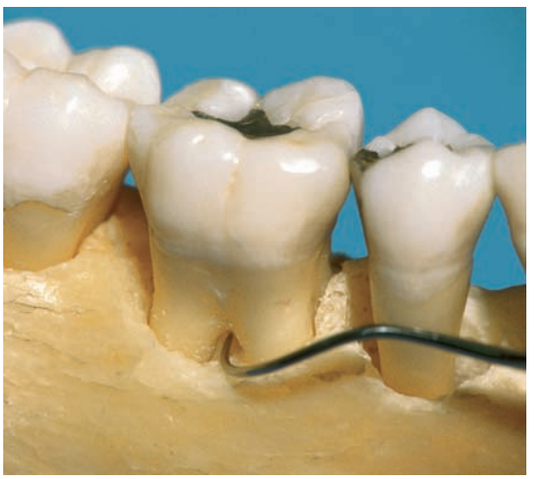
Vertical bone loss in a furcation can also be classified (subclasses A-C, Tarnow & Fletcher 1984). It is measured in millimeters from the roof of the furcation (see also pp. 172, 383):

Subclasses—Vertical

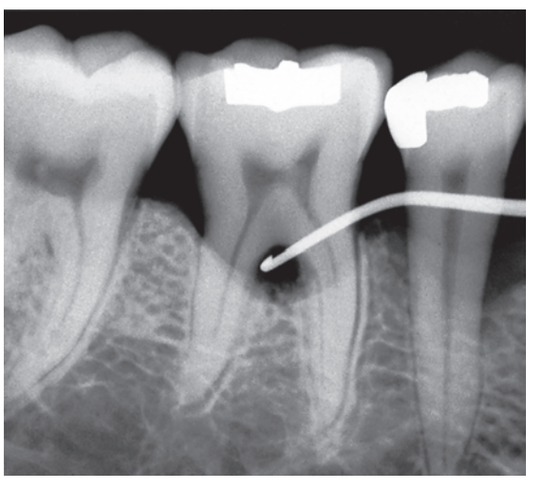
Subclass A:	1–3 mm
Subclass B:	4–6 mm
Subclass C:	>7 mm

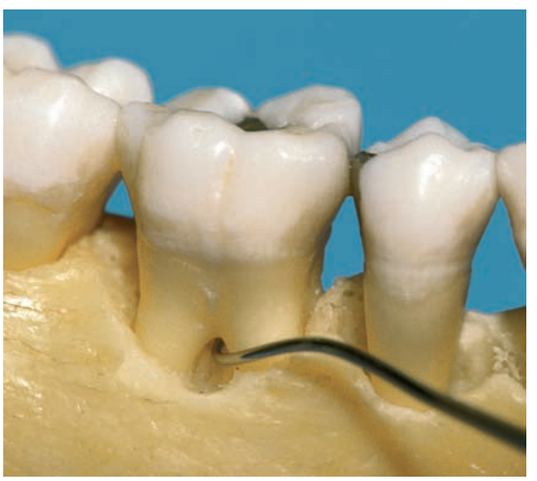
Therapy: Class F1 and F2 furcation involvement may be successfully treated by root planing alone or by flap procedures (conventional and regenerative). Class F3 furcations are usually treated by means of hemisection or by amputation of one root (p. 381).



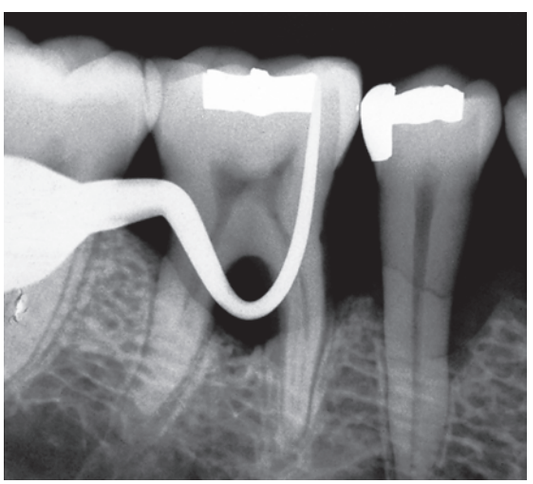


205 Furcation Involvement—F2
The explorer can probe more than 3 mm into the furcation, which is not yet through-and-through.





206 Furcation Involvement—F3, Mild, Subclass A
Narrow, through-and-through bifurcation involvement in the face of relatively minor bone loss (Nabers-2 probe).
Less than 3 mm of bone loss in the vertical dimension; this corresponds to subclass A.





207 Furcation Involvement—F3, Severe, Subclass C
Wide, through-and-through bifurcation in a case exhibiting severe horizontal and vertical bone loss (Cowhorn explorer).
The vertical extent of the furcation exceeds 6 mm: Subclass C.

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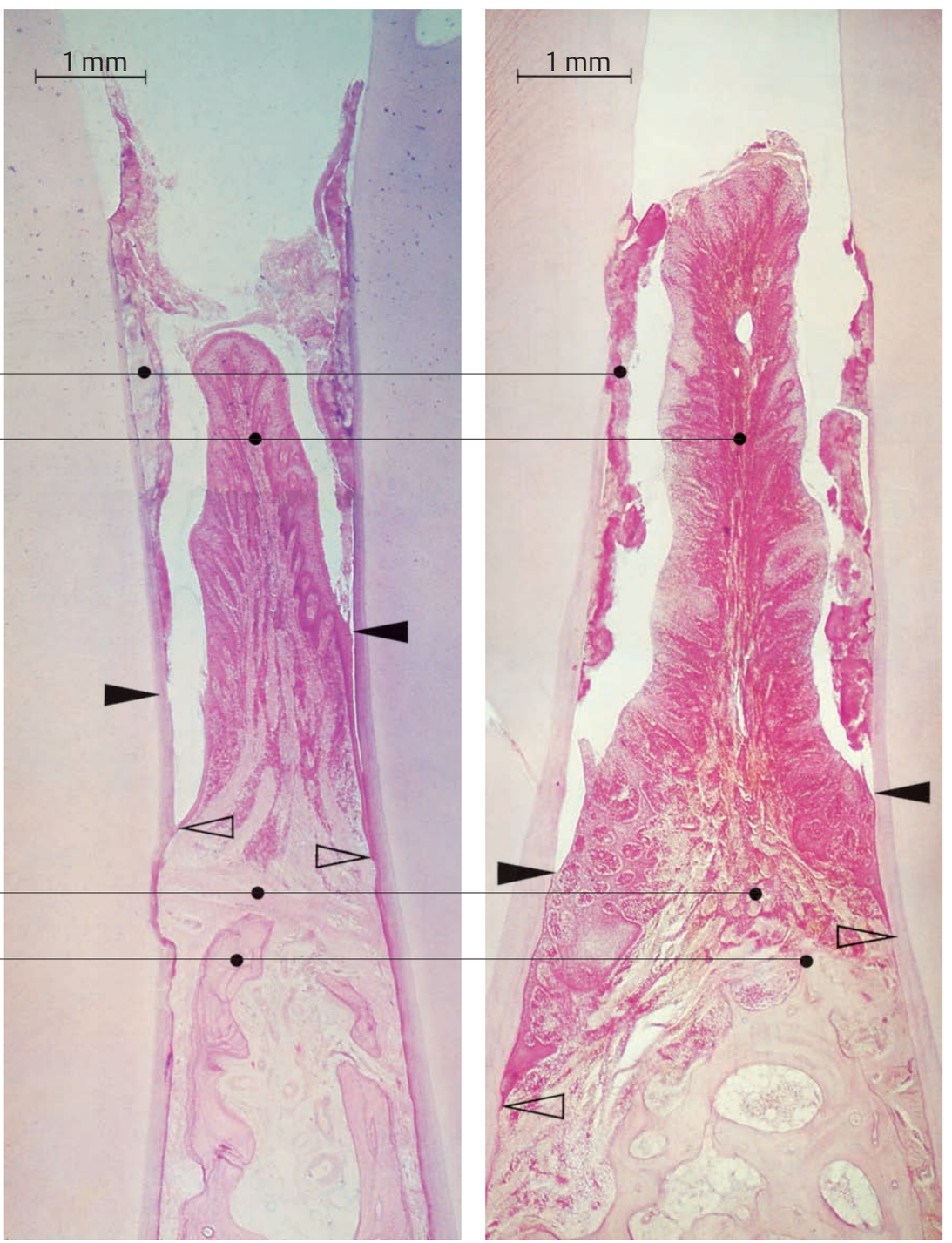
Histopathology

The primary symptoms of periodontitis are attachment loss and pocket formation. *Pocket epithelium* exhibits the following features (modified from Müller-Glauser & Schroeder 1982):

- irregular boundary with the subjacent connective tissue, exhibiting rete pegs; toward the periodontal pocket, epithelium is often very thin and partially ulcerated
- in the apicalmost region, the pocket epithelium becomes a very narrow and short junctional epithelium
- transmigration of PMNs through the pocket epithelium
- defective basal lamina complex on the connective tissue aspect.

Within the *subepithelial connective tissue,* collagen is lost and numerous inflammatory cells invade. In acute stages, pus formation and microabscesses occur. Bone is resorbed, and deeper osseous marrow is transformed into fibrous connective tissue.

Supraalveolar Pocket | **Infraalveolar Pocket**



208 Suprabony Pocket, Gingival Pocket (left)
Pocket epithelium with distinct rete ridges. In the apicalmost region (between the arrows) one observes intact junctional epithelium (exhibiting artifactual separation from the tooth surface in this section). Subepithelial inflammatory infiltrate extends into the area of the transseptal fibers (HE, x40).

209 Infrabony Pocket (right)
Intact junctional epithelium (open arrow) persists on the tooth (left), apical to the interdental bony septum. The pocket epithelium displays pronounced rete ridges and areas of ulceration. Inflammatory infiltrate extends into the periodontal ligament and marrow (HE, x40).

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Additional Clinical and Radiographic Symptoms

Primary Symptoms

- Inflammation (gingivitis)
- True periodontal pockets
- Bone resorption

} Attachment Loss

These *obligate* primary symptoms of periodontitis must be present simultaneously for a diagnosis of "periodontitis" (= inflammation-induced attachment loss); they can occur in different forms and in measurable degrees of severity.

Additional Symptoms

The additional symptoms, listed below, are *not obligate* in every case of periodontitis, but they may modify or complicate the disease picture:

- Gingival shrinkage
- Gingival swelling
- Pocket activity: bleeding, exudate, pus
- Pocket abscess, furcation abscess
- Fistula
- Tooth migration, tipping, extrusion
- Tooth mobility
- Tooth loss

Gingival Shrinkage

During the course of periodontitis, especially the slowly progressive, chronic type in adults, gingival shrinkage may occur with time. Gingival shrinkage can also occur after spontaneous transition of an acute exacerbation into a chronic, quiescent stage, or following comprehensive periodontal therapy or after draining of abscesses. Whatever the cause, shrinkage leads to exposure of root surfaces.

This type of shrinkage must not be confused with true gingival *recession*, which can occur in the *absence* of clinical inflammation. True recession occurs without the formation of periodontal pockets and is most often observed on the facial aspect of roots. On the other hand, gingival shrinkage due to periodontitis may also be quite pronounced in the papillary regions.

If, as a result of shrinkage, the gingival margin is located apical to the cementoenamel junction, clinical probing of any pockets will underestimate the actual loss of attachment. True attachment loss must be measured from the cementoenamel junction to the base of the pocket.

Gingival Swelling

Enlargement of the gingiva is a symptom of gingivitis that may remain if progression to periodontitis occurs.

If the gingivae are edematous or hyperplastically enlarged beyond the cementoenamel junction, pocket depth (probing depth) may be overestimated, while true attachment loss may be underestimated (Fig. 212).

Pocket Activity

The activity of a pocket and the frequency of active episodes are of more importance than absolute pocket depth in mm, especially in regard to treatment planning and prognosis.

Bleeding on probing, presence of exudate, and suppuration after application of finger pressure are all signs that an *active phase of periodontitis* is in progress (Davenport et al. 1982). Such signs are often observed in the aggressive forms of periodontitis, but may also be seen in older patients with chronic periodontitis and reduced host immune response.

Pocket and Furcation Abscess

An additional symptom of active periodontitis is the pocket or furcation abscess. This develops *during an acute exacerbation* if necrotic tissue cannot be either resorbed or expelled (for example, due to closure of the coronal gingiva above deep pockets, furcations and retentive areas). An abscess (macronecrosis) may also be the consequence of *injury*, for example biting upon hard, sharp foodstuffs, improper oral hygiene efforts (e.g., a broken toothpick), or iatrogenic trauma. In rare instances a periodontal abscess can develop into a submucosal abscess (Parulis).

An abscess is one of the few manifestations of periodontitis that may elicit *pain*. If an abscess is expansive, extending to the apical region, the tooth may become sensitive to percussion. A painful abscess must be drained on an emergency basis, either by way of the pocket orifice itself or via incision through the lateral wall. An abscess may release spontaneously through a fistulous tract or via the gingival margin.

Fistula

A fistula may be the result of spontaneous opening of an abscess if the gingival margin is sealed. If the underlying cause (active pocket) is not eliminated, the fistula may persist for an extended period of time without any pain. The orifice of the fistula is not always located directly above the acute process; this can lead to improper diagnosis of the location of an abscess (probe the fistulous tract with a blunt probe!).

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Shrinkage—Swelling

In advanced periodontitis, additional clinical symptoms include shrinkage (gingival recession) or swelling of the gingiva; tooth migration and tipping of individual teeth or groups of teeth can occur. The result of such tooth movements is the creation of diastemata, which can present an esthetic problem. There are many factors that could be responsible for tooth migration and it is not possible in every case to determine the specific cause. It is nevertheless clear that compromised tooth-supporting apparatus is a prerequisite for tooth migration, tipping etc. Numerous other factors may also play a role: missing antagonists, functional occlusal disturbances, oral parafunctions (lip biting, cheek biting, tongue thrust etc.).

A tooth exhibiting a deep pocket on one side and intact periodontal fiber structure on the other side may migrate not so much as a result of pressure exerted by granulation tissue in the pocket, as by forces deriving from the still intact collagenous supracrestal fiber bundles in the healthy tissues. The fact that migrated teeth usually exhibit their unilateral pocket on the side opposite the direction of wandering would appear to support this hypothesis.

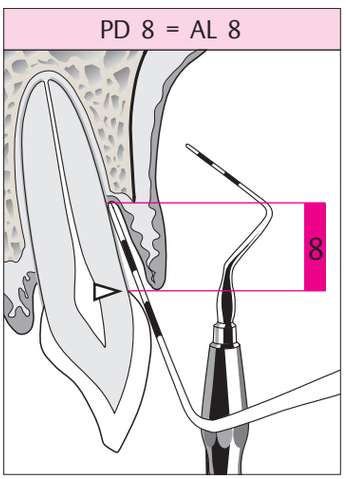
The figures below present typical *clinical* measurements (probing depth, recession of the marginal gingiva, *clinical attachment loss*) (see also p. 171).

Clinical Attachment Loss

210 Pocket Measurement: Probing Depth (PD) = Attachment Loss (AL)
The measurement (8 mm) is made from the gingival margin, which in this case is still in its normal position near the cementoenamel junction; only in such cases does probing depth correspond identically to attachment loss.

Right: The schematic clearly depicts that the pocket depth corresponds to the attachment loss.

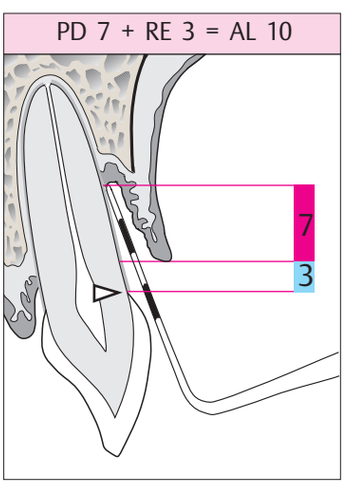




211 Pocket Measurement: Probing Depth Underestimates Attachment Loss
The measurement (7 mm) is made from the gingival margin, which is 3 mm apical to the cementoenamel junction. Thus, the true attachment loss is 10 mm.

Right: The schematic diagram reveals that the probing depth underestimates the attachment loss because of the recession (**RE**) of the gingival tissues.

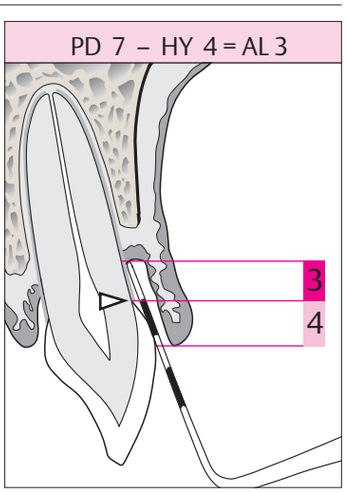




212 Pocket Measurement: Probing Depth Overestimates Attachment Loss
The measurement (7 mm) is made from the gingival margin, but the hyperplastic gingivae (**HY**) extend beyond the CEJ, creating a pseudopocket. The clinical attachment loss (**AL**) is 4 mm less than probing depth.

Right: The schematic diagram reveals that the attachment loss is overestimated as a result of the gingival swelling.





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Pocket Activity, Tooth Migration and Tooth Mobility

Pocket activity and tooth mobility are symptoms of severe, advanced periodontitis. Elevated tooth mobility must, however, be interpreted carefully because it can be influenced by numerous factors.

Even in a healthy periodontium, the teeth exhibit physiologic differences in mobility depending on number of roots, root morphology and root length (p. 174).

Even in a healthy periodontium, occlusal trauma can lead to an increase in tooth mobility (p. 461). In cases of periodontitis, it is the quantitative loss of bone that is the primary determinant of tooth mobility, but superimposed occlusal trauma can increase tooth mobility still further. In such cases, one may observe a continuously increasing (progressive) tooth mobility, which is very unfavorable in terms of single tooth prognosis.

Tooth Loss

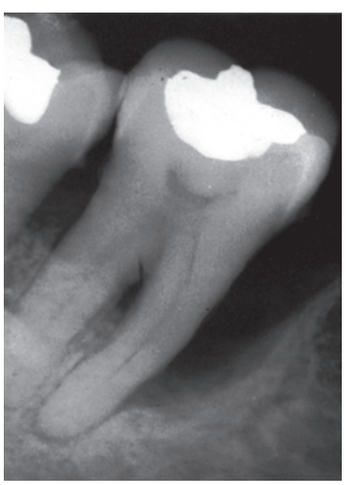
The final "symptom" of periodontitis, the one which ultimately stops the disease process, is tooth loss. It seldom occurs spontaneously because extremely mobile teeth that are no longer functional are usually extracted prior to spontaneous exfoliation.





213 Periodontal Fistula—Pus
13 mm pocket on the distal of tooth 11, which is a candidate for extraction. After probing, pus escapes from both the fistulous tract and the gingival margin.

Left: Pus exudes from an active pocket on tooth 11 after finger pressure is applied.





214 Periodontal Abscess
Originating from a 12 mm pocket on the mesial aspect of the tipped, vital tooth 47, an abscess has developed, which is just about to open spontaneously.

Left: The radiograph reveals a severe vertical bony defect on the mesial of tooth 47.





215 Tooth Migration and Tipping
Creation of a diastema by severe tipping of tooth 41 after loss of 42. Patient exhibits a heavy tongue thrust when swallowing.

Left: Tooth mobility. Increased tooth mobility can be caused by functional disturbances and/or by periodontal attachment loss. Mobility is measured clinically by using two instruments or one instrument and a fingernail (degrees of mobility, see p. 174).

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Chronic Periodontitis—Mild to Moderate

This 51-year-old male had received restorative dental care at irregular intervals. The dentist had never performed any periodontal diagnostic procedures, nor any therapy. The patient himself complained of occasional gingival bleeding and calculus that bothered him. He was unaware of any periodontal disease and felt that he was completely functional in mastication.

Findings: See charting and radiographic survey (p. 109).

Diagnosis: Slowly progressing, mild to moderate chronic periodontitis (Type II).

Therapy: Motivation, oral hygiene instruction and follow-up initial therapy. Closed scaling and root planing. After *re-evaluation*, modified Widman procedure at several sites. No systemic therapy. Possible bridgework in mandibular posterior segments.

Recall: Every 4–6 months.

Prognosis: Even if the patient's compliance is only average, the prognosis is good. In cases such as this, the dentist or periodontist is always "very successful."



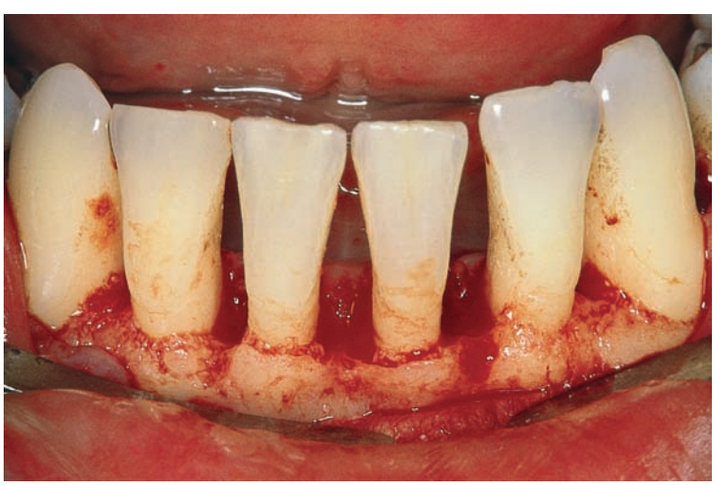




216 Clinical Overview (above)
A cursory inspection reveals only gingivitis and the absence of interdental papillae. The mandibular first molars were extracted 30 years previously, with resultant slow tooth migration, tipping and diastemata. Occlusion is poor.



217 Plaque Disclosure, Oral Hygiene (right)
The labial surfaces exhibit almost no plaque accumulation, while the interproximal areas are filled with plaque and calculus.

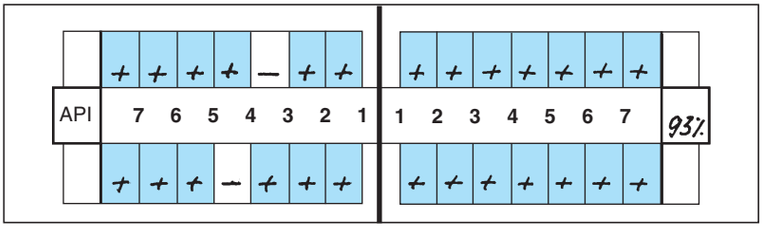


218 Mandible During the Flap Procedure
The facial crest of bone is somewhat bulbous but shows no signs of active destruction. Interdental 3 mm craters were detected.

Treatment consisted solely of root planing, minor recontouring of the bulbous bony margin (osteoplasty) and repositioning of the flaps at their origin position (no apical repositioning).

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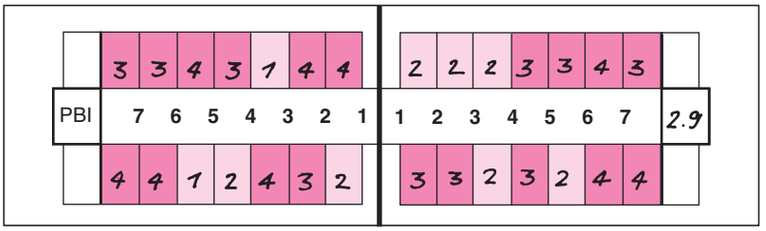
API



219 Aproximal Plaque Index (API) and Papilla Bleeding Index (PBI)

API 93 %. Oral hygiene is poor. Almost all of the interdental spaces harbor plaque.

PBI



PBI 2.9. The index is very high. All papillae bled during performance of the PBI.

The procedure for performing this index is described on pp. 68–70.

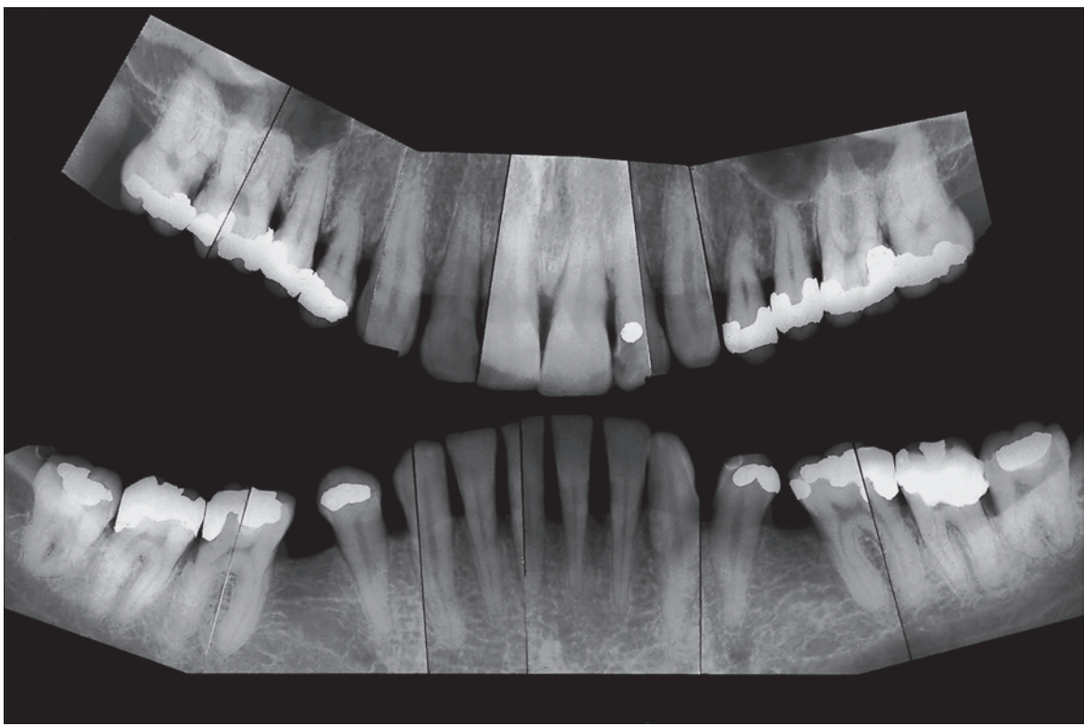
Name Mr. W. F.	Age 51	Date	CAUTION/ALERT!
Chief complaint: Gingival bleeding, calculus	Pockets (mm), furcation (F) severity grades 1 3, tooth mobility TM grades 1–4 Recession (Re) in mm, gingival width (Ag) in mm, frenula, exostoses (E), secretion S)		
Oral hygiene: Insufficient. Halitosis			
Attitude?			
Centric relation (CR): Initial contact 2 / 2 Deviation in initial contact vo re 1 mm right after articulation Contacts during lat. excursions Right 7/8 | 567/678 B — | 237/3 8 Left from (B) 7/8 | 23 67/34 78 (B)			
Parafunctions History: — Clinical: + (Attrition)	Etiology + Pathogenesis Microbial infection: ☐ Mild ☒ Moderate ☐ Severe Resistance: ☒ Good ☐ Moderate ☐ Weak Occlusal trauma: ☐ Slight ☒ Moderate ☐ Weak Clinic. Course: ☒ Slow ☐ Rapid ☐ Very rapid		
TMJ Bilateral clicking	Diagnosis CP Mild to moderate, slowly progressing, localized; some interdental gingival recession		
Morph. and funct. peculiarities Crossbite 2/321	Prognosis Good		

220 Periodontal Charting—I

This periodontal chart (description p. 194, Fig 439) is used in many dental offices, and has convenient places for recording probing depth, recession, furcation involvement and tooth mobility using numerical scores.

This case exhibits uniform probing depths throughout, especially in the interdental areas. For an assessment of the true attachment loss, the millimeter measurements entered by "Re" (recession) must be considered; these indicate the amount of gingival shrinkage.

Tooth mobility is low (grade 0–2). Functional analysis revealed premature contact between the upper and lower right lateral incisors (cross bite, increased mobility). Disturbances in lateral excursion and protrusion.



221 Radiographic Survey

The radiographs confirm the clinical observations: localized, mild to moderate, mainly horizontal bone loss.

Note migration and tipping of several teeth in the mandible. Some of the restorative work is inadequate.

Most important is that the "strategically" important canines and molars will be easy to restore (no furcation involvement).

(Note: This is not a "panoramic radiograph" in the usual sense of that term. Rather this survey was prepared by cutting and fitting individual periapical radiographs into a unified whole. This practice provides a more detailed overview of each individual segment of the dentition than any available panoramic film.

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Chronic Periodontitis—Severe

For decades, this 61-year-old male patient had "scrubbed" his maxillary anterior teeth using a horizontal motion. He had never received oral hygiene instructions, and virtually all of other areas of his dentition were completely neglected. Restorations were inadequate.

Findings: See chart and radiographic survey (p. 111).

Diagnosis: Moderate to severe generalized chronic periodontitis (Type II B). The pronounced gingival shrinkage in the maxillary anterior sextant should not be confused with classical gingival *recession*!

Therapy:

- Immediate: Extraction of teeth 18, 17, 28 and 46 and the root of 41. The crown will be used as a temporary pontic by attaching it to the adjacent teeth (acid etch).
- Definitive treatment: Motivation, modification of oral hygiene, extraction of 26 and 31. Initial periodontal therapy (definitive debridement) and possible subsequent surgical procedures and a cast framework partial denture.

Recall: Every 4–6 months.

Prognosis: For the teeth to be maintained and treated periodontally, the prognosis is good.







222 Clinical Overview (above)
Noticeable in maxillary anterior region are the severe gingival shrinkage and the wedge-shaped defects (hard-bristle toothbrush, abrasive dentifrice). Periodontal pockets were "brushed away" by the patient!



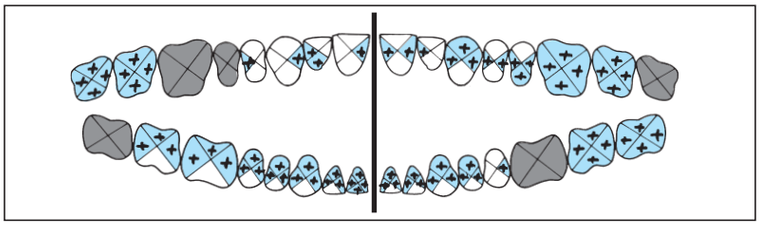
223 Mandibular Anterior Probing Depths (right)
Probing of a 9 mm pocket on the mesial aspect of 41 provoked only slight bleeding. The cervical areas of the mandibular anterior teeth also exhibit abrasion.



224 Retained Root (Tooth 41)
Tooth 41 was no longer functional and had to be extracted. Its root, which had virtually no bony support remaining, was amputated and the natural crown was used as a *temporary pontic* during periodontal treatment, by bonding it to the adjacent teeth using the acid etch technique.

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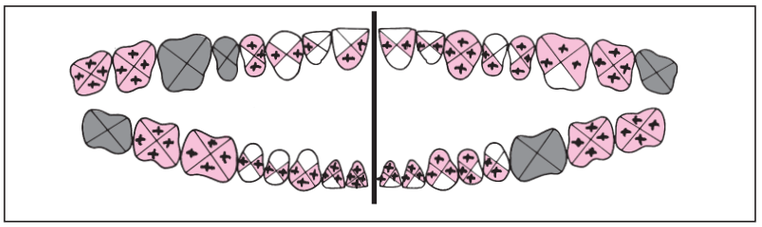
PI



225 Plaque Index (PI) and Bleeding Index (BOP)

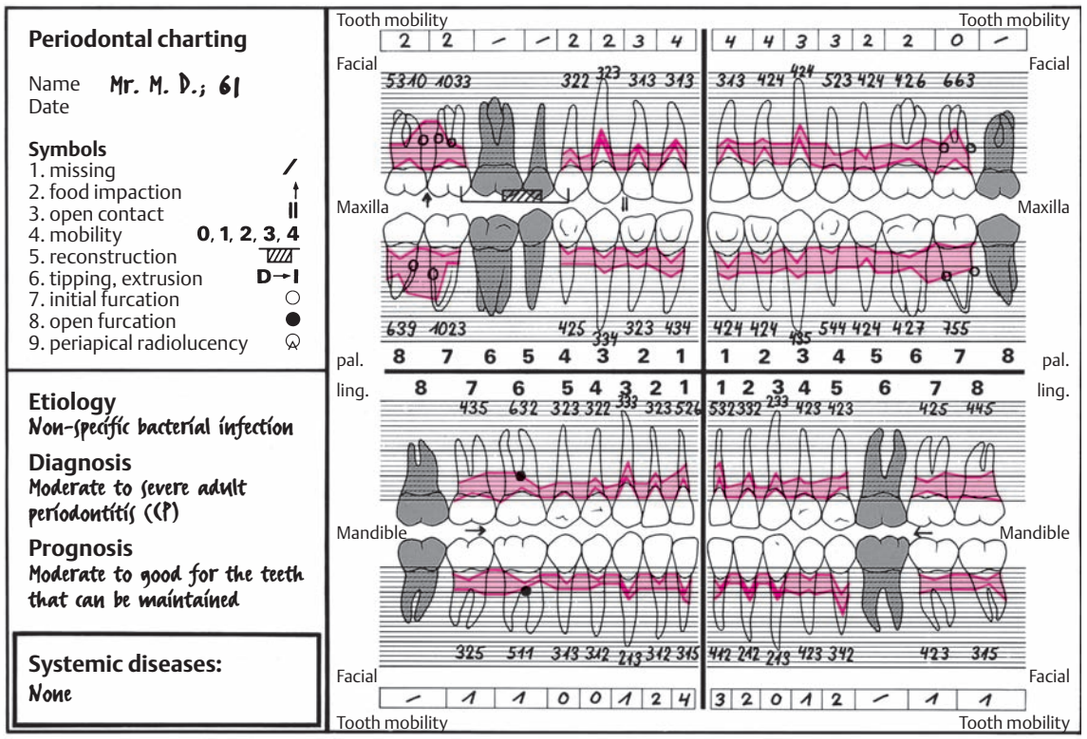
PI 69 %. With the exception of the maxillary anterior and some facial surfaces in the mandibular anterior regions, plaque was present on almost all tooth surfaces.

BOP



BOP 75 % of the pockets examined bled after gentle probing.

All teeth present were probed at all four surfaces (mesial, distal, facial and oral; p. 68–69).



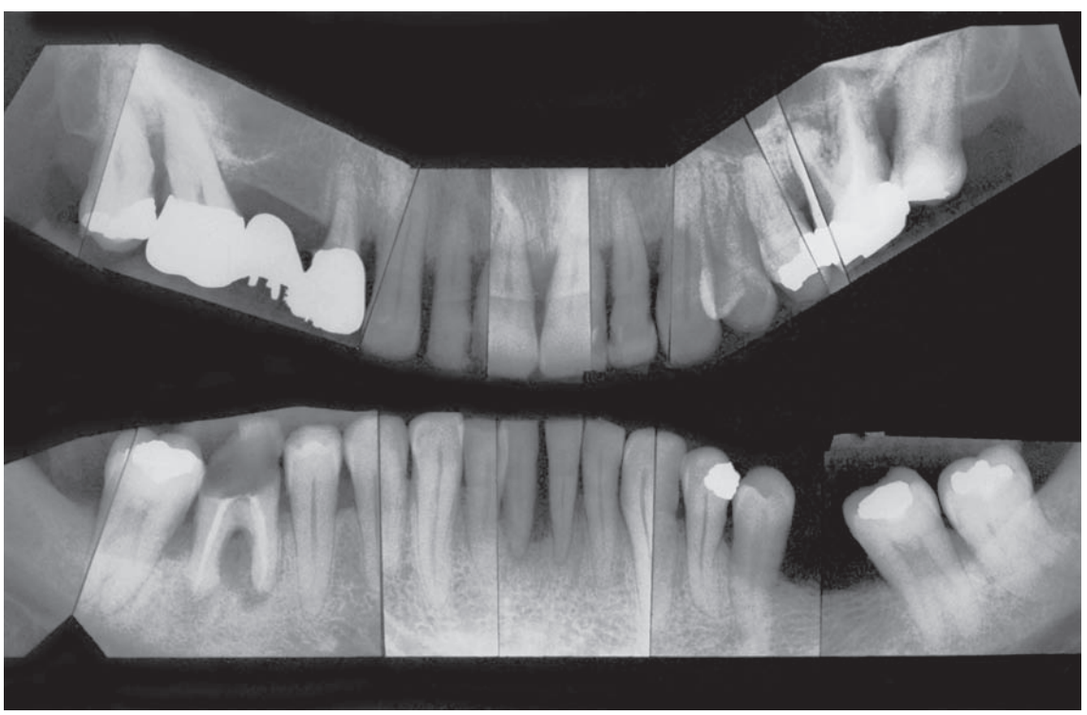
226 Periodontal Charting—II

Using this modified "Michigan Chart," gingival contour, probing depths and attachment loss are represented visually.

Probing depths around each tooth are measured first from the facial aspect at mesial, mid-buccal and distal sites, then from the lingual aspect at 3 similar sites.

Because of the pronounced gingival recession, the pockets in the anterior area are not very deep, despite great attachment loss. All anterior teeth were highly mobile.

Complete information concerning the periodontal chart II and its use is presented on page 194 (Fig. 440).



227 Radiographic Survery

The radiographs reveal a very typical, irregular distribution of bone resorption; this is commonly observed in older patients. While some teeth and/or some tooth surfaces have already lost all attachment, hardly any bone resorption is observed on the mandibular canines and the premolars.

At tooth 46, the apical and periodontal lesions appear to communicate.

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Aggressive Periodontitis—Ethnic Contributions?

This 31-year-old female immigrated to Switzerland from Ethiopia ten years previously. She complained of loose teeth and the formation of a diastema between her maxillary central incisors. Hemorrhage occurred during tooth brushing. She had never undergone any periodontal treatment.

Findings: See microbiology (Fig. 229), charting and radiographs (p. 113).

Diagnosis: Aggressive, moderate periodontitis; an ethnic component must be considered.

Therapy: Motivation, oral hygiene instruction and follow-up initial therapy. Systemic administration of metronidazol and amoxicillin (van Winkelhoff et al. 1989). After re-evaluation, flap surgery in quadrants 1, 2 and 4 (excluding the maxillary anterior). Extraction of teeth 18 and 28.

Supportive Therapy: Recall at 3-month intervals.

Prognosis: With good patient compliance, the prognosis is good.







228 Clinical Overview (above)
Pigmented gingiva; very little sign of inflammation. Interdental plaque and bleeding on probing. Calculus on the mandibular anterior teeth. The diastema between the maxillary central incisors had developed in the previous two years; tooth 11 is slightly supererupted.

Bacterial type		Relative percentage	Number of bacteria
Black pigmenting bacteria		50%	
Aa	*Actinobacillus actinomycetemcomitans*	+ + + +	≥10^6
Pg	*Porphyromonas gingivalis*	+ + + +	≥10^6
Tf	*Tannerella forsythensis*	+ + +	~10^4
Pi	*Prevotella intermedia*	+ +	~10^3
Ec	*Eikenella corrodens*	+ + + +	≥10^6
Fn	*Fusobacterium nucleatum*	–	–

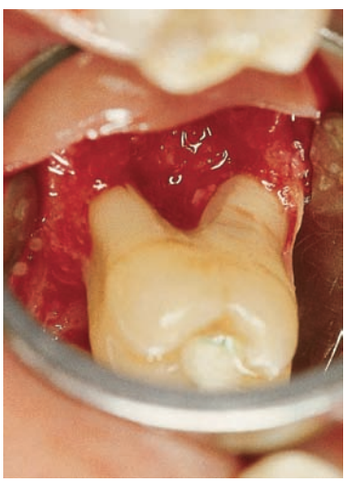
Bacterial Cultures	
Bacterial counts Classes 0 – 5	
0	non-detectable
1	below 10^5
2	≈ 10^5
3	10^5 to 10^6
4	≈ 10^6
5	above 10^6

229 Bacterial Cultures (right)
In deep pockets, high levels of *Aa*, *Pg*, *Ec*.
Right: Classification of the bacterial counts (Socransky et al. 1991).

230 Flap Surgery
Clinical view during flap surgery on teeth 25, 26 and 27, following amputation of the distal roots of teeth 26 and 27.

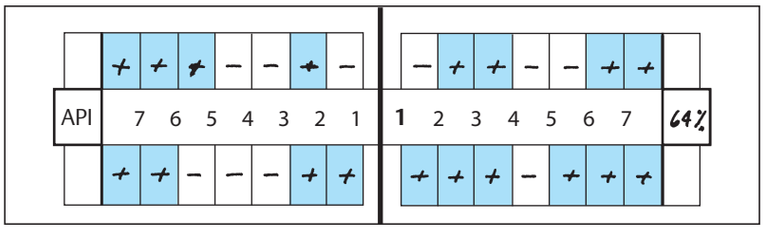
Right: Tooth 27 as viewed from the distal aspect (mirror image).





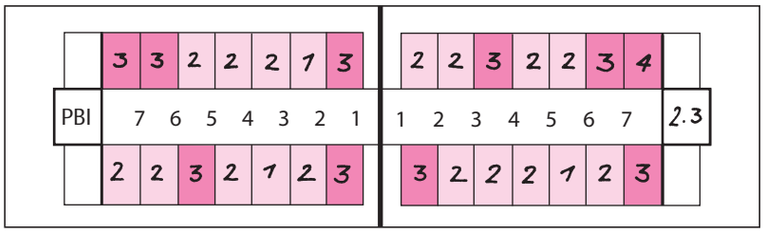
Courtesy *J-P. Ebner*

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231 Approximal Plaque Index (API) and Papilla Bleeding Index (PBI)

API 64 %. Interdental hygiene is inadequate; smooth surfaces are relatively clean.



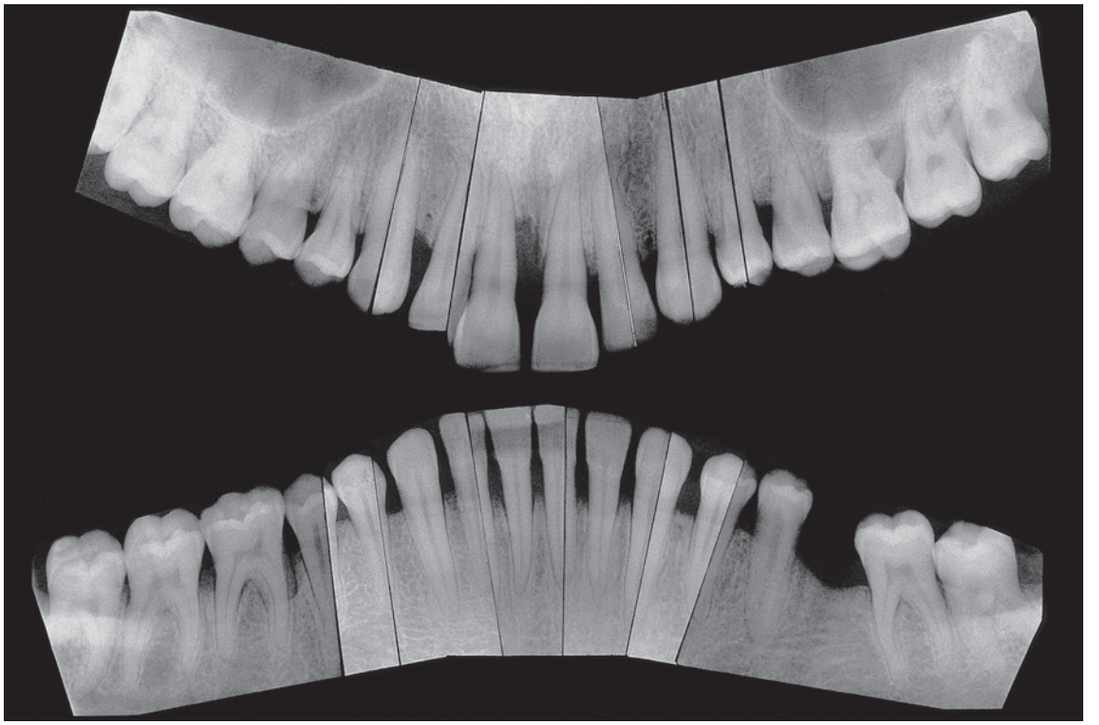
PBI Bleeding was more or less pronounced from all interdental papillae. The papilla bleeding index is very high at 2.3.

Name Mrs. W. F.	Age 31	Date	CAUTION/ALERT! EOP !
Chief complaint: Diastema between 1+1, gingival pain	Pockets (mm), furcation (F) severity grades 1 3, tooth mobility TM grades 1–4 Recession (Re) in mm, gingival width (Ag) in mm, frenula, exostoses (E), secretion S)		
Oral hygiene: moderate Halitosis			
Attitude? Positive. "Keep my teeth!"			
Centric relation (CR): Initial contact Deviation in initial contact dir. 0.5 mm right after articulation Contacts during lat. excursions Right: group-contact — B — Left: group-contact von (B) — (B)			
Parafunctions History: clenching Clinical:	Etiology + Pathogenesis Microbial infection: Mild / Moderate / Severe Resistance: Good / Moderate / Weak Occlusal trauma: Slight / Moderate / Weak Clinic. Course: Slow / Rapid / Very rapid		
TMJ slight popping L and R	Diagnosis AP (formerly RPP/Un-treated LJP; ethnic component)		
Morph. and funct. peculiarities negroid pigmentation	Prognosis ? Attempt initial therapy with/without antibiotics?		

232 Periodontal Charting—I

Probing revealed a very irregular distribution of attachment loss. Remarkable are the deep pockets on teeth 11, 26, 27 and 46. Teeth 26 and 27 exhibit Class III furcation involvement. The furcations are through-and-through from buccal to distal.

Tests of function revealed premature contacts between teeth 26 and 35, with a mild deviating slide of the mandible forward and to the right. This led to trauma on tooth 11. With the exceptions of teeth 11 and 21, the teeth are not mobile. The history revealed that the patient occasionally clenches during the day.



233 Radiographic Survey

The pronounced attachment loss revealed in the periodontal charting is corroborated in the radiographs. The distobuccal roots of teeth 26 and 27 exhibit almost no bony support.

The slightly elongated and distally migrated tooth 11 exhibits a deep pocket on the mesial surface. A typical situation: The most severe attachment loss on a tooth that has migrated is usually on the side opposite the direction of migration.

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Aggressive Periodontitis—Acute Phase

This 32-year-old female was referred by her general dentist because of multiple periodontal abscesses. She complained for years about ever-recurring pain and "pus discharge" from her gingiva.

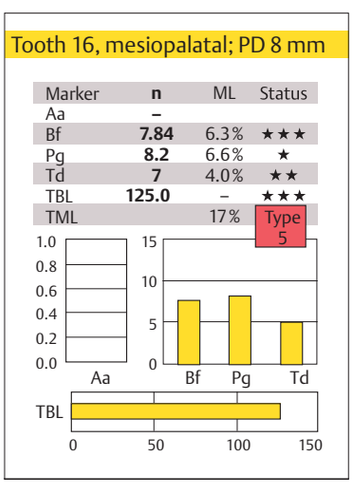
Findings: See charting and radiographs (p. 115) and microbiologic DNA test (IAI Padotest 4.5; Fig. 235 and p. 184).
Diagnosis: Aggressive, moderate, localized profound and acute periodontitis (active stage).
Therapy: Incision of the abscess; motivation, oral hygiene instructions and check-up, immediate extraction of teeth 25, 37, 32, 31, 41 and 42. Temporary replacement for mandibular anterior teeth. Supra- and subgingival scaling. Systemic antibiotic supportive therapy (metronidazol), and topical application of metronidazol (p. 289) at recall appointments. At Phase 1 evaluation, decision concerning additional procedures, depending upon patient compliance (radical, maintenance or regenerative/GTR).
Supportive Therapy: Initially, very short-interval recall.
Prognosis: With the exception of teeth 15, 24 and 37, and with good patient compliance, the prognosis is average.

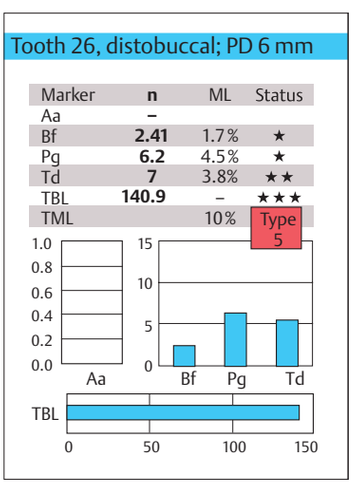




234 Clinical Overview (above)
Acute stage of periodontitis. Localized severe gingival inflammation, abscess formation and pus release from several pockets. Awkward occlusion and severe crowding in the area of the left incisors and canines.

235 DNA Test Results—IAI Padotest 4.5 (p. 184)
Results from the two maxillary teeth that were tested (teeth 16 and 26). Noteworthy is an elevated proportion of *Pg* with a high bacterial load (TBL).





Microbial Test Results

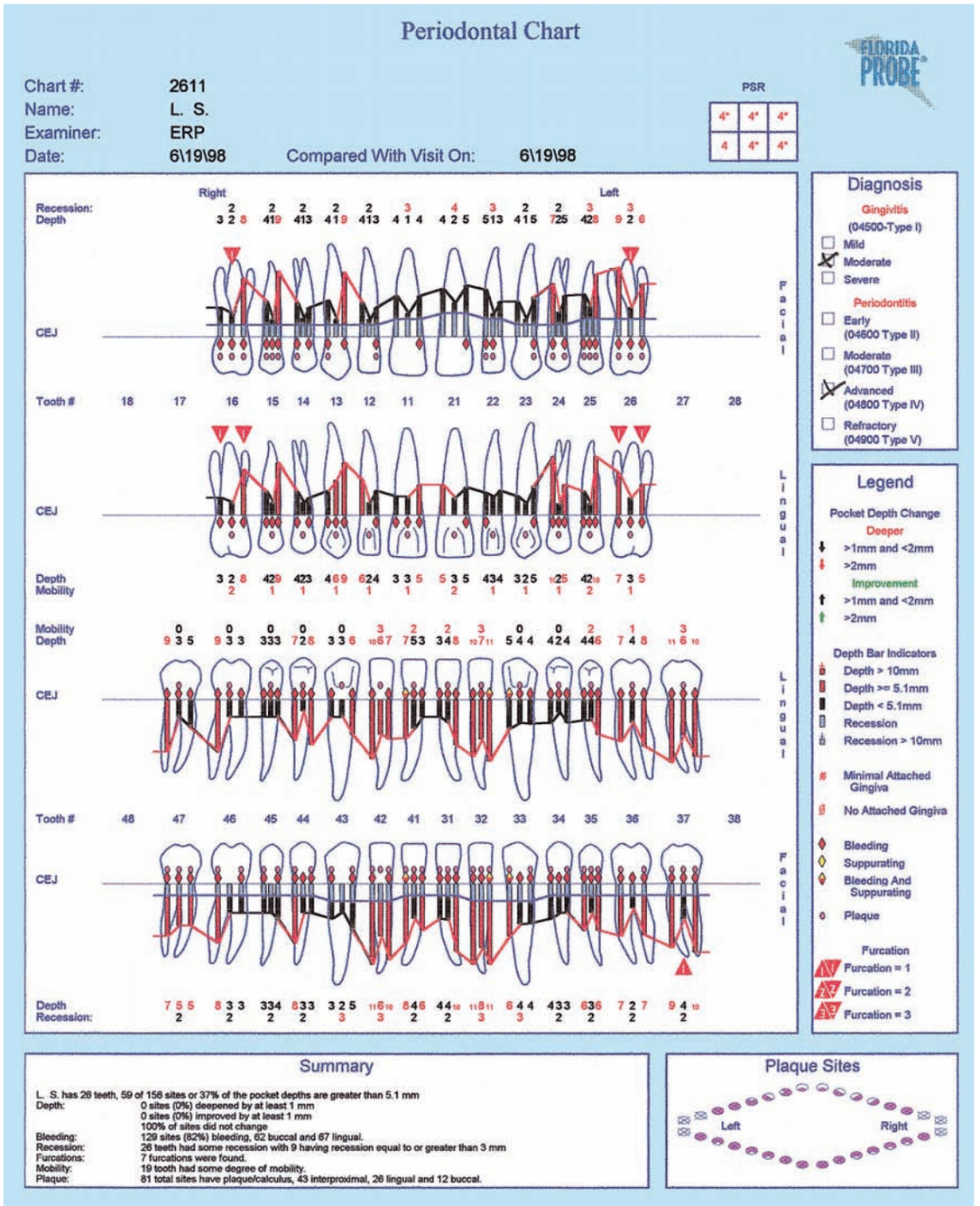
- *Aa:* not detected
- *Pg:* present in Red Complex (cf. pp. 37, 191)
- *TML:* Total marker load, i. e., pathogenic marker bacteria with 17 % and 10 % very high
- Finding: Pocket type 5 (p. 185)
- Treatment recommendation: Mechanical pocket therapy plus Metronidazol

236 Abscess Formation and Pus Release
Finger pressure applied to the gingiva elicited release of pus from the deep pocket between teeth 32 and 33.

Right: Clearly visible is the abscess between teeth 23 and 24, which emanated from the 10 mm pocket between these two teeth. The abscess will soon break through the muscosa.




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237 Periodontal Charting III—Florida Probe Charting

In the cases previously described, and in those which follow, probing depths were determined using a manual periodontal probe (e. g., CP-12 or CP-15 UNC; Hu-Friedy), and the data entered by hand into prepared charts.

With progressive computerization of the dental practice, electronic devices such as the Florida Probe are being used more and more often for clinical data collection (description p. 195).

The Florida Probe (Gibbs et al. 1988) functions with a standardized pressure (0.25 N). The measured values for recession, probing depth etc. are stored by means of a foot pedal. The findings can be seen on the monitor (information, motivation) and can be expressed in color.

In addition to recession, probing depths and attachment level, other parameters such as furcation involvement, tooth mobility, plaque accumulation, bleeding and suppuration can be noted tooth-by-tooth. At recall appointments, new software in this system (FP 32) permits a long-term comparison of the findings: “progression” or “improvement” are depicted graphically.

The present case is one of generalized, aggressive periodontitis (III B). Probing depths up to 5 mm are shown by black bars, deeper pockets by red bars.



238 Radiographic Survey

The radiographic picture corroborates the clinical examinations. Pronounced bone loss, almost to the apex of the rooth, is clearly visible on those teeth that were determined to be non-salvageable from the very beginning: teeth 25, 37, 32, 31, 41 and 42.

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Aggressive Periodontitis—Initial Stage

This 15-year-old female was referred from her private dentist because of "suddenly occurring" periodontal defects on all first permanent molars.

Bitewing radiographs had been taken periodically to check for dental caries, and gingival findings were noted. Thus, the localized osseous defects were not "serendipitous findings."

Findings: See charting and radiographs (p. 117).

Diagnosis: Incipient LJP (Type III A). Typical involvement of first molars, with as yet no pocket formation around incisors.

Therapy: Improvement of interdental hygiene, especially in the molar areas. After initial therapy, modified Widman surgery with intensive root planing (direct vision!) on all involved molars.

Microbiologic DNA test (e.g., PadoTest): *Aa* present, pocket type 4 (p. 185).

Systemic medicinal support with *tetracycline* (p. 289).

Possible filling of osseous defects.

Recall: Repeated professional plaque control during the wound healing phase; subsequent 6-month recall.

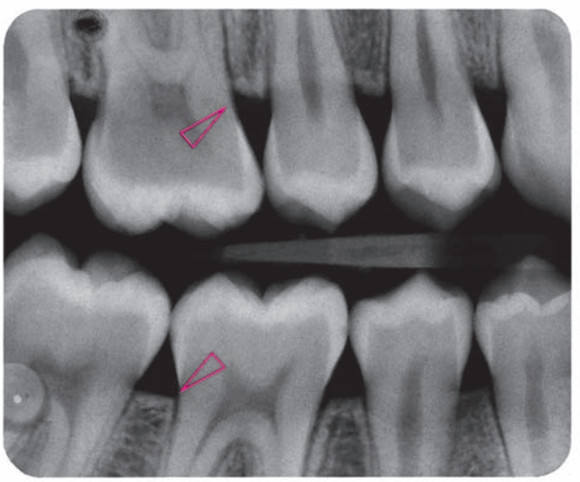
Prognosis: Good.



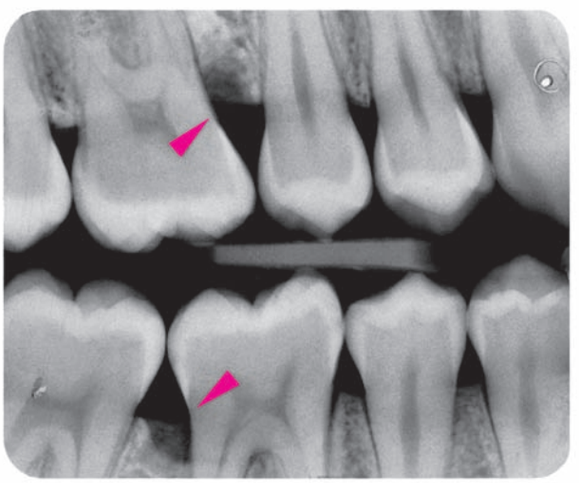




239 Clinical Overview—15-year-old Female (above)
Caries-free dentition.
Upon cursory inspection, the gingivae also appear healthy; however, numerous sites bled after gentle probing.



240 Bitewing Radiograph; Healthy 13-year-old (right)
The radiograph reveals healthy interdental septa of compact bone around the first permanent molars (empty arrows).



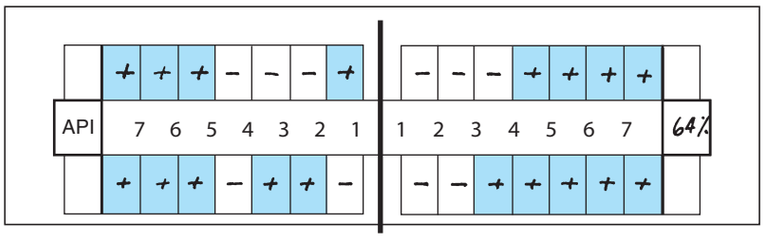
241 Bitewing Radiograph; 15-year-old with LJP (Type III A)
Two years later, obvious bony defects are visible mesial to tooth 16 and distal of 46 (red arrows), with identical defects on the opposite side: early diagnosis—**LJP**!
The significance of regular clinical and radiographic examination of young adults is clear.



Right: Osseous crater distolingual to tooth 46 during surgery.

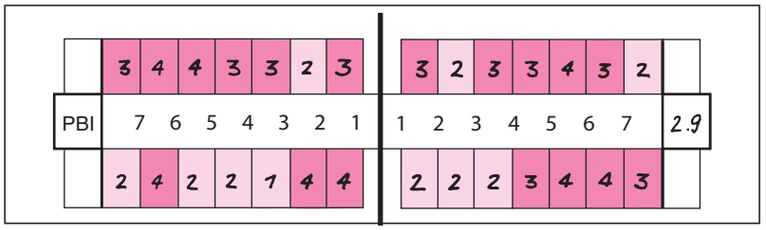
Radiographs courtesy
U. Hersberger

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242 API and Papilla Bleeding Index (PBI)

API 64 %. Only ten of the 28 interdental spaces examined were free of plaque (-), despite the fact that the dentition appeared relatively clean (Fig. 239).



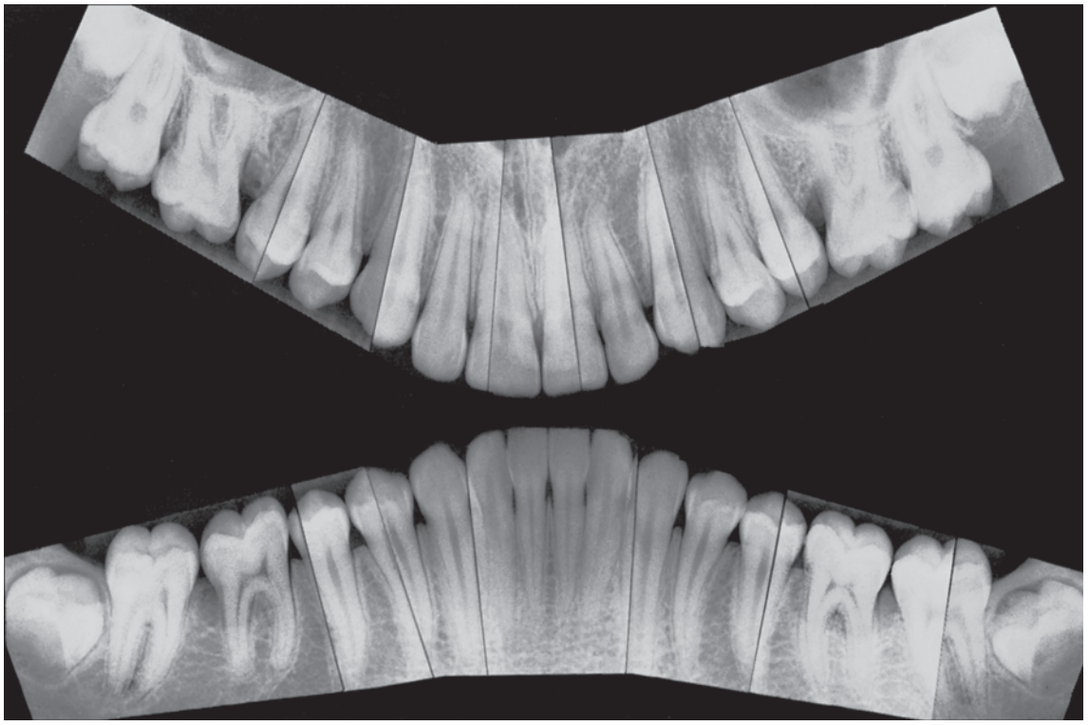
PBI 2.9; number of bleeding sites, 80!
Upon cursory inspection, the gingiva appeared to be inflammation-free. Only after recording the PBI was the massive gingivitis identified.

Name Mrs. W. F.	Age 15	Date	CAUTION/ALERT!
Chief complaint: None (referred)			
Oral hygiene: average Halitosis—			
Attitude? good			
Centric relation (CR): Initial contact Deviation in initial contact mm right after articulation Contacts during lat. excursions Right Left 432/543 B 7/7 34/34 von (B) 1 1 / 21 12 (B)	Pockets (mm), furcation (F) severity grades 1 3, tooth mobility TM grades 1–4 Recession (Re) in mm, gingival width (Ag) in mm, frenula, exostoses (E), secretion S)		
Parafunctions History: Clinical: —	Etiology + Pathogenesis Microbial infection: ☐ Mild ☒ Moderate ☐ Severe Resistance: ☐ Good ☒ Moderate ☐ Weak (low.) Occlusal trauma: ☒ Slight ☐ Moderate ☐ Weak Clinic. Course: ☐ Slow ☒ Rapid ☐ Very rapid		
TMJ WNL	Diagnosis AP/Type III A (LJP Localized juvenile periodontitis with generalized severe gingivitis. Initial stage of LJP.)		
Morph. and funct. peculiarities —	Prognosis With appropriate Tx – good		

243 Periodontal Charting—I

All four first permanent molars have probing depths up to 7 mm, which corresponds to about 5 mm of true attachment loss (see bitewing radiograph, Fig. 241). The two maxillary first molars exhibit Class F1 furcation involvement from the mesiopalatal aspect. There is no evidence at this time of pocket formation around the incisors, an area that is often affected in such patients.
The occlusion is normal, without parafunctions or occlusal trauma! The mobility of tooth 36 is slightly elevated.

Caution: In three of the four 7 mm pockets, *Aa* was identified (color circles).



244 Radiographic Survey

Routine periapical radiographs do not portray the osseous craters—especially on the mandibular first molars—as well as the bitewings (x-ray projection angle). No bone loss is apparent on any teeth other than the first permanent molars, especially the maxillary and mandibular incisors exhibit no bone loss.

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Prepubertal Periodontitis—PP (Aggressive Periodontitis)

This $2^1/_2$-year-old boy was referred from his private dentist. The medical history, gathered from his parents (Japanese mother, Swiss father) revealed that the mandibular deciduous incisors and the maxillary right deciduous incisor had "fallen out" in the previous few months. The left deciduous incisor and also the lateral incisor and canines in the maxilla were highly mobile, but their roots exhibited no resorption.
This case is difficult to classify in the absence of an identification of possible hematologic defects. Even though most of the teeth are affected, the relatively mild gingival inflammation and the absence of gingival hyperplasia speak against the generalized form of PP (Type IV B). Laboratory studies should be performed to rule out hypophosphatasia.

Findings: See charting and radiographs.
Diagnosis: Localized, pre-pubertal periodontitis (PP, Type IV B).
Therapy: Palliative or extraction (Page et al. 1983b). Intensive periodontal preventive maintenance upon eruption of the permanent teeth.
Prognosis: Poor for the deciduous dentition, questionable for permanent teeth.

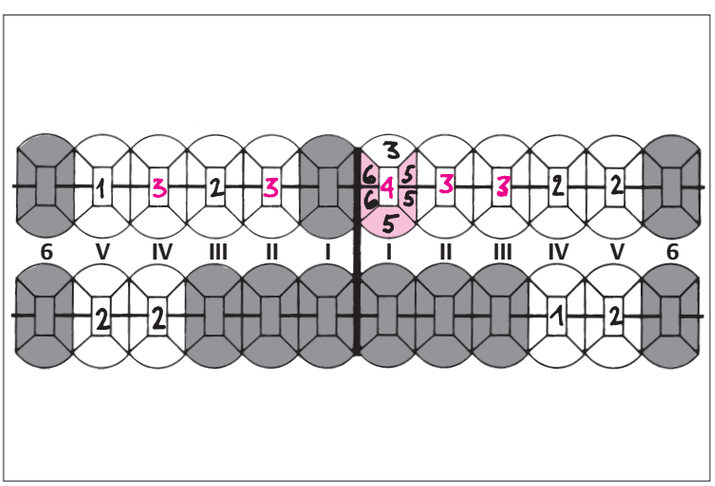
245 Clinical Picture—$2^1/_2$–year-old boy
Maxillary anterior tooth 51 and all mandibular anterior teeth, as well as the canines, exfoliated spontaneously.
The gingiva is unimpressive. Aphthous-like lesion near 73.

Right: The radiograph clearly depicts the pronounced attachment loss on the anterior teeth, in the presence of complete roots.
The pulp chambers appear to be above average in size (hypophosphatasia?).





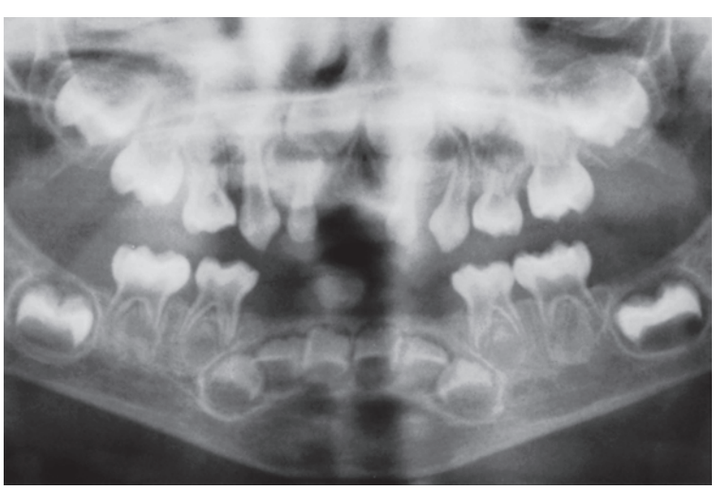
246 Probing Depths and Tooth Mobility
Pocket probing was only possible at four sites around tooth 61, because the $2^1/_2$-year-old boy was very sensitive and understandably impatient.
All deciduous teeth exhibited elevated mobility. Teeth 52, 54, 61, 62 and 63* were highly mobile.



**Tooth numbering* of deciduous teeth *see book-marker.*

247 Panoramic Radiograph
Irregular and in some areas extreme attachment loss is revealed on all maxillary deciduous teeth.
The mandibular deciduous molars appear to be only slightly involved at this time.
Tooth 61, which was present in the clinical picture above (Fig. 245) was spontaneously exfoliated before the panoramic radiograph was taken, some two weeks after the initial clinical visit.

Panorex courtesy *B. Widmer*



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Oral Pathologic Alterations of Gingiva and Periodontium*

As they traverse their clinical course, plaque-induced inflammatory gingival and periodontal diseases may be enhanced or co-initiated by hormonal disturbances and the side effects of systemic medications. Additional oral pathologic alterations accompanying systemic diseases are frequently observed on the gingiva and in the periodontium. In this milieu, clinical differentiation between gingivitis and periodontitis on the one hand and diseases of the oral mucosa on the other may become blurred.

It is impossible in this *Color Atlas of Periodontology* to depict and describe each and every oral pathologic alteration that also manifests on the *gingiva* or in the *periodontium*. A comprehensive treatment of *all* such oral lesions can be found in the *Color Atlas of Oral Pathology* (Reichart & Philipsen 1999).

In the pages that follow, several of the relatively frequently occurring diseases or alterations will be described:

Primarily Gingival Alterations

- Hormonal complications (p. 91)
- Hyperplasia caused by medicaments
- Gingival overgrowth, tumors
- Autoimmune diseases, desquamative and bullous gingival alterations, anomalies of keratinization, dermatologic diseases
- Specific infections
- Allergies
- Toxic manifestations
- Injuries, chemical injuries

Gingival and Periodontal Disorders

- Metabolic disturbances
- Nutritional deficiencies
- Genetically-related systemic syndromes
- Blood cell disorders
- Immune deficiency, AIDS

* In the new AAP Classification (Armitage 1999, pp. 519–522), a portion of the oral pathologic alterations on the *gingiva* are classified under Type I A and B, and in the *periodontium* under Type IV, "periodontitis as a manifestation of systemic diseases."

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Primarily Gingival Alterations (Type I B)

In the following pages, the disorders shown below with a black dot are described and depicted.

Hormonal Complications
- Pregnancy gingivitis (p. 92)
- ○ Gingivitis due to the "pill"
- Puberty gingivitis (p. 92)
- ○ Gingivitis menstrualis and intermenstrualis
- ○ Gingivitis climacterica

Medicament-Elicited Hyperplasia
- Phenytoin gingival overgrowth (p. 121)
- Dihydropyridine-induced gingival overgrowth (p. 122)
- Cyclosporine-induced gingival overgrowth (p. 123)
- Medicament combinations (cyclosporine/nifidepine, p. 124)

Gingival Overgrowth, Tumors
- Epulis (p. 125)
- Idiopathic and hereditary fibrosis (p. 126)
- Neoplasms
  - – benign tumors (p. 126)
  - – malignant tumors (p. 127)

Autoimmune Diseases, Desquamative and Bullous Gingival Alterations, Anomalies of Keratinization, Dermatologic Diseases
- Gingivosis (p. 128)
- Pemphigoid (p. 128)
- Pemphigus vulgaris (p. 128)
- ○ Epidermolysis bullosa
- ○ Exudative erythema multiforme
- Lichen (p. 129)
- Leukoplakia—precancerous lesions (p. 130)
- Erythroplakia (p. 130)
- ○ Dermatomyositis, scleroderma, psoriasis etc.

Specific Infections
- Herpes (p. 131)
- Aphthae ? (p. 131)
- ○ Toxoplasmosis
- ○ Actinomycosis, candidiasis
- ○ Gonorrhea, syphilis etc.

Allergies
- ○ To medicaments
- ○ To metals, mercury

Toxic Reactions
These may occur locally in the oral cavity through release of metal ions possessing high toxic potential, from dental materials (nickel, cadmium, bismuth, beryllium, vanadium etc; Wirz et al. 1997 a, b):
- ○ Dental materials of varying composition
- ○ Lead and other metals

Injuries, Chemical Injuries

Gingival and Periodontal Alterations (Type IV A/B)

Metabolic Disturbances
- Diabetes (p. 132)
- ○ Acatalasemia (Takahara's Disease)
- ○ Eosinophilic granuloma
- ○ Pre-leukemic syndrome

Nutritional Deficiency
Nutritional deficiency as a cause or co-factor in gingivitis or periodontitis is practically no longer observed in the western/northern hemispheres. In extreme conditions (e. g., in the Third World) one may observe:
- ○ Ascorbic acid deficiency (scurvy)
- ○ Kwashiorkor (protein deficiency) etc.

Genetically-Elicited Systemic Syndromes
It is not uncommon that rare, partially inherited systemic syndromes are characterized by severe periodontitis:
- Down syndrome (p. 134)
- Papillon-Lefèvre syndrome (p. 136)
- ○ Chediak-Higashi Syndrome
- ○ Hypophosphatasia (Rathbun Syndrome)
- ○ Pelger-Huet nucleus anomaly
- ○ Ehlers-Danlos syndrome etc.

Blood Cell Diseases
Every blood cell disorder reduces the immune response and, therewith, local defense reactions.
- ○ Leukemia
- ○ Panmyelopathy—Fanconi anemia
- ○ Cyclic neutropenia
- ○ Agranulocytosis
- ○ Erythroblastic anemia etc.

Immune Deficiencies
Any weakening of the immune system may elicit or enhance periodontitis. Perhaps most important today is infection by the human immunodeficiency virus HIV.
- HIV infection, AIDS (p. 139)

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Phenytoin-Induced Gingival Overgrowth

Phenytoin (hydantoin) prevents or reduces the incidence or severity of seizures associated with most types of epilepsy (grand mal), with the exception of petit mal. Phenytoin is also often prescribed following neurosurgical operations or cranial trauma. The anticonvulsive effect is probably due to the inhibition of the spread of nerve potentials in the brain cortex (Hassell 1981).

Systemic side effects of phenytoin are relatively minor. Some bone pathology may be observed after long-term therapy. The patient's mental capacity and reaction time may be negatively influenced (thus, no driver's license!).

The most important *oral side effect* is an often pronounced and secondarily inflamed gingival overgrowth.

Therapy: Motivation, repeated oral hygiene instructions, professional plaque and calculus removal. Once inflammation subsides, fibrous tissue can be excised. Lesions frequently recur.

In collaboration with the patient's physician, in some cases it may be possible to change to a different medication, e.g., valproic acid, benzodiazepine, barbituric acid derivatives, etc.

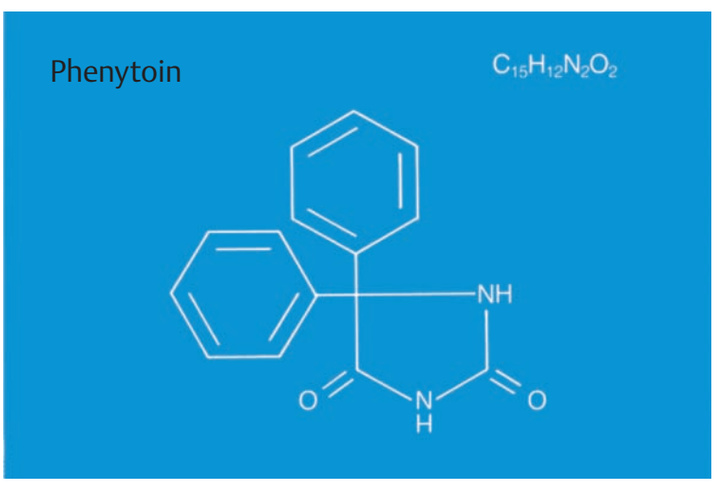


248 Mild Phenytoin-Induced Gingival Overgrowth
Fibrous form of phenytoin induced overgrowth in a 19-year-old female with epilepsy. Following initial periodontal therapy, a gingivoplasty was performed. The oral hygiene of this patient was relatively good, and this made it possible to eliminate secondary inflammation for the most part.

Phenytoin—Trade names

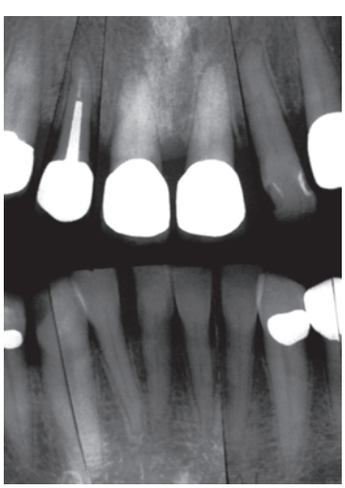
– Dilantin
– Antisacer
– Danten
– Diphantoin
– Diphenin
– Diphenylan Sodium
– Epanutin
– Minetoin
– Solantyl
– Tacosal

The Merck Index no. 7475 (12th ed. 1996, p. 1259)



249 Phenytoin—Structural and Chemical Formula
The medicament phenytoin (diphenylhydantoin) is a 5,5-diphenyl-2,4-imidazolodinedione.

The overgrowth occurs in only approximately 50 % of patients, usually young individuals (pharmacogenetic factor; Hassell 1981). Participating in the etiology are probably macrophages and fibroblasts that stimulate catabolic mediators, growth factors and collagen (Type IV; Sinha Morton & Dongari-Bagtzoglon 1999).





250 Severe Phenytoin Overgrowth—Severe Secondary Inflammation
This 44-year-old female had taken phenytoin on a chronic regimen for six years, since a neurosurgical procedure. She was mildly debilitated and therefore unable to perform adequate oral hygiene. Following initial periodontal therapy, gingivoplasty was performed (p. 367).

Left: There is radiographic evidence of bone resorption at the interdental septa.

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Dihydropyridine-induced Gingival Overgrowth

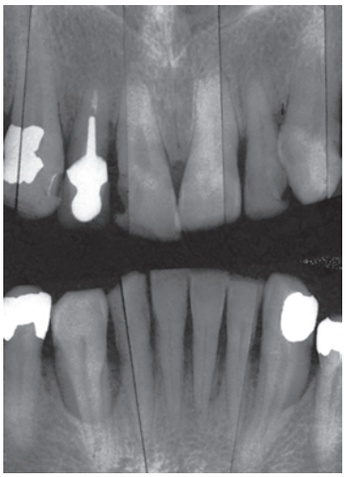
The substituted dihydropyridines (nifedipine, nitrendipine etc.) are calcium antagonists that reduce the influx of calcium ions into heart muscle and thereby reduce the strength of contraction as well as the vascular resistance. This reduces oxygen consumption by the heart while simultaneously increasing cardiac circulation. Thus, dihydropyridines exert both anti-anginal and anti-hypertensive effects. Some general side effects as well as interactions with other drugs are observed. In contrast to phenytoin, dihydropyridines are taken by primarily by *older* cardiac patients, who often have a pre-existing periodontitis.

The most important *oral* side effect is an often pronounced, secondarily inflamed gingival overgrowth. The pathogenesis of the overgrowth is suspected to be similar to that observed in phenytoin-treated individuals (connective tissue accumulation), but an increase in acid mucopolysaccharides (ground substance) also appears to occur (Lucas et al. 1985, Barak et al. 1987).

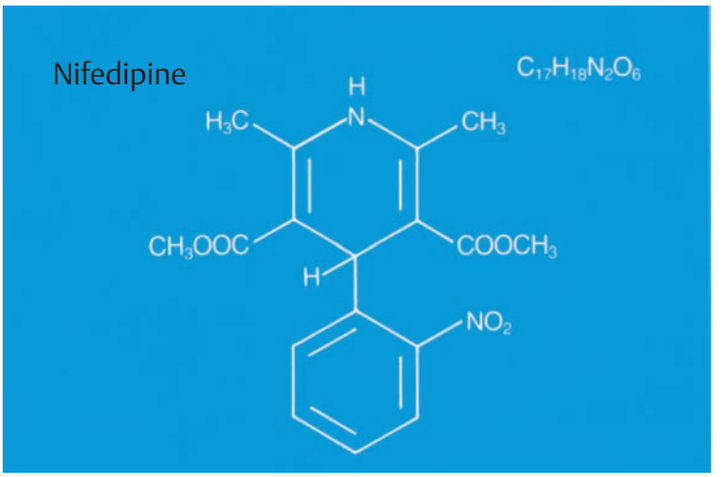
Therapy: Following patient motivation, repeated oral hygiene instruction and initial periodontal therapy, severe lesions may be eliminated surgically (gingivoplasty).

251 Mild to Moderate Nifedipine-induced Gingival Overgrowth
This 55-year-old male exhibited lesions of varying severity, with evidence of secondary inflammation. He had been taking "Adalat" (nifedipine) for two years because of his high blood pressure. The physician changed his medication to a different drug.
Right: The radiograph clearly shows that the gingival alterations were superimposed upon an existing periodontitis; the latter is *not* due to the drug.





252 Nifedipine—Structure and Chemical Formula
This drug is a 1,4-dihydro-2,6-di-methyl-4-(2-nitrophenyl)-3,5-pyridine-dicarbonic acid dimethyl ester.



Nifedipine—Trade names (from 31 available preparations)

– Procardia
– Adalat
– Adapress
– Aldipin
– Alfadat
– Anifed
– Bonacid
– Hexadilat
– Nifelan
– Zenusin

Merck Index no. 6617

253 Severe Nifedipine-induced Gingival Overgrowth
This 58-year-old Black female presented with severe, secondarily highly inflamed gingival overgrowth. The patient had taken "Procardia" (nifedipine) for four years. Note also the mild gingival pigmentation.



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Cyclosporine-induced Gingival Overgrowth

The immunosuppressive effect of cyclosporine-A ("Sandimmune," Novartis Co.) derives from the suppression of antibody formation against *T cell*-dependent antigens, the suppression of cell-mediated immunity, and interference with the production of cytokines (IL-2 etc).

Systemic side effects occur frequently: increased blood pressure, increase in body hair (hirsutism), formation of lymphoma, as well as *nephro*- and *hepatotoxicity*.

The oral manifestation of cyclosporine is the side effect of gingival overgrowth, which is usually secondarily inflamed. The incidence and severity of the gingival lesions are strictly dose-dependent and correlated to blood levels of the drug. New medicines (e.g., Prograf, Cellcept etc.) are reported to have fewer side effects.

Therapy: Good oral hygiene coupled with initial periodontal therapy can reduce both the inflammation and the overgrowth. If instituted early, such measures may actually prevent development. In severe cases, surgical gingivoplasty may be indicated. Organ transplant patients should receive comprehensive dental care *before* organ transplant (Rateitschak-Plüss et al. 1983a, b).

Cyclosporine-A—Trade names

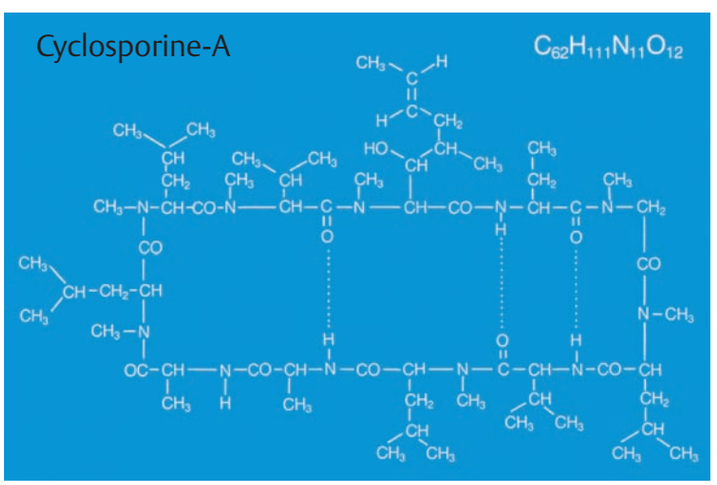
– Cyclosporine-A
– Sandimmune
– Neoral

Merck Index no. 2821



254 Mild to Moderate Cyclosporine-induced Gingival Overgrowth
This 45-year-old female began taking cyclosporine-A two years previously, following kidney transplantation. The gingival overgrowth is pronounced only in the maxilla; secondary inflammation is in evidence.
In order to avoid excessive doses of cyclosporine-A, this medicament is often combined today with azathioprine and/or cortisone.

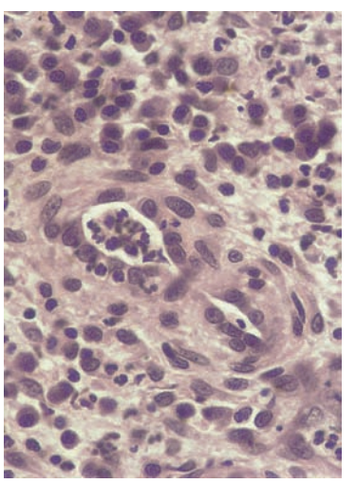




255 Cyclosporine-A—Structure and Chemical Formula
The drug is a cyclic peptide (undecapeptide) consisting of eleven amino acids. It is used to prevent rejection reactions following solid organ and bone marrow transplantation.

Left: Tolypocladium inflatum is the fungus from which cyclosporine was originally isolated during research on antibiotics.

SEM courtesy *R. Guggenheim*





256 Severe Cyclosporine-A Induced Gingival Overgrowth: Overdose
Dramatic enlargements such as in this 51-year-old female are infrequently observed today. This patient had received approximately three times the dosage of cyclosporine-A that is common today. In addition, the patient exhibited poor oral hygiene.

Left: PMN granulocytes and plasma cells are observed histologically in the infiltrated subepithelial connective tissue.

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Gingival Hyperplasia Following Combined Drug Therapies

Dihydropyridine and Cyclosporine

When systemic medications are prescribed, every effort should be made to reduce adverse side effects by setting the dose as low as possible. In certain cases, the use of a *combination of medications* can also serve to reduce the dosage. For example, today, following organ transplantation (kidney, heart etc.), cyclosporine is often combined with azathioprine and prednisone.

On the other hand, the necessary combination of two medications that may *both* possess the same side effect (e.g., gingival overgrowth) may massively increase the adverse effect.

Therapy: In the case presented here, a 30-year-old male (kidney transplant), cyclosporine was prescribed. This medicament can increase blood pressure, so she was also treated with the calcium-antagonist nifedipine (p. 122). Very severe gingival overgrowth was the consequence of this co-medication. In such cases, alternative antihypertensive medications should be employed.

257 Severe Gingival Overgrowth
Simultaneous use of Sandimmune (cyclosporine-A) and Adalat (nifedipine), in conjunction with inadequate oral hygiene, led to severe and secondarily inflamed gingival overgrowth (with formation of pseudopockets) throughout the entire dentition.



258 Following Gingivoplasty in the Maxilla
The "contouring" gingivectomy shown here in the maxillary anterior and canine regions was subsequently performed throughout the entire dentition.
Unfortunately, no particular efforts were expended to improve the patient's oral hygiene.



259 Recurrence in the Maxilla
Eight months after the surgery, a recurrence of the tissue overgrowth was obvious in the maxilla. This occurred despite changing the patient's antihypertensive medication to a different class of drugs (beta blocker). The patient's oral hygiene remains less than optimum.
Patients who have undergone a serious medical procedure such as organ transplantation have more important "problems" to deal with than the achievement of "maximal" oral hygiene!



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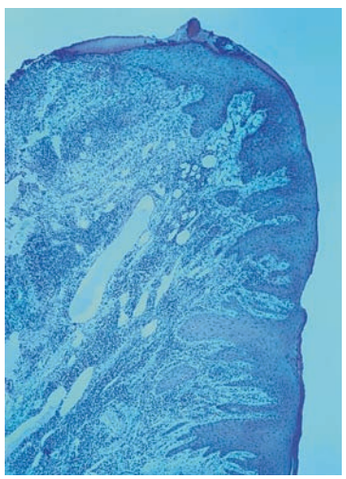
Benign Tumors—Epulis

Gingival epulis represents a family of benign tumors. The classification includes:

- Granulomatous epulis, pyogenic granuloma
- Giant cell epulis
- Fibrous epulis

Giant cell epulis and granulomatous epulis may develop relatively quickly; fibrous epulis grows slowly. The etiology of such tumors is not completely understood, but marginal irritation is one likely cause. Some pathologists contend that *giant cell epulis* is the *only true epulis*. It is a fact that no histologic differences exist between fibrous epulis and fibromas in other areas of the oral cavity.

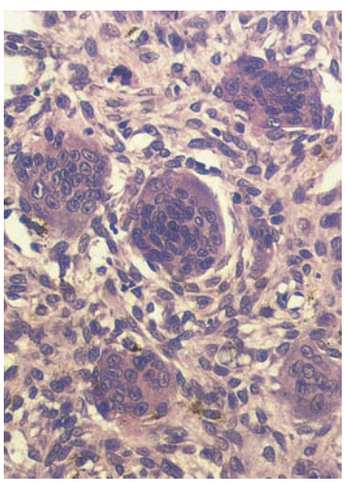
Therapy: Pyogenic granuloma and fibrous epulis can be removed by simple excision.
The giant cell epulis has a tendency to recur; following excision of such tumors, a gingival flap should be reflected, the tooth (and root) surfaces thoroughly cleaned and planed, and the bone filed.





260 Pyogenic Granuloma, Granulomatous Epulis
Localized, tumor-like bright red, soft mass on the labial gingival margin in a 34-year-old female. Epulis is usually seen in the papillary region, and less frequently, as in the present case, on the gingival margin. When probed or injured, the lesion exudes a copious mixture of blood and pus.

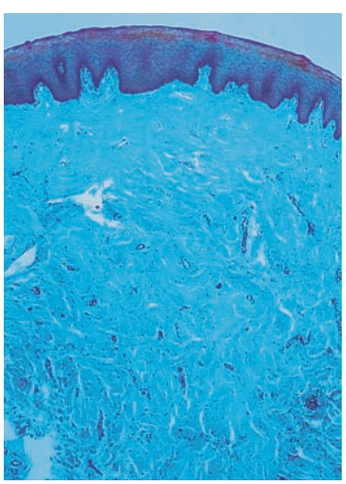
Left: The histologic view exhibits a loose granulation tissue that is highly vascularized (HE, x40).





261 Giant Cell Epulis—"True" Epulis
Clinically resembling the granulomatous epulis, the giant cell epulis can only be differentiated and diagnosed histologically. Such lesions can become very large and, as in this 50-year-old female, can cause displacement of adjacent teeth.

Left: The histologic section reveals an inflammatory infiltrate including *multinucleated giant cells* in the subepithelial connective tissue (HE, x 400).





262 Fibrous Epulis
This 45-year-old female exhibited a localized, fibrous, firm mass upon the gingiva between the central and lateral incisors. The etiology of such lesions can only seldom be ascertained.

Left: Histologically one observes an accumulation of fibrous connective tissue. If the mass becomes secondarily inflamed, a typical inflammatory infiltrate can be expected.

Courtesy *B. Maeglin*

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Benign Tumors—Fibrosis, Exostosis

The list of benign tumors of the oral cavity is long (Pindborg 1987, Reichart & Philipsen 1998). Mention will be made here of only *gingival* tumors, as well as gingival and osseous lesions that must be distinguished from the plaque-elicited, inflammatory swellings (gingivitis) and epulis:

- Fibrosis
- Exostosis
- ○ Verrucous hyperplasia, papilloma, hemangioma, gingival cysts, peripheral ameloblastoma, nevi

Fibrosis and exostosis may be localized or generalized on the gingiva. Their causes are for the most part unknown. Hassell & Jacoway (1981a, b) described a genetically determined (autosomal dominant) form of gingival hyperplasia (Elephantiasis gingivae).

Therapy: True gingival hyperplasia (histologically determined) is treated by simple gingivoplasty. Osseous thickening can be reduced by osteoplasty following flap reflection. These procedures may also be combined (Fig. 264, right). Recurrence is, however, frequent.

263 Hereditary Gingival Overgrowth
This 28-year-old male exhibited generalized gingival overgrowth with regionally varying severity. His family history revealed that similar gingival alterations had been observed in his father (who today wears a complete denture). The tissue overgrowth occasioned pseudopocket formation; these act as niches for plaque accumulation and therefore can lead to secondary inflammation. Following surgical resection, such overgrowth often recurs.



264 Idiopathic Gingival and Osseous Thickening
This 26-year-old female exhibited pronounced gingival overgrowth *and* osseous thickening. The latter could be diagnosed by passing a sterile injection needle through the soft tissue ("sounding").

Right: The treatment consisted of a combined procedure including gingivoplasty and osteoplasty following flap reflection. Depicted is the suture closure of the flap, which was previously thinned by external gingivectomy.





265 Exostosis
Exostosis is a harmless "idiopathic" thickening of bony tissue, whose cause is unknown (bruxism?). Such osseous lesions can be left untreated if they do not negatively influence function, well-being or periodontal health (niches?).
The maxillary right segment exhibits a particularly pronounced idiopathic osseous thickening, which could render oral hygiene more difficult.

Courtesy *B. Maeglin*



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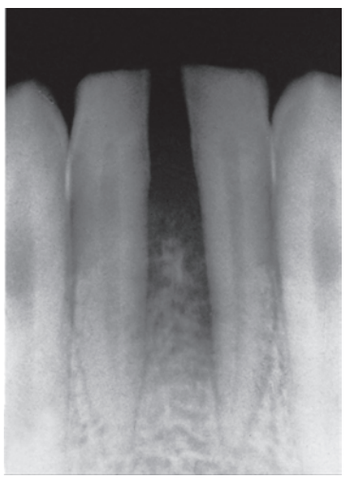
Malignant Tumors

- Carcinoma
  - Melanoma
- Sarcoma (chondrosarcoma, fibrosarcoma, rhabdomyosarcoma, lymphoma etc.)

Malignant epithelial and mesenchymal tumors are frequently observed on the mucosa of the oral cavity. In the Western world, oral carcinoma accounts for 1–5 % of all carcinomas (Pindborg 1987). Malignant tumors are, however, seldom observed on the *gingiva*.

In addition to *primary tumors*, the gingiva may also be the recipient site for metastases, from the kidneys, lungs, prostrate, breast or other organs.

Therapy: If there exists even the slightest suspicion of malignancy, the patient should be referred immediately to the oral surgeon, who may perform diagnosis, biopsy and frozen-section diagnosis, as well as radical surgical removal, radiotherapy and/or chemotherapy. The dentist should avoid manipulation of the tumor mass, and should never attempt a biopsy!

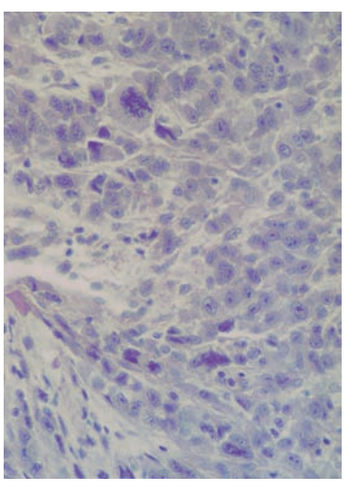




266 Chondrosarcoma
This 25-year-old female complained of a large swelling in the mandibular anterior area. The tumor extended from the gingiva into the oral mucosa and was ca. 2 cm wide. Histopathologic diagnosis: Highly differentiated chondrosarcoma. Metastases had not been detected up to this time.

Left: The radiograph reveals resorption of bone between the mandibular central incisors.

All photos courtesy *B. Maeglin*





267 Rhabdomyosacroma
A large, epulis-like swelling in a 38-year-old female was diagnosed by the pathologist as the rare malignant rhabdomyosacroma. The tumor grows by invading the alveolar bone. Metastasis to other skeletal sites occurs rapidly.

Left: The histologic picture is one of growth of “strands” of the malignant tissue. Note the dramatic number of mitotic figures (HE, x400).





268 Adenocarcinoma—Metastasis
Poor wound healing occurred in this 63-year-old male following extraction of tooth 45. A large swelling developed one month later on the right side of the mandible. Histopathologic diagnosis: Poorly differentiated, clear cell adenocarcinoma. (Metastasis from prostate carcinoma!)

Left: The radiograph depicts the non-healing alveolus of tooth 45.

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Gingivosis / Pemphigoid

Mild forms of gingivosis (desquamative gingivitis) are characterized by spotty gingival erythema. In more severe cases, epithelial desquamation occurs. If blister formation is also in evidence, the clinical descriptor is pemphigoid.

Therapy: True "causal" therapy is not possible. Therapy therefore is polypragmatic and symptomatic (pain medications and vitamin-A containing ointments). In severe cases (pemphigoid) topical (and systemic) corticosteroid preparations may be indicated.

Pemphigus vulgaris

Pemphigus vulgaris may affect the skin, as well as all mucosal surfaces and the gingiva. With or without blister formation, the epithelial layer sloughs, leaving behind expansive and painful erosions. The histologic diagnosis may be substantiated by immunofluorescence serology and by identification of "Tzanck cells."

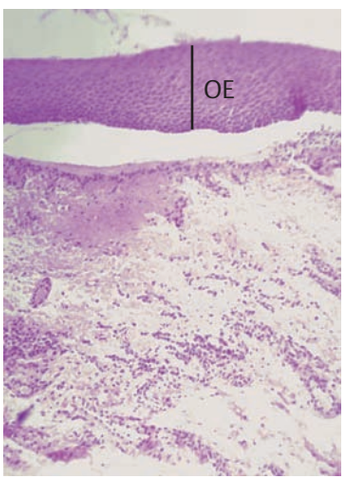
Therapy: Immunosuppressive drugs and systemic corticosteroids. The painful lesions can be treated topically and symptomatically with ointments containing cortisone and antibiotics. The prognosis is relatively poor.

269 Gingivosis/Desquamative Gingivitis
Severe, spotty erythema of the attached gingiva in this 62-year-old female. Epithelium can be easily separated from subepithelial connective tissues. Secondary gingivitis is elicited by plaque.

Right: Histology exhibits a thin oral epithelium (**OE**) devoid of rete ridges and not keratinized. The epithelium has separated (carbol fuchsin, x100).

Courtesy *H. Mühlemann*





270 Pemphigoid
There are no strict criteria for clinical differentiation between gingivosis and pemphigoid. In this 54-year-old female, one observes a severe, marginally localized erythema of the gingiva. The patient reported recurring blister formation, especially on the lingual aspect.

Right: Blister formation and sloughing of the epithelium in another patient with pemphigoid.

Courtesy *U. Saxer*

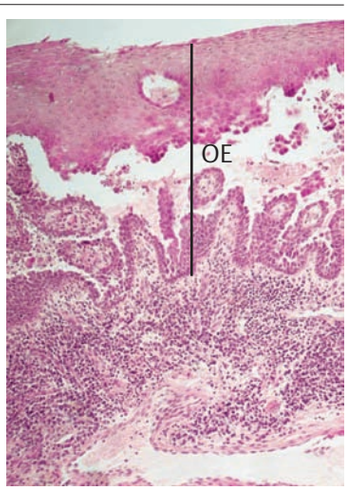




271 Pemphigus vulgaris
Fiery red gingiva with secondary effluorescences (burst vesicles). This 50-year-old female also manifested pronounced symptoms on her skin and on other areas of the oral mucosa.
Right: It is obvious that vesicle formation and sloughing of the superficial layer of gingiva occurs intra-epithelially. The basal cell layer remains attached to the connective tissue.
OE = oral epithelium (HE, x250).

Courtesy *B. Maeglin*





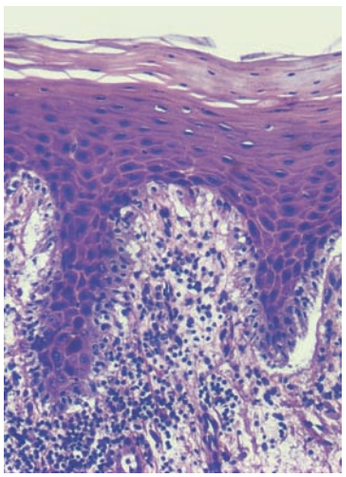
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Lichen planus: Reticular and Erosive

Lichen (intertwined) is a general term for a family of similar-appearing yet differentiable skin (lichen ruber) and mucosal alterations (lichen planus *reticularis, erosivus, nitidus, pilaris, acutus, verrucosis* etc.). Lichen occurs relatively frequently; morbidity figures of 0.2–1.9 % of the adult populations have been reported (Axéll 1976).

The symptoms of reticular lichen planus are milky-white, pebbly, hyperkeratotic effluorescences and/or net-like coatings, the so-called "Wickham's striae." The white lesions may also be expansive (plaque-forming type) and resemble a leukoplakia. The affected mucosa may atrophy (atrophic form), and may subsequently erode (erosive lichen planus; pre-malignant?).

Therapy: There is no true causal therapy. The lesions should be monitored closely. Treatment for erosive forms include systemic administration of corticosteroids, often combined with retinoids.





272 Lichen Planus
This 42-year-old male presented with generalized erythema as well as a whitish, hyperkeratotic, net-like coating (Wickham's striae) on the gingiva and oral mucosa, as well as secondary gingivitis.

Left: Histologically the epithelium exhibits rete ridges and appears to be hyperkeratinized. A subepithelial inflammatory infiltrate is visible (HE, x400).

Courtesy *B. Maeglin*





273 Reticular Lichen Planus (Wickham's Striae)
In addition to the red and whitish gingival lesions, the pronounced striae of this lesion are impressive, extending from the vestibular fold and covering the entire mucosa of the internal surface of the lip.

Courtesy *B. Maeglin*

Left: Bullous lichen with secondary gingivitis.





274 Erosive Lichen
Red-whitish spots, some of which show initial, quite painful erosions (rendering oral hygiene difficult!). Secondary plaque-induced gingivitis.

Left: The lesions extend over the entire gingiva into the molar region.

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Leukoplakia, Pre-Cancerous Lesions—Oral Granulomatoses

Axéll et al. (1984) and Pindborg (1985) defined leukoplakia as white spots "that cannot be classified clinically and pathologically as any other type of lesion, and which are unrelated, except for the use of *tobacco*." The lesions are characterized histopathologically by hyperkeratotic epithelial thickening. There are two clinical appearances (Bengel & Veltmann 1986):

- the more common homogenous form
- the speckled (verrucous) form.

The gingivae are seldom affected, and the etiology is largely unknown. A causal relationship has only been shown with smoking and chewing tobacco. Smokers exhibit leukoplakia three times more frequently than non-smokers.
Secondary infections and malignant transformation (ca. 10 %) are possible.

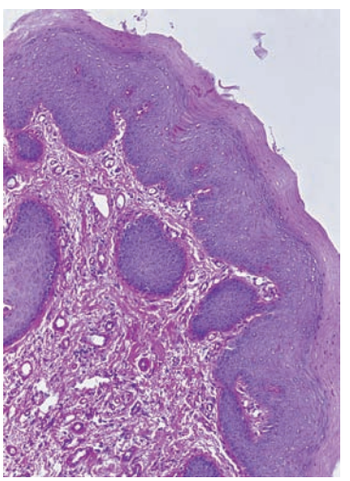
Therapy: Vigilant observation of the leukoplakia. In mild cases, retinoids may be employed. Surgical intervention in pronounced cases where malignant transformation is suspected.

275 Expansive Leukoplakia
In this 65-year-old male cigar smoker, expansive leukoplakia is observed on the attached gingiva adjacent to teeth 15–17.

Right: The histologic picture is one of a slightly thickened, hyperkeratotic (leukoplakic) gingiva.

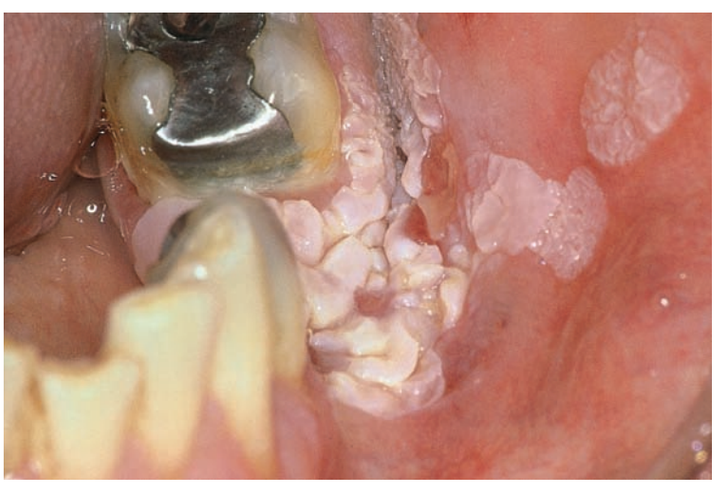
Figs. 275 & 276 courtesy *B. Maeglin*

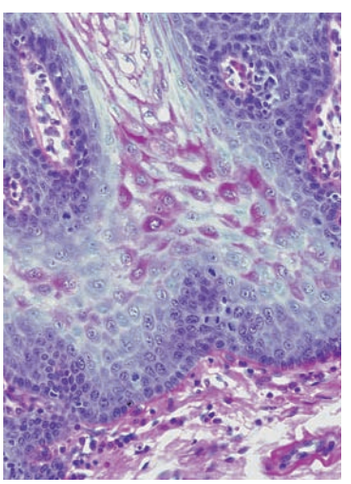




276 Precancerous Lesion
This 40-year-old female had not noticed the long-standing leukoplakia. It transformed into a precancerous situation (verified histologically).
The verrucous, papillomatous lesions were localized to the gingiva in premolar and molar regions, the vestibulum and the cheek.

Right: The histologic section reveals epithelial acanthosis and keratinization of individual cells, as well as numerous mitotic figures.





277 Oral Granulomatosis or Erythroplakia?
Severe erythema of all attached gingiva, especially near tooth 32. The appearance is similar to oral granulomatosis (general term for local phenomena), which often occurs in various diseases such as Crohn's desease, Melkersson-Rosenthal Syndrome etc.
By definition, *erythroplakia* is an oral precancerous lesion (severe epithelial dysplasia or carcinoma in situ). Differentiation: histology

Right: Enlargement, 21–22.





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Herpes—Herpetic Gingivostomatitis

This viral infection is most commonly detected in children and in young adults between the ages of 20 and 25. Once primary infection has occurred, the patient experiences *fever* and painful swelling of the lymph nodes. Intraoral examination reveals an acute, painful gingivitis with blister-like aphthae, erosive lesions on the attached gingiva and not infrequently on the oral mucosa and lips as well. The differential diagnosis must include NUG and common recurrent aphthous ulcer. The etiology is a *Herpes simplex virus* infection (usually HSV-1; see Fig. 278, left).

Predisposing factors include mechanical trauma, sun exposure, inadequate diet, hormonal disturbance and psychic stress. The lesions generally disappear spontaneously within 1–2 weeks without any treatment.

Therapy: Topical application of palliative ointments, and possibly a plaque-inhibitory agent to prevent bacterial superinfection. In general, aciclovir preparations may be prescribed systemically and topically. In severe cases, antibiotics may be used to combat any bacterial superinfection.

HHV—Human Herpes Viruses

HHV-1	Herpes simplex v. 1	**HSV-1**
HHV-2	Herpes simplex v. 2	**HSV-2**
HHV-3	Varicella Zoster v.	**VZV**
HHV-4	Cytomegalovirus	**CMV**
HHV-5	Epstein-Barr v.	**CBV**
HHV-6		
HHV-7		
HHV-8	Kaposi sarcoma v.	**KSV**

Therapy: Vaccines
Aciclovir
Ganciclovir

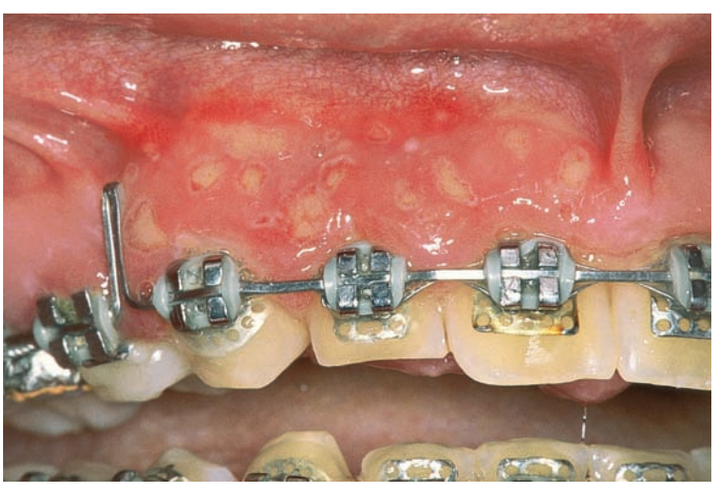


278 Mild Herpetic Gingivostomatitis
Whitish patches and erosive alterations, especially on the attached gingiva. This female patient exhibited good oral hygiene, very little plaque accumulation and hardly any signs of marginal inflammation. The possibly that the Herpes infection occurred secondary to a toothbrush injury cannot be ruled out.

Courtesy *N. Lang*

Left: Human Herpes viruses.





279 Herpetic Gingivostomatitis
The young patient's poor oral hygiene, coupled with the plaque retention fostered by orthodontic brackets and wires led to gingivitis which became superinfected with Herpes.

Left: Differential diagnosis: Solitary aphthae. Whitish ulcer surrounded by fiery red mucosal tissue; often recurrent. Solitary and multiple aphthae should not be confused with the effluorescences of Herpes.



280 Severe Herpetic Gingivostomatitis
This 20-year-old male experienced fever and swollen cervical lymph nodes. A severe gingivitis had been present even before the Herpes infection. This acute clinical picture is reminiscent of ulcerative gingivitis, which should be included in the differential diagnosis.

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Periodontitis with Systemic Diseases (Type IV)—Diabetes Type I and Type II

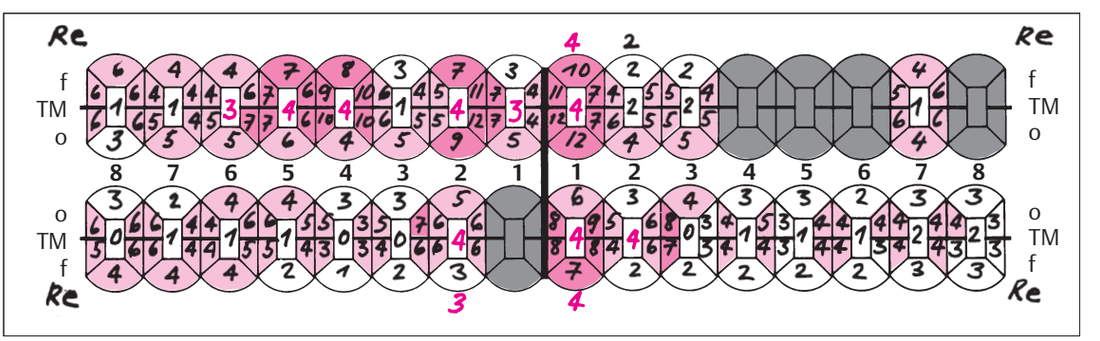
Numerous investigations of the relationships between Diabetes mellitus and gingivitis/periodontitis have been published. Most investigators found correlations between uncontrolled or poorly controlled diabetes and gingivitis/periodontitis (Firatli 1997; Salvi et al. 1997; Tervonen & Karjalainen1997; Katz 2001). Today, diabetes is an established risk factor.

One explanation for this could be the defects of polymorphonuclear granulocytes (PMNs) that are regularly observed in diabetes patients (Manouchehr-Pour et al. 1981a, b). Furthermore, the hyperglycemia increases the presence of AGE (advanced glycated end-product), which stimulates the macrophage receptor (RAGE) toward increased synthesis of TNFα, IL-1β and IL-6. Both the form and function of extracellular matrix components, such as collagen, become altered.

It is possible that the vascular pathology associated with diabetes (cf. retinopathy, Fig. 285) may also play a role in terms of periodontal blood supply, but this has yet to be proven (Rylander et al. 1987).



281 Clinical Overview (above)
Localized acute inflammation of the gingivae, which exhibit some edematous swelling as well as areas of shrinkage. Plaque and calculus are abundant. Almost all of the deeper pockets exhibit signs of activity (pus).



282 Probing Depths, Gingival Recession (Re) and Tooth Mobility (TM, right)
Immediately apparent is the extraordinarily irregular attachment loss. Probing depths in the interproximal areas ranged from 4 to 12 mm. Some teeth exhibited extreme mobility (cf. radiographic survey).



283 Radiographic Survey
The radiographs confirm the clinical findings. Teeth 15, 14, 12, 21 and 32 had to be extracted on an emergency basis.

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A 28-year-old male had developed severe, *insulin-dependent juvenile diabetes* at age 15. The associated periodontitis had never been treated.

Findings :

API: 69 % PBI: 3.2

Probing depth, gingival recession and tooth mobility: see Fig. 281.

Diagnosis: Rapidly progressing, advanced periodontitis in conjunction with juvenile diabetes.

Therapy: The treatment plan for patients with juvenile diabetes is usually rather radical.

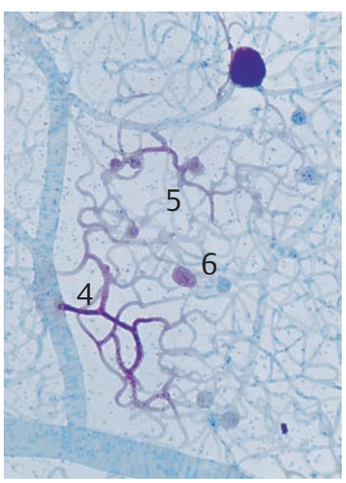
- Maxilla: Extraction of all teeth with the exception of teeth 13 and 23 (periodontal treatment); temporary partial denture.
- Mandible: Extraction of the remaining anterior teeth and the third molars. Periodontal treatment of the remaining teeth, cast framework partial denture. Dental implants are not indicated in diabetes patients.

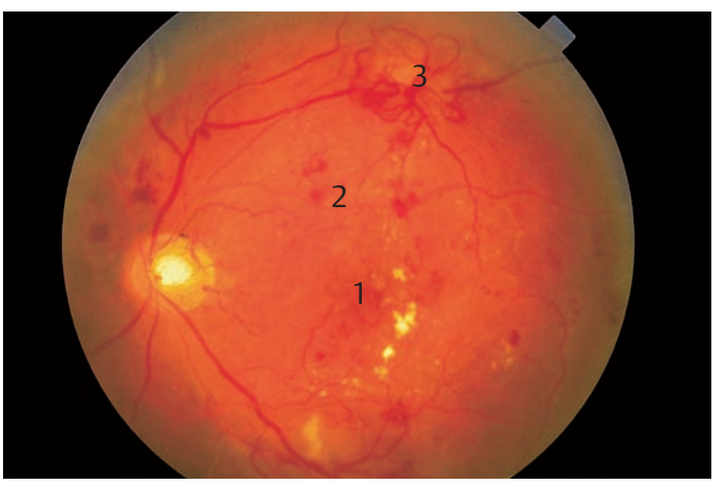
Recall: Initially, every 3 months.

Prognosis for the abutment teeth: "Guarded" at best, in view of the proposed radical therapy.



284 Maxillary Anterior Area—Initial Findings
Diastemata had formed over the course of the previous years. Tooth 21 appears elongated, exhibits no bony support, is highly mobile and painful (see radiographic survey, Fig. 283). It was extracted along with the other teeth after a temporary removable partial denture was constructed.

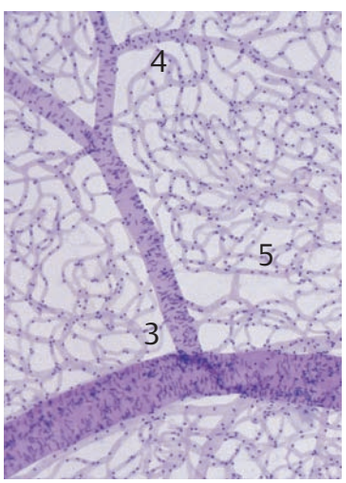


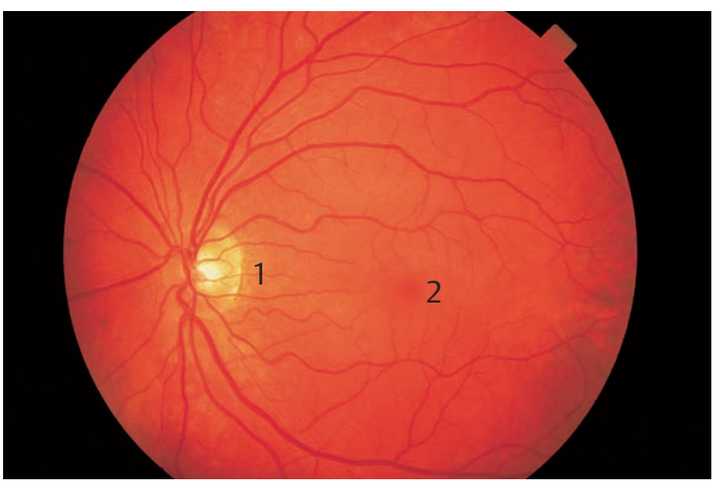


285 Retinoscopy in Diabetic Retinopathy
1 Yellow lipid bodies in the retina
2 Disseminated hemorrhage and microaneurysms
3 Neovascularization bundle due to ischemia

Left: Histologic view of diabetic retinal microangiopathy
4 Closure of a precapillary
5 Atrophic capillaries, cell-free zone
6 Microaneurysms

(Trypsin digestion, x25)





286 Normal Retinoscopy, Healthy Eye
1 Optic nerve papilla with exiting retinal vessels
2 Macula free of vasculature

Left: Normal histology of the retina
3 Retinal arterioles
4 Precapillaries
5 Capillaries

(Trypsin digestion, HE, x25)

Courtesy *B. Daicker*

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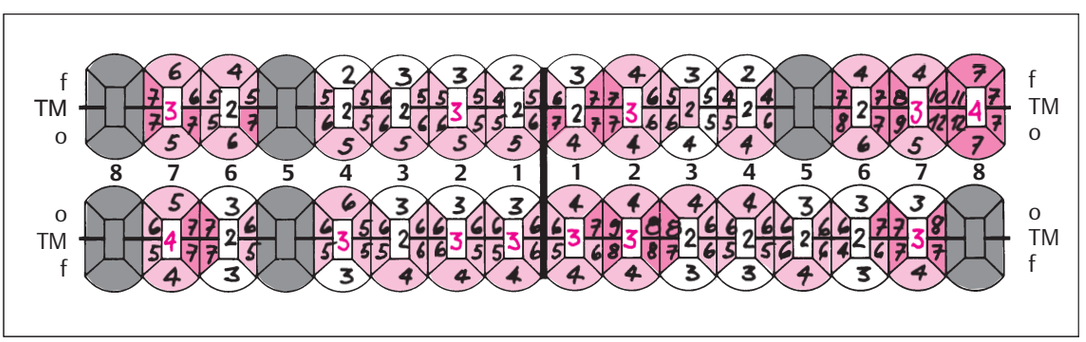
Periodontitis Associated with Systemic Diseases (Type IV B)
Down Syndrome, Trisomy 21, "Mongolism"

Trisomy 21 (previously: Mongolism) carries the name of John Langdon Down who first described the condition in detail in 1866 (Rett 1983; literature review by Reuland-Bosme and Van Dijk 1986). The basis for this condition is a chromosomal aberration: During meiosis, which is the cell division process for reproductive cells, the separation of the paired chromosome 21 does not occur during nuclear division. Two homologues fail to disjoin at the first meiotic division, and therefore two of the resulting gametes carry a "double dose" of the chromosome. Thus, instead of a zygote containing one chromosome 21 from the male and one chromosome 21 from the female, the genotype of the zygote contains three chromosomes at position 21 (trisomy 21; see karyotype, Fig. 290).

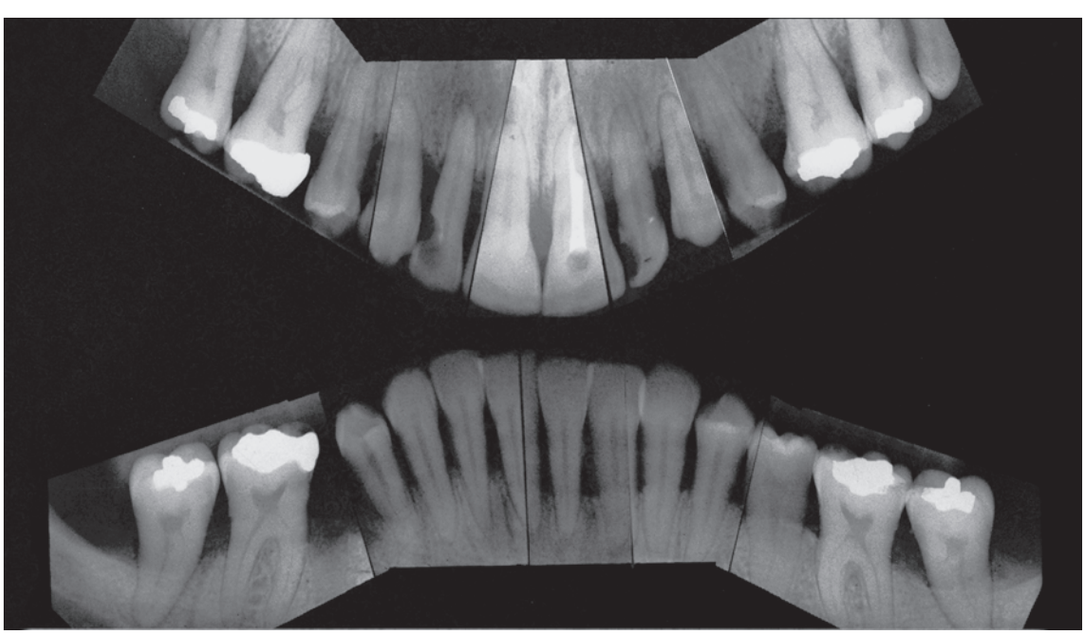
Mongolism occurs at a rate of one in about every 700 live births. It is likely, however, that the prevalence of mongolism will diminish significantly in the future as a result of increased use of ultrasound prenatal analysis and amniocentesis, as well as more liberal abortion laws (Schmid 1988).



287 Clinical Overview (above)
Poor plaque control, severe gingivitis, anterior open bite, crossbite and end-to-end occlusion in molar segments.



288 Probing Depths and Tooth Mobility (TM, right)
The deeper pockets exhibit signs of activity. As a result of the massive attachment loss, all teeth exhibit varying degrees of mobility.



289 Radiographic Survey
The radiographs corroborate the generalized and profound attachment loss: Horizontal and in some areas vertical bone loss of up to two-thirds of the root length. The widened periodontal ligament space anticipates the elevated tooth mobility.
Tooth 21 is non-vital and exhibits an apical radiolucency.

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This 27-year-old female (without cardiac complications) has the mental development of a 6-year-old. She was raised in her parent's home, is well cared for and can converse readily. For these reasons every attempt was made to maintain as many teeth as possible.

Findings:

API: 100 % PBI: 3.8

Probing depths and tooth mobility: See Fig. 288.

Diagnosis: Advanced, rapidly progressive periodontitis in this patient with Down Syndrome.

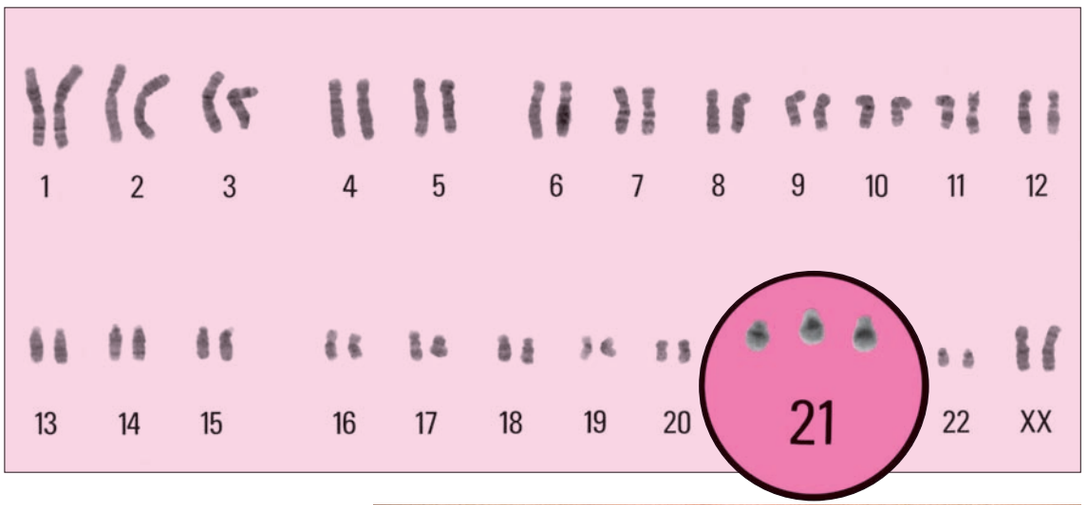
Therapy: Professional debridement and oral hygiene instruction. (Tooth brushing by the patient, interdental cleaning by her mother).

Modified Widman surgical procedures in all quadrants; extraction of teeth 17, 27, 28, 37 and 47.

Recall: Frequent but brief appointments.

Prognosis: Patient compliance will always be poor. Plaque control will depend for the most part on her caretakers. The prognosis is therefore guarded.

Thanks to today's modern medical treatments, the life expectancy for Trisomy 21 patients has increased dramatically in the past few decades.



290 Karyotype in Trisomy 21
The abnormal chromosomal picture results from triplication of the small autosome 21. This "Trisomy 21" occurs in about 94 % of all "mongoloid" patients.

Modified from *H. Müller*



291 Symptoms of Down Syndrome—Scrotal (Fissured) Tongue
The deeply fissured tongue is one of the typical symptoms in Down Syndrome.

Symptoms of Down Syndrome:
– Scrotal tongue
– Ocular peculiarity
– Small head
– Stocky physique
– Short, soft hands
– Cardiac defects (one-third have a short life expectancy)



292 7 Years After Treatment
Following initial therapy, the entire dentition was treated by means of modified Widman surgical procedures. The end result, 7 years after the final surgery, was an acceptable result in comparison to the initial situation. A short-interval recall is recommended, and possibly also the use of a custom tray for daily application of chlorhexidine gel.

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Pre-pubertal Periodontitis Associated with Systemic Disease Papillon-Lefèvre Syndrome (Type IV B)

Papillon-Lefèvre Syndrome (PLS) is a rare, inherited, *autosomal recessive*, "dermatologic" disease (Haneke 1979). Obligate symptoms include severe periodontitis and hyperkeratoses, usually localized to the palms and soles of the feet, as well as other skin areas that commonly absorb minor trauma (HPP = *Hyperkeratosis palmaris* and *plantaris*).

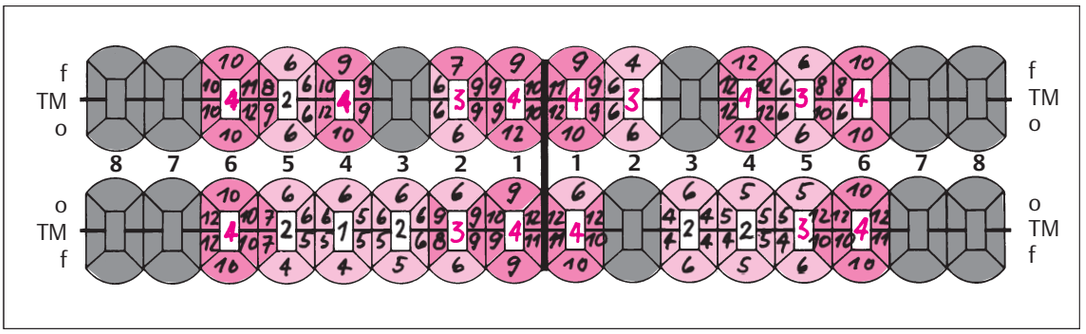
The deciduous teeth are lost prematurely in most cases. The permanent teeth are always periodontally involved.

The primary etiologic factors include a mutation of the cathepsin-C gene (chromosome 11q14-q21; Hart et al. 1998), which regulates epithelial cells and immune cells, as well as a particularly aggressive pocket flora (gram-negative anaerobes).

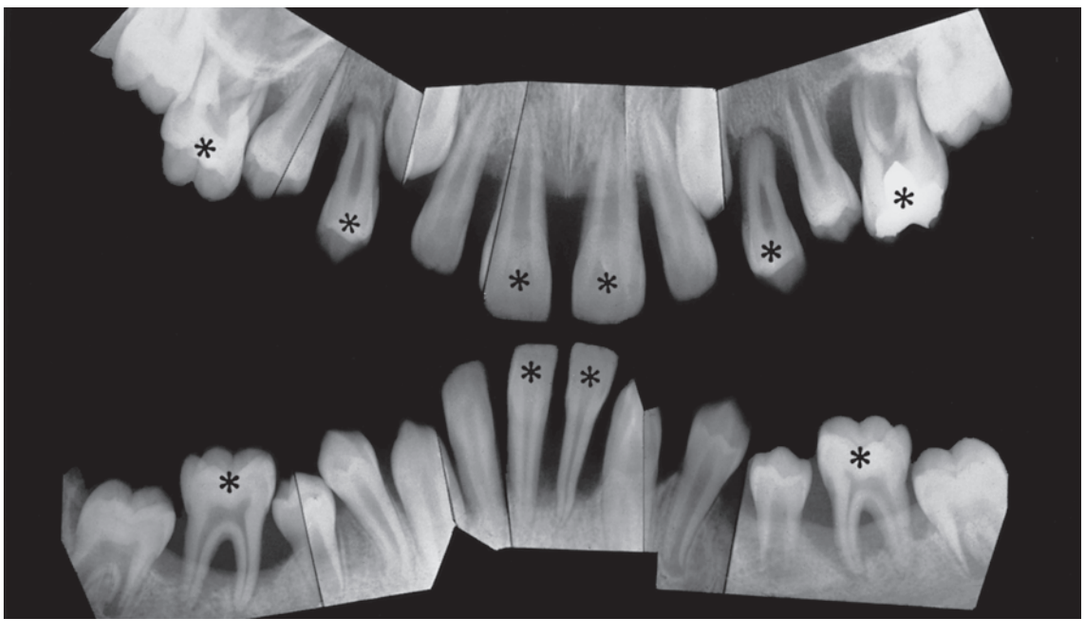
Earlier therapeutic attempts were without success. During the 1980's, however, Preus & Gjermo (1987) as well as Tinanoff et al. (1986) reported that extraction of the deciduous teeth as well as the already present permanent teeth in a 9-year-old patient led to maintenance of the subsequently erupting permanent teeth.



293 Clinical Overview (above)
Extremely severe gingivitis and periodontitis, plaque, spontaneous hemorrhage, exudate and suppuration from the pockets. Initial abscess formation on the facial of 11 and 21. Poor occlusion, severe overbite.



294 Probing Depths and Tooth Mobility (right)
All of the deeper pockets exhibit signs of advanced activity (pus).



295 Radiographic Survey
The marked (✱) teeth

· · 6 · 4 · · 1	1 · · 4 · 6 · ·
· · 6 · · · · 1	1 · · · · 6 · ·

are all in the process of being spontaneously exfoliated, and will be immediately extracted (plaque-retentive niches). Periodontal destruction appears to begin after eruption of the teeth, and subsequently progresses rapidly. Vertical defects (infrabony pockets) predominate. Class F3 furcation involvements.

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A 9-year-old boy was referred from his pediatrician because of severe halitosis and extreme tooth mobility.

Findings:
API: 100% PBI: 3.9
Probing depths, radiographic survey, tooth mobility and skin lesions: See figures below.

Special findings: PMN defects, specific pocket flora (*Porphyromonas* and spirochetes).

Diagnosis: Acute, severe pre-pubertal periodontitis associated with Papillon-Lefèvre Syndrome.

Therapy: Extraction of the hopeless teeth (Fig. 295; note asterisks on teeth to be extracted).
Mechanical periodontal debridement and simultaneous topical (chlorhexidine) and systemic (metronidazol/tetracycline) treatment; temporary removable partial denture (hygiene).

Recall: Very short interval.

Prognosis: Teeth that can be "saved" beyond puberty may be maintained over the long term (see next case, p. 138).

Papillon-Lefèvre Syndrome PLS with Palmar and Plantar Hyperkeratosis



296 Hyperkeratosis on the Palm of the Hand
The hyperkeratotic area exhibits cracks and fissures, which are actually wounds that have occurred due to normal function. These heal poorly and slowly. The patient suffers from these palmar lesions especially in winter.



297 Hyperkeratosis on the Elbows



298 Hyperkeratosis on the Foot
The sharp line of demarcation between hyperkeratotic areas and normal-appearing skin on the lateral border of the foot correspond to the outline of the shoe worn by this patient.

Minor trauma to the skin elicits this type of severe hyperkeratotic response. Even dermatologists treat this disease only symptomatically and polypragmatically.

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Papillon-Lefèvre Syndrome—"An Exception for Every Rule"

A 7-year-old girl was referred to the dental clinic because of severe mobility of her recently erupted permanent incisors and first molars. The patient remained under dental care for 24 years thereafter.

As a teenager, the patient was reconciled to wearing a cast framework partial denture. At age 18, she underwent surgery to correct her mandibular prognathism (note osteosynthesis wires in the radiograph, Fig. 301). At age 25, the remaining dentition was treated with splinted total reconstructions in both mandible and maxilla, which were cemented temporarily.

Diagnosis: Acute, severe periodontitis in a case of Papillon-Lefèvre Syndrome (PLS).

Course of the disease, and treatment: In this exceptional case, it was possible to retain a large number of permanent teeth as a result of timely extractions, intensive periodontitis therapy, frequent recall and the excellent cooperation of the patient (Fig. 301).

It is worthy of note that the treatment rendered 24 years ago was purely mechanical and did not include any systemic or topical supportive medicinal therapy.

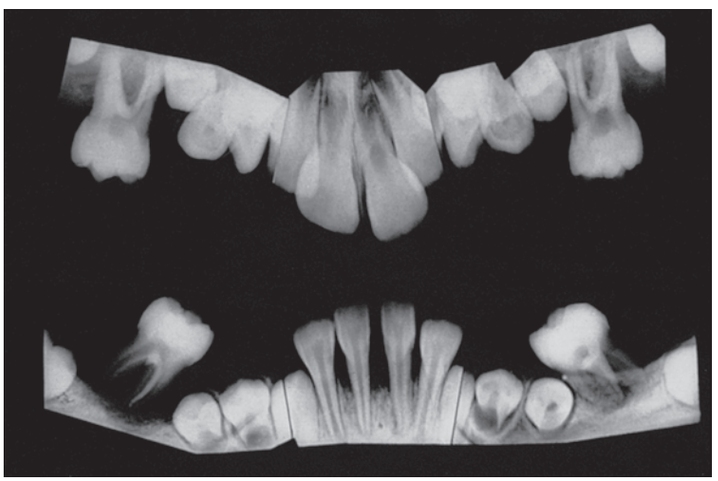
299 Radiographic and Dental Survey of the 7-year-old Female

Erupted permanent teeth:

·	·	6	·	·	·	2	1	1	2	·	·	·	6	·	·				
·	·	6	·	·	·	2	1	1	2	·	·	·	6	·	·				

The first permanent molars were exfoliated one year after eruption. The mandibular anterior teeth exhibit severe periodontitis. All permanent teeth are present (as yet unerupted).

The primary teeth were lost prematurely.



300 Same Patient, Age 31

The patient smiles with (many of) her own teeth, which were deemed to be "hopeless" due to her Papillon-Lefèvre syndrome. She displays advanced hyperkeratosis on both palms, and painful cracks and chapping on her heel. The skin on the back of her hands is paper thin, hyperkeratotic, dry and visibly erythematous.



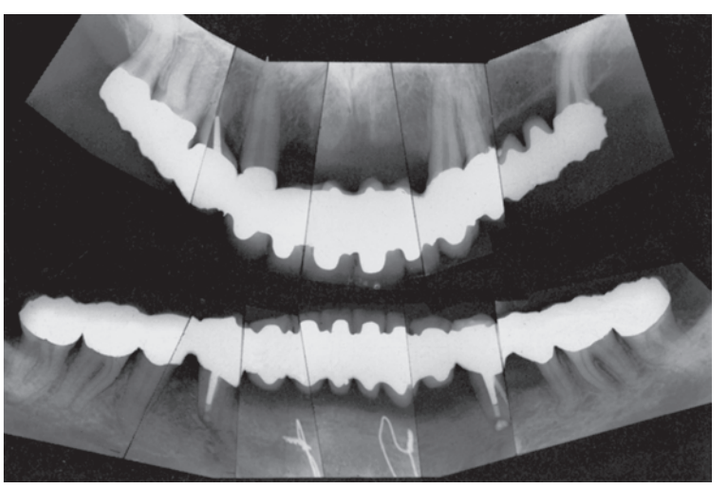


301 Radiographic Survey of the 31-year-old Female

Destructive periodontitis came to a halt after the patient traversed puberty. The remaining teeth served as abutments for fixed bridgework:

8	7	·	·	·	3	·	·		·	·	3	·	5	·	·	8			
8	7	·	5	4	·	·	·		·	·	·	4	5	·	7	8			

This bridgework is seated only temporarily (Temp-Bond), and is periodically removed, cleaned and re-cemented.



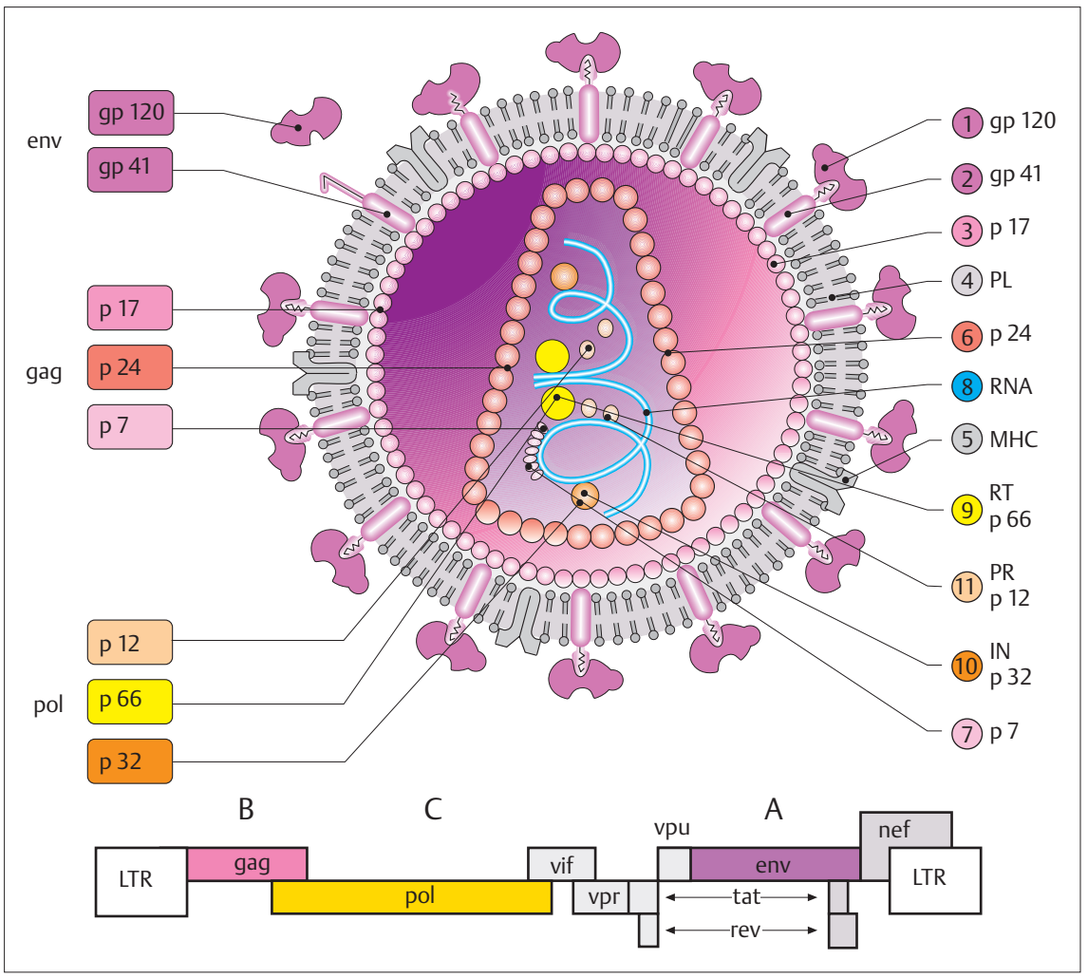
Radiographs courtesy *U. Saxer*

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HIV Infection—AIDS

The immune deficiency disease AIDS (Acquired Immunodeficiency Syndrome) is caused by the HIV virus 1, a retrovirus. It is a complex virus, whose RNA genome (8,700 bases) contains at least nine genes. The three most important of these nine are *env* (virus capsule protein), *gag* (group-specific antigen) and *pol* (polymerase, enzyme-coding portion in the retrovirus genome; Fig. 302).

The virus exhibits the following structural characteristics: an RNA genome at the center on which the reverse transcriptase (RT, p66 *pol*-gene product) is bound; *gag*-coded proteins determine the virion structure; the phospholipid membrane (PL) provided by the host; two *env* gene products: a membrane-penetrating (gp 41) glycoprotein and an externally localized glycoprotein (knobs, gp 120) anchored to it. Three genes (*tat, rev, nef*) have regulatory functions. The specific functions of the remaining genes have not yet been completely elucidated.



302 HI Virus
The *env*-, *gag*- and *pol*-coded proteins (**p**) and glycoproteins (**gp**) are shown at the left, and all other structures (**1–11**) on the right.

HIV Envelope
– Virus-coded

1	Knob bodies	gp120
2	Transmembrane portion	gp41
3	Capsule matrix	p17

– Host-coded

4	Phospholipid bilayer
5	MHC-proteins (receptors, p. 46)

Capsid/Core

6	Capsomere proteins	p24
7	RNA capsule protein	p7

Genome/Enzymes

8	HIV-RNA, 2 single strands	
9	**RT** reverse transcriptase	p66
10	**IN** integrase	p32
11	**PR** protease	p12

HIV Genome (below)
A ***env***-coded viral surface proteins (binding sites for CD4 and membrane fusion)
B ***gag***-coded nucleocapsid and nuclear membrane
C ***pol***-coded for the enzymes RT, IN, PR and ribonucleases

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HIV Disease—Epidemiology

The world wide dissemination of the HIV disease continues to increase. At the end of 2002, over 41,000,000 persons were infected with the HI virus. There are grave differences between the industrialized nations on the one hand and the developing countries on the other: While in the USA, Western Europe, Japan, Australia and New Zealand the new infections of adults have generally stabilized, the rates for the African countries south of the Sahara Desert, and in Russia continue to increase. At the present time, two thirds of all the HIV-infected adults are found in sub-Saharan Africa and Russia, and 90 % of all infected children!

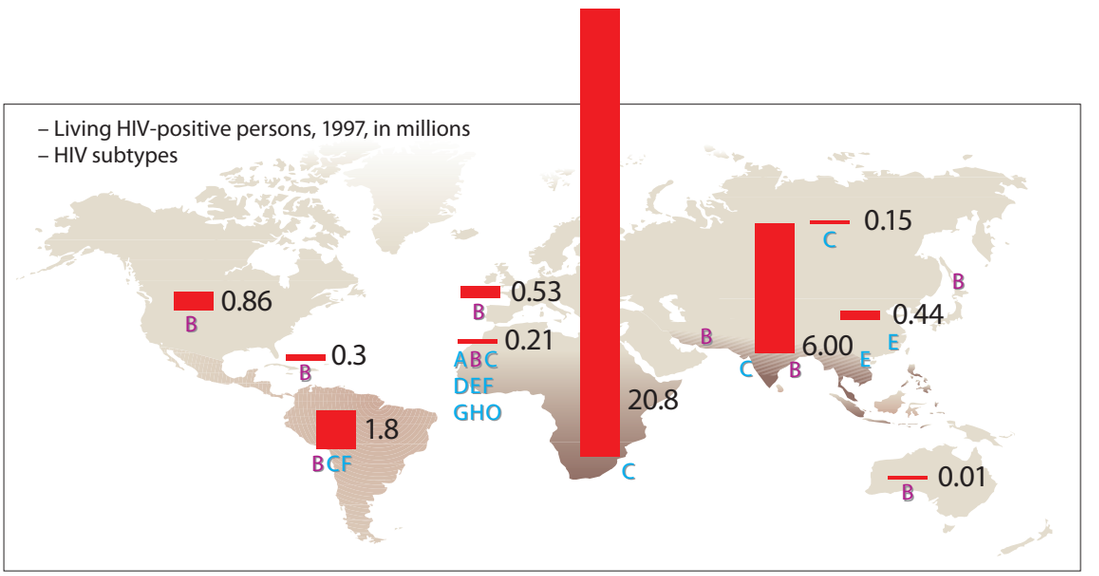
Unprotected heterosexual intercourse, the dissemination of uncontrolled blood products and above all the lack of prevention (information), as well as the enormous costs of treatment may be responsible for the current situation. Even more perplexing is the fact that the HI virus appears in various types and subtypes (Reichart & Gelderblom 1998, Reichart & Philipsen 1999), which makes efforts toward treatment or even immunization infinitely more complex.

The battle against the AIDS pandemic must also be waged against the socioeconomic backdrop.

Ninety percent of all HIV-infected individuals world wide live in developing countries, but 90 % of all monies spent for information, prevention and treatment are expended in the industrialized countries—ca. $10,000 per infected person per year.

Epidemiologic research has shown that we must differentiate not only between countries and their various stages of economic development. Even within the industrialized nations, there are clear differences between persons of different socioeconomic classes. For example, in the USA the infection rate for Blacks is almost five times higher than for white males. The difference between Afro-American and Caucasian females in the USA is even more dramatic (Fig. 304, right).

Because it is extremely unlikely that the differences between industrialized and developing nations, as well as between different socioeconomic standards will ever be reconciled, the highest priority must be given to the development of effective and inexpensive medicines, and above all an effective vaccine (Mann and Tarantola 1998).



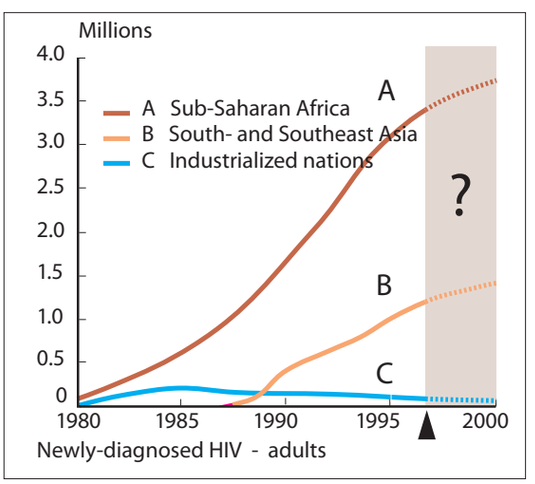
303 World Wide Distribution of HIV-infected Individuals, 1997

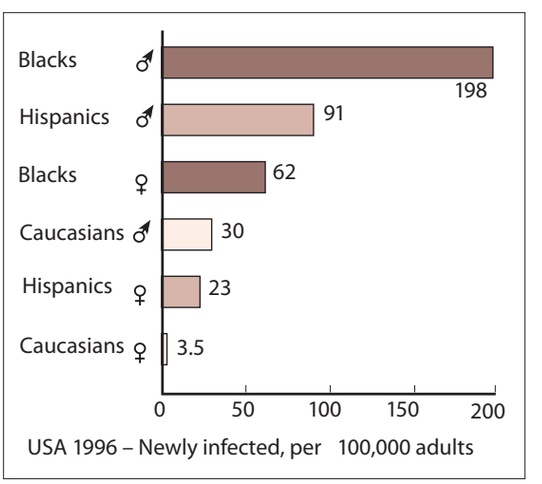
By far the majority of HIV-infected person live in sub-Saharan Africa (20,800,000), as well as in south and southeast Asia (6,000,000), followed by Latin America (1,800,000).

Various HIV subtypes (A–G) can be differentiated.

Type B predominates (80 %) in Europe and North America.

By the end of 2002, over 41,000,000 persons world wide were infected with HIV.





304 Increase in HIV-infected Adults, 1980–1998

Left: In the industrialized countries, the number of infected adults has decreased somewhat, following the initial increase. South of the Sahara Desert—but also in south and southeast Asia—a dramatic increase occurred.

Right: New infections per 100,000 adults and adolescents in the USA, 1996; the numbers are higher in individuals in lower socioeconomic groups.

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Classification and Clinical Course of HIV Disease

With every-increasing knowledge, the classification of the HIV disease has already been modified many times, and additional variations will surely be implemented in the future. The classification derives from data from the USA *Centers for Disease Control and Prevention* (CDCP). This classification is based on the number of CD4 cells (stages 1–3) in relation to clinical symptoms (stages A-C) (Fig. 305).

Besides this classification, today the most significant parameter of the clinical course of disease is the number of free virus copies per milliliter of blood plasma, the so-called "viral load." This is determined using the polymerase chain reaction (PCR).

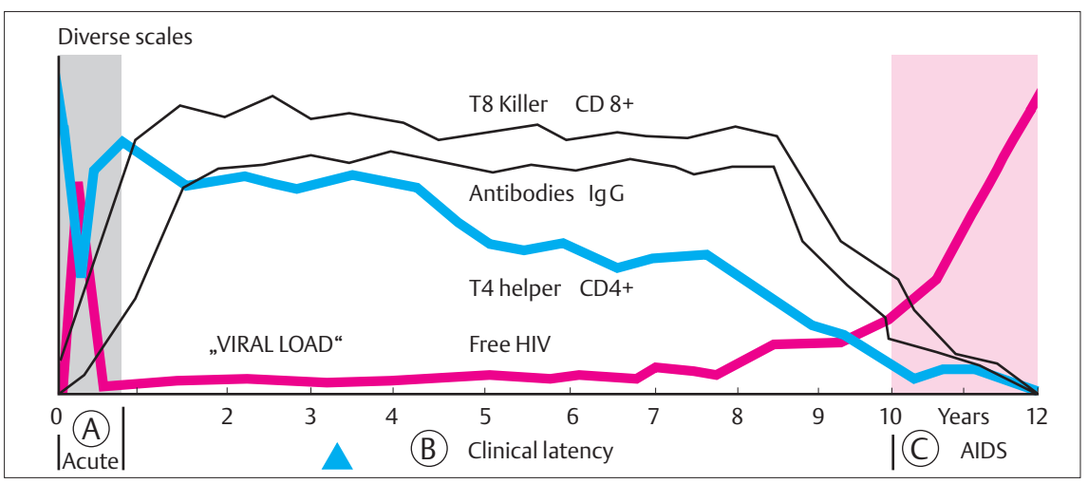
HIV disease develops very rapidly following the initial infection. Billions of HIV particles destroy millions of CD4 lymphocytes. This bitter "war of attrition" between HI viruses and host cells goes on continually for years. During the further (average) course of the disease—about 6 months after infection—the number of freely circulating viruses decreases dramatically and the number of immune cells again increases. A balance between attack and defense can remain constant for ca. 10 years, until the number of viruses again increases dramatically and the host defense breaks down (Fig. 306).

Even from patient to patient, the disease can assume extraordinarily different clinical courses. "Long survivors" are those individuals who live longer than 10 years after infection, while others with the disease have only a short post-infection life span. A prognosis concerning the course of the disease is possible by measuring the viral load. Mellors (1998) measured the viral load in 1,600 untreated HIV-infected males, and reported that 70 % of his study population who exhibited more than 30,000 virus copies per milliliter of blood plasma died within six years (mean lifespan: 4.4 years). In contrast, less than 1 % of the untreated patients died within six years if their viral load was below 500 copies/ml. In the latter group, the mean life expectancy was more than 10 years.

It is now clear that the determination of viral load provides important information concerning prognosis and therapy. Using today's anti-retroviral medicinal polytherapy (p. 149), the number of virus particles can be held below the level of detection.

Clinic. symptomatic ▶ Grade – $CD4^+$-cell no.	A Asymptomatic or acute HIV infection	B ARC syndrome, e. g., oral candidiasis	C AIDS indicator diseases
1 >500	A1	B1	C1
2 200–500	A2	B2	C2
3 <200	A3	B3	C3

305 CDCP Classification
Close scrutiny of the CDCP classification clearly reveals that patients with a relatively high number of CD4 cells may already exhibit AIDS symptoms (**C2**), while, on the other hand, infected individuals with only low levels of CD4-cells may remain asymptomatic (**A2**).



306 "Average" Clinical Course of HIV Disease
Immediately following HIV infection, there follows a ca. 6-month acute phase (bouts of fever, lymphadenitis etc), followed by a years-long asymptomatic period. This is characterized by a low *viral load* and a positive immune response (T-helper and T-killer cells, antibodies etc.). On average, 8–12 years later, the viral load increases and host defense collapses. The "average" clinical course depicted here varies significantly from patient to patient.

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Oral Manifestations of HIV Disease

In addition to numerous somatic symptoms, the HIV disease can also be characterized by pronounced oral manifestations. The progress in systemic medical therapy has fortunately reduced many oral symptoms, e. g., bacterial and fungal infections, viral infections, neoplasms and other pathology of unknown etiology (Fig. 307).

The oral lesions are often painful and can compromise the patient's quality of life. The time of appearance of oral manifestations will be determined by the CD4 cell count and the viral load (Fig. 308). Occasionally, however, oral alterations—especially linear gingival erythema (LGE) and necrotizing periodontitis (NUP)—occur at unpredictable times during the course of the HIV disease.

The dentist must be fully aware of the oral manifestations of HIV disease; in numerous cases, a dentist has diagnosed oral alterations that lead to suspicion of HIV infection, only to have those suspicions subsequently corroborated after medical examination by the physician.

307 Some Oral Manifestations of HIV Disease
Most of the alterations (listed to the right) can be diagnosed by the dentist. LGE, NUG and NUP can only be treated in the dental practice (p. 151). All other disease manifestations must be treated in collaboration with the physician.

Non-specific Bacterial Infections

- Linear gingival erythema (LGE)
- Necrotizing ulcerative gingivitis (NUG) and periodontitis (NUP)
- Exacerbation of periapical processes

Specific Bacterial infections

- MAI (Intracellular Mycobacterium avium)
- Enterobacter cloacae

Fungal Infections

- Candidiasis
- Histoplasmosis

Viral Infections

- Human herpes viruses (HHV)
- Herpetic stomatitis
- Human papilloma virus (HPV)

(Details, see pp. 131, 145)

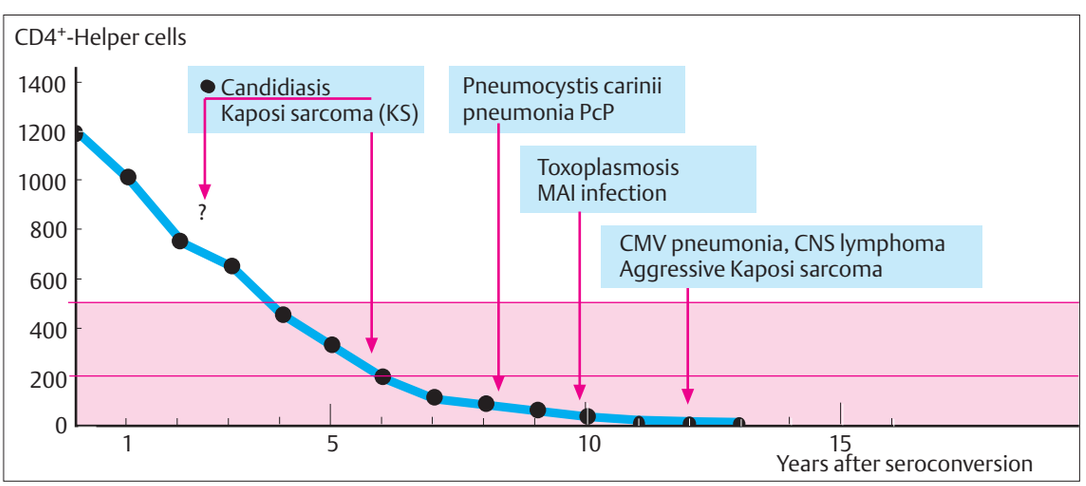
Neoplasms

- Kaposi sarcoma
- Non-Hodgkin lymphoma
- Spinal cell carcinoma

Unknown Etiology

- Delayed wound healing
- Aphthae
- Ulcerations
- Pigmentations
- Idiopathic thrombocytopenia (hemorrhage)
- Xerostomia
- Salivary gland disorders

308 Occurrence of Disease in Correlation with the CD4 Cell Number ("Marker" Diseases)
While *Candidiasis* can occur relatively early, the severe systemic diseases such as *Pneumocystis carinii pneumonia* (PcP), *Toxoplasmosis*, *infections with intracellular Mycobacterium avium* (MAI), *Cytomegalovirus (CMV) pneumonia* etc. occur only when the CD4 cell count drops.



309 Association Between Certain Oral Symptoms and HIV Disease
The frequency of occurrence of these opportunistic diseases is also dependent on the stage of the HIV disease (Weinert et al. 1996).

Most Frequent Occurrence +++	Occasionally Occurring ++	Seldom Occurring +
• **Candidiasis** • **Hairy Leukoplakia** • **LGE** • **NUG/NUP** • **Kaposi Sarcoma** • **Non-Hodgkin Lymphoma**	• Bacterial infections such as tuberculosis (frequent in S. Africa) • MAI • Viral infections (Herpes simplex virus, Varicella-zoster virus, Papilloma virus) • Atypical ulcerations • Thrombocytopenic purpura • Salivary gland alterations • Pigmentations	• Fungal infections (except Candida) • Other poorly understood infections • Neurologic disturbances, viral infections (cytomegalovirus etc.) • Recurring aphthous stomatitis • Reactions to medicaments

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Bacterial Infections in HIV

- Linear gingival erythema (LGE)
- Necrotizing gingivitis/periodontitis (NUG/NUP)

The disease states marked with a solid bullet (•) on this page and the pages that follow are depicted and described.

LGE is clearly differentiable from simple plaque-elicited gingivitis. It is characterized by a clearly demarcated band of reddening in the marginal gingiva. Classic periodontitis—both "chronic" and "aggressive" forms—do not occur in HIV-disease patients any more frequently than in other individuals; on the other hand, *NUP* occurs much more frequently, often exhibiting an extremely rapid clinical course of attachment loss.

Th significance of certain microorganisms in the etiology of LGE and NUP remains unclear. One finds periodontopathic microorganisms as seen in aggressive forms of periodontitis (p. 96), but often also significant increases in *Candida albicans* (*Ca*). The spotty erythema on the attached gingiva, and the frequent observation of this fungus in niches and pockets can be attributed to *Ca* In those not responding to mechanical therapy, the cytomegalovirus is often detected.



310 Linear Gingival Erythema (LGE)
Note the even band of erythema along the gingival margin and papillae. It is unclear whether such LGE (in the absence of treatment) can develop into NUP. The latter appears to be much more closely associated with ulcerative gingivitis (Fig. 311). Treatment consists of mechanical cleaning with betadine irrigation, motivation to improve oral hygiene and, in severe cases, CHX rinses.

Courtesy *J. Winkler*





311 Ulcerative Gingivitis—NUP, Initial Stage
23-year-old female drug addict. The inflammation and ulcerations are similar to those observed in classic ulcerative gingivoperiodontitis. In the absence of treatment, the destruction of gingival tissue can progress rapidly.

Left: Severe, painful, ulcerative gingivitis in a 28-year-old female drug addict. The case was treated successfully (Figs. 329–335); she returns for regular recall and is recurrence-free for 7 years.



312 Very Advanced NUP
45-year-old homosexual male with extremely severe and painful NUP. Exposed bone could be probed in the interdental areas. In such severe cases, osseous sequesters may form. This patient was in the end stage of AIDS, and died 3 months later.

The "normal" NUP therapy is described on pages 151–154.

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Fungal Infections

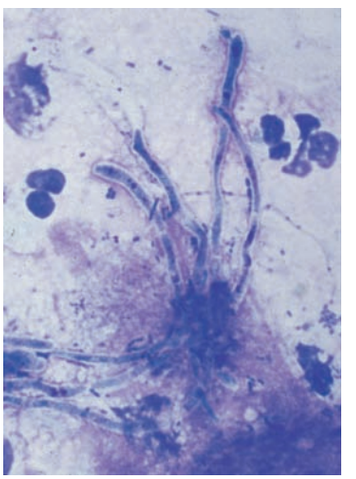
- Candidiasis
  - Atrophic/Erythematous
  - Angular cheilitis
  - Pseudomembranous
  - Hyperplastic
- Histoplasmosis

The most common and earliest appearing fungal infection in HIV disease is candidiasis in its many and varied forms. Approximately 95 % of all fungal diseases are caused by *Candida albicans*; other fungi have only minor medical significance. *Candida albicans* is also found in a high percentage of healthy individuals, without causing any clinical symptoms. If host defense mechanisms are reduced, as is the case in HIV disease, proliferation of the fungi can occur by growth of hyphae and the formation of mycelia. The latter can invade the mucosa and lead to clinical manifestations of the types listed above. Oral Candida infections have a tendency to recur. Spread into the respiratory tract or the gastrointestinal tract is an indication of progression of the HIV disease, and is a complication to be taken very seriously by the patient and the physician.

313 Pseudomembranous Candidiasis
The whitish, painless layer on the gingiva and mucosa of this 27-year-old drug-addicted HIV patient can be easily wiped away. Treatment for cases limited to the oral cavity generally consists of careful removal of the pseudomembrane, and in severe cases systemic antimycotics.

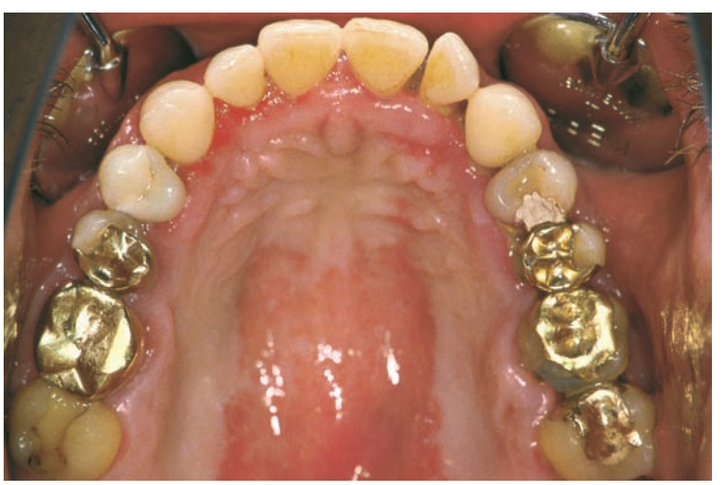
Right: Candida mycelia in culture.





314 Atrophic, Erythematous Candidiasis
This 45-year-old, HIV-infected homosexual male exhibits reddish lesions in the middle of the palate. Similar alterations can also be observed on the edentulous alveolar ridge, attached gingiva and the dorsum of the tongue. The lesions may be painful.

Medicinal treatment involves *topical* rinsing with CHX, and *systemic* administration of the antimycotic Diflucan (Fluconazol).



315 Angular Cheilitis (Perlèche)
Typical fissures at the corner of the mouth in a 32-year-old heterosexual, HIV-positive patient (promiscuity). These lesions are very painful and render dental treatment difficult. There is an association between *Candida albicans* and *Streptococcus aureus*. Perlèche is also observed in immunodeficient elderly individuals, as well as patients with severe overbite. The treatment consists of topical antimycotic agents (see above).



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Viral Infections

Human Herpes Viruses (HHV)

- H. simplex virus Type 1 (HSV-1) HHV-1
- H. simplex virus Type 2 (HSV-2) HHV-2
- Varicella zoster virus (VZV) HHV-3
- Epstein-Barr virus (EBV) HHV-4
- Cytomegalovirus (CMV) HHV-5
- Human herpes virus type 6 HHV-6
- Human herpes virus type 7 HHV-7
- Human herpes virus type 8 HHV-8

Viruses that are latent and which can also be found in "healthy" tissue usually are limited to ectodermal structures (skin, mucosa, retina etc). The primary infection usually occurs early in childhood through contact or droplet transfer. It can remain as a so-called latent infection throughout life.

The clinical manifestations of viruses in the oral mucosa are extraordinarily variable. Blisters, leukoplakic lesions on the tongue ("hairy leukoplakia"), localized and often expansive ulcerations and wart-like lesions have been described.



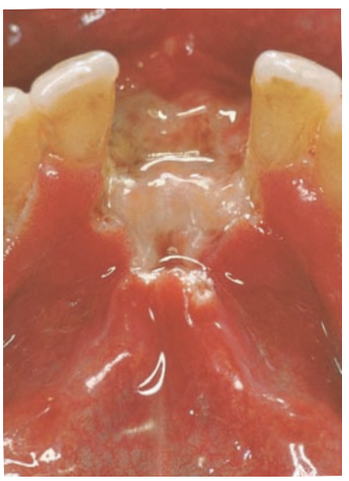


Human Herpes Viruses

316 Herpetic Stomatitis
Multiple blisters surrounded by erythema were caused by HSV-1 in this 40-year-old, HIV-positive homosexual patient. When the blisters burst, painful ulcerations remain.
Therapy: Topical analgesics and anti-inflammatory medicaments; systemic Aciclovir (e. g., Zovirax).

Left: Hairy leukoplakia on the lateral tongue border (Epstein-Barr virus, EBV).







317 Expansive Ulceration
Left: 28-year-old, HIV-positive homosexual with expansive ulceration and advanced attachment loss in the mandible.

Middle: After gross debridement of the soft tissue and extraction of 41 and 31. Despite treatment, the ulcer became larger. Suspicion of cytomegalovirus infection (CMV, smear).

Right: CMV infection confirmed. Extraction of tooth 42 and systemic therapy. Ulcer healed.





Human Papilloma Virus (HPV)

318 Verrucae (Wart)
Wart on the gingiva of a 42-year-old, HIV-positive homosexual, caused by HPV. This lesion was removed using the CO_2 laser.

Left: Similar wart, caused by the same virus on the fingertip and nail bed.

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Neoplasms

- Kaposi sarcoma
  - Non-Hodgkin lymphoma
  - Spinal cell carcinoma

The most common neoplasm in HIV disease is the *Kaposi sarcoma*. It is an angiosarcoma of the endothelium of blood and lymph vessels, and can appear ubiquitously. The affected sites are dark red to bluish, with varying color intensity; they may be flat or exophytic in appearance; they are painless. Kaposi sarcoma of the oral cavity is observed frequently bilaterally on the hard palate corresponding to the course of the palatine arteries (Fig. 319), but is also observed on the soft palate, the gingiva (Fig. 320) and the buccal mucosa.

The etiology has been only partially elucidated. While the participation of the human herpes virus type 8 (HHV-8) is certain, the influence of angiogenic factors from mononuclear cells is only suspected. The Kaposi sarcoma is found in 10–20 % of all HIV disease. It is more common in homosexuals than in drug dependent patients (Grassi & Hämmerle 1991, Reichert & Philipsen 1998).

319 "Flat" Kaposi Sarcoma on the Palate
34-year-old homosexual with end-stage AIDS.
There is no proven therapy. Laser surgery has been recommended for lesions in the oral cavity, and radiotherapy for skin lesions. Systemically administered drugs such as cytostatics and Interferon may be considered.



320 Aggressive, Exophytically Growing Kaposi Sarcoma on the Gingiva
40-year-old homosexual with end-stage AIDS. Several months after this photograph, the patient died.

Right: Less pigmented and pronounced Kaposi sarcoma on the gingiva between teeth 11 and 21. The exophytic tissue can be "lifted."

Courtesy *M. Grassi*

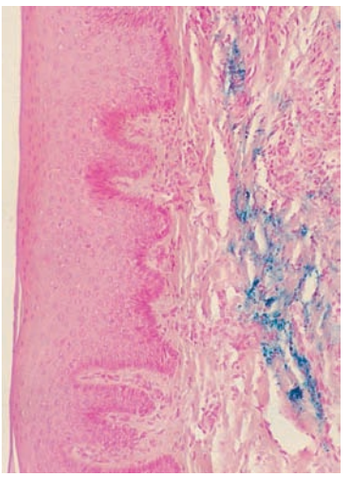


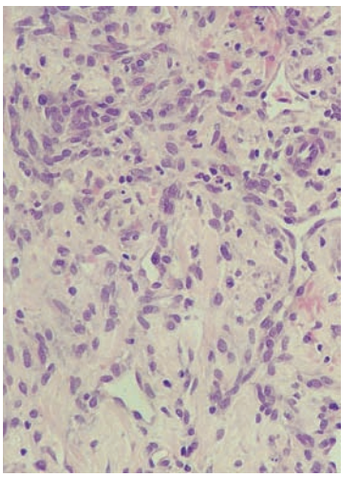


321 Histology of Kaposi Sarcoma
Left: Biopsy from the palate. Note keratinized epithelium. Blue-stained hemosiderin is observed within the connective tissue. The pigment derives from extravascular, dead erythrocytes (Berlin blue, x32).

Right: Large tumor cells exhibiting a vortex arrangement. Note also the proliferating capillaries (HE, x80).

Courtesy *S. Büchner*





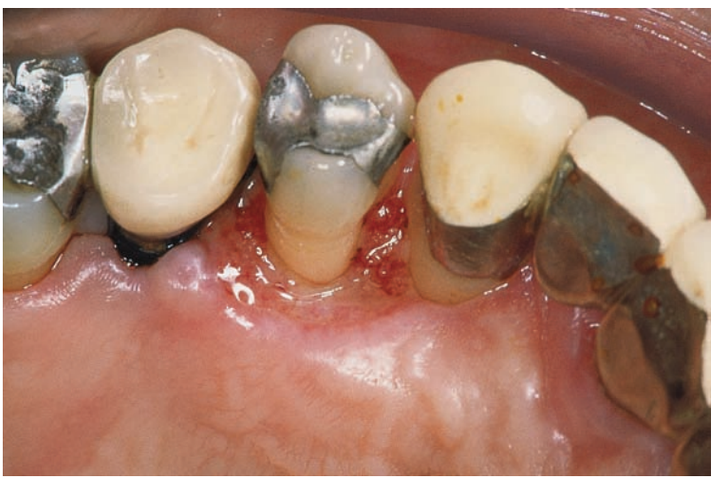
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HIV-Associated Lesions of Unknown Etiology

- Ulcers
- Aphthae
- Saliva gland disorders (xerostomia)
  - Tissue hemorrhage (thrombocytopenia)
  - Oral pigmentations

Ulcers of unknown etiology may occur in the region of the marginal gingiva as localized, expansive and sharply demarcated lesions. They can persist for long periods of time and, like aphthae, be extremely painful. The etiology of some ulcerous alterations is known, e.g., the necrotizing, ulcerative periodontitis (NUP), and the cytomegalovirus infection. Large, recurrent *aphthae* are more frequently observed in patients with compromised immune function. The etiology is unknown.

Saliva gland disorders and the resultant xerostomia as well as rampant caries occur in up to 50 % of HIV-infected children. The saliva glands in adults are less frequently affected. Especially the parotid gland is often enlarged. Such glands produce less saliva. This symptom is frequently coupled with lymphadenopathy.



322 Ulceration
In this 40-year-old male one observes a broad healing ulceration on the palatal margin of tooth 14. Because of this lesion and the expansive aphthae in this patient (Fig. 323), the dentist requested an HIV test. The result was positive.



323 Expansive Aphthae
The ulcer on the mucosa is coated with fibrin and the margins are erythematous. Causal treatment is not possible. Therapy is polypragmatic and symptomatic, involving rinsing solutions. Positive effects may also be obtained using tinctures and ointments that disinfect, anesthetize, cauterize or reduce inflammation (corticosteroids).



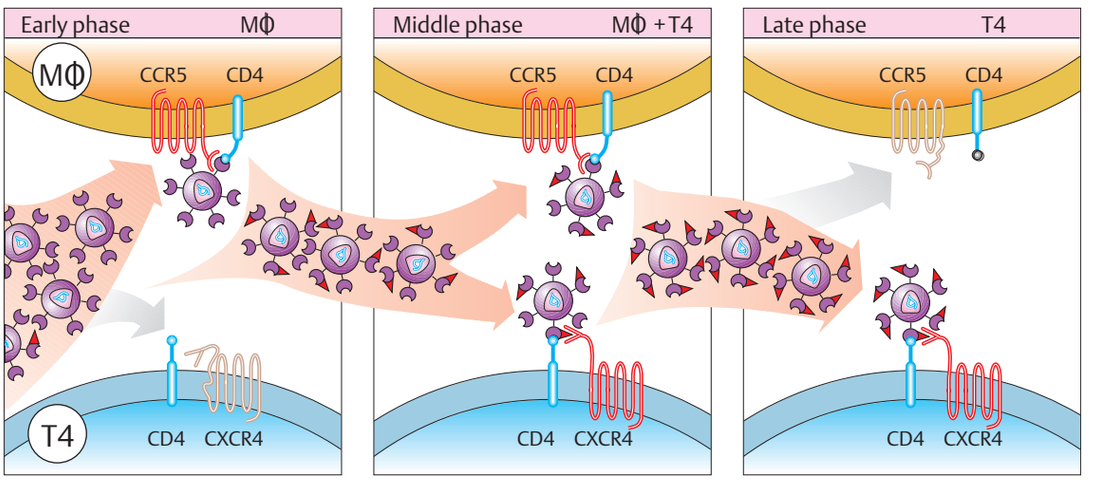
324 Caries with Xerostomia
This 42-year-old, HIV-positive homosexual exhibited bilateral enlargement of the parotid glands, which led to pronounced xerostomia. As a result, rampant caries developed throughout the dentition.
Restorative treatment and intensive preventive measures are highly indicated (oral hygiene, fluoridation, nutritional counseling, and possibly also saliva-stimulating sugar-free chewing gum).

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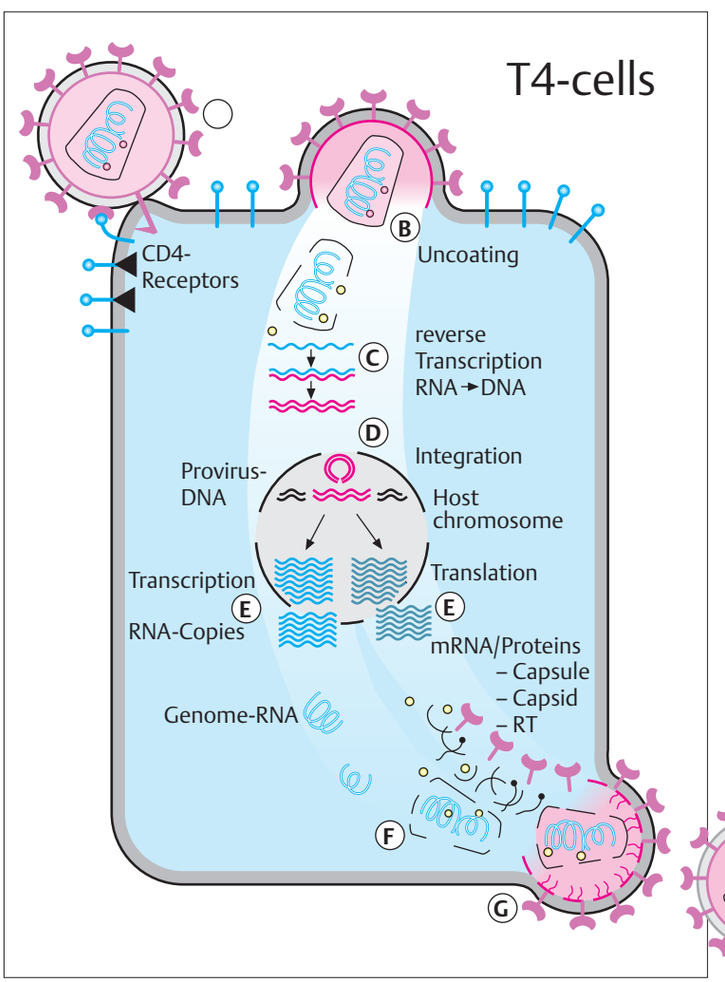
Invasion and Replication of the HI Virus—Hurdles for Systemic Medical Treatment

HIV affects monocytes/macrophages (MΦ) and T4-lymphocytes via CD4-receptors and co-receptors (Fig. 325). The replication within the T4-lymphocytes is depicted in Fig. 326.

The best way to conquer the HIV disease worldwide, and in a financially feasible manner, would be immunization. This is not yet possible (p. 149, vaccines). The pharmaceutical treatments used today and in development are targeted to inhibit the "docking" of the virus on target cells, and breaking the replication cycle. In the foreground today is blocking the reverse transcriptase (RT), which transforms viral RNA into DNA. An additional therapeutic possibility is the inhibition of proteases. These are necessary at the end of the replication cycle, for modification of the synthesized virus proteins. Other theoretical possibilities for therapy are depicted in Fig. 328.



325 Tropism of HIV
HIV strains exhibit affinity for macrophages and T-cells, and following infection they bind specifically via the HIV surface glycoprotein gp/20 (Fig. 302) to CD4 receptors on T4 cells and monocytes/macrophages.
A consequence of this binding is that the "knob" is removed, and a harpoon-like binding area (gp41) on the viral membrane is exposed. Endocytosis then occurs if corresponding co-receptors are present (MΦ: **CCR-5**, T4 cell: **CXCR4**; Chemoreceptors!).



326 HIV Replication Cycle in the T4 Cell

- **A** HIV "docking" on specific receptors.
  Example: T-helper cell
  – CD4 receptor
  – Co-receptor CXCR4
  – "Viral harpoon" binds the virus to T4 cells
- **B** Endocytosis and release of the single-strand viral RNA
- **C** Transcription of the RNA into host-readable DNA by **Reverse Transcriptase (RT)**
- **D** Integration of the "pro-virus" DNA into the host DNA by **Integrase (IN)**
- **E** Induced copying of ...
  – virus RNA = transcription
  – viral structural elements = translation
- **F** Initial congregation of viral RNA and capsule proteins
- **G** Budding of the immature HIV, modification of the structural proteins via **proteases (PR)**
- **H** Maturation into the infectious virus

Anti-retroviral Therapy—Possible Attack Points

- **A** Damage the free HI virus:
  – *Outside the organism:* disinfectants, solvents, detergents
  – *Inside the organism:* neutralizing antibodies, blocking the receptors (fusion inhibitors)
- **B** Inhibit endocytosis (Interferon)
- **C** **RT-inhibitor*** (Fig. 328):
  – nucleocidal
  – non-nucleocidal
- **D** **IN-inhibitor**
- **E** Anti-sense RNA
- **F** Glucosidase inhibitors
  IFNα = inhibits budding
- **G** **PR-inhibitor***
- **H** Immunization:
  – cytotoxic cells
  – neutralizing antibodies

* Used clinically since 1998.

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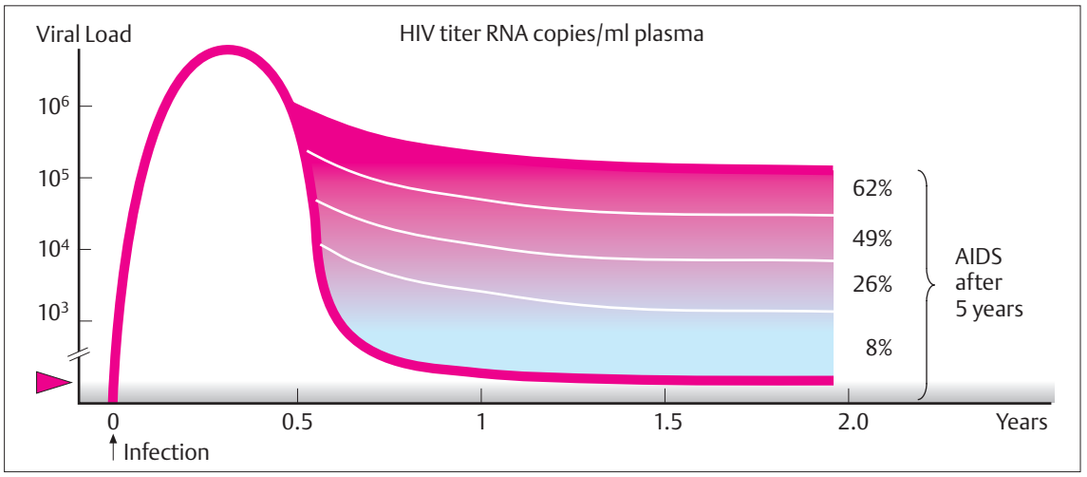
Treatment of the HIV Patient—Pharmacologic Aspects

Antiretroviral Medicines

Groups of drugs (Fig. 328) have existed for several years that have the capacity to reduce the viral load in blood plasma; especially the "triple therapy", a combination of three drugs (today even four and more). This drug regimen must be taken over the long term; it is extremely cost intensive, brings with it many serious side effects and is therefore compliance dependent. This treatment regimen is referred to as "HAART" (*highly active anti-retroviral therapy*). For compliant HIV-infected patients, this drug regimen can significantly increase post-infection lifespan.

Vaccines

Active or passive vaccines with the capability of immunizing healthy individuals or stopping further spread of HIV remain in the research stages and are not yet ready for use. But the clock is running quickly: Only an effective vaccine will be financially feasible worldwide and promise the capability to reduce the developing AIDS pandemic in Third World countries. To date the most promising are genetic vaccines, e.g., directly injected plasmid-bound DNA of certain HIV genes, which elicit a potent host immune response (Kennedy 1997, Weinert & Kennedy 1999).



327 Viral Load and Average Five-year Survival Rate for HIV-positive Persons (Mellors 1996)
The goal of anti-retroviral therapy is the elimination of virus particles from the blood (i. e., below the level of detection, which is becoming lower and lower because of more precise test methods). Success is acknowledged today when the level of viral RNA copies is less than 5,000 per ml of plasma

RT I Nucleoside analogs		
Videx	Didanosine	**ddI**
Epivir	Lamivudine	**3TC**
Zerit	Stavudine	**d4T**
Hivid	Zalcitabine	**ddC**
Retrovir	Zidovudine	**AZT**
Combivir	AZT + 3TC	
Ziagen	Abacavir	
Sustiva	Efavirenz	

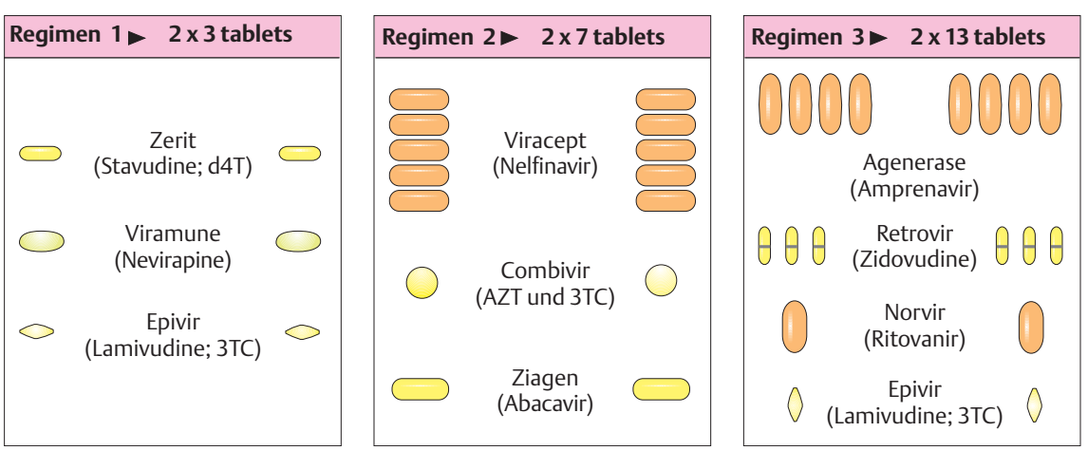
RT I Non-nucleoside analogs	
Rescriptor	Delavirdine
Viramune	Nevirapine
Stocrin	Efavirenz

PR I Protease inhibitors	
Crivixan	Indinavir
Viracept	Nelfinavir
Norvir	Ritonavir
Invirase	Saquinavir a (Hartgel)
Fortovase	Saquinavir b (Softgel)
Agenerase	Amprenavir

328 Antiretroviral Medicaments
Most widely employed today are agents that inhibit the viral enzyme *reverse transcriptase (RT)* and *proteases (PR)*. RT inhibitors prevent the translation of the viral RNA into DNA that can be read by host cells. PR inhibitors prevent the proper structural configuration of viral proteins, which—immediately following budding of the immature virus particles—are absolutely necessary for the definitive structure of the infectious virus.

The high mutation rate of HIV leads quickly to mutants that are resistant to an individual medicament.

On the other hand, the so-called "varying therapy" has been effective because it better prevents the development of resistance; these therapies, however, are often almost unbearable for the patients, who must consume up to 20 different tablets per day in precise intervals. In addition, as of 1998, most of these medications are associated with severe side effects.



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HIV—Treatment of Opportunistic Infections

In addition to the direct combat against AIDS via the antiretroviral polytherapy (see previous page), medicines must also brought to bear against frequent opportunistic systemic infections that accompany HIV disease:

- Bacterial infections
- Mycotic infections
- Viral infections
- Parasitic infections
- Additional combined infections
- Neoplasms etc.

Treatment for Opportunistic Infections in the Oral Cavity, Periodontitis Therapy

The systematic dental/oral examination of each patient with regard to mucosal surfaces, teeth and the periodontium has taken on additional significance because of the dissemination of the HIV infection and its oral manifestations. Not every person who is infected with HIV is aware of that infection, while others attempt to conceal their infection. Therefore, the "hygiene concept" must be high and vigilant in every dental practice and be applied for each and every patient. Because it is the law:

"Healthy" HIV positive individuals must be treated routinely in every dental practice.

If oral symptoms of the HIV infection are observed, these must be discussed with the patient's physician and appropriate treatment alternatives applied. Topical, oral therapy—especially the treatment of periodontal lesions (NUP)—remains in the dentist's hands.

Periodontal treatment for an HIV patient is similar to the procedure for the treatment of aggressive periodontitis in otherwise healthy individuals (p. 96): In parallel with the primary mechanical-instrumental therapy, there must also be topical medicinal supportive therapy, and sometimes systemic drugs. If systemic drug therapy is to be used, it *must not* employ only drugs that are reserved for the treatment of systemic life-threatening, opportunistic and classic infections (e. g., tuberculosis).

It must be kept in mind that additional systemic medication will only put an additional burden upon the HIV infected patient (additional medications, dosages, temporal sequence, as well as possible additional side effects).

A particularly unhealthy peculiarity in patients who are already immune suppressed is the *unavoidable recurrence of disease* following cessation of the systemic medication. The opportunistic infection is only suppressed, not eliminated. Effective healing rarely occurs.

A comprehensive synopsis of the dental problems of HIV patients was provided by Reichart & Gelderblom (1998).

The following medicaments have been proven effective for oral problems in HIV patients (p. 235, 283, 289):

- Topical — CHX (chlorhexidine)
  Iodine/Povidone (betadine)
- Systemic — Metronidazol, Ornidazole

Acute Phase

Periodontal lesions in HIV patients are often ulcerated and painful. Necroses and dull osseous pains are common. It is almost always impossible for these patients to perform adequate oral hygiene. Betadine rinses do not elicit pain (alcohol-free!), have antibacterial effects, are analgesic and therefore usually permit professional instrumentation without local anesthesia. Necrotic tissue must be removed.

During the first days following active therapy, most patients cannot perform mechanical plaque control at home: Therefore, chlorhexidine mouthwash should be prescribed.

Consolidated, Sub-acute Phase

Treatment of periodontal pockets in an AIDS patient does not differ from usual and conventional therapy (p. 151). Whether or not to cover the patient with a systemic antibiotic should be discussed with the patient's physician. The medication employed may be different depending upon the stage of the HIV disease, the type and severity of periodontitis and the already prescribed drugs.

The treatment of a patient with complex periodontitis (NUP) is depicted and described on pages 151–154.

Infection Prevention and Post-exposure Prevention—The Dental Team

The dental team protects itself and its patients by adhering to the hygiene regulations that are valid for all patients (gloves, mask and eye protection etc.)

Note: *HI-viruses are much less infectious than influenza or hepatitis viruses!*

If there is suspicion of HIV infection (e. g., needle stick wound), the protocol for "*post-exposure prevention*" must be followed: normally a 4-week, two-to-three medicament regimen (anti-retroviral) with various RT inhibitors. If the patient has an advanced HIV disease with a high viral load or if he is already being treated with RT inhibitors, normally an additional protease-inhibitor is prescribed (Reichart & Gelderblom 1998).

The technical aspects of medical insurance must be clarified.

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Treatment of HIV-Periodontitis

Clinical Procedure—Step-by-Step

HIV periodontitis usually occurs as necrotizing periodontitis (NUP). It can usually be successfully treated by local mechanical means (scaling and root planing) combined with medicinal therapies.

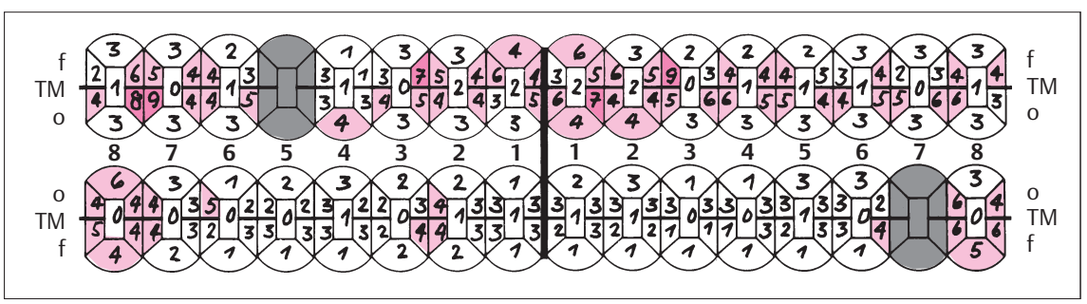
A 32-year-old male drug addict presented with severe pain in his gingiva. As emergency care, the necrotic tissues were carefully removed using universal curettes in a 2-hour appointment, using constant irrigation with betadine solution (Fig. 333). Betadine (10 % iodine) inactivates (oxidizes) chlorhexidine (CHX); the two should therefore never be used simultaneously. The second appointment included prophylaxis and oral hygiene instructions. The patient was told to rinse twice daily at home with a CHX solution (0.2 %). A 7-day regimen of systemic metronidazol (Flagyl) was prescribed (1 gm/day).

While the treatment in the mandible was quite successful, in the maxilla between teeth 11 and 21 a large ulcer formed with an expansive bony sequester. The four anterior teeth had to be extracted.



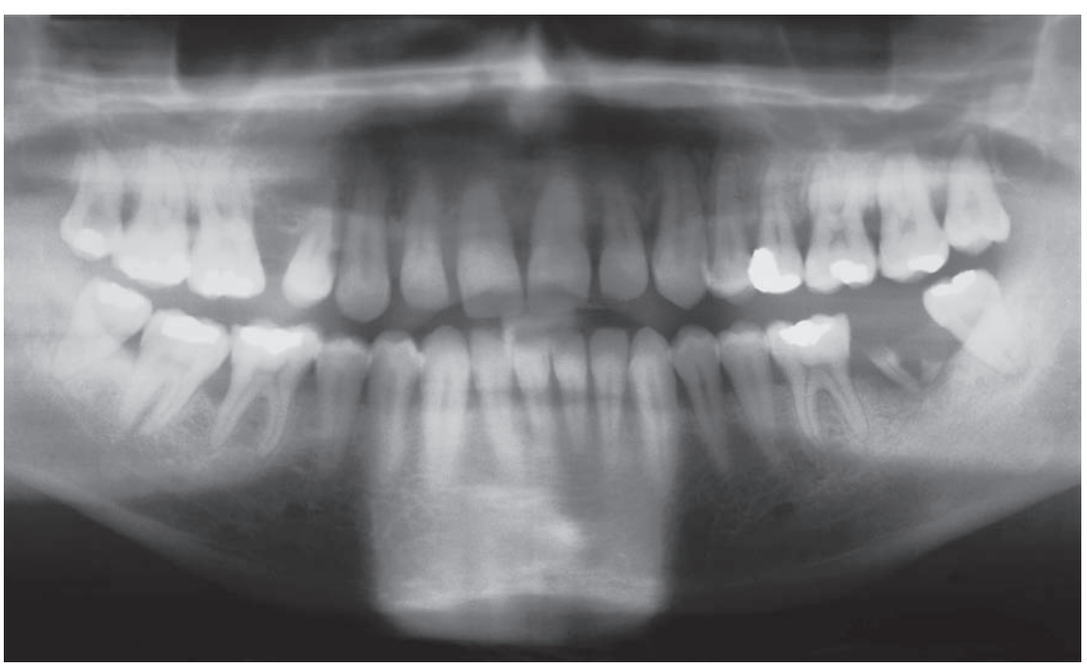






329 Clinical Overview (above)
Ulcerative gingivoperiodontitis (NUP). Immediately apparent is the severe band of necrosis in the mandibular anterior, canine and premolar regions. One notes "reverse architecture" in all other areas, as well as interdental craters.

330 Probing Depths and Tooth Mobility (Table)
Pockets are especially pronounced in the maxillary anterior and molar regions. The maxillary anterior teeth exhibited elevated tooth mobility.

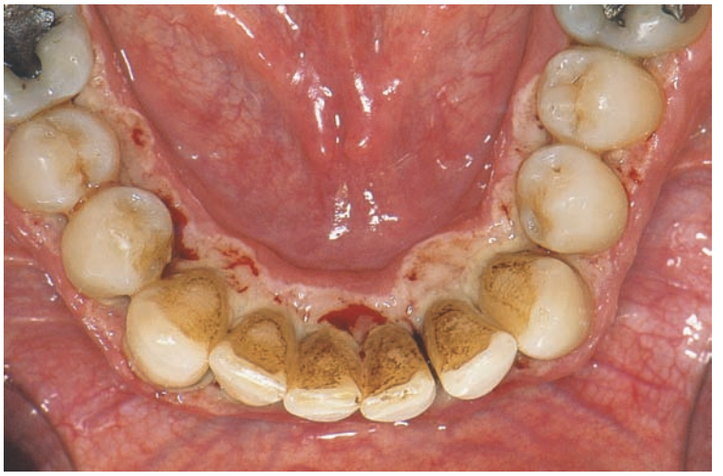


331 Panoramic Radiograph
The attachment loss is most pronounced in the maxillary anterior and molar regions. Tooth 15 was missing, and only the roots of tooth 37 remained.

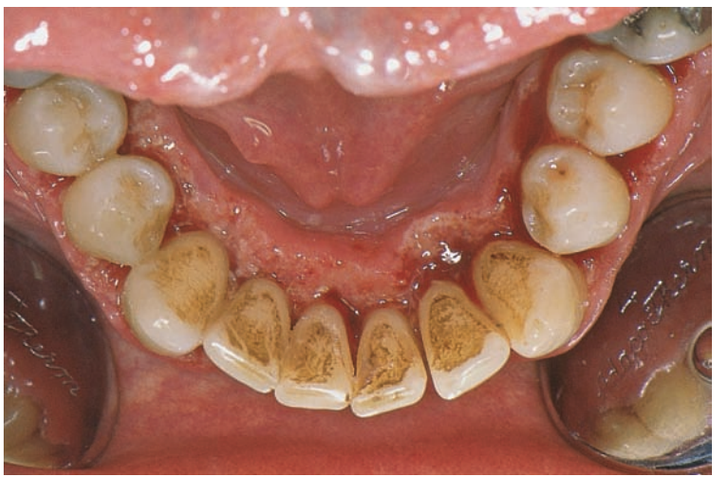
Three weeks following emergency treatment, teeth 18 and 28, as well as the root fragments of tooth 37 were extracted.

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Lingual View of the Mandible



332 Initial Findings
This lingual view clearly reveals the extensive band of necrosis in the anterior, canine and premolar regions.



333 Following Removal of the Necrotic Tissue and Initial Calculus Removal
This photograph was taken immediately following removal of the necrotic tissue and supragingival calculus. The careful soft tissue treatment was performed with continuous irrigation using betadine solution (10 %). This agent not only has antimicrobial effects, it also reduces hemorrhage and provides mild surface anesthesia.

Right: Betadine solution (10 %) and syringe for irrigation.





334 Four Days after Treatment
Tissue healing began immediately following the initial treatment. Four days later, the formerly ulcerated tissue has already begun to re-epithelialize. The patient was now free of pain.

Right: He continued daily rinses with CHX and took his metronidazol (Flagyl) for an additional 3 days (total of 7 days). Purely mechanical oral hygiene was re-instituted, checked and corrected.





335 Four Years Later
Clinical photograph taken during a recall appointment.
In the interim, new restorations were placed.

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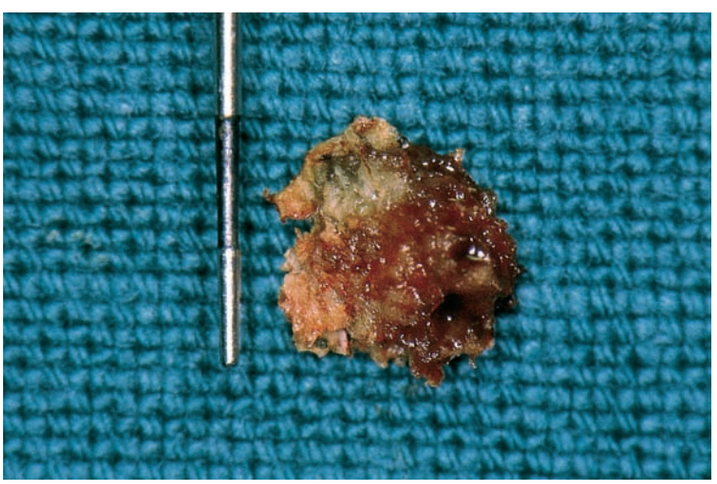
Palatal View of the Maxillary Anterior Region

336 Initial Situation; Same Patient
Pronounced ulceration in the region of teeth 11 and 21; both were highly mobile. Expansive ulceration such as this could be caused by the cytomegalovirus. These can only be successfully treated in combination with a systemic anti-viral (herpes-) approach. When this case was first seen (1990), that approach was not available.



337 Following Soft Tissue Debridement
The clinical view clearly shows that the necrotic tissue has been mechanically removed. A deep crater remains between the incisors. Subepithelial connective tissue has been denuded over a large area.





338 Bone Sequester
An osseous sequester with a diameter of ca. 6 mm was removed from between teeth 11 and 21.

Left: Periapical radiograph taken 5 months after initial therapy, immediately following removal of the bone sequester. The radiolucency in this film is considerably larger than the size of the sequester itself.



339 Maxillary Anterior Palatal View Immediately after Sequester Removal
Deep interdental crater with gingival retraction. Tooth 11 exhibited a mobility of grade 4, tooth 21, grade 3. The treatment plan included extraction of all four anterior teeth.

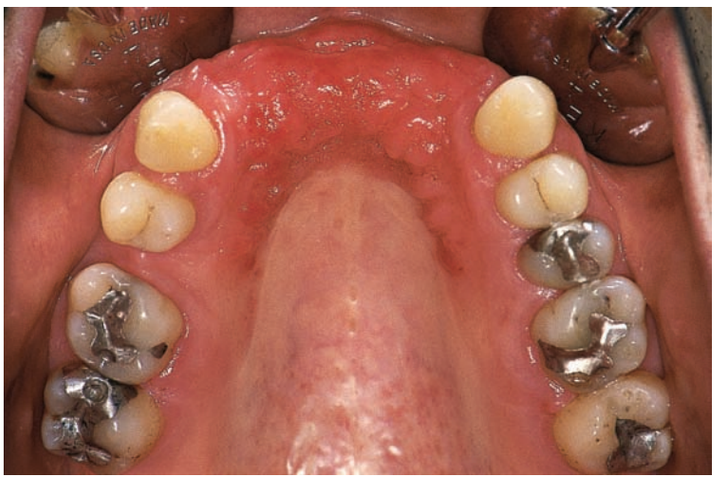
The expansive primary ulceration, the sequester formation and the remaining large defect could be attributed to infection with cytomegalovirus.

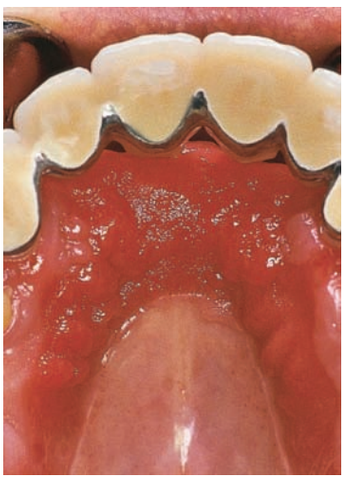
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Prosthetic Restoration of the Maxilla



340 Cast Framework Partial Denture
Immediately after extraction of the anterior teeth, a removable acrylic resin temporary denture was seated. It was replaced 8 months later by a cast framework partial denture (chrome-cobalt).

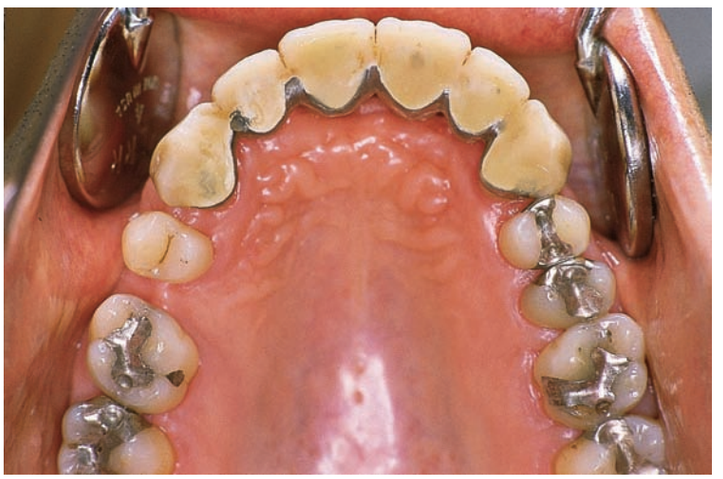




341 Irritation of the Mucosa by the Prosthesis
Mucosal erythema beneath the prosthesis became more severe over time. Allergic and toxicologic tests were *negative*. It is likely that mechanical and microbial irritations were the causes of this severe, localized inflammation in a patient with reduced immune response capacity.

Right: Fixed bridge (titanium-acrylic). The bridge was constructed to make plaque control as simple as possible.

Seven Years after Completion of Treatment



342 Maxillary Bridge
The mucosal erythema completely disappeared a months after seating the fixed bridge. This type of anterior bridge has been completely free of complications.



343 Mandible
With “more or less” regular recall every 4–6 months, the patient’s home care is of varying quality. Since the beginning of his dental treatment, he also participated in a methadone program; this drug leads to reduced saliva flow and, despite regular fluoride application, new caries developed. When this photograph was taken, the patient had been on a “triple therapy” with anti-retroviral medicaments for a 2-year period (p. 149).

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Gingival Recession

Type VIII B 1

Recession of the marginal gingiva (classified as Type VIII B 1) may result from various etiologies, and can occur in various clinical manifestations, including combined forms:

- *"Classical" recession* occurs in the absence of any infection, is inflammation-free, and is usually localized to the facial surfaces. It is the most common type of gingival recession, normally without loss of the interdental papillae (Figs. 349–350).
- Recession attendant to *untreated periodontitis* (most often the chronic forms): It progresses slowly, often over many years, and involves both marginal gingiva and papillae (Fig. 358).
- Loss of the marginal and interdental gingiva often occurs following *periodontitis therapy*, especially when resective treatment methods are employed (Fig. 359).
- Recession is a manifestation of the involution of aging, with retraction/recession of marginal and usually also the interdental gingiva (Fig. 360).

"Classical" Recession

Classical recession accounts for 5–10 % of all periodontal attachment loss. The term recession is defined as an *inflammation-free* clinical condition characterized by apical retreat of the facial and less often of the oral gingiva. Despite recession of the gingival margin, the interdental papillae usually fill the entire embrasure area in younger patients. Recession is usually localized to one or several teeth; generalized gingival recession is rare. Teeth exhibiting classical gingival recession are not excessively *mobile*. The periodontal supporting structures are generally of excellent quality. Teeth are never lost due to classical gingival recession alone! If the patient's oral hygiene is inadequate, secondary inflammation may occur and eventually pocket formation (periodontitis) may ensue.

Etiology: A primary factor is purely the morphology and the anatomy of the situation. The facial plate of bone overlying the root is usually very thin. Not infrequently, the root surface is completely denuded of alveolar bone (*dehiscence*), or exhibits *fenestrations* in the thin osseous lamella. Anterior teeth and premolars are most frequently affected.
Recession is initiated as a consequence of the morphologic/anatomic situation, and the following etiologic factors:

- Improper, traumatic tooth brushing, e.g., horizontal scrubbing, excessive force (Mierau & Fiebig 1986, 1987)
- Mild, chronic inflammation that may be only slightly visible clinically (Wennström et al. 1987a)
- Frenum pulls, especially when fibers of the frenum attach near the gingival margin
- Orthodontic treatment (tooth movement labially; arch expansion; Foushee et al. 1985; Wennström et al. 1987a)
- Excessive periodontal scaling (Caution at recall!)
- Functional disturbances (e.g., bruxism) as the cause for gingival recession continue to cause heated discussion.

"Classical" recession is illustrated in the following pages graphically, on skull preparations and clinically.

Radiographically, pure gingival recession localized to the facial surfaces of teeth cannot be diagnosed.

Therapy: With scrupulous and *proper* oral hygiene, recession can be halted. An atraumatic tooth brushing technique with a soft manual brush or a sonic device should be recommended.
Treatment for severe types of recession by means mucogingival surgery is described in detail beginning on page 397.

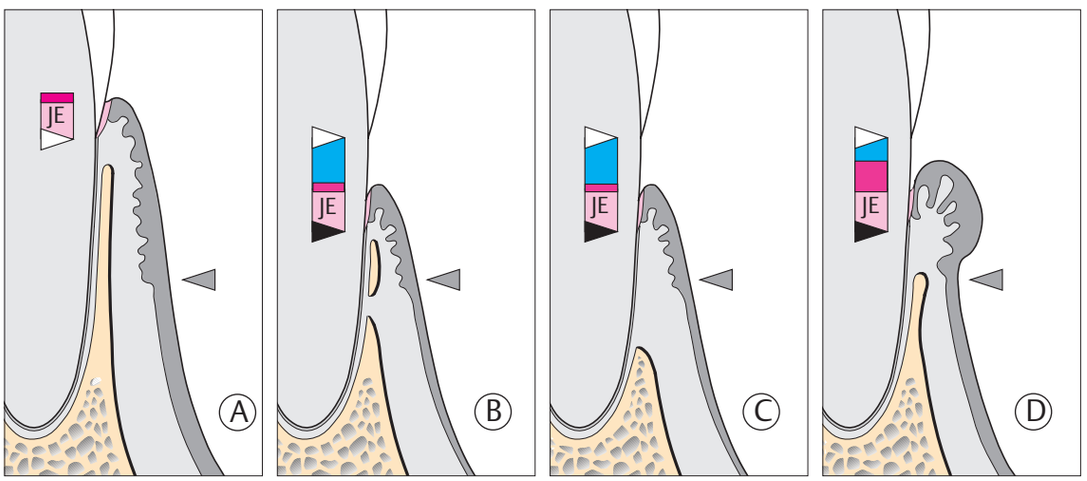
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Fenestration and Dehiscence of the Alveolar Bone

In a healthy periodontium the facial margin of the alveolar crest lies approximately 2 mm apical to the gingival margin, which courses near to the cementoenamel junction. The facial aspect of the alveolar bone covering the root is usually very thin. As revealed by a flap operation or on a skull preparation the coronal portion of the root often is not covered by bone (*dehiscence*) or there is a *fenestration* of the facial bony plate. Towards the apex, the facial plate of bone becomes thicker and trabecular bone fills the interval between the facial cortical plate and cribriform plate. In these thicker areas, recession generally stops spontaneously.

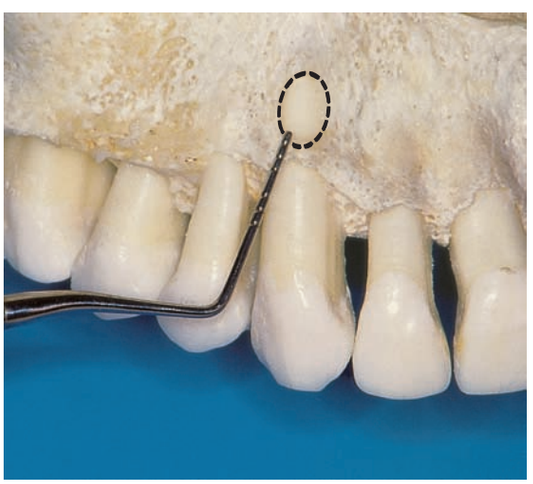
In *elderly* individuals, especially in those who have practiced excessive interdental hygiene for many years, recession of facial periodontal tissues may appear in combination with horizontal bone loss in the interdental area. In such cases, the interdental papillae usually also recede. Nevertheless, no *true* periodontal pockets are in evidence.

344 Normal Periodontium and Various Manifestations of Recession as Viewed in Oro-facial Section
Recession (blue), junctional epithelium (**JE**), minimal PD (red). The mucogingival line (arrowheads) and the CEJ are indicated.
A Gingiva and normal bone
B Simultaneous recession of bone and gingiva, fenestration
C Bony dehiscence more pronounced than gingival recession
D Recession with formation of McCall's festoon (Fig. 350).



Skull Observations

345 Fenestration (left)
Adjacent to the fenestration on tooth 13 (circle), dehiscences and horizontal bone loss in the interdental areas.



346 Dehiscence (right)
A pronounced dehiscence that extends almost to the apex is observed on the facial of 13. The other teeth exhibit dehiscences of lesser severity. Generalized interdental bone loss is also in evidence (right).

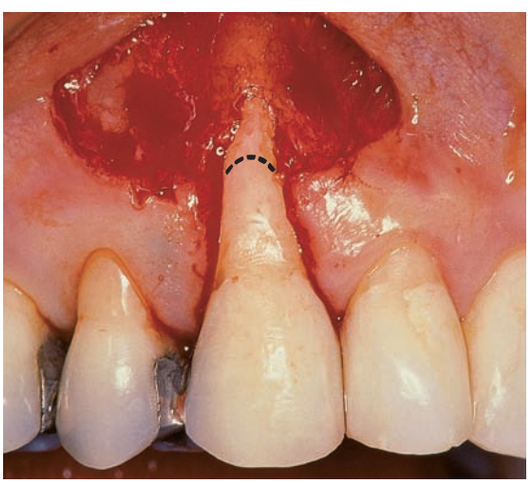


Findings During Surgery

347 Multiple Fenestrations
During the course of an Edlan operation, large fenestrations on teeth 16, 15 13 and 12 became visible after flap reflection (left).



348 Dehiscence on Tooth 13
During an extension operation using FGG, an unexpected osseous dehiscence was encountered, which had not been detected by probing. The dehiscence was more severe than the orginal gingival recession (right).



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Clinical Symptoms

- Gingival recession (of the entire gingival margin)
- Stillman cleft
- McCall's festoon

The clinical manifestations of recession are numerous. Gingival recession usually begins with a gradual apical migration of the entire *facial aspect* of the gingiva, revealing the CEJ. Less frequently, the first sign of recession is the relatively rapid formation of a small groove in the gingiva, a so-called *Stillman cleft*. This can expand into pronounced recession. As a consequence of recession, the remaining attached gingiva may become somewhat thickened and rolled, a non-inflammatory fibrotic response known as *McCall's festoon* (Fig. 350).

If the recession progresses to the mucogingival line, secondary inflammation of the gingival margin often occurs (Fig. 351).

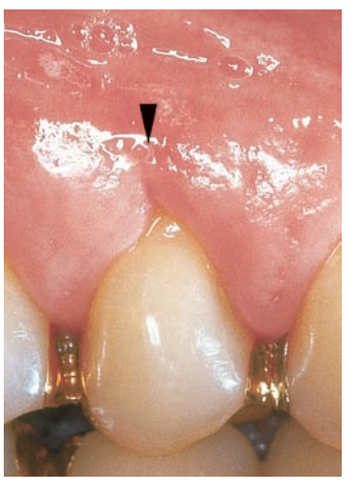
Gingival recession can lead to esthetic considerations in the maxillary anterior segment. As root surfaces are exposed, cervical sensitivity may also become a problem. Gingival recession is often observed on teeth that exhibit wedge-shape defects at the cervical area (p. 164).

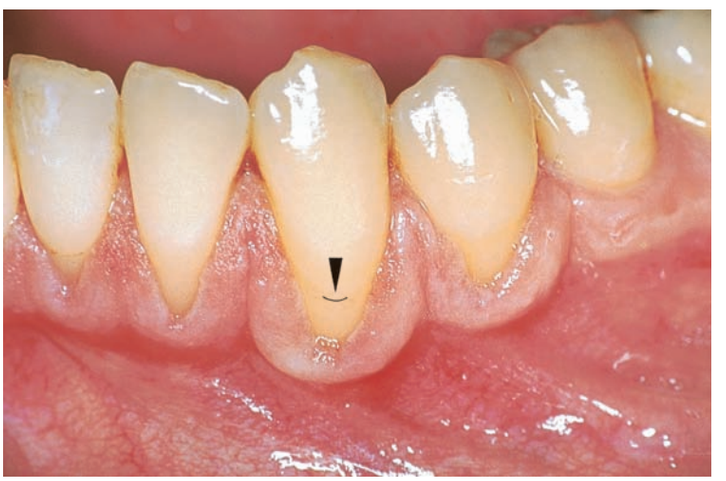




349 Initial Recession
Early exposure of the cementoenamel junction (arrows) due to recession of the gingival margin. The mobile oral mucosa has been stained with Schiller iodine solution (see Fig. 363).

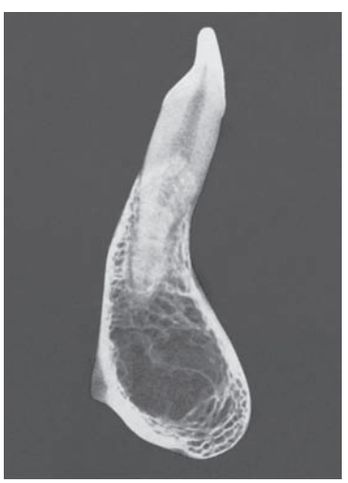
Palatal Recession (left)
Gingival recession on the palatal or lingual surfaces is considerably less common than on facial surfaces (morphology).





350 McCall's Festoons
The attached gingiva consists of nothing more than a collar-like, fibrous thickening (arrow). This *may* be a tissue response to further recession beyond the mucogingival line.

Stillman Cleft (left)
Cleft-like defect of traumatic etiology. Such clefts may spread laterally, creating an area of gingival recession. The exposed root surface may be extremely sensitive. Such clefts are often covered with plaque.





351 Severe Localized Recession
The root of this tooth has been denuded all the way down to the mucogingival line. The gingival margin is secondarily inflamed. Following initial therapy, a mucogingival surgical intervention was indicated in this case (p. 397).

Dehiscence of the Alveolar Process (left)
Orofacial section through an anterior tooth, as viewed in the radiograph. Remarkably little bone surrounds the tooth, facially and lingually.

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Recession—Localized

A 26-year-old male presented to the clinic with a chief complaint of recession on his maxillary canines. His oral hygiene was impeccable; he claimed to brush his teeth four times a day. Neither a dentist nor a dental hygienist had ever observed or corrected his tooth brushing technique.

Findings:

API: 10% PBI: 0.8

Figure 353 depicts probing depths, tooth mobility and areas of recession.

Diagnosis: Pronounced facial gingival recession on canines. Initial generalized gingival recession.

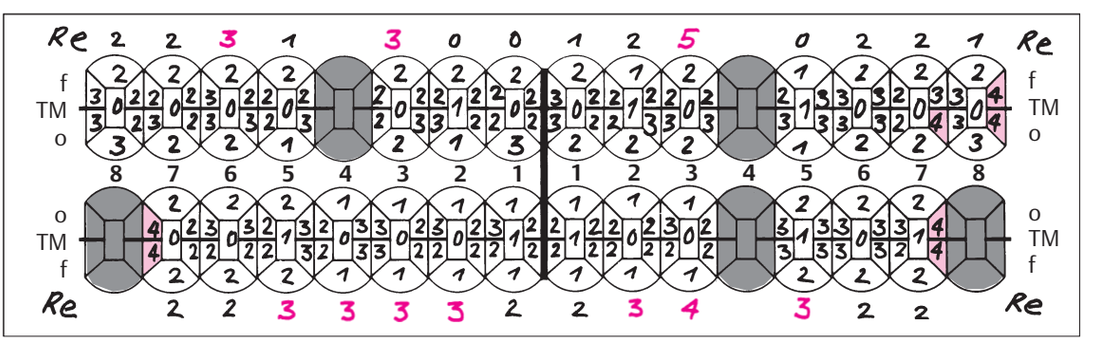
Therapy: Oral hygiene instruction, emphasizing a gentle technique. Study models provide a way to measure any progression of the gingival recession. If the esthetic situation is of great concern, one may consider surgical intervention to cover the recession, e.g., with a connective tissue graft on tooth 23 (p. 419).

Recall: 6 months or longer.

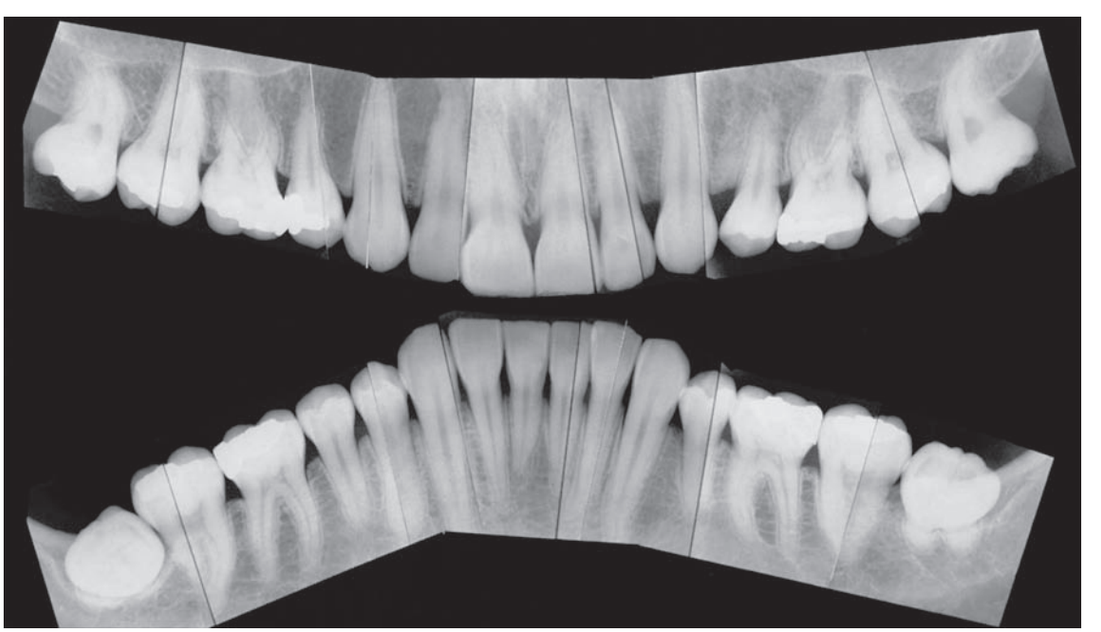
Prognosis: Good.



352 Clinical Overview (above)
The gingival recessions are of varying severity, with pronounced areas over the canines. The papillae still fill the interdental spaces. In the molar segments, mild marginal inflammation is noted. Tooth 35 exhibits a wedge-shaped defect.



353 Probing Depths, Recession (Re), Tooth mobility (TM; right)
The classical recession is associated with neither pathologically deepened pockets nor elevated tooth mobility.



354 Radiographic Survey
Facial osseous dehiscences are not visible on the radiograph (see clinical picture of the canines). Recession cannot be diagnosed using radiographs alone. In young patients, no loss of interdental septal height is noted.

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Recession—Generalized

This 43-year-old female was worried that her teeth appeared to be getting longer. Wedge-shaped defects were in evidence on all canines and premolars; the patient experienced cervical sensitivity in these areas from time to time. She expressed the desire to have her "gum condition" treated, if possible.

Findings:

API: 20% PBI: 1.0

Probing depths, recession, tooth mobility: see Fig. 356.

Diagnosis: Generalized, advanced facial recession. Slight loss of periodontal supporting structures in the interdental areas, but without significant pocket formation.

Therapy: Change home care technique; fabricate study models; monitor at regular intervals of 3–6 months. If the recession progresses, mucogingival surgery to halt or cover the recessions (p. 397).

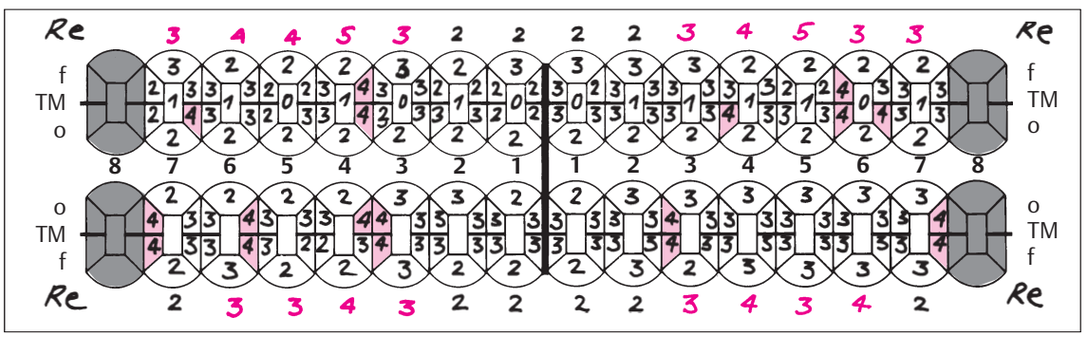
Recall: 6 months or longer, comparing measurements on sequential study models.

Prognosis: Good, if secondary inflammation is avoided and proper oral hygiene is practiced.



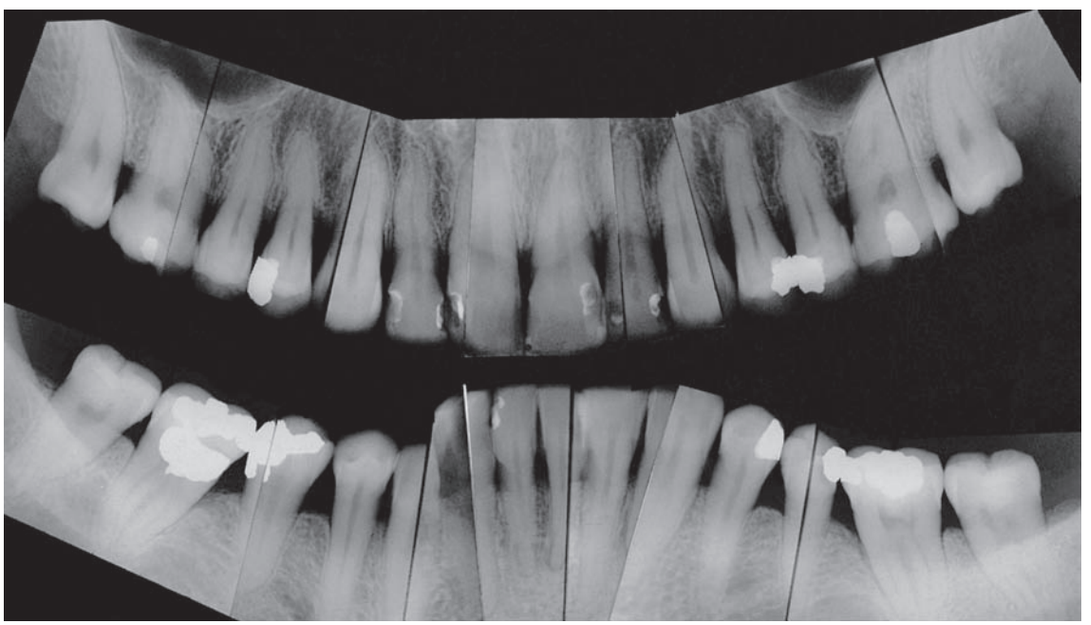






355 Clinical Overview (above)
Generalized gingival recession is apparent in this middle-aged female. The interdental papillae have also receded slightly. The interdental spaces have begun to "open."

356 Probing Depths, Facial recession (Re), Tooth Mobility (TM; schematic, left)
In the mandibular premolar area, the facial attached gingival is almost completely absent due to the gingival recession. In this area, attachment loss continued slowly despite changing the patient's brushing technique.



357 Radiographic Survey
Mild generalized, horizontal reduction of the interdental septal height is noted. The more advanced bone loss on the facial surfaces is not visible radiographically.

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Clinical Situations Resembling Recession

In addition to classical gingival recession, there are other forms of *clinically observable* attachment loss:

- *Shrinkage (recession) of the gingiva as a consequence of untreated periodontitis:*
  - ○ Symptom of periodontitis (p. 105).
- *Condition following periodontal therapy:* "Long teeth," open interdental spaces and cervical sensitivity are often the uncomfortable consequences of treatment for advanced periodontal disease.
- *Recession of the entire periodontium in the elderly:* This condition is *not* the rule. It may be caused by mild chronic inflammation and shrinkage; it can be enhanced by improper (overzealous) tooth brushing technique or other iatrogenic irritation.
  - ○ *Classical recession with a superimposed secondary periodontitis* (p. 359): This combination seldom occurs, because patients who practice proper oral hygiene usually exhibit neither plaque accumulation nor inflammation.

358 "Recession" (Shrinkage) in a Case of Untreated Periodontitis
This 32-year-old female with periodontitis presented with 5–6 mm pockets in the interdental areas. In addition, she exhibited 4–5 mm of gingival recession; thus, the true measure of attachment loss was 9–11 mm. This uncommon degree of gingival recession appeared to have been enhanced by improper tooth brushing technique (note wedge-shaped defects).



Similar clinical manifestations:
– at various ages
– with varying etiologies

32-year-old

Periodontitis

359 Clinical Picture Following Periodontal Therapy
In this 36-year-old female with advanced periodontitis (9 mm pockets) the esthetic consequences of radical surgical intervention (primarily resective, radical surgical operations) were severe. Although the "pocket reduction" was successful, the esthetic result was considerably less than favorable.



36-year-old

Therapy

360 Gingival "Shrinkage" in an Elderly Male
This 81-year-old man exhibited no periodontal pockets. Throughout the patient's life, the dentist had performed only restorative treatment. The patient had taken care of the periodontal "treatment" by himself, through vigorous brushing and a coarse diet (note wedge-shaped defects and abrasion).

Courtesy *G. Cimasoni*



81-year-old

Age

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Recession—Diagnosis

For many patients, gingival recession is the primary reason for seeking dental care. These patients are disturbed by the poor esthetics of gingival recession, they confuse recession with periodontitis, and they fear tooth loss.
Both dentist and patient can readily recognize gingival recession. Nevertheless, especially for treatment planning, precise data collection must be made, for example, using exact measurement methods. The dentist must clarify whether the recession is "classical" and without inflammation or pocket formation, or whether the clinical symptom is of untreated periodontitis (shrinkage), or the result of periodontal therapy (radical surgical procedures). The width of the remaining attached gingiva is of less importance, but if it is completely absent and if the mobile mucosa (labial or buccal frena) extends directly over the area of recession, the result can be an uncontrollable progression of the gingival recession.

Probing of the sulcus, use of the "roll test" with probe or finger, and staining the mobile mucosa are examinations that can be performed to predict whether or not complications will ultimately result from the recession.





361 Attachment Loss Due to Recession—Toothbrush Injury
In this case, the facial gingival recession from the cementoenamel junction to the gingival margin was 5 mm. It appeared as though absolutely no attached gingiva was present. In addition, the typical McCall's festoon was not in evidence, which would usually be viewed as a reparative response to the minimal gingival width.

Left: The radiograph does not permit diagnosis of facial bone loss.



362 "Roll Test"
Using the finger or a periodontal probe, the mobile mucosa is displaced toward the area of recession. This permits verification of whether or not attached gingiva provides any resistance against the roll test.
In the case depicted here, the mobile oral mucosa extends all the way to the gingival margin.





363 Iodine Test
Both gingiva and oral mucosa are painted with Schiller iodine solution (diluted aqueous iodine-calcium iodide) or diluted Lugol solution. The glycogen-containing mobile mucosa takes on a brown coloration, while the attached, glycogen-free gingiva remains unstained.
In this case, the iodine test clearly shows that there is *no* attached gingiva over tooth 23.

Left: Schiller iodine solution.

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Measurement of Recession (Jahnke)

Occasionally more precise methods are needed to more exactly define areas of recession, e.g., for scientific clinical studies. In an effort to quantitate gingival recession, Jahnke et al. (1993) measured not only the *vertical* extent of recession in millimeters from the cementoenamel junction to the gingival margin (measurement 1), but also the probing depth (4), which together with the recession provides a measure of attachment loss or the attachment level (2). In addition to the width of the keratinized gingiva (3), the *horizontal* expanse of the recession (5) and the adjacent papillae (6) are of significance for surgical treatment of the recession.

Classification of Recession (Miller)

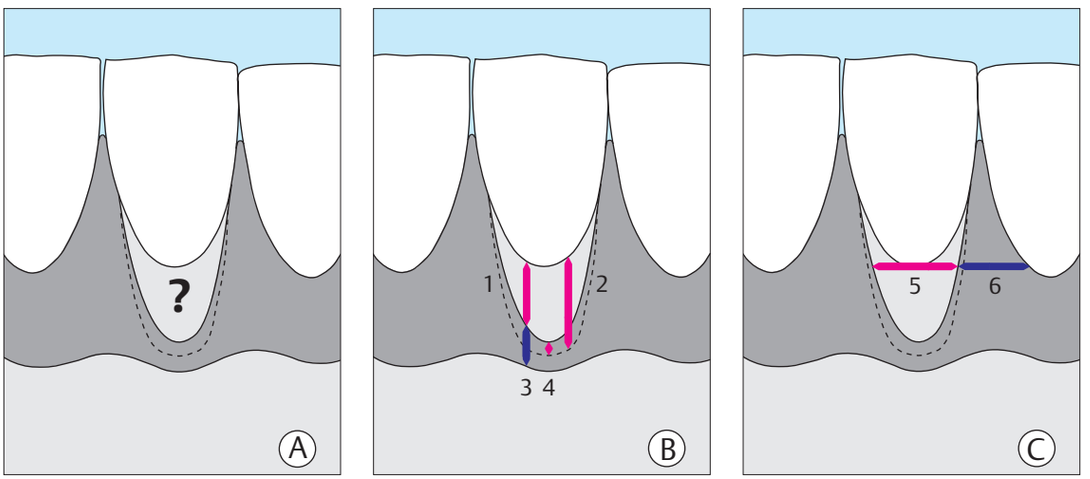
Also with an eye toward treatment, Miller (1985) devised a "classification of gingival recession." Without precisely measuring the extent and the various localizations of gingival recession, Miller described recession on the one hand with regard to its breadth and depth in relation to the gingival margin and the remaining attached gingiva; on the other hand, he described the loss of papillae, i.e., the interdental tissues. The Miller classes I-IV are significant in defining the possibilities and boundaries of surgical therapeutic modalities (e.g., covering recession, see p. 413).

364 Measurement of the Recession and the Surrounding Gingiva Tissue

This figure considers only the "classical" recession (length and width) and its relationship to the attached gingiva, the sulcus depth and the width of the adjacent papillae. These parameters are of significance for any anticipated surgical procedures.

Recession of the papillae themselves, which is important both for treatment and especially for the prognosis following treatment, is not considered. The "Jahnke measurements" are primarily used in scientific (computerized) investigations of gingival recession.

Modified from *P. Jahnke et al. 1993*



Recession: Millimeter Measurements (Jahnke et al. 1993)

- **Vertical Measurements**

  1 vertical recession
  2 probing attachment level
  3 width of keratinized tissue
  4 probing (pocket) depth

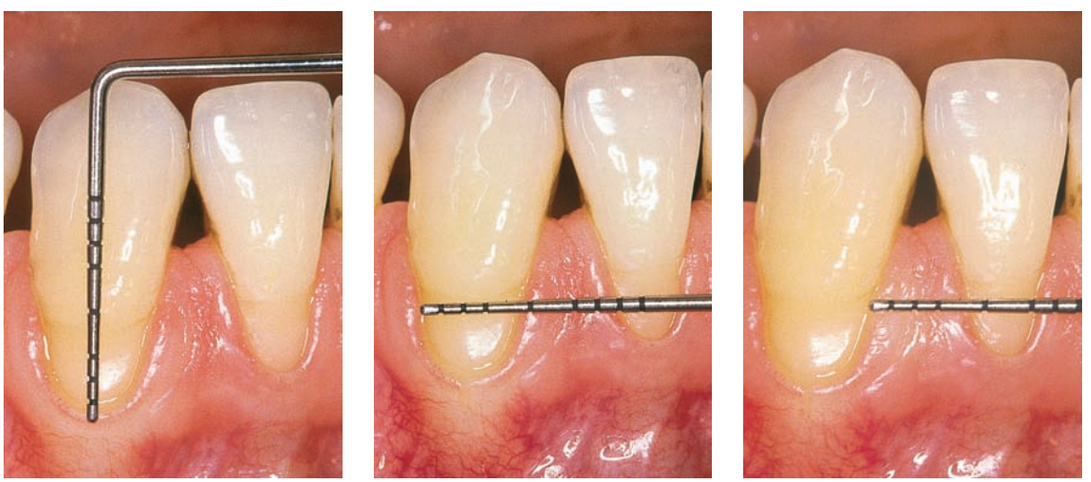
- **Horizontal Measurements**

  5 defect width at the level of cementoenamel junction (CEJ)
  6 width of interdental papilla at the CEJ

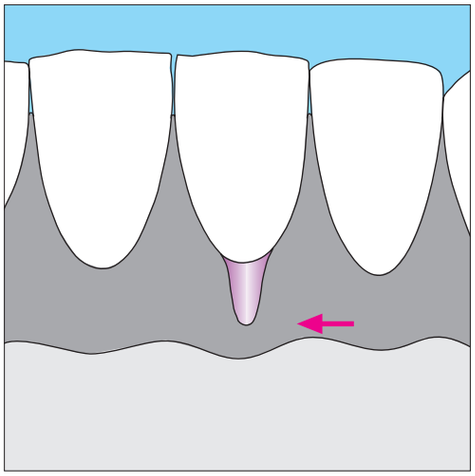
365 Clinical Measurement of the Three Most Important Parameters

1 & 2 Vertical recession and attachment level (left)
5 Horizontal recession/width (middle)
6 Width of papilla (right)

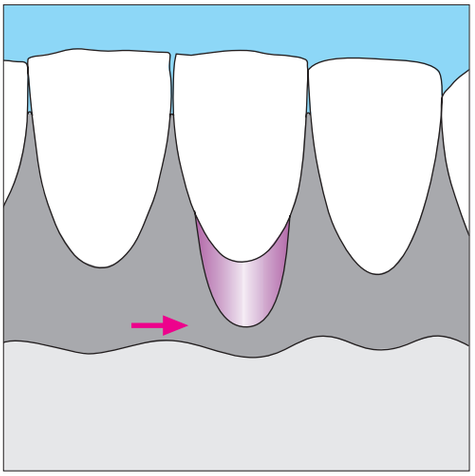
For a surgical procedure to cover areas of recession, it is the mass of surrounding tissue and not the expanse of the recession itself that is most important (nutrition for the graft).



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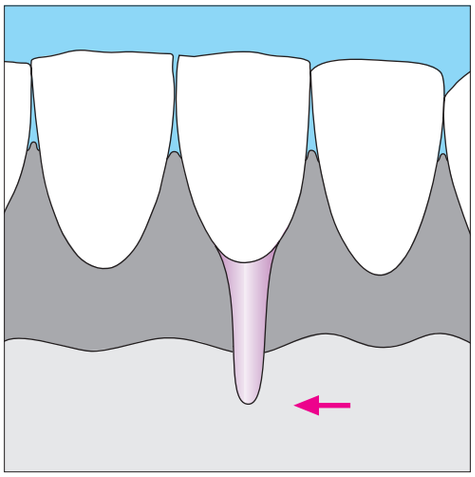
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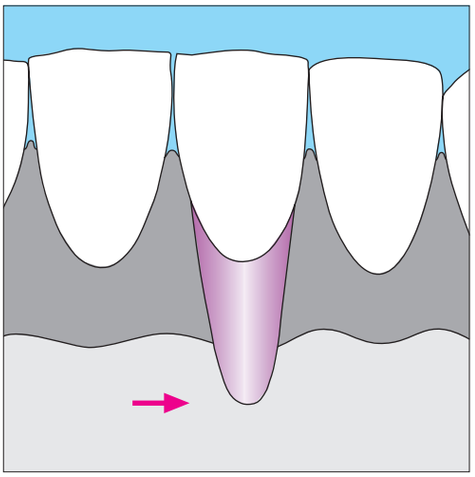
Classification of Recession (Miller, 1985)

366 Class I
Narrow *(left)* or wide *(right)*, localized "classical" recession isolated to the facial surface, with papillae filling the interdental areas. The defects do not extend to the mucogingival line.

Therapy: Complete, 100 % coverage of this type of recession can be achieved, e. g., by use of a free connective tissue graft.

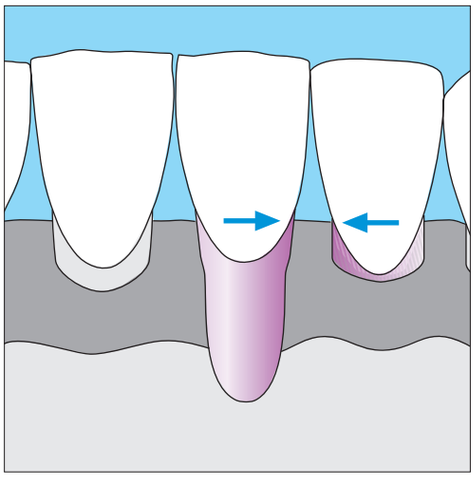


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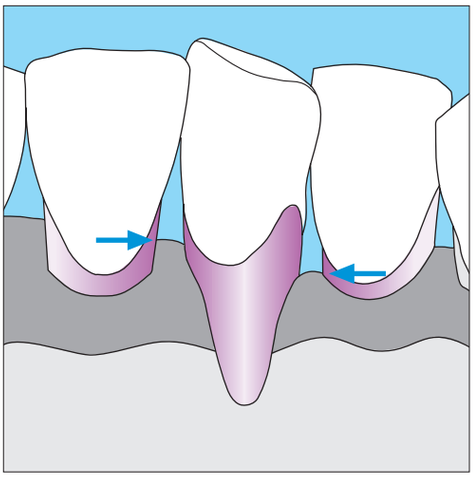


367 Class II
Narrow and wide, facially localized "classical" recessions, which extend beyond the mucogingival line into the mobile mucosa. The papillae remain essentially intact.

Therapy: Complete coverage of the root surface can still be achieved. With such deep recessions, guided tissue regeneration (GTR) with membranes can be employed (p. 435) instead of a connective tissue graft.

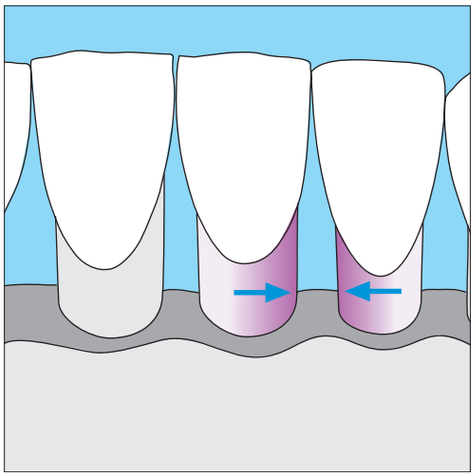


III

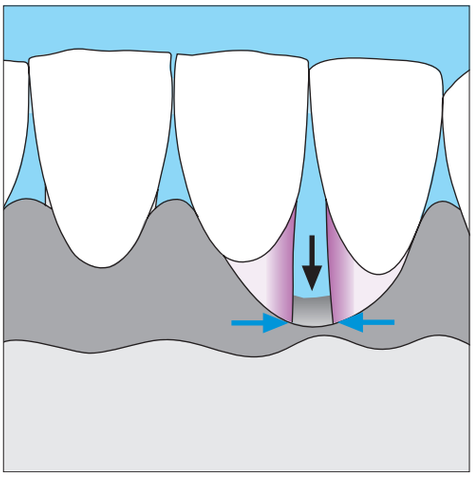


368 Class III
Broad recessions that extend beyond the mucogingival line into the mobile mucosa. The interdental papillae may be lost due to "shrinkage" and malpositioned teeth.

Therapy: Complete regeneration of such tissue defects is not possible; the facial root surface can, at best, be partially covered, and rebuilding the papillae is hopeless.



IV



369 Class IV
Left: Loss of periodontal hard (bone) and soft tissues around the entire tooth. The loss of tissue may be due to periodontitis, or to radical, resective periodontitis therapy.

Right: This type of tissue loss is frequently observed after repeated acute exacerbations of ulcerative gingivoperiodontitis.

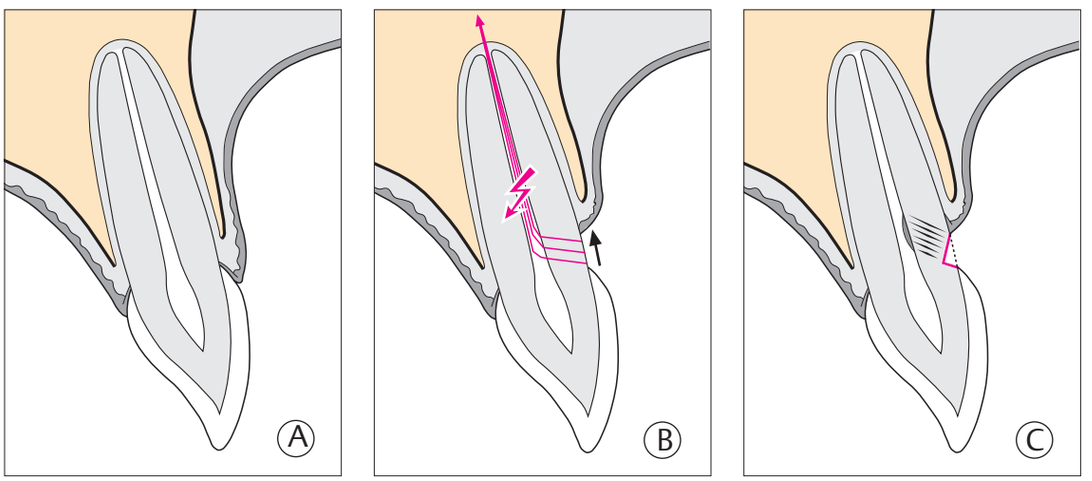
Therapy: Regeneration of the lost tissues by surgical procedures is only rarely possible.

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Consequences of Recession: Cervical Hypersensitivity, Wedge-Shape Defects, Class V Caries—Differential Diagnosis for "Erosion"

The consequences of gingival recession are myriad. In the visible regions, e.g., in the maxillary anterior, canine and premolar regions, patients often complain of the esthetic situation. *Hypersensitivity* may occur on teeth with gingival recession, and in some cases this problem is difficult to combat (Fig. 458). A further consequence, especially if the patient continues improper tooth brushing (horizontal) *wedge-shaped defects* may form, which are often highly sensitive. In elderly patients, especially those with advanced dementia (e.g., Alzheimer's), who usually exhibit poorer oral hygiene, the exposed root surfaces are highly susceptible to dentin caries, the treatment for which presents numerous problems (p. 456).

The classic wedge-shape defect must be differentiated from *erosion*. Erosion occurs coronal to the tooth neck in the enamel, primarily on maxillary anterior teeth and canines. The etiology usually involves a very acidic, calcium-binding diet (fruit, acidic drinks etc.) and the insufficient salivary flow in these areas (inadequate remineralization).

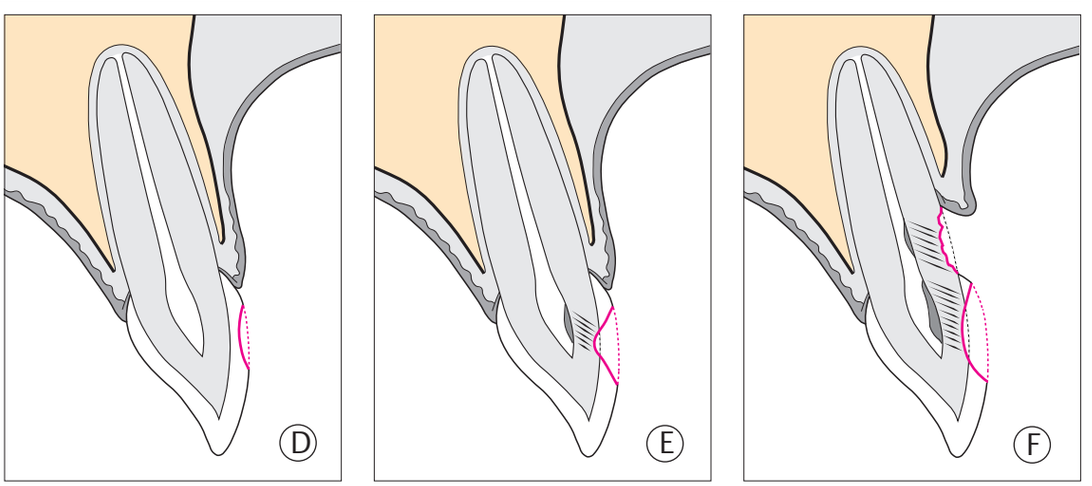


370 Facial Recession and its Consequences—Wedge-shaped Defects

A Healthy situation in a young patient.

B Recession caused by traumatic oral hygiene. Esthetic problems and sensitive root surfaces are common consequences; with poor oral hygiene in the elderly, root caries may also occur.

C If improper oral hygiene continues (horizontal scrubbing) deep, wedge-shaped defects will result.



371 Differential Diagnosis: Erosion

D Loss of superficial enamel from the clean, plaque-free tooth surface occurs due to the effects of acids over long periods of time (nutrition).

E Pronounced erosion into the darker dentin.

F *Combination* of pronounced erosion and a wedge-shaped defect with gingival recession. Both are caused by improper hygiene, especially after the consumption of acid-containing food or beverage.

Therapy: Gingival recession and enamel erosion can be prevented or successfully treated by therapeutic measures.

The best prevention for gingival recession and the consequential *wedge-shape defects* is a gentle and properly instructed oral hygiene technique. If recession occurs despite good oral hygiene, or progresses, it can be preemptively halted by a free gingival graft (FGG, p. 401). If the patient's esthetic concerns are primary, an attempt can be made to cover the exposed root surfaces by mucogingival surgical techniques, e.g., connective tissue grafting (p. 419).

Pronounced wedge-shaped defects that are not indicated for soft tissue coverage can be treated using bonding-composite materials after minimal tooth preparation.

Erosions should be diagnosed at the earliest possible stage (when limited to the enamel). Nutritional advice (e.g., immediate rinsing after ingestion of acidic foodstuffs), careful oral hygiene and fluoride use to enhance remineralization can bring erosion to a halt. Deep erosions, extending into the dentin (yellowish coloration) should be additionally treated using the acid etch technique, dentin bonding and composite materials. Only in rare cases are combinations of wedge-shaped defects and true erosions observed.

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Data Collection—Diagnosis—Prognosis

Before any therapy is undertaken, clinical and other relevant data must be collected, to serve as the basis for diagnosis and a preliminary prognosis. The clinician must determine whether the case is to be comprehensive care or whether the patient simply seeks a solution to a local, specific problem. Even in the latter case, it is nevertheless always necessary to gather succinct *systemic* and *special* histories, in order to detect systemic disorders, medical risks, current medications etc.
Most important today is the *comprehensive care patient*, for whom a comprehensive treatment concept and definitive treatment plan must be painstakingly considered. This is only possible if the patient's own wishes and notions are taken into consideration. In patients with severe periodontitis, exhibiting large bony defects, it may be necessary to obtain consultations from other specialists, e. g., oral surgeons, prosthodontists, orthodontists or even general medical practitioners.
The initial diagnosis and prognosis should always be viewed as a temporary or working diagnosis/prognosis. Of primary importance is the differentiation between chronic and aggressive forms of periodontal disease.
On the basis of the initial data collection, the preliminary treatment plan can be established, as well as alternative plans.

In the chapter on diagnosis, the following topics will be presented:

Findings
- systemic and special patient history
- "classical" findings
- additional findings such as microbial tests and examination of the host response

Risk clarification
- acquired and genetic factors
- risk estimation

Charting
- "classic" clinical data collection forms
- digitized findings, electronic data collection

Diagnosis—preliminary/definitive
- overall diagnosis, e. g., chronic periodontitis/Type II
- diagnosis for individual teeth or surfaces of individual teeth (see Charting)

Prognosis—preliminary/definitive
- for the patient as a whole
- for individual segments of the periodontium

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Data Collection—Examinations

Before performing extensive periodontal examinations, every patient should be subjected to a screening exam (e. g., PSR index, p. 73); this takes only a few minutes, and reveals whether periodontal lesions are present or whether other oral problems predominate.

If periodontal tissue destruction is detected, more extensive examinations are indicated. First among these are the "classic" examinations such as periodontal probing or measurement of attachment level (Fig. 383).

If there is evidence of special forms of disease, their etiologies and clinical course, additional facultative examinations should be performed. Such exams are especially indicated when one encounters:

- copious hemorrhage with slight plaque accumulation
- symptoms of disease activity (pus)
- advanced attachment loss in young patients
- elevated tooth mobility with mild bone loss
- suspicion of a systemic disease.

Only after collection and evaluation of these exams will it be possible to establish a more precise differential diagnosis (e. g., aggressive periodontitis).

372 Checklist of Obligatory and Supplemental Clinical Findings

Obligatory

The obligatory, "classical" clinical findings must be recorded for every periodontitis patient before initiating therapy. This requires a special periodontal chart, which may be enhanced by additional forms for the history, hygiene index, gingival indices and functional analyses. Such data collection can be performed using traditional data collection forms or, today, using computer-enhanced and -printed data collection forms. In either case, most important is that the obligatory examinations be performed systematically, and that data be collected for each individual tooth.

Supplemental

In severe cases, e. g., in aggressive, progressive forms of periodontitis, and/or with the existence of severe functional disturbances and anticipated major reconstructions, *supplemental* examinations will be necessary. Such examinations, and their necessity, will be determined and selected for each individual patient.

Beyond data collection, contemporary tests for specific bacteria and for host response in aggressive periodontitis cases provide an improved estimation of the risk potential and more targeted treatment planning (additional systemic medication?) and a more precise prognosis.

Examination/Findings		Patient Chart
Obligatory Findings		
General Medical History		Health History Form
Special history	→	Periodontal status, charts
Gingival inflammation	(PBI, BOP) →	Periodontal status (and recall charts)
Plaque score	(API, PI, PCR) →	Periodontal status
Probing pocket depths	→	Periodontal status
Clinical attachment loss	→	Periodontal status
Gingival recession	→	Periodontal status
Furcation involvement	F1—F3 →	Periodontal status
Clinical bone level	→	Periodontal status
Pocket activity	(exudate, pus) →	Periodontal status
Tooth mobility	TM →	Periodontal status
Minor functional analysis	(intraoral analysis) →	Periodontal status
Radiographic findings		Panoramic film, bite-wings, full radiographic survey
Supplemental, facultative findings		
Impressions for study models	→	Mounted study models
Facial and intra-oral photographs	→	Photographic survey, special findings
Microbiologic examinations		
Microscopy (dark field or phase contrast)	→	Protocol
Bacterial culture	→	Laboratory report
DNA tests	→	Laboratory report
Antigen-antibody tests	→	Laboratory report, protocol
Enzymatic tests	→	Laboratory report, protocol
etc.		
Examination of host response		
Interleukin-1 polymorphisms (IL-1 positive genotype)	→	Laboratory report
AST—Test of the enzyme marker for cell death	→	Protocol ("chair side")
Pocket temperature test	→	Protocol
etc.		
Tissue biopsy	→	Laboratory and/or physician's report
General medical/physical examination	→	Physician's report (cooperation)
Blood status (e. g., PMN defect analysis)	→	Laboratory report
Extensive intra-oral functional analysis	→	Functional analysis charts
Functional analysis and registration in the articulator	→	Mounted study models Functional analysis chart

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General Patient Health History

Each and every patient examination begins with the collection of the systemic, medical history. This is simplified by use of a health questionnaire, but such questionnaires do not replace a face-to-face discussion of the patient's history. The health questionnaire must be rendered complete through targeted questioning. It is very important, today, to ascertain acquired and genetic risk factors.

The general medical history serves to protect patients with systemic diseases, and also protects the dentist and dental team from potentially dangerous infections (p. 211).

Special Patient Health History

In addition to questions concerning the general health of the patient, special areas of the patient's background must be investigated: What was the patient's motivation to seek out the dentist? What oral complaints does the patient have, and what does she/he expect from the dentist? Are caries, periodontal, or prosthetic problems of prime importance? Is the oral mucosa diseased? Is the patient experiencing pain? And last but not least: Does the patient complain of esthetic problems with teeth which are too dark, or large interdental spaces, tooth positional anomalies, "long teeth," or excessively visible gingivae?

Medical History Questionnaire

Please fill out this form correctly and completely—thank you! Your responses are important and will be kept in complete confidence.—You are aware of the physician's code of confidence –

Family name ..... Given name .....

Street address ..... City and zip code .....

Profession ..... Date of birth .....

Home telephone ..... Business phone .....

Physician .....

Reason for this consultation .....

Do you now have, or have you experienced ...	**Yes**	**No**
– Chest pain on exertion (Angina pectoris)	❑	❑
– A heart attack ... (when?)	❑	❑
– Heart valve failure/artificial heart valve	❑	❑
– High blood pressure	❑	❑
– Elevated bleeding tendency ... (quick?/INR?)	❑	❑
– Stroke	❑	❑
– Epilepsy	❑	❑
– Bronchial asthma	❑	❑
– Lung problems, chronic coughing	❑	❑
– Allergic reactions ... (medicines?, other allergies?)	❑	❑
– Diabetes mellitus	❑	❑
Do you require insulin?	❑	❑
– Thyroid disease	❑	❑
– Liver disease	❑	❑
– Kidney disease	❑	❑
– Malignant diseases (leukemia, carcinoma, other)	❑	❑
– Infectious diseases	❑	❑
Hepatitis/"jaundice"	❑	❑
HIV—positive (AIDS)	❑	❑
Others, specify	❑	❑

Additional information ...

– Do you require antibiotic coverage before dental treatment?	❑	❑
– Are you currently taking any medications prescribed by a physician?	❑	❑
Other medications?	❑	❑
– Are you a smoker? How many cigarettes per day?	❑	❑
– For women only: Are you currently pregnant? (week, month?)	❑	❑

Date: ..... Signature: .....

373 Medical History Form
For new patients, the top section of the questionnaire contains the important personal data, which can subsequently be entered into the comprehensive, computerized office database.

The medical history form should be completed by the patient in the waiting room, to save time.

The medical history form comprises ca. 20 questions about the patient's medical history, which can be answered *yes* or *no*. By signing the form, the patient acknowledges the validity of her/his answers.

As mentioned previously, the dentist or dental hygienist should discuss the medical history form with the patient in order to enhance information.

In the case of severe systemic diseases, the prudent dentist will consult the patient's physician.

All information provided by the patient must be held in strict confidence.

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Classic Clinical Findings

Following collection of the systemic and special medical histories, the clinical examinations are performed. Recording of the "classic" findings (gold standard) remains of primary importance. It begins with a visual diagnosis. The entire oral cavity is inspected using a mirror. Even such a cursory inspection can reveal numerous manifestations of potential disease, e.g., plaque accumulation, gingivitis, gingival recession (Fig. 374). Brief inspection, however, can also lead to incorrect conclusions. For example, severe gingivitis may be incorrectly interpreted as periodontitis, or healthy appearing gingiva may mask true attachment loss. The important early diagnosis of periodontitis, including the existence of true periodontal pockets (clinical attachment loss, alveolar bone defects) can only be determined using the periodontal probe.

The clinical examination must be enhanced by radiographic diagnosis as well as vitality testing of all teeth. Elevated tooth mobility should be compared to the clinical findings (functional analysis, p. 174).

374 Cursory Inspection – Useless?
A brief inspection of the oral cavity reveals what appear to be healthy gingival conditions. However, such inspection can reveal only superficial alterations of the oral mucosa and teeth (see right), but reveal nothing in the *periodontal area*. On the other hand, visual inspection can be life saving: a *carcinoma screening*, e. g., floor of the mouth, palate, lateral lingual borders should be routinely performed, especially in heavy smokers (stomatitis, leukoplakia).



Teeth
- Condition of the hard structures
- Plaque accumulation
- Restorations (oral hygiene)

Gingiva
- Erythema
- Swelling
- Ulceration
- Recession

Oral Mucosa
- Effluorescences
- Discolorations
- Precancerous areas
- Tumors

375 Pocket Probing
In the same case (above), periodontal probing reveals that advanced periodontitis (labial attachment loss) is present in this area of healthy appearing gingiva.

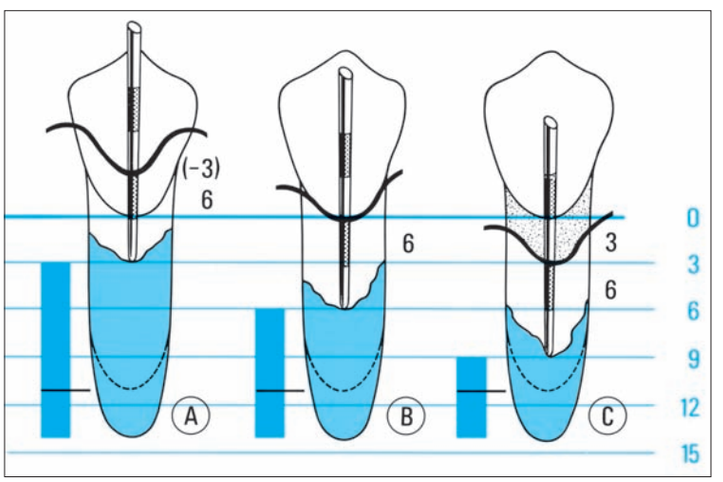
Right: The surgical site reveals:
- Loss of 3 mm of bone
- Burnished calculus in . . .
- . . . a shallow buccal groove on the root surface

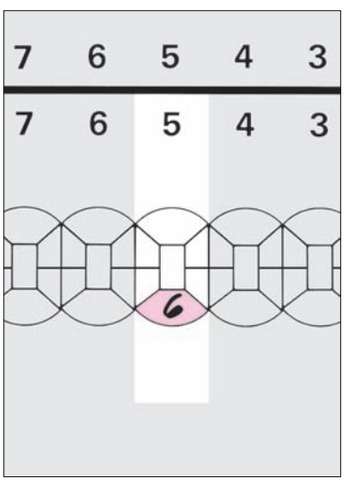




376 "Probing Depth 6 mm . . ."
This statement provides no information concerning attachment loss or how much attachment remains on a tooth (blue columns):

A	6 mm probing depth – 3 mm pseudopocket = 3 mm true attachment loss
B	6 mm probing depth = 6 mm true attachment loss
C	3 mm gingival recession + 6 mm probing depth = 9 mm true attachment loss



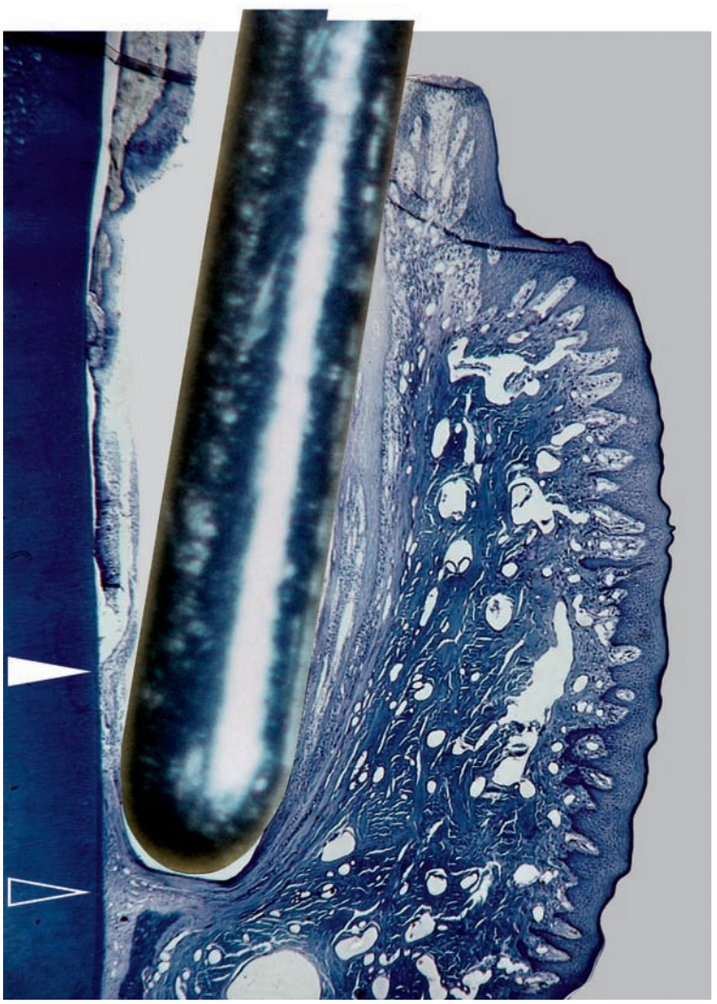


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Pocket Probing—Probing Depth, Clinical Attachment Loss

The primary symptoms of periodontitis are loss of tooth-supporting tissues ("attachment loss") and the formation of true gingival and/or bony pockets. It is for this reason that any clinical examination of a periodontitis patient must include the measurement of pocket probing depths and attachment loss. Unfortunately, the significance of these *clinical* measurements is only relative, and not always congruent with the anatomic-histologic realities (Armitage et al. 1977, van der Velden and Vries 1980, van der Velden et al. 1986); the clinical measurements are much more dependent upon the state of health of the periodontium (tissue resistance).

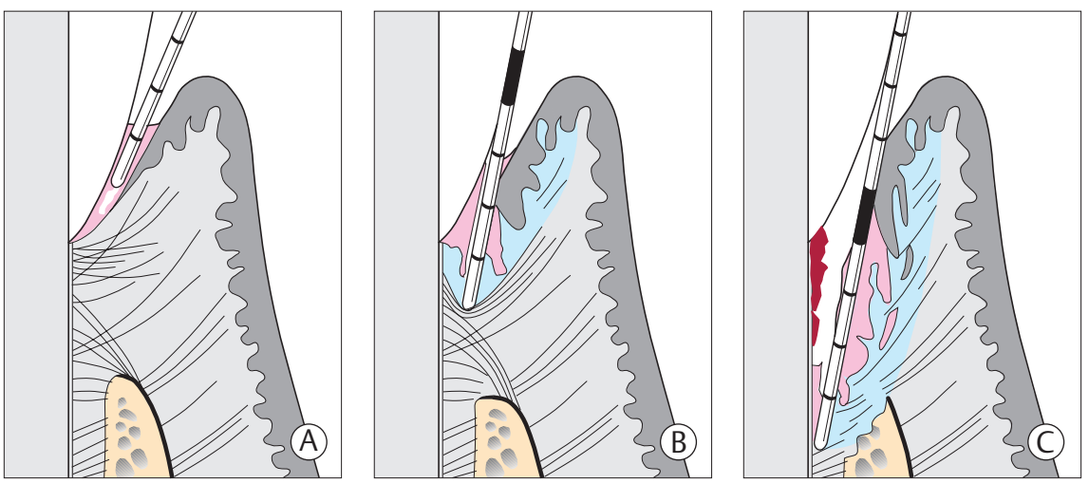
The tip of the periodontal probe always penetrates into tissue that is below the sulcus or pocket fundus, even when the recommended probing force of 0.20–0.25 N is applied. With healthy gingiva and a normal junctional epithelium, the sulcus is histologically a maximum of 0.5 mm deep, but periodontal probing routinely yields a 2.5 mm measurement. The probe penetrates intraepithelially *into* the junctional epithelium. If gingivitis or periodontitis are present, the probe tip perforates the pocket epithelium and the infiltrated, vascular connective tissue (hemorrhage!) to the first intact collagen fibers that insert into cementum.



377 Probing Depth versus Pocket Depth
This photomontage depicts a periodontal probe within a shallow, supracrestal pocket, with anatomically accurate spatial relationships:
The pocket epithelium is perforated, and the gingiva is severely deflected laterally. It is only the healthy collagenous fiber bundles and/or alveolar crest of bone that stop the probe tip from further penetration.

White arrow: pocket fundus
Empty arrow: probing depth

The *measurement error* between the true, histologic pocket depth and the clinical measurement (probing depth) can be up to 2 mm in severe periodontitis. For initial data collection, such error is usually of no consequence, but must be considered for the "before and after" comparisons following periodontal therapy. In most cases, the therapeutic result will be overstated, revealing an exaggerated reduction of probing depth.
Courtesy of *G. Armitage*



378 Probing Depth
A Healthy Gingiva
The probe tip remains in the junctional epithelium (pink) and no hemorrhage is elicited. PD ca. 2.5 mm.
B Gingivitis
The probe tip perforates the junctional epithelium (bleeding) and stops only when it encounters collagen fibers.
C Periodontitis
The probe tips perforates the junctional epithelium (bleeding) and is stopped only by contact with bone. PD 7.5 mm.

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Pocket Probing—Periodontal Probes

For the measurement of pocket probing depths, an enormous variety of instruments is available. When used properly, most of these instruments fulfill their purpose; however, probes for use in routine daily practice and for scientific investigations can be differentiated.

Special plastic probes are used when "pockets" around a dental implant must be measured (mucositis, peri-implantitis).

The trend today is toward periodontal probes with simple millimeter markings, e. g., at 1 mm intervals. It is important that the readability of the probe is maintained.

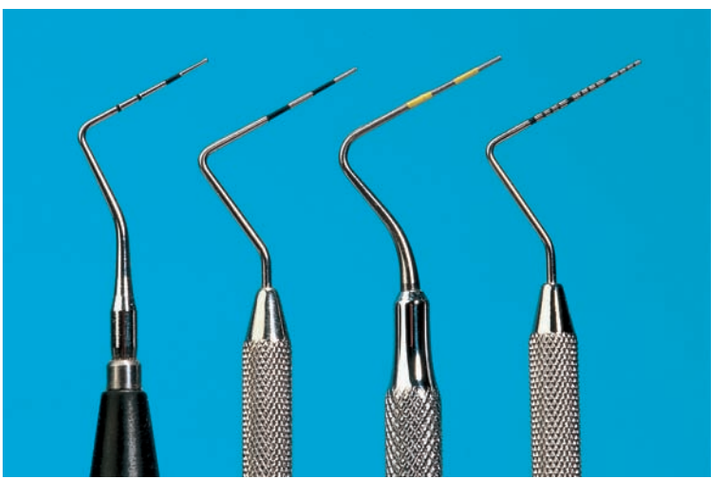
Also today, periodontal probes with a tip diameter of 0.5–0.6 mm and a *rounded* tip are preferred.

The recommended probing force is ca. 0.20–0.25 Newtons (ca. 25 gm). Each dentist and dental hygienist should test her/his probing force using a laboratory scale, or if this is not available then using the nail bed (pain!): Research has shown that the probing force is generally too great, leading to incorrect measurements. A further important clinical observation is that the periodontal probe tip may be impeded by subgingival calculus and therefore not reveal the true and maximum pocket probing depth.

379 Periodontal Probes with Various Millimeter Indications
Metal probes with precise measurement indicators in mm.
From left to right:

- **CPITN/WHO Probe** (Deppeler): 0.5 (ball): 3.5, 5.5, (8.5, 11.5)
- **CP 12** (Hu-Friedy): 3, 6, 9, 12
- **GC-American:** 3, 6, 9, 12
- **UNC 15** (Hu-Friedy): millimeter markings, and a wide, black marking at 5, 10 and 15 mm

Right: UNC 15, enlarged.

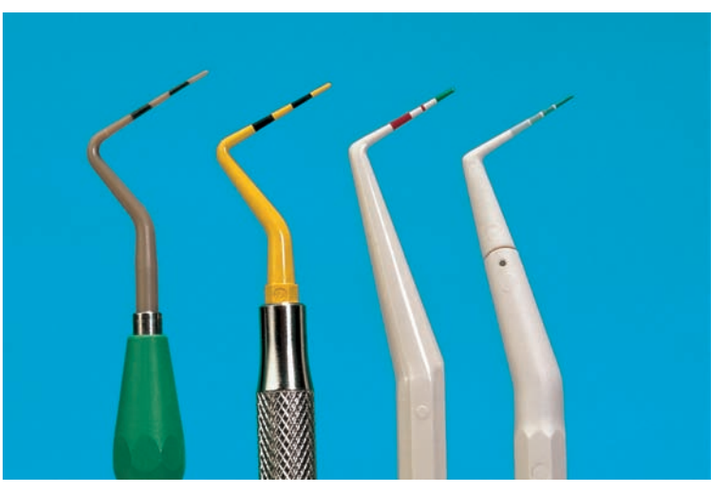




380 Plastic Probes
Sterlizable probes for pocket depth measurement around dental implants.
From left to right:

- **Deppeler:** 3, 6, 9, 12
- **Hu-Friedy:** 3, 6, 9, 12
- **Hawe:** 3, 5, 7, 10
- **Hawe "Click Probe":** 3, 5, 7, 10

Right: Esro **"Peep Probe"**: 3, 6, 9, 12. When a force of ca. 0.20 N is applied, this probe provides an acoustic signal.

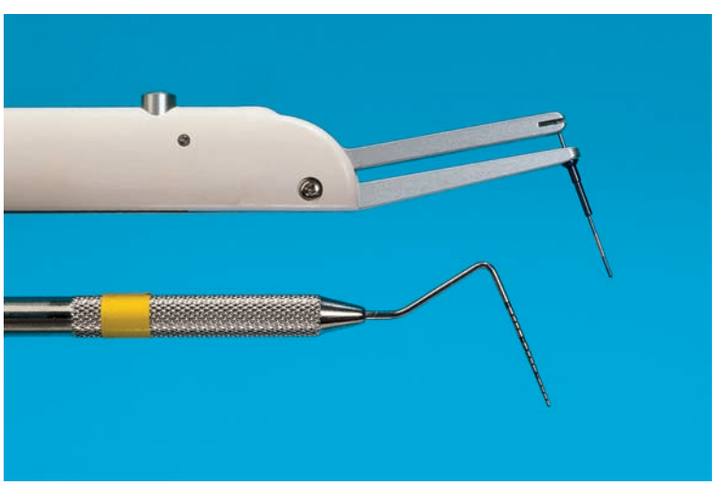




381 Florida Probe System
The *titanium tip* (diameter 0.45 mm) of this electronic probe measures pockets around teeth and implants with a normalized force of 0.25 N and with a precision of 0.2 mm.
Comparison: UNC 15 probe.

Right: The three probes of the Florida System measure from different reference points:

- **Disc Probe**
- **Stent Probe**
- **PD Probe ("Pocket Depth")**





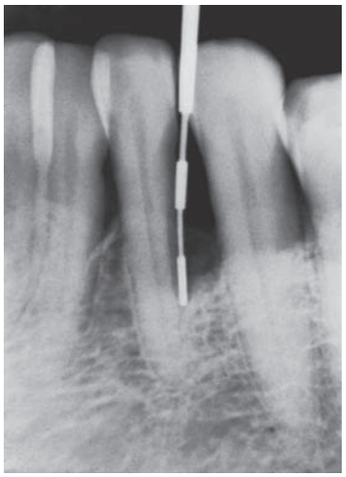
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Pocket Probing Depths—Interpretation of the Measured Values

During the clinical examination of a periodontitis patient, the consideration of *pocket depth* receives high consideration, but the actual histologic depth of pockets *cannot* be determined clinically, because the probe tip always penetrates into the tissues. In the final analysis, the clinical probing results depend greatly upon the severity of the inflammation, i. e., the resistance offered by the periodontal tissues. On the other hand, the probe tip may also be inhibited by calculus, root irregularities etc.

In general, the following parameters can be ascertained with a periodontal probe, either mechanical or electronic:

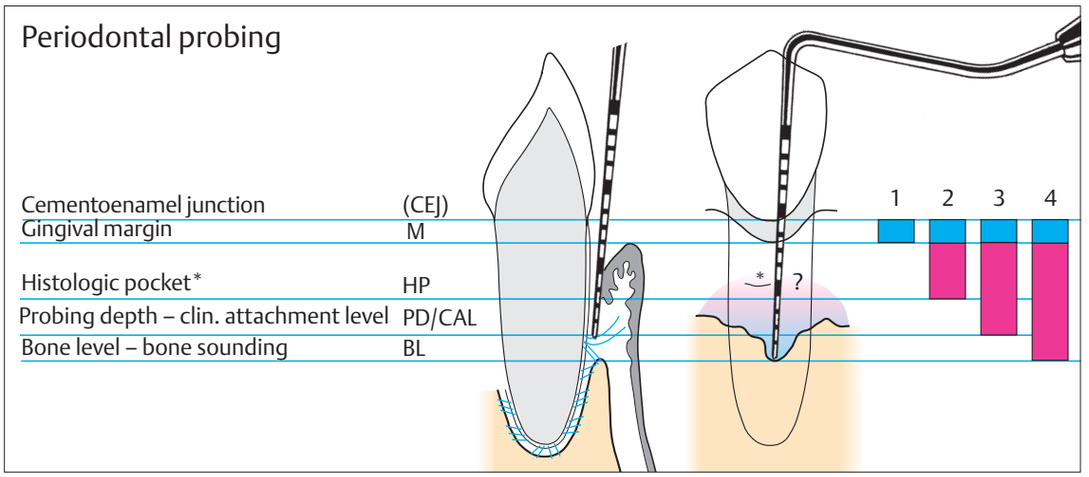
- *Probing pocket depth*: Measurement from the gingival margin to the point at which the probe tip stops; inaccurate
- *Clinical attachment level* (CAL): Measurement from the cementoenamel junction (CEJ) to the point at which the probe tip stops (PDL fibers); more accurate
- *"Bone sounding," under anesthesia*: Measurement from the gingival margin to the alveolar crest
- *Recession*: Measurement from the cementoenamel junction (CEJ) to the gingival margin
- *Gingival swelling*: Measurement from the CEJ (often difficult to ascertain) to the coronal gingival margin.





382 9 mm Deep Bony Pocket—Distal of Mandibular Left Lateral Incisor
The probe tip penetrates the remaining junctional epithelium and fundus of the pocket, and reaches the alveolar bone with only minimal pressure (ca. 0.25 N) in this case of inflamed periodontal tissues.
Advanced horizontal bone loss, with initial vertical, cup-shaped bone resorption.

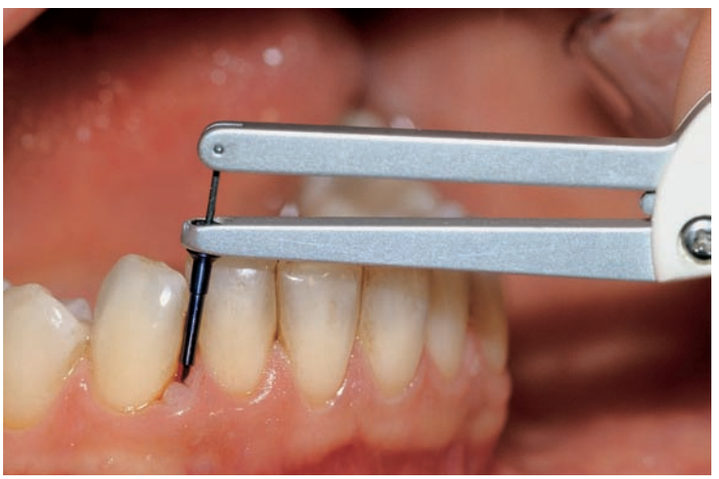
Left: The probe tip appears to approach the osseous niveau.



383 Schematic Representation of Measurable Parameters
With the exception of gingival swelling, the above-mentioned measurement possibilities with a periodontal probe are depicted. The measurements shown here on the buccal aspect can be performed on at least six sites around each tooth (p. 194)!

The bar graph depicts:
1 Gingival recession (blue)
2 Histologic pocket fundus
3 Clinical probing depth
4 "Bone sounding"





384 Florida Probe in situ
The probe exhibits a probing depth of 7 mm. It is clear that the guiding tube must approximate the gingival margin during this measurement.

Left: The thin (0.5 mm diameter) Florida Probe is depicted in a radiograph; the radiographic image is relatively small because the probe consists of titanium.

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Furcation Involvement—Horizontal and Vertical Furcation Invasion

Root surface irregularities, root fusion, enamel projections

Treatment would be greatly facilitated if all tooth roots exhibited a round-oval profile! Very often, however, even single-rooted teeth exhibit concavities and the roots exhibit an hourglass shape. In the case of multirooted teeth, the practitioner must ascertain the exact location of the furcation (root trunk length, furcation entrance angle) and to what degree the furcation is already involved by attachment loss. On the other hand, one must also determine to what degree the individual roots are fused, a situation that often is accompanied by deep, narrow grooves along the root surface in the furcation area. Enamel projections (p. 382) and "pearls" in or near the furcation also deserve attention. Even the healthy root (cementum) surface is never smooth, rather it exhibits rough areas, and lacunae often occur in the apical region as well as in the furcations, where they are often pronounced (Schroeder & Rateitschak-Plüss 1983, Schroeder 1986).

The detection of such morphologic and pathologic peculiarities becomes more difficult as pocket depth increases, becomes narrower and more tortuous in the furcation region.

385 Special Probes for the Diagnosis of Furcations, Depressions and Grooves:

- **EX 3 CH** (HuFriedy): Fine and pointed, paired left-right, bent/curved, for checking surfaces, narrow grooves
- **PC-NT** 15 (HuFriedy): Right-angle probe; millimeter markings, with color-coding each 5 mm (5, 10, 15)
- **PQ 2N** (Nabers; HuFriedy): Color-coded furcation probe (markings at 3, 6, 9 and 12 mm)



Severity of Furcation Involvement:

Horizontal (Grade 0–3)
F0 –
F1 up to 3 mm
F2 > 3 mm
F3 through-and-through between two roots

Vertical (Subclasses A–C)
A up to 3 mm
B 4–6 mm
C ≥ 7 mm

The length of the root trunk from the CEJ to the furcation roof must also be considered.

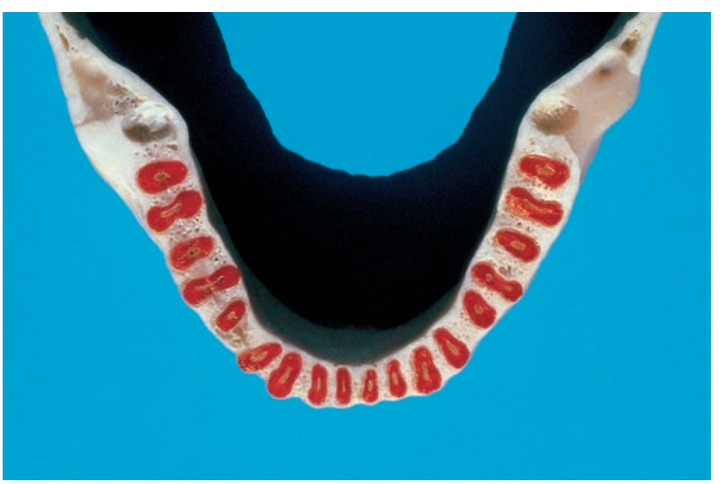
386 Section Through Maxilla

This view, with the cut root surfaces colored red, clearly depicts the enormous variations in root morphology. The narrow furcations, root fusions and hourglass shapes of some roots can also be observed. The interdental and interradicular osseous septa are also of varying dimensions.



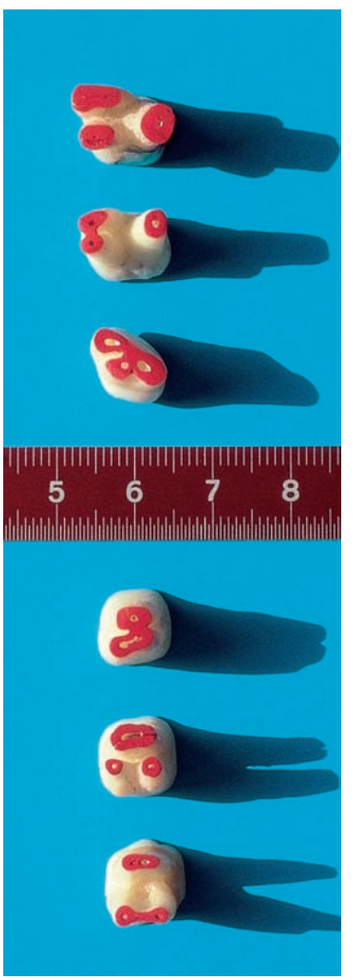
387 Many Forms of Maxillary and Mandibular Molars (right)

The roots were sectioned horizontally about 4 mm apical to the cementoenamel junction. This view depicts clearly the variability of furcation areas as well as root fusions. It is easy to imagine the difficulties associated with root planing in such areas!



388 Section Through Mandible

Root morphology in the mandible is more uniform and less complicated than in the maxilla, but almost all of the roots exhibit some depressions labiolingually. Many molars also exhibit enamel projections ("pearls"). Particularly the mandibular molars exhibit variability in the length of the root trunk.



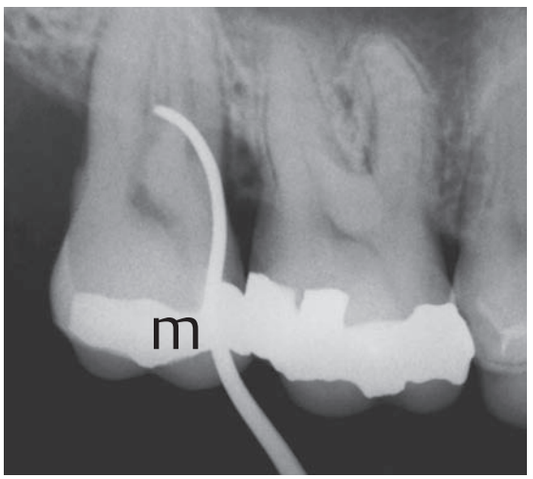
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For diagnostic purposes, the blunt, straight periodontal probe alone is inadequate. Adjunct probes that are specially curved, blunt or pointed, are required (Fig. 385).

The clinical recognition and ascertainment of such root peculiarities is of importance, because furcations are some of the most difficult to treat plaque-retentive areas and retention areas for microorganisms.

Furthermore, when a flap procedure permits direct vision of the exposed root surfaces, the practitioner quickly realizes that nature has even more morphologic fantasy than was perhaps apparent at the initial examination. Morphologic peculiarities and the attachment loss picture are usually more pronounced than anticipated during the original data collection.

The radiograph can provide additional evidence of root peculiarities, but cannot precisely portray the variations in root morphologies that occur from tooth to tooth. The radiograph cannot replace the comprehensive examination of the root surface with a fine probe. Only high resolution computer tomography (CT) could display and reveal the three-dimensional characterization of this region.

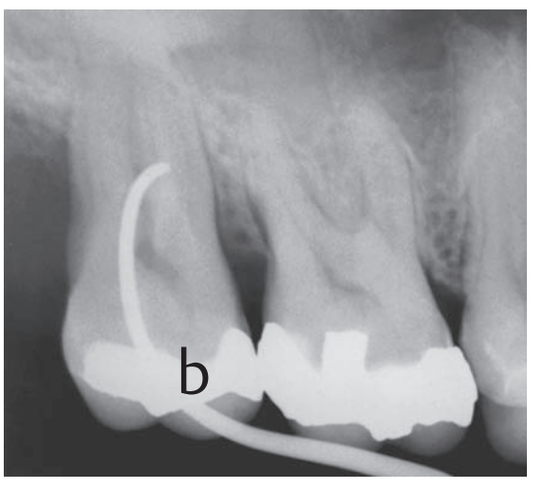




Probing the Trifurcation of Tooth 17

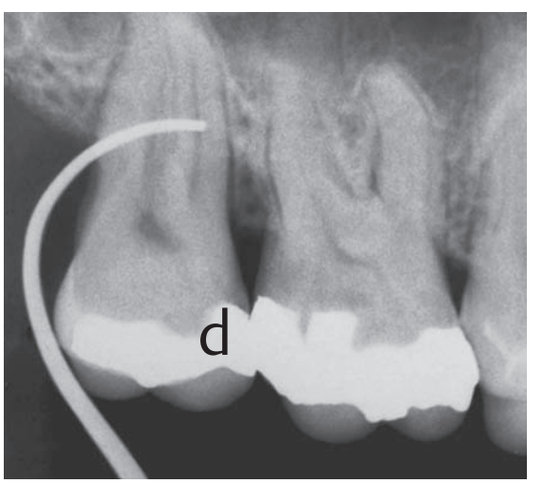
389 "Mesial" Furcation—m
In the radiographic, no furcation involvement is visible. However, using a Nabers-2 probe it is possible to probe the interradicular area of tooth 17 via the mesial furcation.

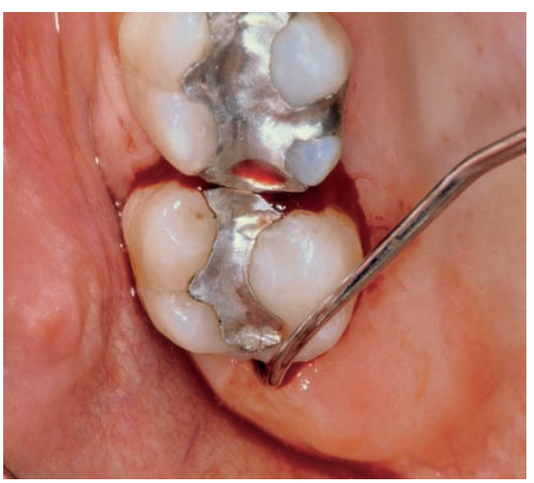
The mesial furcation can only be probed with certainty from a mesiopalatal approach.





390 "Buccal" Furcation—b
Through the narrow buccal furcation, between the mesiobuccal and distobuccal roots, the probe tip reaches the roof of the furcation, and can also be guided into the interradicular area of the same tooth.





391 "Distal" Furcation—d
When the second molar is the most posterior tooth in an arch, the distal furcation can be probed from either distobuccal or distopalatal approaches. As shown, the Nabers-2 probe achieves the interradicular area from the distal approach.
Thus, tooth 17 is shown to exhibit three different class F3 furcation involvements: from the mesial, distal and buccal aspects.
These F3 lesions were treated periodontally and have remained stable for twenty years.

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Tooth Mobility—Functional Analysis

Parafunctions (clenching and/or bruxing) do not cause gingivitis or periodontitis (p. 459). Parafunctions can, however, lead to occlusal trauma and pathologic alterations within the periodontium, and therefore can accelerate the course of an *existing periodontitis* (Svanberg & Lindhe 1974, Polson et al. 1976a, b). Unphysiologic loading of the dentition can lead to elevated tooth mobility, without attachment loss. Parafunctions may be elicited by local *premature contacts*; parafunctions are frequently related to phases of stress.

According to Ramfjord (1979), "Occlusion-related periodontal trauma may elicit a deviation from periodontal health; therefore, periodontal comprehensive therapy should include treatment of traumatic lesions in the periodontium." Beyond their periodontal implications, functional disturbances may play a significant role in the etiology of myoarthropathology, and must therefore be eliminated before any prosthetic rehabilitation is initiated (Bumann & Lotzmann 2000).

392 Fremitus—Localization of Tooth Mobility
Any tooth mobility that is caused by premature contacts or traumatization of individual teeth can be clinically ascertained if the patient is asked to close in centric intercuspation and "tap" the teeth together while the clinician palpates the individual teeth with her/his fingertip. The etiology of locally elevated tooth mobility must be evaluated using additional tests. Here, the patient uses her own finger to "feel" her tooth mobility.



393 Manual Tooth Mobility Tests—TM Severity
With the patient's mouth open, each individual tooth is examined for its mobility, whereby the tooth is manipulated between an instrument and the clinician's fingernail using a force of ca. 5 N (ca. 500 g) in the oro-facial direction (periodontal tooth mobility; p. 460)

Right: In this *Atlas*, tooth mobility will be classified in four distinctly categorized degrees of severity, on the basis of previously performed electronic measurements.



Tooth mobility (TM)—Degrees of tooth mobility
(H.R. Mühlemann 1975)

0	**normal** physiologic mobility (p. 460)
1	**detectable mobility** elevated mobility
2	**visible mobility** up to 0.5 mm
3	**severe mobility** up to 1 mm
4	**extreme mobility** vertical tooth mobility; tooth no longer functional

394 Hyperfunction and Inappropriate Function
Long-term and persisting parafunctions can exert negative influences upon the periodontium, tooth structure or the TMJ and masticatory musculature.

Left: Elevated tooth mobility and tooth migration, resulting from damage to the periodontal supporting structures.

Right: Excessive abrasion from severe parafunctions.



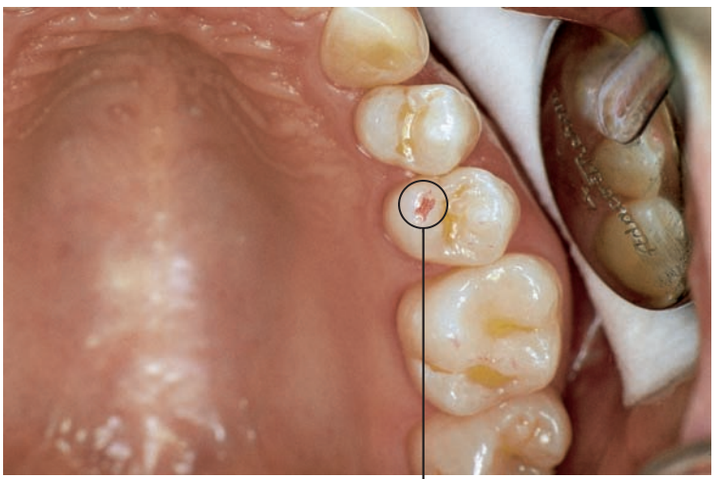


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395 Manual Test for Premature Contacts in the Retruded Contact Position
With the patient seated upright, the clinician guides the mandible into its retruded position vis-à-vis the maxilla.

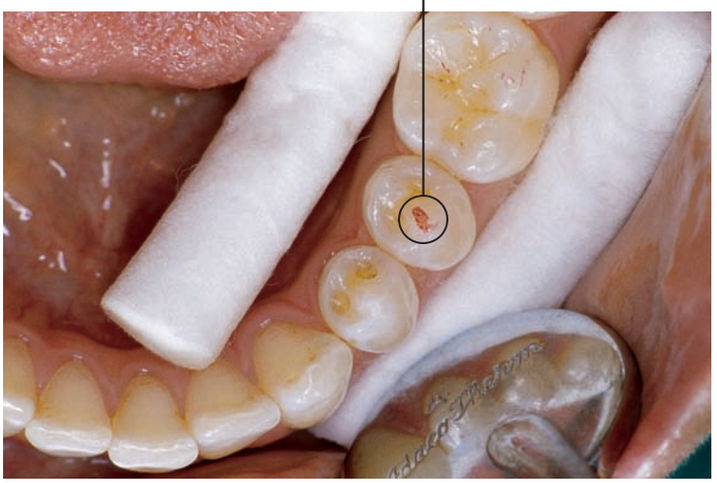
Excessive force in the retral direction can force the condyle backwards and downwards into an unphysiologic position. During this test, the mandibular condyle should achieve its zenith in the fossa.



Marking a Premature Contact in Centric Relation (Retruded Contact Position) between Teeth 25 and 35.

396 Marking in the Maxilla on the Mesial Cusp of the Palatal Aspect of Tooth 25
This is a location in which premature contacts are relatively frequently observed.





397 Marking in the Mandible on the Buccal Cusp of Tooth 35
Following the "tapping test" in retruded centric position, one notes contact solely between the left second maxillary premolar and its antagonist in the mandible.
When the patient bites together, the result is a *deviating movement* of the mandible anteriorly into *habitual occlusion* (intercuspation; centric occlusion).

Left: Lateral excursion to the left; no contact on tooth 25.

Centric Relation (CR)
– Initial "premature" contact — 5 / 5
– Deviation in **intercuspation** — 1.5 mm forward and right

Articulation
– Contacts during lateral excursions

3 2 1	6 7	B	7	1 2 3 4
3 2 1	6 7		7	1 2 3 4

toward..... right left

anteriorly (B) 2 1 | 1 2 / 2 1 | 1 2 (B)

Parafunctions
– Dental history
– Clinical examination

Morphologic and functional peculiarities: None

TMJ mild crepitus, left

398 Most Important Entries into the "Minor Functional Analysis"
The chart entries shown here represent the case of a periodontally healthy female.

A minor "prophylactic" selective odontoplasty could, in this case, achieve an improvement of centric relation, by removing the premature contacts, as well as a reduction/elimination of the *balancing contacts* (**B**) between teeth 26, 27 and 36, 37 as well as 17, 47.

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Radiography

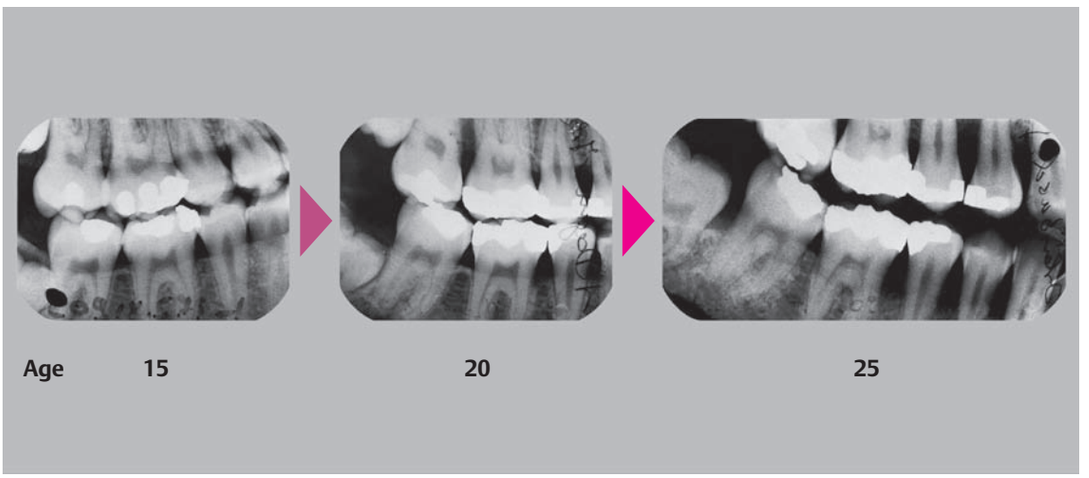
The clinical data described thus far *must* be supplemented by a radiographic examination. Comparative studies have shown that clinical measurement of probing depths and attachment loss does not always provide a complete and precise picture of the periodontal destruction. On the other hand, a diagnosis of periodontitis should never be made *solely* on the basis of radiographic findings. A radiograph depicts only a two-dimensional *alteration* of the *interproximal bone*. Additional changes in the hard tissues (caries, endodontic complications, reconstructive considerations) will be critical during treatment planning.

Intra- and Extraoral Radiographic Techniques

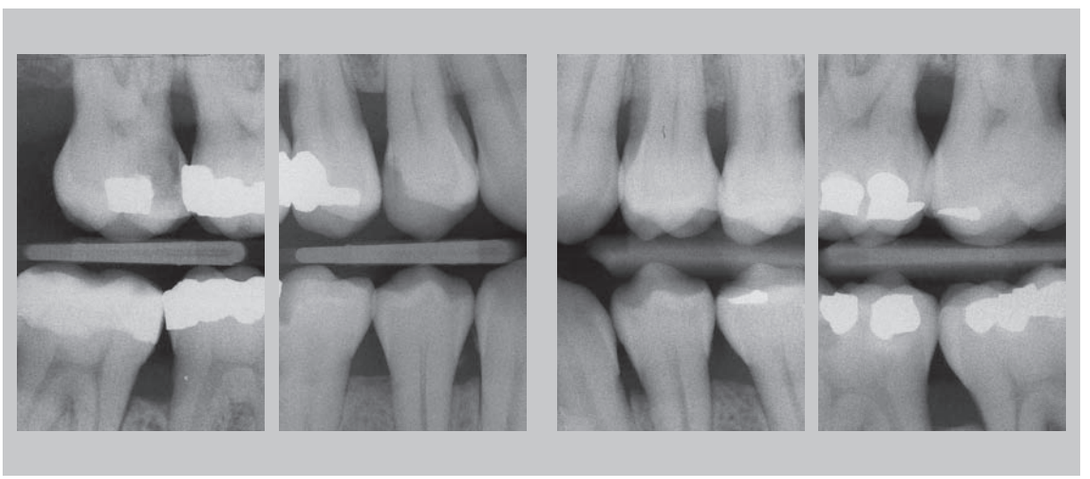
- *Screening—Panoramic radiography*: The slightly magnified film can provide a good overview of the dental structures, and often clarify incidental observations.
- *Single film survey*: The panoramic film cannot yet completely replace the classical radiographic survey, especially in complex cases. For the optimum depiction of periodontal structures, the parallel, right-angle technique, using the long cone is recommended (Pasler & Visser 2000).

Screening

399 Horizontal Bitewing Film
Dependent upon *caries activity*, bitewing radiographs are indicated in children and adolescents every 1–2 years. The case depicted here shows a 15-year-old patient with scarcely detectable juvenile periodontitis (LJP; new: type III A), but at 20 years of age revealed (local) "visible" periodontitis, and at age 25 a generalized periodontal problem. Regular use of a periodontal probe could have prevented this circumstance!



400 Vertical Bitewing Radiograph
In most periodontitis patients, four radiographs of this type can provide a satisfactory overview of the interdental bony defects (including caries and calculus accumulation) in the *posterior segments*. In addition, the above-described initial *juvenile periodontitis*, and above all the more severe and advanced types of bone loss can be depicted with vertical bitewing radiographs (cf. Fig. 399).



401 Panoramic Radiograph—Incidental Findings
Even though the diagnostic sharpness of individual periapical radiographs cannot be achieved, the panoramic radiograph is an excellent screening mechanism, especially in patients with a severe gag reflex. Frequently, unanticipated anatomic circumstances are detected.

Note: Retained, severely impacted mandibular canine, and a large retention cyst (cf. p. 437).



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Radiographic View of Pathologic Alterations and Their Causes

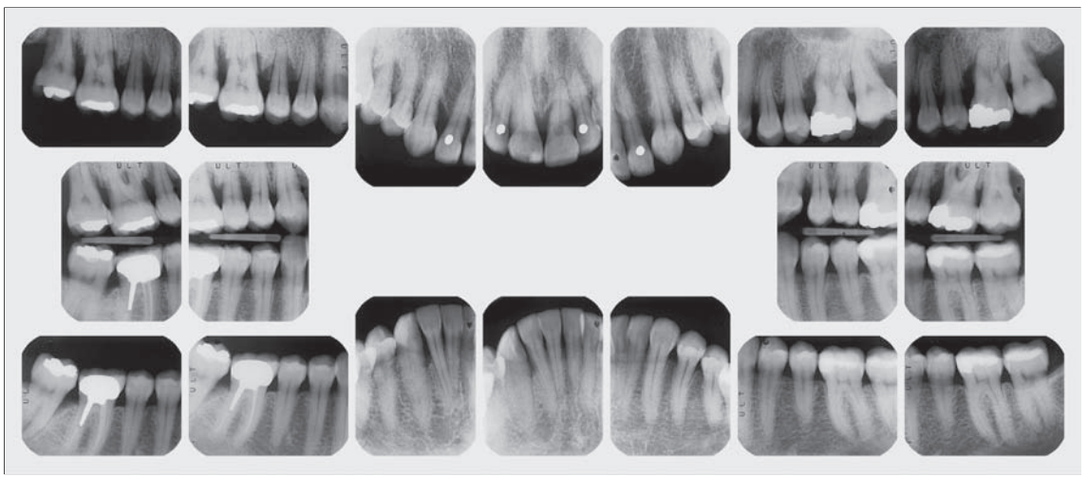
Distribution and localization of periodontal bone loss, the hard tissue defect:
- Changes in the entire dentition
- On individual teeth (septa) and root surfaces.

Type of bone loss:
- Demineralization (maintained matrix, reversible)
- Resorption at the alveolar margin
- Horizontal bone loss (with narrow septa)
- Vertical bone loss (with wide septa; Fig. 195)
- Vertical cup-like bone loss.

Extent of destruction:
- Distance from bone to the CEJ
- Furcation involvement
- Remaining attachment in relation to root length.

Etiology of destruction:
- Supra- and subgingival calculus, iatrogenic irritants
- Tooth (root) form and position (niches).

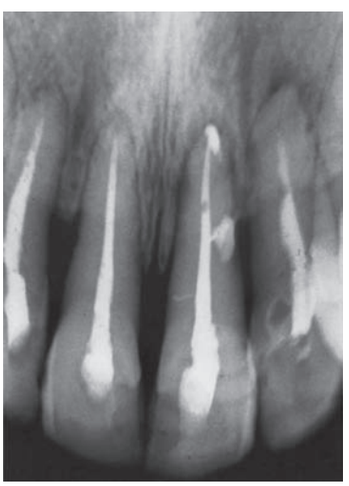
Radiographs depict destruction that has already occurred. Regular radiographic monitoring may clarify the dynamics of the course of disease.

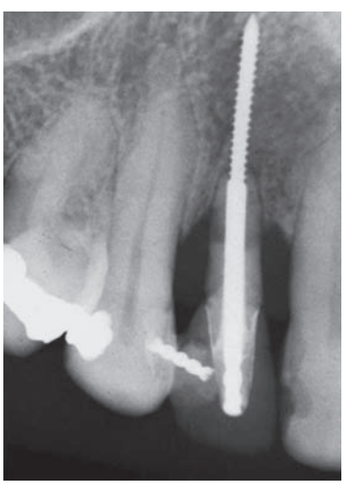


Detailed Radiographic Findings

402 Radiographic survey for Periodontal Diagnosis
In a complete dentition, a minimum of 14 periapical radiographs is necessary to depict all interdental and interradicular septa. The 14-film series should be enhanced by two or four bitewing films in patients with multiple restorations or crowns in order to ascertain any iatrogenic problems.







403 Special Radiographic Findings
Left: Apical abscess around 2-rooted tooth 14, and pulpal necrosis (cf. endo-perio problems, p. 445).

Middle: Severe periodontal bone loss, extending from tooth 21 into the region of a filled lateral pulpal canal.

Right: Transfixation screw (Wirz 1983) and splinting explain low tooth mobility despite minimal osseous fundament.





404 Digital Radiography
Digital radiography provides the benefits of computerization (time saving) and environmental concerns (lack of toxic chemicals). Intraoral mechanisms include either *chips* (*CCD* or *CMOS*) or *phosphorizing foils*. Digital imaging is virtually free of artifacts, which provides enormous advantages in endodontics and implantology. The figure depicts “scanning in” of the digitized image.

Left: The digital image on the monitor.

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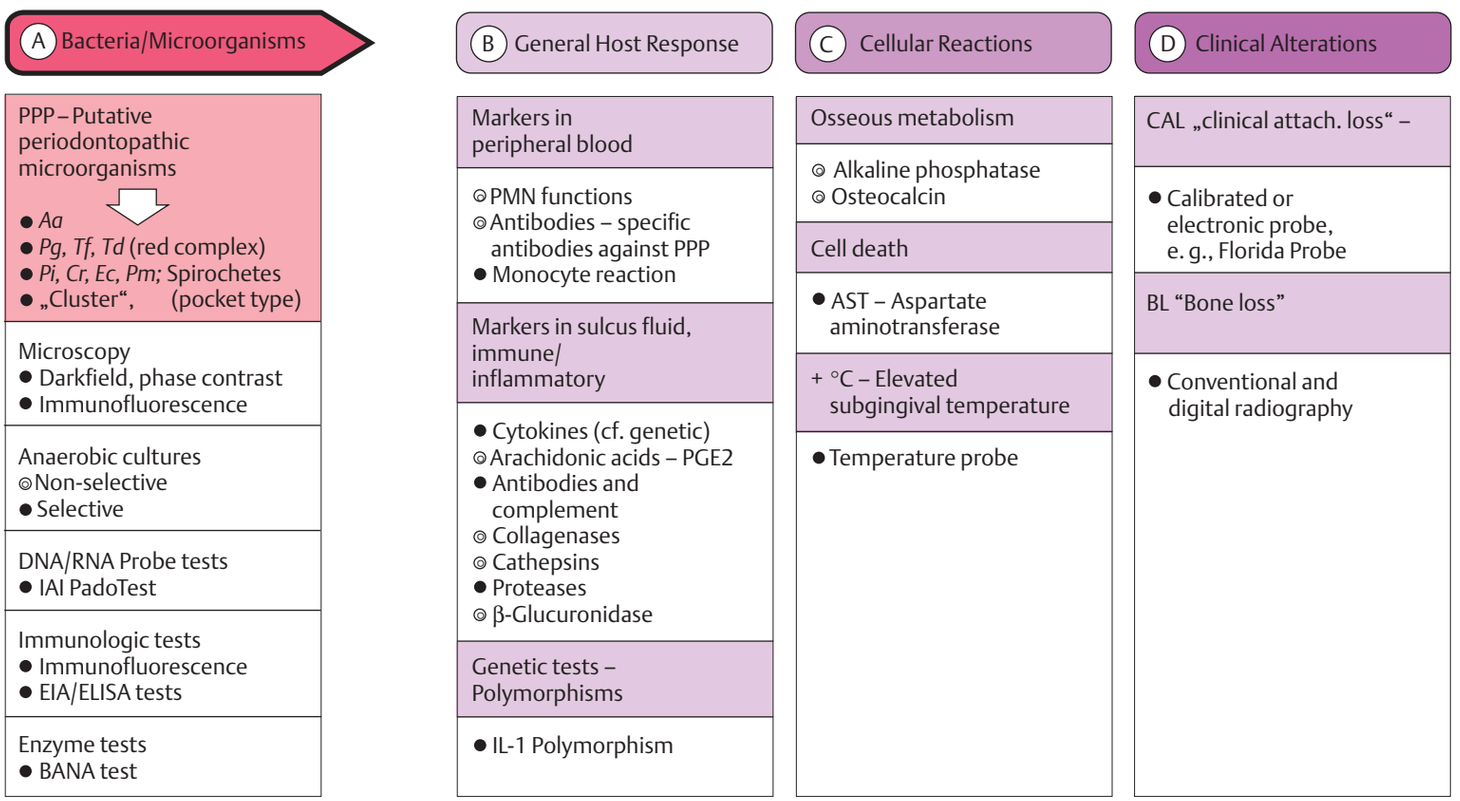
Additional Diagnosis—Tests

- **Microbiologic Tests**
- **Tests of Host Response**

The *classical* clinical examination methods were presented in the previous pages (pp. 168–175). New tests have become available in recent years, which should permit improved diagnosis and therefore also improved prognosis.

Figure 405 (modified from Lindhe et al. 1997) presents a summary of all these tests; those marked with a *solid* bullet are presented in detail in the following pages.

405 Additional Diagnostic Tests



The use of these diagnostic test methods is often time-consuming and expensive, and therefore not indicated or not necessary for uncomplicated chronic periodontitis cases. Such tests can only be recommended for establishing the prognosis in difficult-to-classify individual cases.

Tests for Microorganisms

The primary periodontopathic microorganisms can be identified using various methods (see Fig. 405).

Tests of the Host Response

The composition of the bacterial pocket flora is important, but of equal importance is the general host response to the infection (see Etiology, p. 39), including the markers and mediators in peripheral blood, in sulcus fluid, as well as genetic, non-alterable risk factors, e. g., gene polymorphisms.
The pathogenic bacteria and the host's reaction to them or to their metabolites elicit further cellular reactions and lead to destructive processes in the periodontal tissues. This includes above all clinical attachment loss and resorption of alveolar bone.

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Microbial Diagnosis—Test Methods

Microbiologic tests for the type and amount of marker bacteria are indicated primarily in aggressive periodontitis, refractory forms and patients manifesting severe periodontitis in conjunction with a systemic disease (e.g., diabetes, AIDS). Such tests can also provide clues concerning whether the mechanical therapy should be enhanced with antimicrobial supportive therapy. In addition, these tests can indicate the success of therapy.
The following will be presented:

- Microscopic techniques—dark field, phase contrast
- Bacterial cultures—non-specific/specific
- Use of DNA or RNA probes—amplification of DNA using PCR
- Immunologic methods—fluorescence microscopy, EIA-/ELISA tests
- Enzymatic bacterial tests (BANA)







406 Subgingival Plaque Removal
Left: Supragingival cleaning and drying of the harvest site.

Middle: Inserting the paper point (average size, # 30–50) into the pocket.

Right: Placing the paper point into the transport container.

Collection Technique

Various methods have been proposed for harvesting bacteria from the pocket (subgingival plaque). Most common today is collection using paper points of moderate strength. One can collect a single, site-specific probe, e.g., from the deepest pocket in a quadrant, or "pool" several probes from various pockets.

It is important to record the site and the date of the bacterial probe in the patient's chart (or via photography).

The site to be tested must be cleaned of all supragingival plaque and then thoroughly dried (*not* with an air syringe!). Then the paper strip is inserted to the bottom of the pocket and left *in situ* for 10 seconds. During removal, the strip must not come into contact with saliva, pus or the oral mucosa.

Depending on the test method, the paper point is placed into the transport container with transport fluid (for RNA probes, e.g., IAI PadoTest 4.5) or without any fluid (DNA probes, DMDx/Anawa etc.). The container is then tightly closed and sent to the special laboratory for further processing.

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Microbial Pocket Diagnosis—Dark Field and Phase Contrast Microscopy

These methods permit limited bacterial diagnosis directly at chairside. They do not require fixation or Gram-staining and are therefore quick and simple to perform. However, only the *morphotypes* can be identified, i.e., the shape of the bacteria and their *motility*. The determination of these criteria permits a limited conclusion about the pathogenicity of the microorganisms (Listgarten & Helldén 1978).

If the sample reveals primarily cocci and non-motile rods, it is an indication of only few active pathogens.

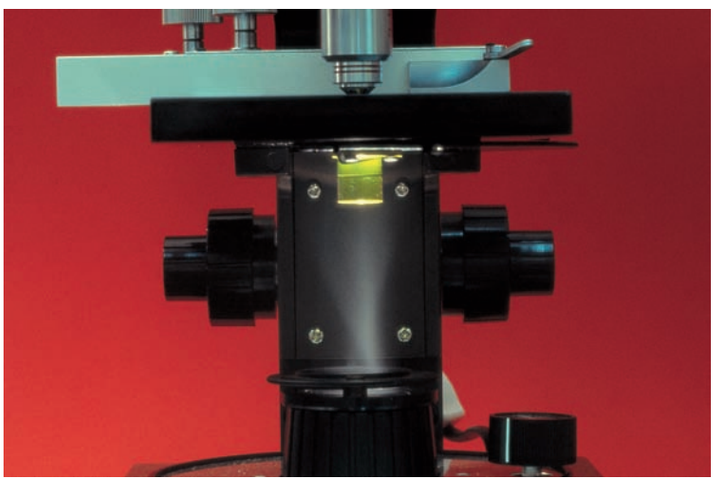
If the field exhibits numerous motile bacteria (e.g., rods and spirochetes) it is an indication of an active phase within the pocket, and elevated levels of potentially pathogenic pocket flora.

If the microbiologic picture is displayed on a screen and reveals numerous motile rods and spirochetes, it can provide a high level of *motivation* for the patient.

Even this simple and rapid diagnostic method is associated with costs (the microscope), and the clinician must determine the cost/benefit ratio in comparison to the classical examination techniques or other test procedures.

407 Dark Field Microscopy
This method reveals the shapes of bacteria and their motility, but does not permit any identification of bacterial classification or species. The method is therefore limited in its informative potential: It permits differentiation between *inactive* and *active* pockets. Its greatest value is in patient motivation.

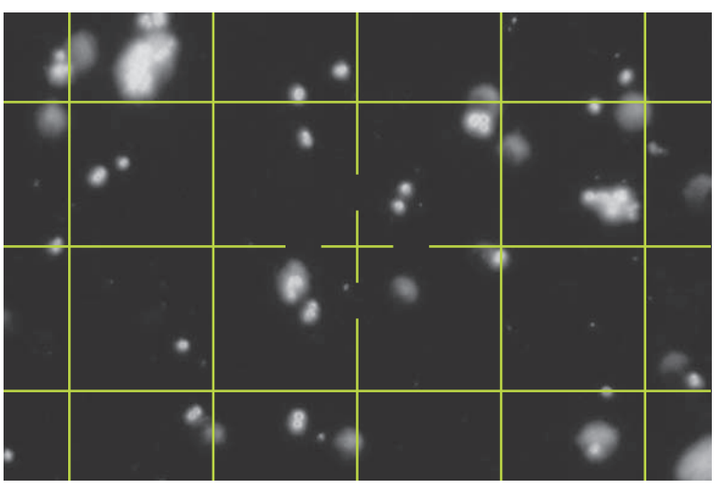
Right: The identifiable vital bacteria as seen in dark field and phase-contrast microscopes are identified according to the criteria listed.



Possible Classification of Bacterial Morphotypes

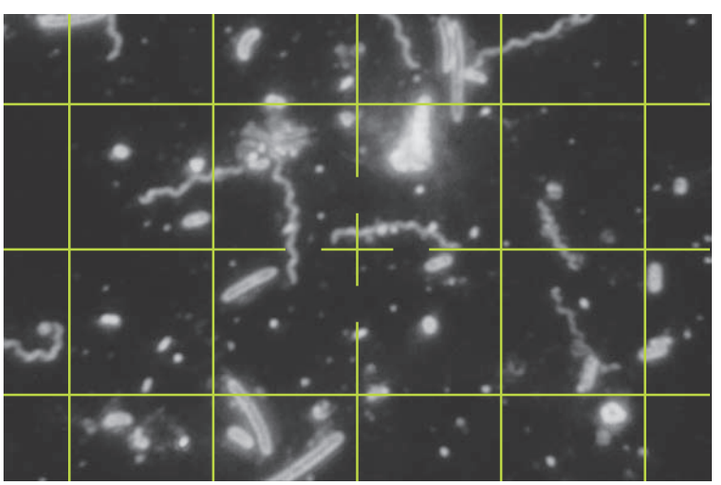
A	Cocci
B	Non-motile rods
C	Motile rods
D	Bent rods
E1	Small spirochetes
E2	Moderate spirochetes
E3	Large spirochetes
F	Others

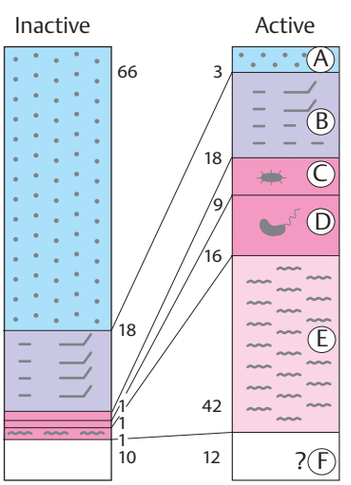
408 Bacteria from an Inactive Pocket—Cocci
In this photomicrograph one observes a few cocci and some non-identifiable particles; virtually no motile rods or spirochetes (proportion of spirochetes to cocci ca. 1:40).
Generally, in all segments of this microscopic field there is but a low number of bacteria from this inactive pocket.



409 Bacteria from an Active Pocket—*Spirochetes*!
Motile rods and spirochetes predominate. Their relationship to cocci is about 4:1, with a massively increased bacterial count. In contrast to the inactive pocket, there has been a "shift" from cocci to rods, and from *non-motile* to *motile* microorganisms.

Right: Comparative depiction and typical distribution (in %) of the morphotypes (see Fig. 407, right) in an inactive and an active pocket.





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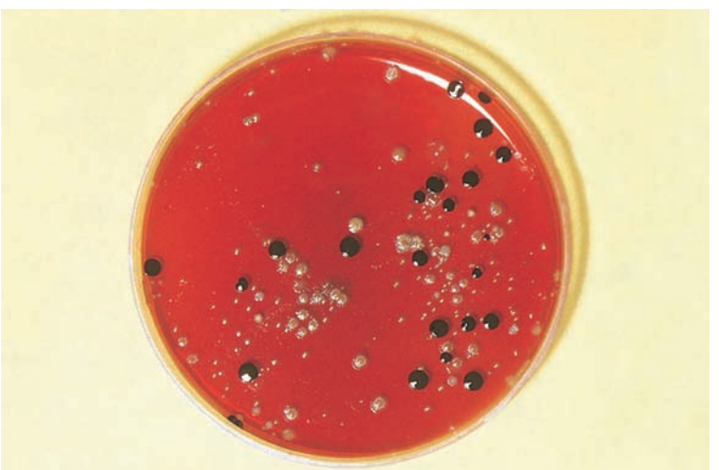
Microbial Pocket Diagnosis—Cultures

The preparation of bacterial cultures is one of the oldest "classic" diagnostic methods. For culturing, the bacteria must be maintained in the vital state.

Periodontopathic marker bacteria are usually *anaerobic*, which presents problems for the removal of the organisms and their transport to the laboratory (O_2, temperature, culture medium). In addition, preparing cultures is time-intensive, demands microbiologic knowledge as well as adequate laboratory equipment. All of this leads to appropriate costs! Oral anaerobic bacteria grow very slowly, and a definitive result is often possible only after ca. 3 weeks.

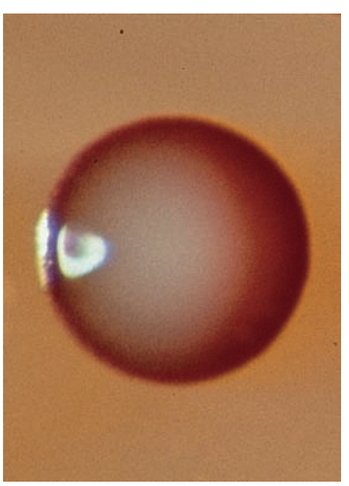
Both selective and non-selective culture media can be used to demonstrate the bacterial strains of interest, e.g., *Actinobacillus actinomycetemcomitans (Aa), Porphyromonas gingivialis, Bacteroides* species, *Capnocytophaga, Eikenella corrodens,* Fusobacteria etc. On the other hand, to date it has been virtually impossible to grow spirochetes on the usual artificial culture media. One advantage of the culture technique is the possibility to create an antibiogram, to test whether a certain marker organism is *sensitive*, or more importantly, *resistant* to specific antibiotics.

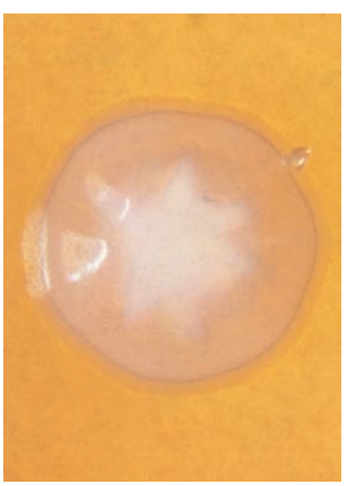




410 Anaerobic Bacterial Culture on a Blood Agar Plate
Various bacterial colonies can be identified on a blood agar plate that was incubated *anaerobically* for 10 days. In addition to the colonies of black pigmented Bacteroides species, many others are observed.
From the *form* of the colonies, their *metabolism* etc., the microorganisms can be determined and classified (*Bergey's Manual of Systematic Bacteriology* 1984).

Left: Magnified colonies.



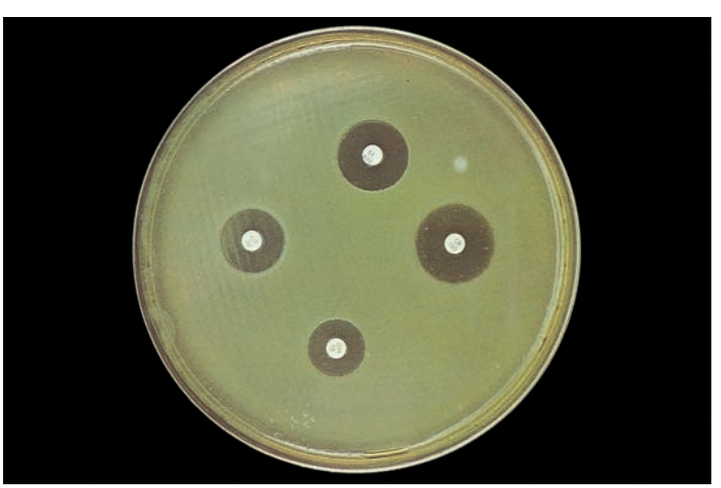


411 Isolated Colonies
Left: *Porphyromonas gingivalis* colony.

Right: Actinobacillus actinomycetemcomitans colony in its typical star-shaped configuration.

Courtesy *A. Mombelli*





412 Resistance Determination via Antibiogram
The clear field surrounding each antibiotic-soaked pellet determines the extent of *inhibition* of bacterial growth, and therefore permits a targeted use of the most effective systemic medication. (Courtesy *A. Mombelli*)

Left: Yellow? Red? Orange? Medicaments should not be empirically administered on the basis of their "colors"; rather, they should be selected only after more in-depth diagnosis (p. 287).

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New Diagnostic Tests—Evaluation

The classical data collections, including the just-described bacteriological culture examinations, still represent the *gold standard* (GS), but these demonstrate only processes that have already occurred in the tissues. Because relatively imprecise, conventional diagnostic findings have often led to under- and over-treatment, new and improved diagnostic tests would, in the ideal situation, make possible a more definitive prognosis and an individually appropriate and targeted treatment.

New *microbiological* (and immunological, p. 186) tests in the field of periodontal diagnosis have already become valuable diagnostic aids. In certain "difficult" cases, these new tests represent important components of diagnosis, and especially in the selection of a therapeutic regimen, e.g., with aggressive (Type III) or refractory periodontitis, with periodontitis combined with systemic diseases (Type IV), with severely advanced CP-cases (Type II), and in dental implantology.
Such additional diagnostic methods provide information concerning:

- The future progression of periodontitis
- The *identification of patients at risk*
- Criteria for an individualized recall program.

Periodontal Test Procedures

Modern test methods should be better, simpler, faster, more exact, non-invasive etc., and if at all possible *low cost*. In general they should provide more information than the old and often error-plagued gold standard, and must represent procedures that are acceptable to the practitioner! Attachment loss or attachment gain can only be precisely determined histologically, i.e., after extraction of the tooth!

Therefore, in routine clinical practice, this invasive standard must be replaced by a procedure that provides results that are equal to or better than those resulting from the gold standard (Müller 2001). The following must be evaluated:

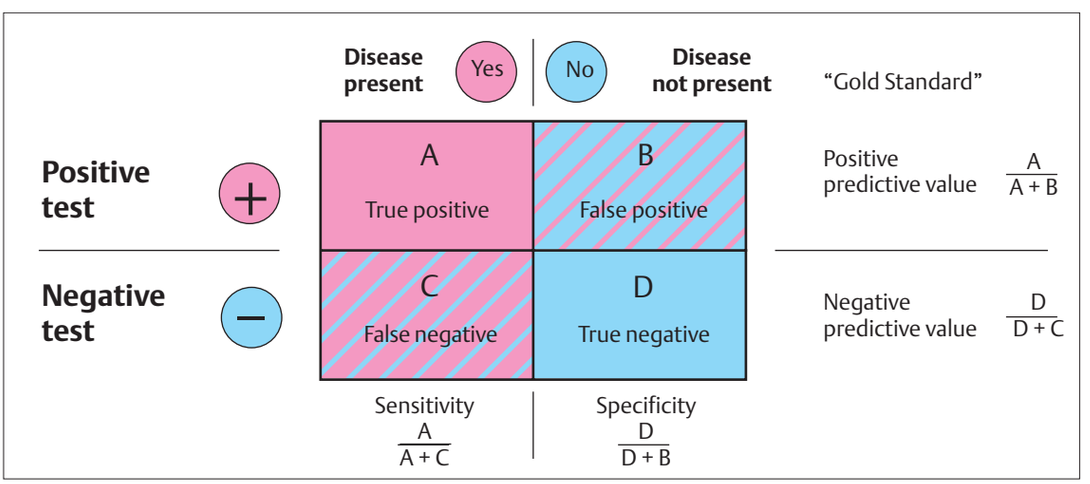
- Most important is the *negative* or *positive predictive value*
- *Sensitivity* and *specificity*; these are defined in the Glossary (below)
- The *validity* of the test results; this determines their practical use.

From a practical point of view, all of the test results are placed into a 2x2 schematic ("decision matrix") and the desired parameters are calculated (Fig. 413).

413 Decision Matrix—Evaluation of a Diagnostic Test
The parameters include

- Sensitivity
- Specificity
- Positive predictor
- Negative predictor

Using this matrix, the *validity* of any test can be determined.



Glossary
In the example "diseased—healthy," the following expressions must be defined and clarified (cf. also AAP 1995; Diagnosis of Periodontal Diseases; Pihlstrom 2001):

- *Gold standard*: Measurement of the true condition—yes/no: disease present (= positive) or not present (= negative).
- *Sensitivity*: Probability (in %) that an existing disease is scored as positive in the test. High sensitivity indicates that the test will provide only few false negative results. Thus true disease will not be overlooked. This is important with contagious conditions and life-threatening diseases (Hepatitis B etc.).
- *Specificity*: The probability (%) that a healthy patient will be shown to be healthy by the test (= "disease *negative*"). *High specificity* indicates that no healthy person will be incorrectly shown as diseased (= false positive). This is important, for example, with severe diseases that would demand unbearable therapy in a healthy individual (carcinoma, HIV).
- *Periodontitis*: A test with a sensitivity of, e.g., 70% (all diseased individuals were not detected!) and a specificity of, e.g., 90% (few healthy individuals were diagnosed as diseased) would suffice for this rarely contagious, non life-threatening disease, which is therapeutically not demanding of invasive procedures.

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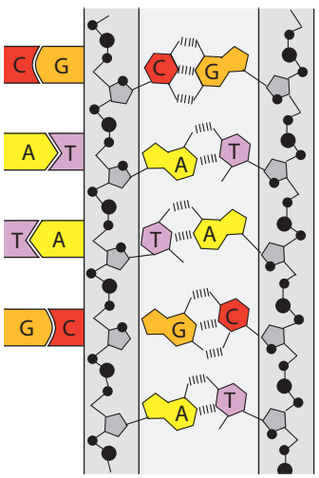
Molecular Biological Tests

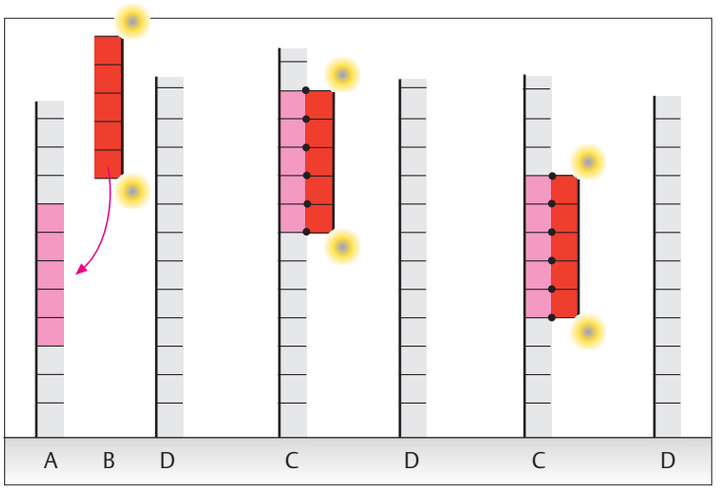
DNA Probe Hybridization—Gene Probes

In recent years, commercially-available tests entered the marketplace, involving species-specific usually *radioactively* tagged *oligonucleotide sequences* (DNA probes with 24–30 bases), which bind to complimentary bacterial DNA or RNA sequences (hybridization, Fig. 414). These tests do not require living bacteria, and therefore transport to the specialized laboratory is unproblematic. To perform the test, the bacteria are lysed, the DNA or RNA double strands separated, fragmented and affixed to a membrane.

Polymerase Chain Reaction (PCR)

Within each bacterium, only one set of DNA is present; on the other hand, ribosomal (r)RNA molecules are present 1,000 times more! For *DNA/RNA hybridization*, the bacterial rRNA need not be further amplified. For DNA/DNA *hybridization*, on the other hand, the few target molecules must be enhanced through PCR. Even the tiniest DNA amounts can be *amplified a million times* with only 20–30 copying cycles.





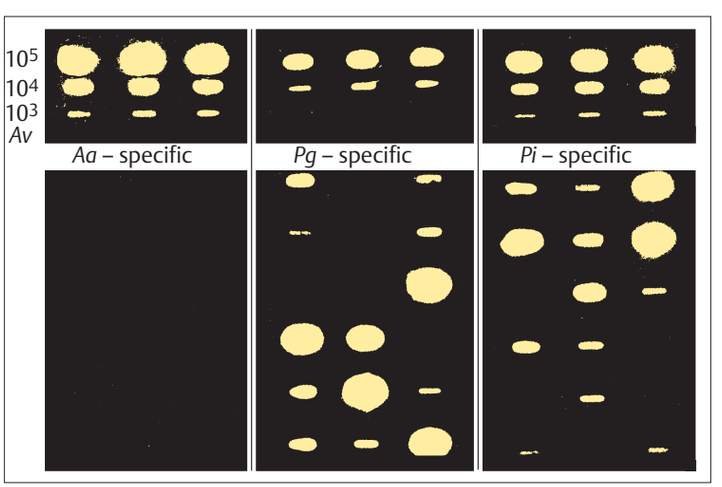
414 Hybridization Technique
After separating the double helix, the individual strands are fragmented and affixed to membranes. *Tagged gene probes* find the complimentary segment of the target DNA, and hybridize with it. The identification can be performed *autoradiographically* or *enzymatically* (color reaction).

Left: Complimentary base pairs between A and T, as well as G and C:
A Adenine **G** Guanine
T Thymine **C** Cytosine

DNA or RNA Probe Tests—Marker Bacteria

- **DMDx PathoTek (DNA)**
  Standards: *Aa, Pg, Pi*
  plus: *Tf, Cr, Td, Fn, Ec*
- **Meridol DNA probe test 3/8,** s. **DMDx**
- **MicroDent Test (DNA)**
  Aa, Pg, Tf, Pi, Td
- **Perio Bac test (DNA)**
  Aa, Pg, Tf, Pi, Td

- **IAI PadoTest 4.5 (RNA*)**
  Aa, Tf, Pg, Td
  *plasmid RNA



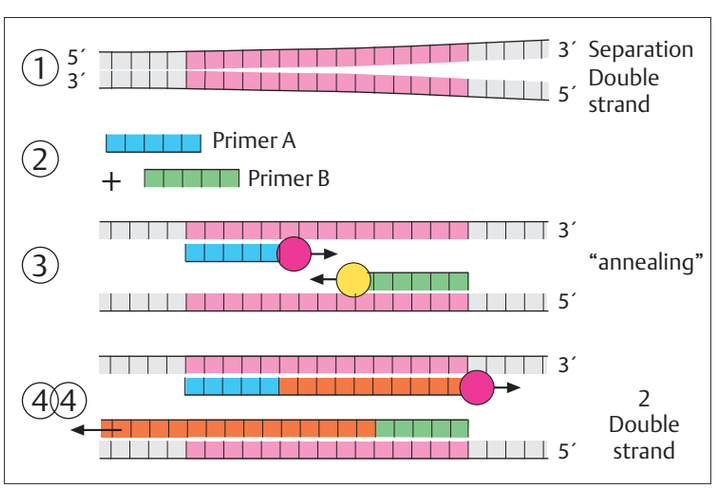
415 Autoradiography of a DMDx Test
Following hybridization, all non-bound probe fragments are rinsed away, the patient samples are applied in triplicate to a filter paper, and the autoradiograph is prepared.
Upper Section:
Positive controls with three quantitative standards (10^5, 10^4 and 10^3). Negative control with *Av*.

Lower Section: The result is negative for *Aa*, but positive for *Pg* and *Pi*.

PCR in Schematic

1. Separation of the individual DNA strands
2. Primer-A and primer-B specific oligonucleotide sequence
3. Annealing of the primer on the target sequence (reddish section)
4. Polymerase (Taq) doubles the target sequence at each pass

Number of the target sequence: 2^n where n = number of the passages.
Example: 20 cycles = 2^{20} = ca. 1,000,000 copies



416 Polymerase Chain Reaction
In order to multiply a determined section of DNA, one requires a heat-resistant *Taq-polymerase* (Bacterium *Thermophilus aquaticus* is intact and active even at 95° C!), two appropriate primers (each at the beginning of both DNA strands) and the four bases (nucleotides) A, T, G and C in excess.

Separation of the DNA strands occurs at 95° C; cooled to 55° the *tagged* primers accumulate, then the polymerase fills the "gaps."

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Bacterial Probe Test—Practical IAI PadoTest

The available DNA or RNA tests permit demonstration of three to eight of the most strongly associated marker bacteria for periodontitis.

Using the IAI PadoTest 4.5—an RNA test, cf. p. 183—the following four periodontopathogens can be demonstrated either site-specific or as pooled probes:

- *Aa/Actinobacillus actinomycetemcomitans*
- *Tf/Tannerella forsythensis* (formerly *Bacteroides forsythus*)
- *Pg/Porphyromonas gingivalis*
- *Td/Treponema denticola*

In addition, this test provides supportive data regarding diagnosis and therapy (Fig. 420 and 421):

Quantitative:

- ML: Number and percentage composition of marker bacteria
- TBL/TML: Total load of all bacteria, i. e., the marker bacteria
- Statistical comparison of the results*

Qualitative:

- Typing of the pocket (Types 1–5)*

* Typing according to large field studies (Baehni et al. 1993).

417 Harvesting Pocket Flora Using a Paper Point

In most cases, the probes for bacterial tests are harvested from the deepest pockets in each quadrant (usually on molars). In the present "true" case, the sample is taken from the distal surface of tooth 12. The paper point is inserted to the fundus of the pocket and left *in situ* for 10 seconds.

Right: Thereafter, the paper point is placed into the color-coded transport vial, and the vial is tightly sealed.





418 The IAI PadoTest 4.5 kit for Harvesting Bacterial Samples

Paper points, transport vials and a convenient holder are included gratis in the commercially-available kit. The helpful *color-coding* is perpetuated throughout the four transport vials and the test stand, as well as the information forms and the final report (see p. 185) (red = quadrant 1 etc.).

Right: DNA probes are stable even in the dry condition, but the chemically more fragile RNA requires a stabilizing transport fluid.





419 Transport to the Specialized Laboratory

The transport vials along with the pre-printed protocol form and often a coded patient name, as well as the noted site of sample harvest are sent using regular mail to the laboratory. The test results will be provided within a few days. The microflora of the individual pockets (sites) will be typified according to the reported field studies and the bacterial distribution reported therein (cf. Cluster/Socransky 1998; IAI/pocket types 1–5).





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DNA/RNA Probe Tests—IAI PadoTest 4·5

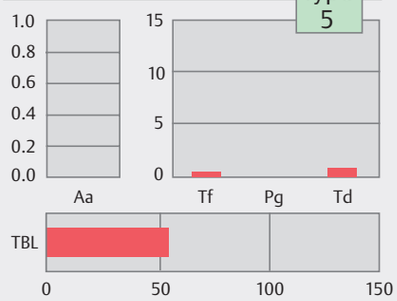
IAI PadoTest 4·5

Test Results

Dentist: **Dr. Wolf, Herbert F., Zürich**
Patient: **Mr. # 1284 (HW)**
Evaluation from: **07/12/00** (pre-treatment tests)

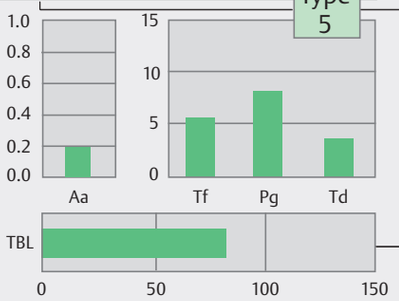
Tooth 17, distobuccal; PD 8 mm

Marker	n	ML	Status
Aa	–		
Bf	0.31	0.6%	
Pg	–		
Td	0.69	1.3%	★
TBL	53.63	–	★
TML		2%	Type 5



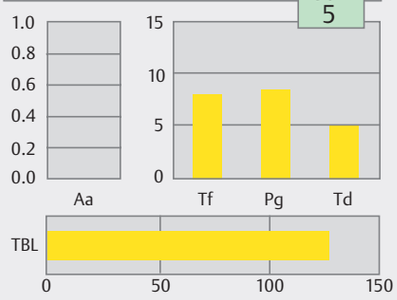
Tooth 12, distofacial, PD 7 mm

Marker	n	ML	Status
Aa	0.184	0.2%	★★
Bf	5.39	6.56%	★★★
Pg	8.04	9.8%	★
Td	3.31	4.0%	★
TBL	82.37	–	★
TML		21%	Type 5



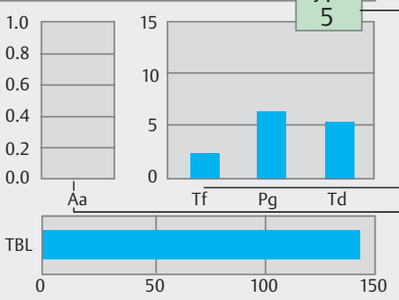
Tooth 42, distobuccal, PD 9 mm

Marker	n	ML	Status
Aa	–		
Bf	7.84	6.3%	★★★
Pg	8.27	6.6%	★
Td	4.96	4.0%	★★
TBL	125.06	–	★★★
TML		17%	Type 5



Tooth 26, distobuccal, PD 7 mm

Marker	n	ML	Status
Aa	–		
Bf	2.41	1.7%	★
Pg	6.27	4.5%	★
Td	5.29	3.8%	★★
TBL	140.97	–	★★★
TML		10%	Type 5



420 Test Results and their Prognostic and Therapeutic Significance

In this case, the three deepest sites in the maxilla (teeth 17, 12, 26) and a very deep pocket in the mandible (tooth 42) were evaluated.

Quantitative Parameters, Pocket on Tooth 12 Distobuccal (green table)

n = number of bacteria in millions, e. g., *Aa* about 180,000 entities; *Bf* about 5.4 million

ML = "marker load" in %. Percentage composition of the marker bacteria vis-à-vis the total bacterial load (**TBL**), e. g., in this case ca. 82,000,000; in comparison: percentage of *Aa* = 0.2 %
Statistical comparison ("Star"). For details, see Fig. 421.

TML = "total marker load" in % of the entire count of *all* bacteria, here 21 %

TBL = "total bacterial load" (Total "pathogens + non-pathogens") The scale ranges from 0 to 150 million

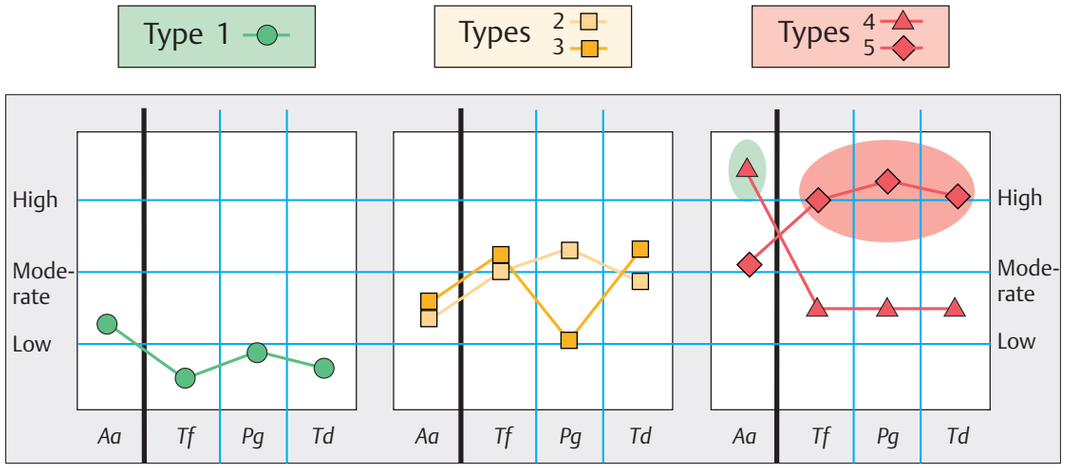
Qualitative Parameters

Characterizing the periodontal pocket according to its colonization and microbial assortment in **Types 1–5**.
This characterization permits a true understanding of the complexity of the microbiological results, and provides a clue to the clinical significance.

Type 5 = pocket type

Tf, Pg, Td—"red complex," p. 37

Scale for ***Aa***: 0–1 million
Scale for *Tf, Pg, Td*: 0–15 x 10^6



421 Pocket Type—Microbiologic Complexes

According to the distribution of the marker bacteria, five types can be distinguished:

	Therapy
Type 1	Simple therapy
Types 2 and 3	Moderately difficult
Types 4 and 5	complex

Types 1–3 can be treated purely mechanically. Types 4 (*Aa*!) and 5 ("red complex") demand adjunctive antibiotic therapy.

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Immunological Tests—Antigen-Antibody Reactions

Antigen-Antibody Reaction

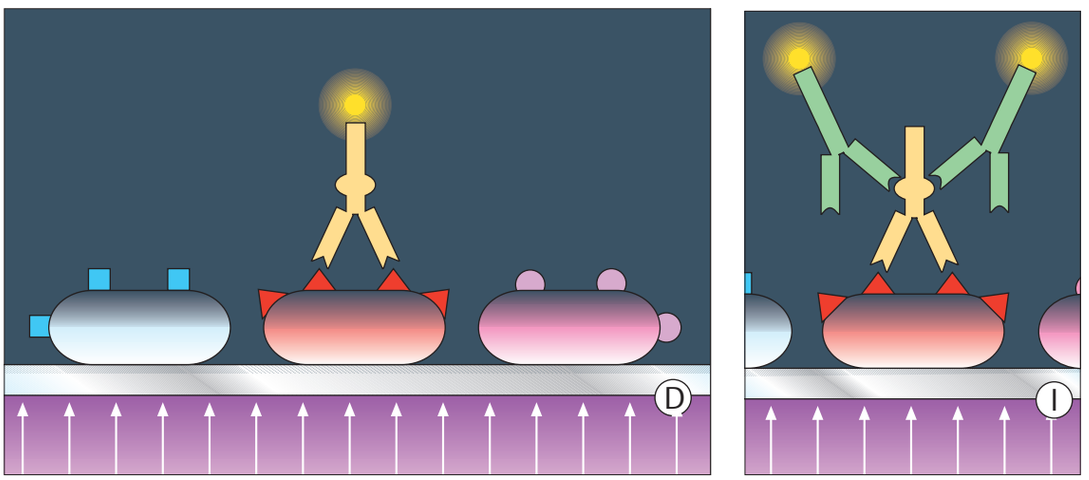
In contrast to the tests just described, the immunologic determination of bacteria can also be accomplished using specific *antigens* (Ag), *surface markers* (structures such as pili, fimbria; usually carbohydrates or glycoproteins), to which specific monoclonal antibodies (Ab) attach. In order to render the Ag-Ab binding visible, specially prepared antibodies with so-called reporter molecules (RM) are tagged. This can be done using enzyme-like colors or substances that fluoresce.

Direct and Indirect Immunofluorescence

Using immunofluorescence, the Ag-Ab reaction can be visualized using a *fluorescence microscope*. The bacteria affixed to the microscope slide bind to the added specific antibodies via their type-specific *surface antigens*, and the antibodies are rendered visible in UV light as fluorescing molecules (Gmür & Guggenheim 1994). Dead *and* living bacteria are seen in this way, and new techniques make it possible to differentiate even on the basis of color (Netuschil et al. 1996).

422 Immunofluorescence
D—direct (left):
The specific antibody (yellow), tagged with a fluorescent enzyme, docks with the red, triangular antigens (Ag).

I—indirect (right)
In a second step, several tagged (green) antibodies bind to the non-tagged yellow; this permits the fluorescence to be clearly visualized in UV-light (Ag-Ab reaction).



EIA/ELISA—Enzyme-Linked Immunosorbent Assay

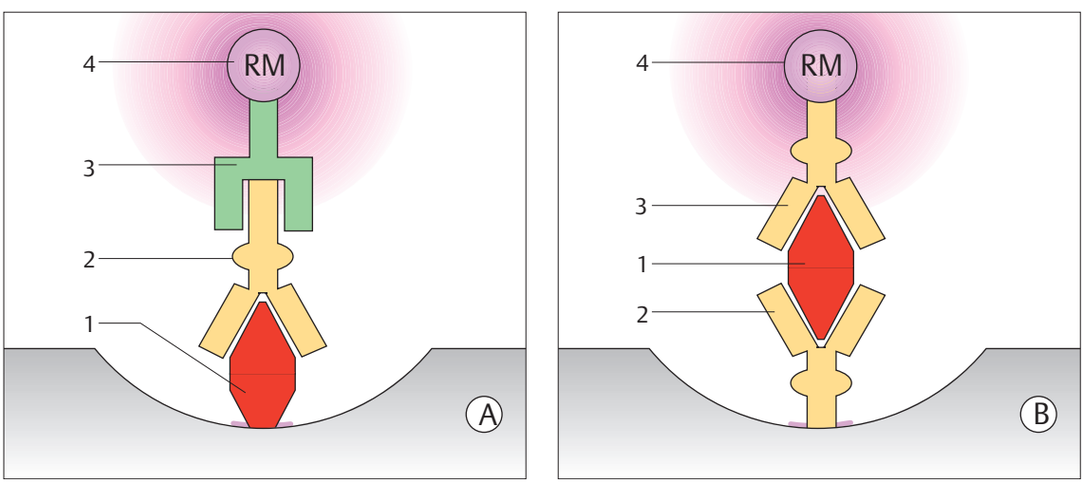
An immediate chairside test for identification of marker bacteria in periodontitis patients is no longer commercially available. In fact, immunologic tests that would elicit a color reaction would be an ideal solution to avoid the expensive and time-consuming laboratory studies and the expensive apparatus needed for immunofluorescence investigations. In recent years, the quite good Evalusite Test was made available (Kodak/semiquantitative color test for the marker bacteria *Aa*, *Pg*, *Pi*), but unfortunately it has been removed from the market. Practitioners are therefore reconciled to waiting for a similar reliable chairside test system. Today, immunological test procedures are performed in laboratories according to the two procedures shown below: "antibody capture" (A) or "antigen capture" (B). In the first case (A) a specific monoclonal antibody (yellow) "captures" the antigen (red) that is fixed in a minicuvette, and upon which a second antibody becomes affixed with the reporter molecule (RM). Following a rinsing procedure, only the Ag-Ab complex participates in the color reaction. Procedure (B) proceeds in a similar manner, but the procedures are reversed.

423 Immunological Tests, Color Reactions—EIA and ELISA

A " Antibody Capture"

B "Antigen Capture"

1 Specific antigen (red)
2 Primary antibody (yellow): Ag-Ab reaction
3 Secondary antibody (green or yellow) carry reporter molecules
4 Reporter molecules (RM) elicit the *color reaction*



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Enzymatic Bacterial Tests—BANA Test

Certain periodontopathic bacteria (including the "red complex": *Tf*, *Pg*, *Td*; pp. 37, 191; but not *Aa*) produce in their metabolism a trypsin-like enzyme, a peptidase, which has the ability to degrade BANA (Loesche et al. 1992). BANA stands for *N-α-benzoyl-DL-arginine-2-naphthylamide*, a synthetic substrate that is hydrolyzed by the above-mentioned bacterial peptidase. One of the breakdown products is β-naphthylamide, which becomes visible due to a color reaction and therefore provides evidence of the pathogenic bacteria.

The BANA test (e.g., Dentocheck; Butler) is relatively inexpensive and can also be performed by auxiliary personnel in the practice. A *strong positive* test is an indication that antibiotics against obligate anaerobes should be used in addition to mechanical *pocket treatment* (pocket Type 5; p. 185). Unfortunately, the robust and difficult to eliminate *Aa* is not identified by the BANA test. An additional disadvantage is that other, less-pathogenic pocket flora may also be BANA positive. These facts demonstrate the limitations of this bacterial test.

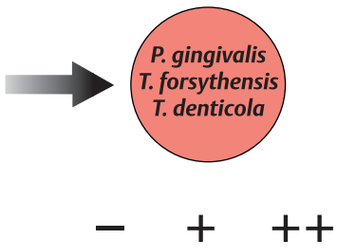


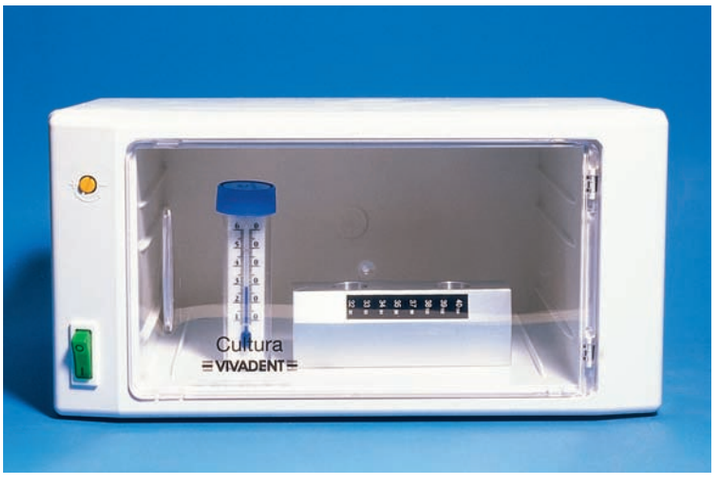


424 DentoCheck
(Butler/Heico Dent)
The kit contains materials for 30 tests/test sites:

- Reagents 1, 2 and 3
- 100 paper points
- 1 pipette
- Color scale
- Detailed instructions

Left: Using 3 paper points, plaque samples from the deepest pockets are harvested and then processed according to the laboratory protocol.

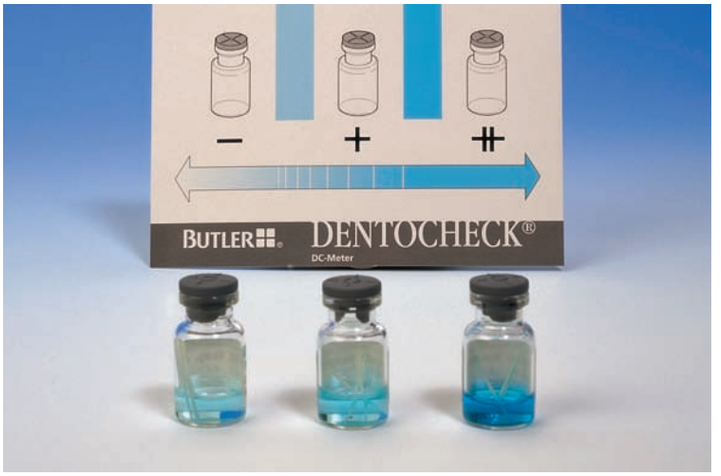




425 Incubation Chamber, Aluminum Block, Thermometer, Stopwatch. . .
... must be available. The incubation chamber and the aluminum block must be brought to 37° C before the test is initiated.

Left: BANA-positive are the "companions of the red complex" (Socransky et al. 1998). Depending on the concentrations of these microorganisms, the test will reveal negative, weakly positive or strongly positive.





426 Evaluation of the BANA-Test
In comparison to the color scale, the presence and quantity of BANA-positive bacterial flora can be estimated.

Left: The reaction time (15 min at 37° C) must be carefully adhered to.

The following tests are commercially available:

- **Dentocheck**
- **Perioscan**
- **Periocheck**

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Tests of the Host Response—Risks

- **Risk factors**
- **Severity factors**
- **Progression factors**

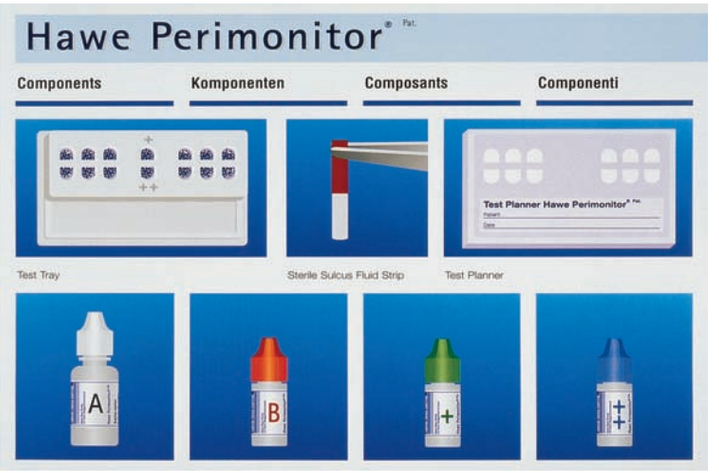
All of the tests described thus far are based on clinical parameters and directly or indirectly on the identification of periodontopathic bacteria. The tests described below provide indications of the host response to the microbial infection.

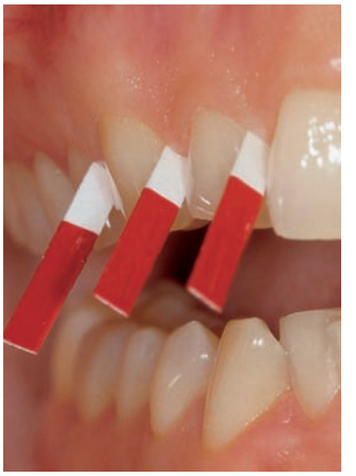
Many indicators can signal on-going periodontal destruction, including PMN defects, high titers of antibodies against periodontopathogens, enzymes such as aspartate aminotransferase (AST; Persson et al. 1995), elevated inflammatory mediators (PGE2) and others. Also, the measurement of subgingival temperature (e. g., PerioTemp System) can indicate the existence of inflammation, but not necessarily provide information about the progression of the disease (Kohlhurst et al. 1991, Fedi & Killoy 1992, Trejo et al. 1994).

None of the current methods for testing host response have yet achieved routine utilization in practice.

427 Enzyme Test—Hawe Periomonitor
This practical test measures the amount of aspartate aminotransferase (AST) in the *sulcus fluid*. AST is an intracellular enzyme, which is released upon cell death and can therefore be correlated with the amount of tissue destruction. The test is based on a color reaction.

Right: Sulcus fluid strips remain *in situ* for 30 seconds.





428 Subgingival Temperature – PerioTemp System (Abiodent, Inc.)
Heat is one of the cardinal symptoms of inflammation (“calor”). The temperature at the base of a periodontal pocket can be measured using a fine, graduated, sterile temperature probe. The instrument provides a color scale depicting all 32 teeth, displaying green-red when the average subgingival temperature exceeds 35.5° C. The measured values can also be printed out (left).



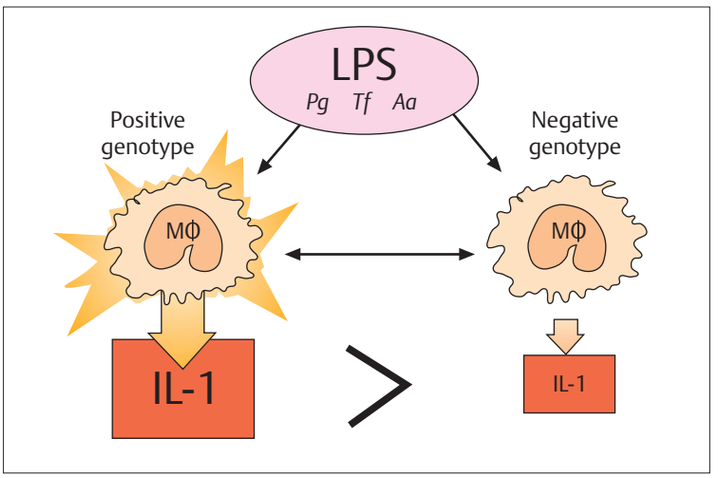
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Genetic Risk—Test for IL-1 Gene Polymorphism

In the chapter on etiology and pathogenesis (pp. 41–66) the *enormous* significance of the various host defense mechanisms against microbial infection was extensively portrayed. It was demonstrated that inflammatory reactions are regulated by numerous messenger substances and enzymes such as prostaglandins, metalloproteinases and certain cytokines. One of these cytokines is one of the most powerful general activators: Interleukin 1 (IL-1). It exists as IL-1α and IL-1β, whose production is coded for by the two genes *IL-1A* and *IL-1B* (both on chromosome 2).

IL-1 Gene Polymorphism

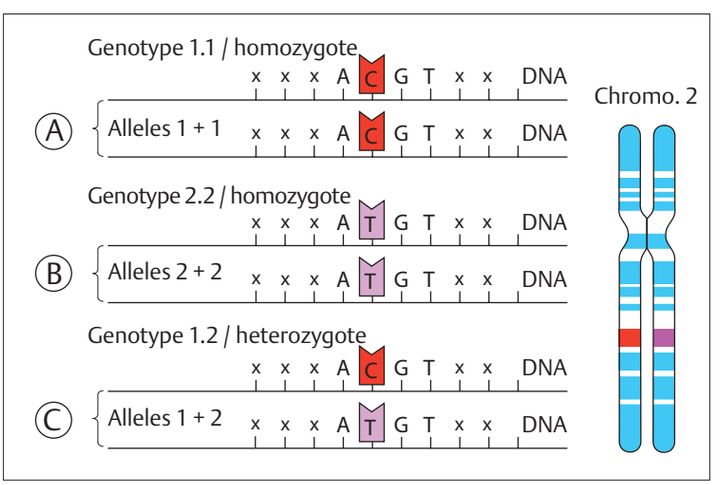
Mutations of an individual base pair ("single nucleotide polymorphism"; SNP) are quite common; the normal base sequence in the gene is termed *Allele 1*, and the less common or altered as *Allele 2*. Both IL-1 genes can exhibit the SNP: The base cytosine (pairing with guanine, Fig. 436 left) is then replaced by thymine (pairing with adenine). Polymorphisms are not representative of major genetic alterations (gene defects, cf. p. 54), and their effects are therefore usually mild.



429 Differences in Monocyte Stimulation with IL-1-Positive and IL-1-Negative Genotype
Monocytes/Macrophages determine for the most part the severity of the local inflammatory reaction to the corresponding irritant.

With an IL-1-positive genotype, the monocytes are provoked to up to 4 times higher production of IL-1 by lipopolysaccharide (LPS) from gram-negative anaerobes (*Pg, Tf, Td*).

Note: This local overproduction by hyperactive MΦ can be additionally elevated as a result of the *autocrine* (self-stimulating) effect of IL-1 (Deschner 2002).



430 Polymorphism—Genotype
The chromosomes of tissue cells are present in two categories (diploid, one each from the mother and the father). Therefore in a SNP, three variants per gene are possible (**A, B** and **C**):

A 2 times **C** (Cytosine: "normal")
= Allele 1 & 1, homozygote
B 2 times **T** (Thymine; *SNP*)
= Allele 2 & 2, homozygote
C 1 time **C** and 1 time **T**
= Alleles 1 & 2, heterozygote

Effect upon the Periodontium

The general effect of a positive IL-1 genotype is depicted in Figure 429.

Definition: The "IL-1-positive genotype" ("positive" with regard to SNP) is allocated to those individuals who possess the Allele 2 homozygote in one gene or at least heterozygosity in both IL genes (genes *IL-1A* and *IL-1B*; see above).

The elevated susceptibility to periodontal inflammation that is associated with the IL-positive genotype does not manifest itself immediately in the young persons, as long as no additional risk factors such as smoking, poor oral hygiene, systemic diseases such as diabetes mellitus (DM) etc. are present (Kornman et al. 1997). But if risks exist over a longer period of time, the IL-1 polymorphism accelerates and accentuates periodontitis to a great degree ("severity factor"; odds ratio ca. 1.5–2).

In difficult cases or in patients with high or multiple risk factors, the test for the existence of the IL-1 gene polymorphism is indicated.

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IL-1 Gene Test—Technique, Evaluation

Today, periodontitis is categorized within the large group of multifactorial diseases. Therefore it is logical to seek out new test systems for the demonstration of genetic factors that could elevate *susceptibility*, *progression* or *severity*. Already recognized are polymorphisms of the genes or gene clusters of TNFα, IL-10 as well as receptors of T-cells, immunoglobulins (Fcγ-RII, -III etc.), cathepsin, vitamin D3 etc. Especially and most well-researched is the effect of a gene polymorphism upon periodontitis that can enhance the inflammatory process via interleukin 1 (IL-1α and β; Kornman et al. 1999).

Several specialized laboratories now offer *interleukin-1 gene tests*; all measure the SNP ("single nucleotide polymorphisms") for the IL-1α at locus +4845 or –889 (equal effects), for IL-1β at locus +3954.

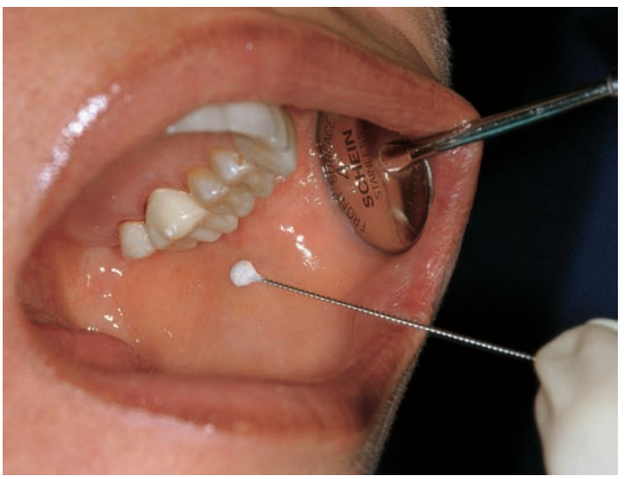
These simple DNA tests require host cells, which can be taken from blood, saliva or from the oral buccal mucosa. The miniscule amount of DNA must be amplified using PCR (polymerase chain reaction; p. 183).

IL-1 gene-positive does *not* immediately indicate "severe periodontitis": Its effect becomes critically important only in combination with additional risk co-factors.

431 IL-1 Gene Test—Harvest Technique

Left: Using a sterile brush, the buccal mucosa is scraped to collect some epithelial cells for the molecular biological analysis.

Right: The tip of the brush is inserted into a transport vial for the laboratory.





IL-1 gene tests that are commercially available:

- **PST—Interleukin Genetics**
- **GenoType PRT—Hain**
- **ParoGenTest—IAI**

Test results: Consequences of a Positive IL-1 Gene Polymorphism

432 Case No. 1

At age 35, this patient exhibits 10 % bone loss as a result of initial, chronic periodontitis.

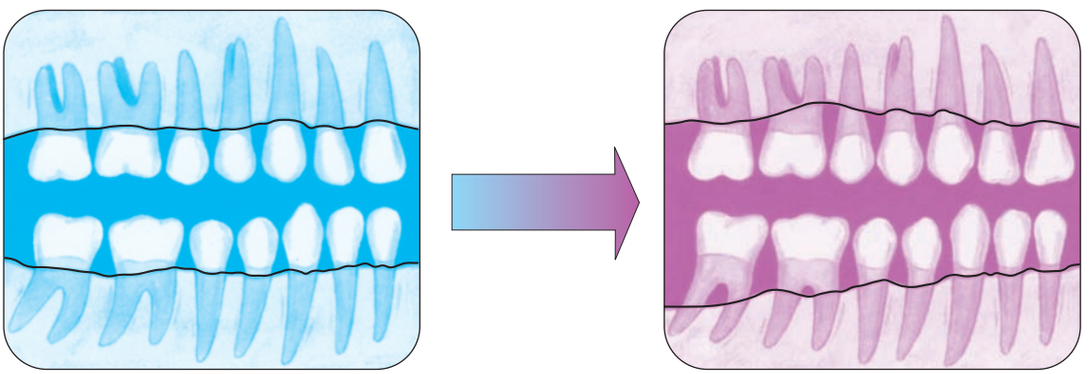
With *very good oral hygiene* and in the absence additional risk factors, 20 years later the 55-year-old individual will have retained all of the teeth; in areas that are naturally predisposed (molars, multi-rooted teeth etc.) bone loss may have advanced.

35 years old

10% bone loss

Very good oral hygiene — IL-1 gene-positive

55 years old



433 Case No. 2

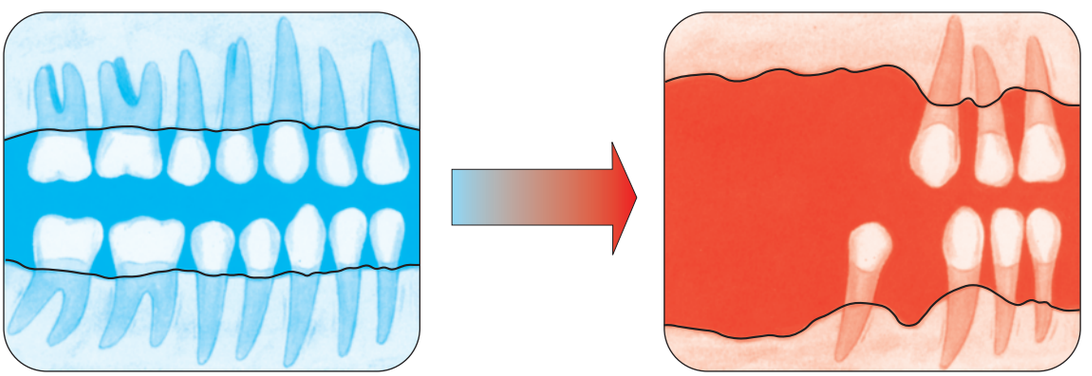
In a patient of the same age and under the same conditions, but with only *average oral hygiene*, within 20 years one can expect pronounced bone loss and tooth loss. The positive IL-1 genotype functions primarily as a "severity factor": It intensifies attachment loss and bone loss.

35 years old

10% bone loss

"average" oral hygiene — IL-1 gene positive

55 years old



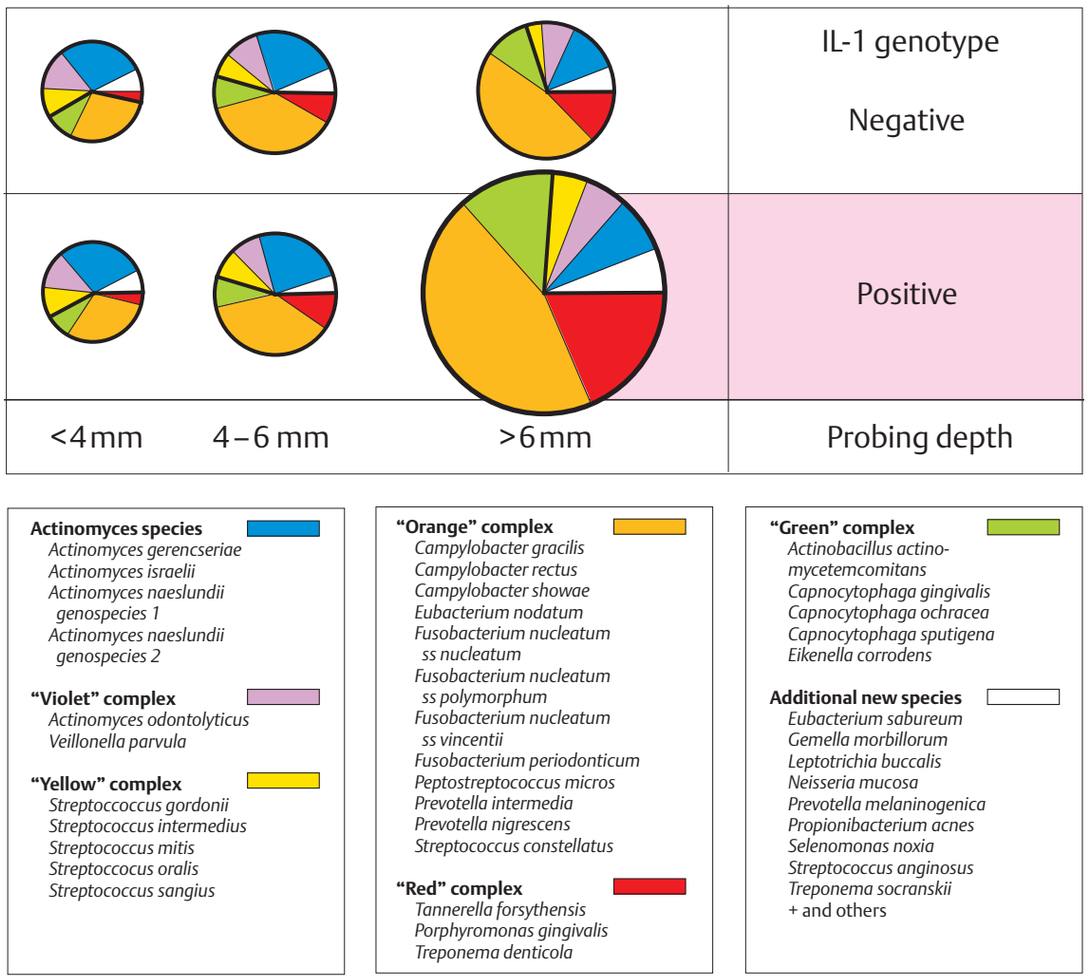
Modified from the *Hain Co.* prospectus.

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Risk Factor IL-1-positive Genotype—Additional Risk Factors

The genetic susceptibility—represented by the already described positive IL-1 genotype—does not, by itself, elicit periodontitis. The *initiation* and *progression* of this disease are, as heretofore, dependent upon bacteria, their pathogenicity and amount; therefore, the depth of the periodontal pockets also plays a major role (anaerobiosis!). The role that a positive IL-1 genotype might play was described by Socransky and coworkers (2000) for a primarily Caucasian population (Fig. 434): The association between positive genotype and severe, chronic periodontitis was observed only in older individuals (Fig. 433) and with deep pockets (Fig. 434). The *positive genotype* should therefore be viewed as a "*severity factor*." It is noteworthy that the positive genotype (Allele 2; p. 189) was detected in only 8 % of the examined Afro-Americans (Walker et al. 2000) and in only 2.3 % of the examined Chinese patients (Armitage et al. 2000), but in every third Caucasian.

Additional important risks for periodontitis, alone or combined, are well acknowledged: Above all smoking and poor oral hygiene, then also systemic conditions (diabetes, HIV), and stress; there is a relationship also to patient age.

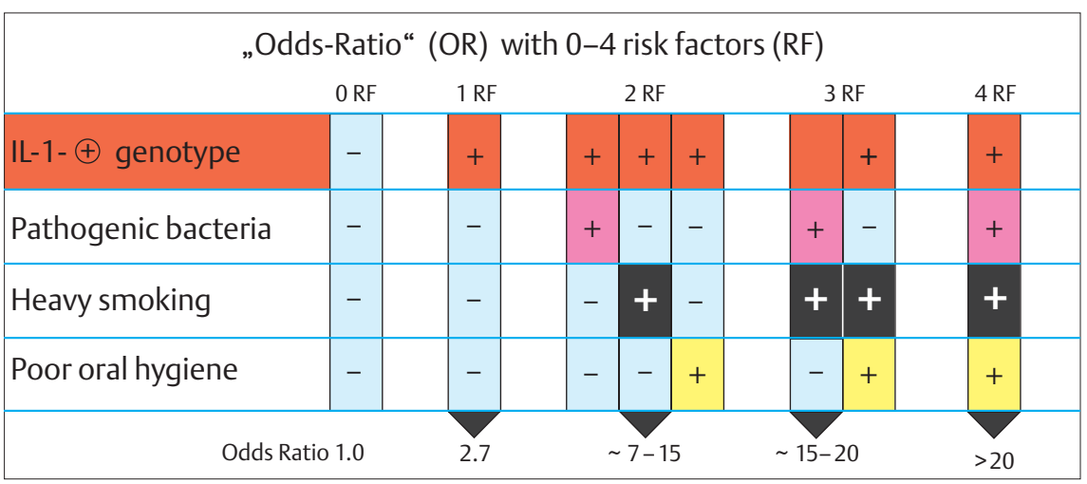


434 IL-1 Genotype—Pocket Depth and Bacterial Flora
With a probing depth of 6 mm, the bacterial colonization is similar in IL-1 negative and IL-1 positive patients (diameter of the circles). In addition, the percentage composition of the various complexes does not vary significantly between the two cases.

Beyond 6 mm probing depth, however, in an IL-1 positive patient one observes a dramatic alteration in terms of both the quantity of microorganisms as well as their quality (size of the circle; *orange* and *red complexes*). The strictly anaerobic milieu certainly plays an important role. The progression in the advanced stage of periodontitis appears to be greater/faster in patients who are genetically susceptible.

The bacterial complexes that have been reported by Socransky et al. (1998) are depicted at the left in their various and most important groupings (p. 37, Fig. 77).

Modified from *Socransky et al. 2000*



More Risk Factors = Elevated Overall Risk

435 Positive IL-1 Genotype in Combination with Additional Risk Factors
In addition to the IL-1 positive genotype, three additional risk factors are portrayed. The individually more or less important factors (cf. odds ratios) become multiplied and as a result for the individual patient provide an estimate of the overall risk. This can be critical for *therapy* and *long-term prognosis*.

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Poor Oral Hygiene as a Risk Factor—Bleeding on Probing (BOP)

In the preceding pages, bacterial test methods as well as several host response tests, especially the IL-1 gene polymorphism and its consequences, were described. Such tests enhance, to a certain degree, the traditional clinical and radiographic findings, and may improve diagnosis and prognosis of the individual yet very different forms of periodontitis, as well as planning for active periodontal therapy.
Because the various forms of periodontitis are neither generalized nor similarly progressing diseases, the detailed tooth-specific and site-specific examinations of plaque or bleeding indices retain their validity.

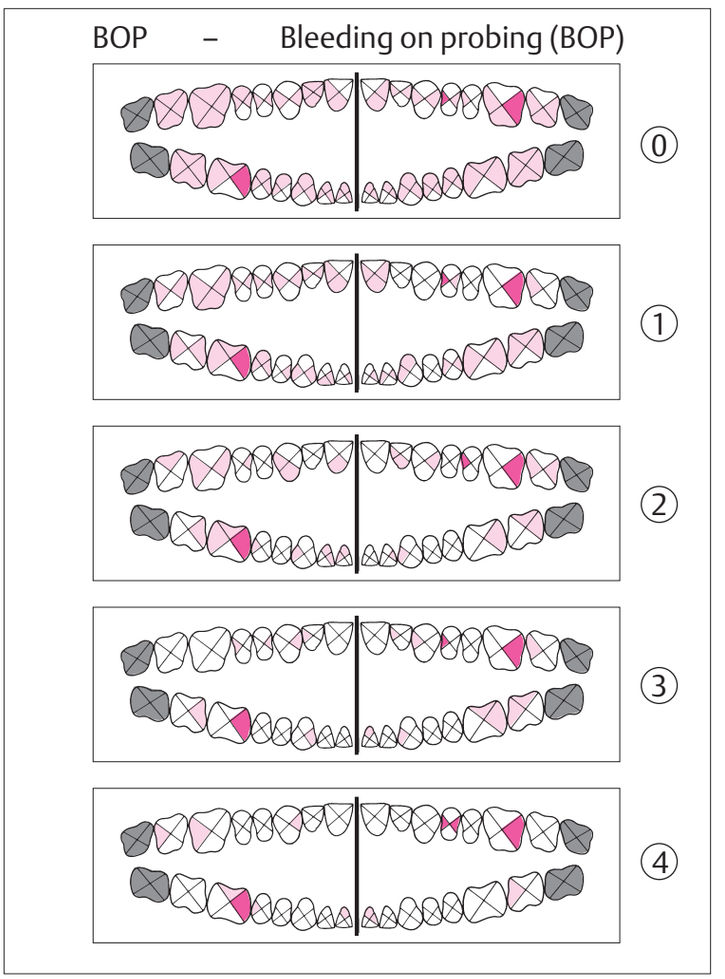
Plaque indices reveal both oral hygiene efficacy and patient compliance, but reveal nothing about the *host response* to the infectious inflammation. These individually differing reactions are finally critical for the severity of the disease, and can be better ascertained using the BOP index. "*Bleeding on probing*" (BOP) *per se* is a poor risk indicator (Lang & Brägger 1991): Bleeding = inflammation = gingivitis is not an indicator of periodontitis progression! *No* bleeding at the same site—measured over time (recall)—can, however, signal "stable conditions, without progression."

436 Long-term Monitoring—BOP Recording throughout Treatment and Recall

Chronology of the measurements:
0 before periodontitis therapy
1–4 during the four subsequent recall appointments

Prior to the active treatment, bleeding on probing was observed on all teeth (*pink* in the diagram).
After initial therapy, active treatment, and during recall therapy, the BOP continuously decreased: Less and less sites tested "positive" for BOP.

It is important to note in this case that on teeth 24, 26 and 46, on each tooth (red) the bleeding on probing persisted over the entire time. These sites—of which two were in the region of furcation entrances—carry a 30 % risk of progressive attachment loss of ≥ 2 mm. The correlation between *bleeding* and *attachment loss* is therefore relatively weak. Sites that do *not bleed* over an extended period of time almost never (98 %) experience attachment loss.

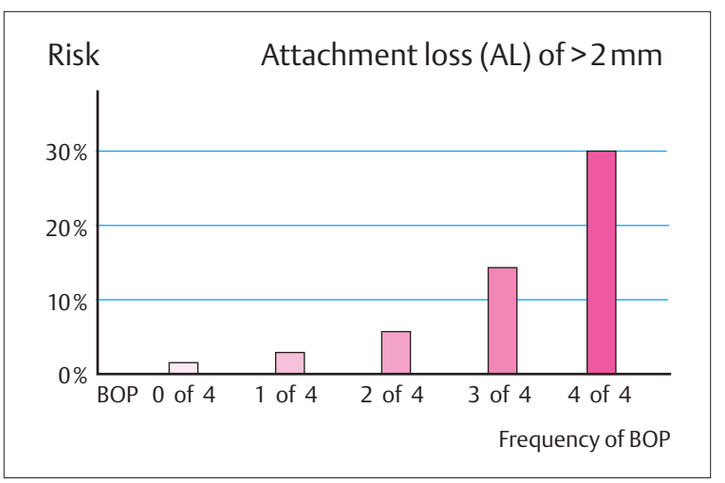


Validity of the BOP test

- *Positive* predictive value 0.3 (30 %)
- *Negative* predictive value 0.98 (98 %)

437 BOP and Attachment Loss – Clinical Study

The more often BOP is recorded at recall appointments at the same site, the greater is the likelihood that attachment loss will occur at that site (Lang & Brägger 1991). If, at four consecutive recall appointments (4 of 4) bleeding occurs, the probability for attachment loss exceeds the 30 % mark. The risk of attachment loss increases with the persistence of chronic inflammation.



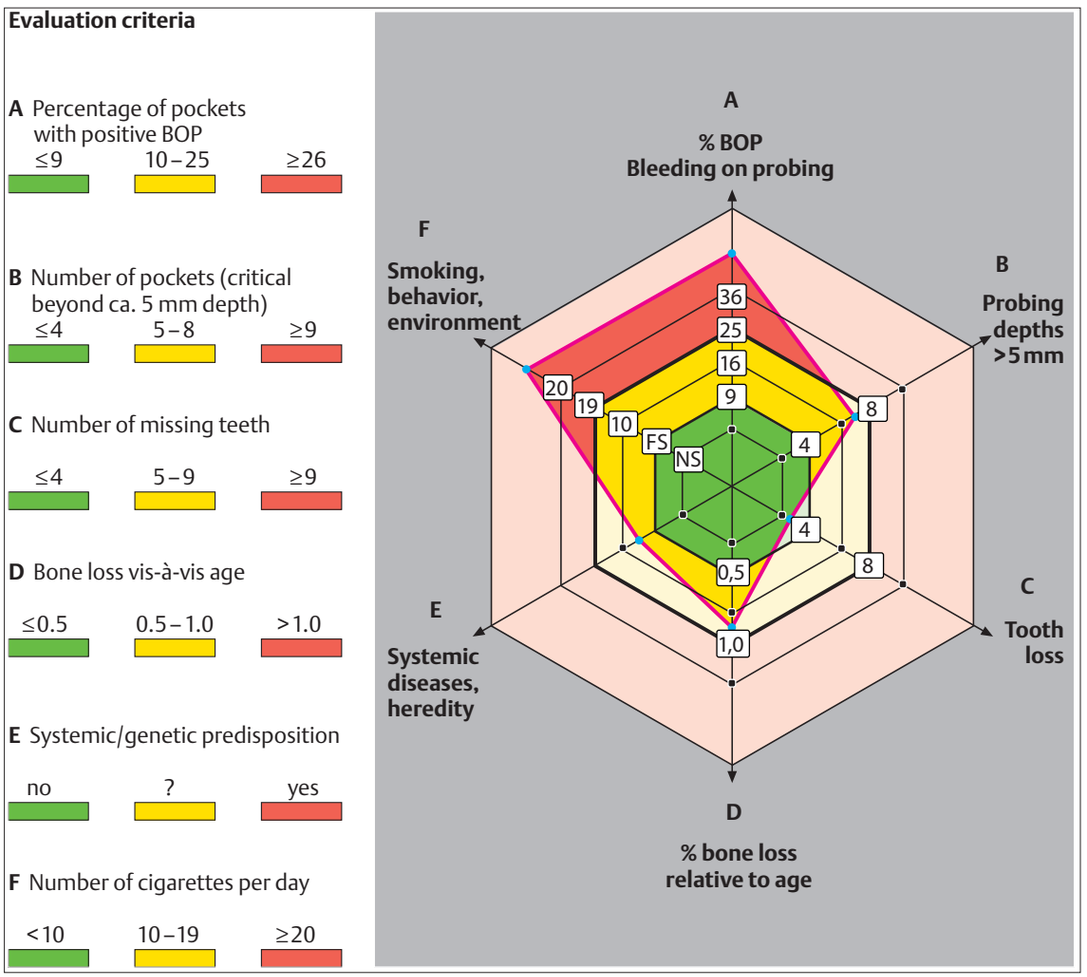
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Periodontal Risk Assessment—Individual Risk Profile

The large number of risk markers and risk factors demands a practice-friendly overview of such findings. The model that was created at the University of Berne (Lang & Tonetti 1996; risk hexagon, the "Bernese spider web") achieves this goal. The model observes six *risk clusters* upon tooth surfaces, teeth and the patient niveau. The resulting individual *risk profile* permits the clinician to establish a definitive diagnosis, prognosis and an optimum treatment plan; the model also provides the patient with valuable information and motivation toward removing or eliminating any alterable major risk factors (smoking, poor oral hygiene, diabetes).

Analytical Criteria—General

The hexagon takes into consideration *local* and *systemic* as well as *alterable* and *non-alterable* factors. For each criterion the risks are evaluated as *high*, *moderate* and *low*.
This risk analysis is not performed only with new patients, rather it is also employed during recall appointments following completion of active therapy. This permits a good overview of the patient's compliance through successive reduction of the overall risk (p. 451).



438 The "Risk Hexagon"—with a Patient Example
The "risk web" is configured in three concentric segments (from the center outwards):

- **Low** risk (*green*)
- **Moderate** risk (*yellow*)
- **High** risk (*red*)

Six parameters are evaluated [●]:

A BOP, number of sites [48]
B Probing depths ≥5 mm, number [7]
C Tooth loss, number of teeth [3]
D Bone loss in relation to age [0.8]
E Systemic diseases, genetics [-]
F smoking [> 20 cigarettes]; additional "lifestyle" risks

This example depicts a 50-year-old female with three high risks (BOP, smoking, bone loss). The areas in red indicate a *high overall risk*.

Analytical Criteria in Detail

Creation of the individual risk profile is simple:

- Missing teeth (C) are noted for each new patient.
- The number of residual pockets ≥ 5 mm probing depth (B) can be easily determined using the CPITN or the PSR index.
- The documentation of BOP (A) is recorded in conjunction with the performance of periodontal probing.
- The average bone loss in comparison to patient age (D) is determined from radiographic observations.
- (E) Systemic diseases (diabetes, HIV), medication side effects and genetically-based elevated susceptibility for periodontitis are determined from the medical history and its regular update at recall appointments.
- Smoking as a risk factor (F) influences in a dose-related manner the initiation and progression of periodontitis: For the non-smoker (NS), the former-smoker (FS) and with consumption of less than 5 cigarettes per day, the risk is calculated as slight; beyond 20 cigarettes a day, severe.

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Diagnostic Data Collection—Periodontal Charting, I and II

Periodontal findings must be recorded in writing, via charting, using radiographs, and in some cases with photographs and study models. Only this combination of data permits a secure diagnosis and detailed treatment planning, as well as the evaluation of long-term results.

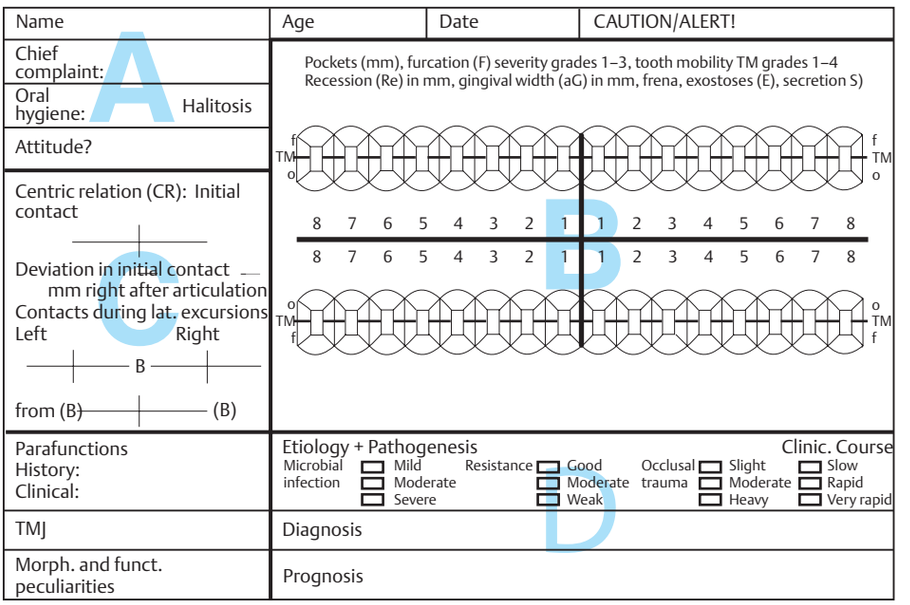
Diagnostic data collections are also imperative for patient information (motivation, compliance) and for the re-evaluation of the case following appropriate active treatment phases, during recall (p. 449), as well as for later evaluations and, hopefully seldom, for legal reasons.

Which of the many available periodontal data collection forms are used by the dentist is of little importance. It *is* important, however, that a good overview be provided, and that the possibility for measuring *probing depths* at six sites per tooth, *recession*, *furcation involvement* and *tooth mobility* can be ascertained and recorded. The clinical attachment loss (CAL) must be calculated! Additional findings (plaque, BOP, pulp vitality etc.) can be integrated into the periodontal data collection form or documented on separate forms, e. g., during repeated recall appointments.

The two charts used exclusively in this *Atlas* are described below.

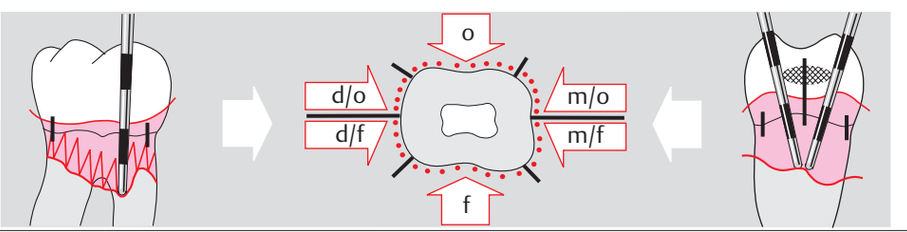
439 Periodontal Data Collection Form I

- **A Medical History**
- **B Periodontal Findings**
  Pocket probing depths, mm, colorations used in this *Atlas:*
  0–3 mm = –
  4–6 mm = pink
  beyond 7 mm = red
  TM = tooth mobility
  Space is available for recording recession (**Re**), furcation involvement (**F**), gingival width (**aG**) etc. The effective attachment loss (**AL**) is calculated from the recession (or hyperplasia) and the probing depths.
- **C Functional Findings (p. 174)**
- **D Etiology, Risk, Diagnosis / Prognosis**



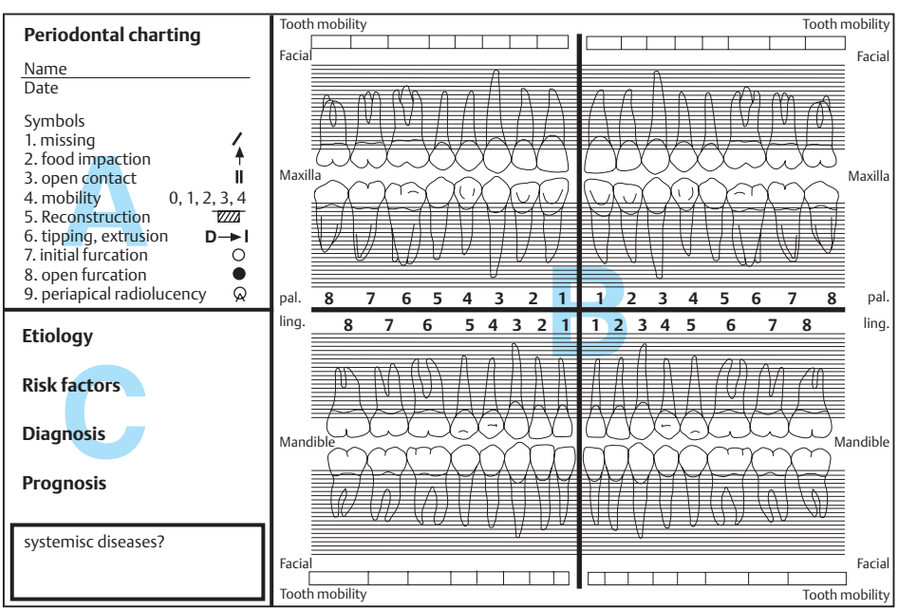
Measurement Technique—Probing Depths

The base of the pocket is assessed from the oral toward the facial, by "walking" the periodontal probe continually. Greatest depth is recorded for six sites.



440 Periodontal Data Collection Form II

- **A Glossary** of symbols for completing the form.
- **B Periodontal** findings: Gingival margin and probing depths are noted and then sketched into the form with millimeter precision (visual representation of the probing depths/attachment loss).
- **C Etiology, Risk, Diagnosis / Prognosis**
  The most important factors as well as the systemic and local risk factors are assessed and a "preliminary" diagnosis is made. This prognosis is only provisional.



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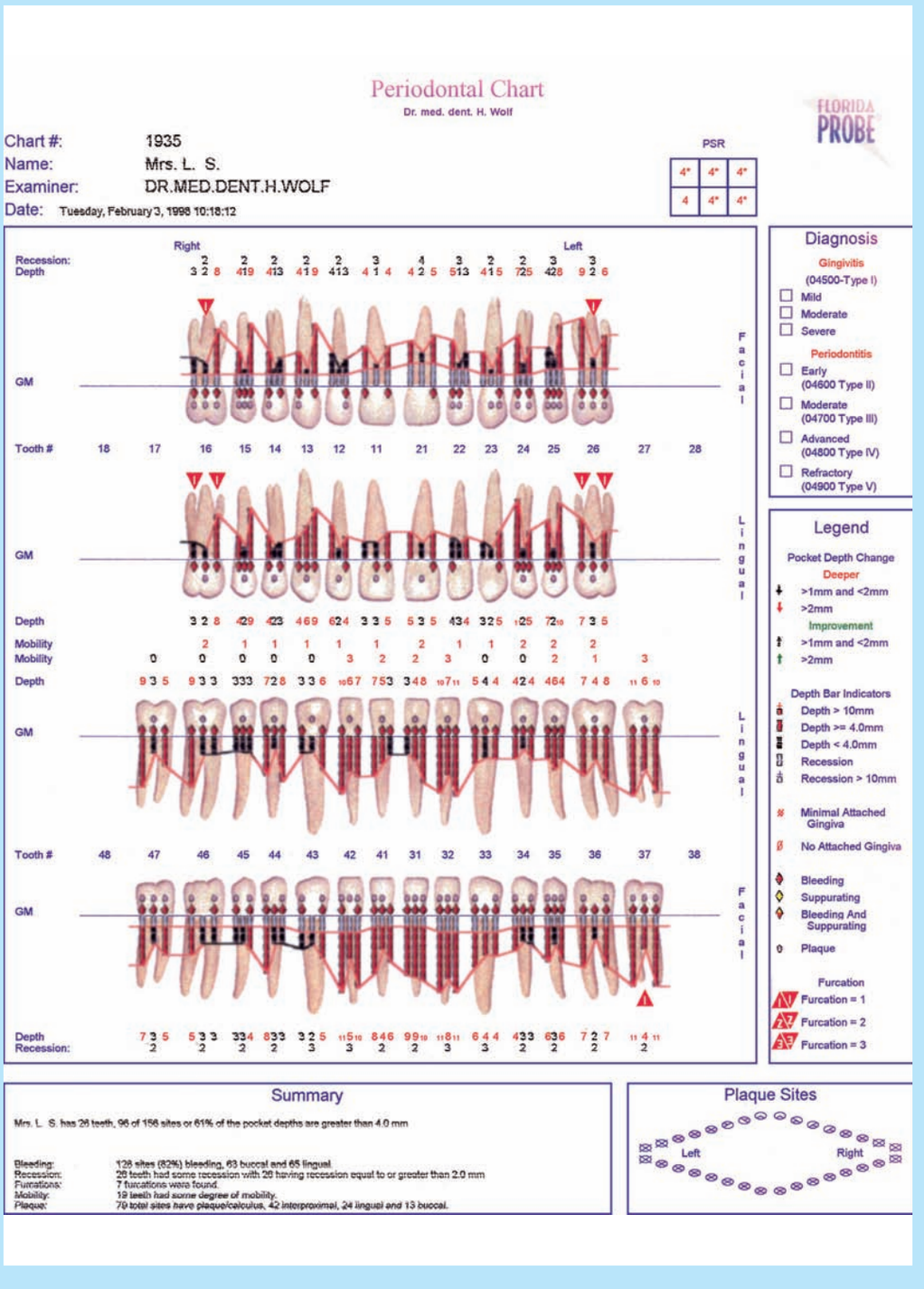
Computer-Enhanced Charting—the Florida Probe System

Recording the *periodontal condition* doesn't mean just measuring the probing depths of all pockets. As a primarily local disease, periodontitis also requires the probing of each individual tooth, and each individual site with regard to "pocket depth" *and* recession in order to calculate the clinical attachment loss. In addition, precisely at these sites, one must also record presence of plaque (PI), bleeding on probing, as well as any suppuration.

With computerization of almost all procedures in the dental practice, it is reasonable to collect and store periodontal data in a software-enhanced data bank.

The advanced *Florida Probe System* (FP32), developed at the University of Florida College of Dentistry (Gibbs et al. 1988) has today become the "gold standard." It consists on the hardware side of a force-calibrated (0.25 N) electronic handpiece with a titanium probe tip (p. 170), the innovative three-function foot pedal, as well as additional components (interface, etc.).

The comprehensive software, which is provided on a CD, permits a complete overview of the periodontal condition of the patient on a computer monitor, and a four-color printout of each patient's clinical condition.



441 Florida Probe (FP)—Charting

This figure represents an actual clinical case. The presentation contains all of the obligatory clinical parameters of a periodontal examination, tooth-by-tooth and color-coded:

- Recession RE** (6 sites)
- Probing depth PD** (6 sites)
- Furcation involvement (3 grades)
- Tooth mobility (definable)
- Bleeding on probing (6/4 sites)
- Suppuration (6 sites)
- Plaque (PI/PCR) (6/4 sites)

** Color-codes of the bars:

RE	**Blue**
PD up to 5 mm	**Black**
PD beyond 5 mm	**Red**

Additional observations such as *inadequate gingival width* can also be recorded.

The *PSR* or *PSI* (indices, p. 73) are automatically noted in the computer program (above right)!

The program is particularly valuable during Phase-3 therapy: At each recall appointment, the *improvement* or the *disintegration* at each site is immediately visible (patient information, motivation, compliance).

An important advantage is that the plaque documentation (below right), in the form of the PCR (O'Leary 1972; p. 68) can be portrayed and given to the patient as a separate print-out.

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Diagnosis

A reliable diagnosis demands not only on the classical local findings combined with bacteriologic tests. Such documentation provides information about the severity and, to a certain degree, also the etiology as well as the clinical course of the disease, the latter in relation to patient age. But the diagnosis depends also upon both the *general* and *specific* medical history, i.e., on the patient's systemic health and above all the presence of risk factors, and the patient's own risk behaviors (e.g., smoking; p. 216).

One must differentiate between the *general diagnosis* of the patient and the entire dentition, and the *diagnosis of individual teeth* and tooth surfaces. According to the newest classification (AAP 1999), one must differentiate between chronic (Type II), aggressive (Type III) and periodontitis associated with systemic diseases (Type IV). In addition, the severity (slight, moderate, severe) and the extent of involvement (localized, generalized) must be determined (for details of classification, see p. 519).

With a new patient, however, despite precise data collection, the diagnosis cannot be made "definitive" immediately.

General Periodontal Diagnosis

A general diagnosis is first made for the entire oral cavity:

- *Gingivitis*:
  Acute, chronic; hyperplastic; hormonal or influenced by medications; as a side reaction to systemic diseases etc.
- *Periodontitis*:
  Type, severity (clinical attachment loss), dissemination
- *Recession*

The *general periodontal diagnosis* reveals and encompasses only the *type* of periodontal disease in the particular patient.

It is, indeed, rare for gingivitis/periodontitis or even recession to be equally severe and generalized throughout the oral cavity. Much more often it is the case that severe pathologic conditions are observed on *individual teeth*, while other, milder symptoms are observed elsewhere, with some sites exhibiting no symptoms whatever.
According to the new classification, the term *localized* is used when 30 % or less of all tooth surfaces are affected; a more disseminated disease process is termed *generalized*.

442 Chart for "Single-Tooth Diagnosis"
In addition to the overall periodontal diagnosis, each tooth is evaluated individually.

- **Gingivitis**
- **Mild periodontitis: < 2 mm attachment loss (AL)**
- **Moderate periodontitis: AL 3–4 mm**
- **Severe/advanced periodontitis: ≥ 5 mm* AL**

* Furcation involvement? Tooth mobility?

Gingivitis																
Periodontitis, mild																
Periodontitis, moderate																
Periodontitis, severe*																
Single-Tooth Diagnosis	**8**	**7**	**6**	**5**	**4**	**3**	**2**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
	8	**7**	**6**	**5**	**4**	**3**	**2**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Gingivitis																
Periodontitis, mild																
Periodontitis, moderate																
Periodontitis, severe*																

Periodontal Single-Tooth Diagnosis

All periodontal charts as well as charts for plaque accumulation and inflammation (bleeding, p. 69) contain, in addition to an overview of the entire case, also the possibility to record single-tooth information, and thus permit single-tooth and "site" diagnosis; the probing depth around each tooth at six sites is measured and noted, either by hand or using computer data storage. The periodontal data collection instrument should provide the possibility to evaluate each tooth, and each surface of each tooth individually, even though the overall periodontal diagnosis is normally presented "generally," e.g., generalized, moderate, chronic periodontitis (CP) with localized severe areas.

It has been demonstrated time and again that periodontitis is not a generalized disease, rather it is a localized and often quite variable event from tooth to tooth. The "generalized" components include genetic predisposition, systemic predisposition, and diverse general "risk factors."

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Prognosis

The prognostic evaluation of a new periodontitis patient is, at first, difficult, and depends upon diverse factors. The *initial prognosis* that is derived based upon the initial clinical findings and preliminary diagnosis must frequently be revised during and after treatment.

Patient compliance and manual dexterity (oral hygiene) must be properly assessed and evaluated before the initiation of treatment. Also the reaction of the tissues to therapy, with regard to wound healing and capacity for regeneration cannot always be anticipated (biologic age, genetic risk factors).

In general terms, chronic periodontitis (CP, Type II) has a good prognosis, whereas the less common, rapidly progressive and aggressive forms (AP, Type III) are usually associated with a poorer prognosis.

The prognosis for the entire case is determined by the *prognosis for each individual tooth* (see below: periodontal single-tooth prognosis).

The clinician must differentiate between:
- *General prognostic factors*
- *Local prognostic factors*

General Prognostic Factors

- General health, resistance, immune status
- Genetic, systemic and acquired risks
- Etiology and clinical course of periodontitis
- Age in relation to attachment loss
- Demands and true possibilities for the patient
- Regularity of periodontal recall appointments
- Motivatibility and oral hygiene (compliance)

Local Prognostic Factors

- Tooth and root morphology, tooth position anomalies
- Amount and microbial composition of the plaque biofilm (virulence)
- Rapidity of plaque formation, independent of quality and intensity of oral hygiene
- Localization, depth and activity of pockets
- Furcation involvement/open furcations
- Extent of attachment loss
- Remaining attachment (root length)
- Type of bone destruction: horizontal or vertical
- Tooth mobility in relation to bone loss
- Tooth mobility in relation to occlusal trauma

Maintainable																
Questionable																
Hopeless																
Single-Tooth Prognosis	**8**	**7**	**6**	**5**	**4**	**3**	**2**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
	8	**7**	**6**	**5**	**4**	**3**	**2**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Maintainable																
Questionable																
Hopeless																

443 Chart for "Single-Tooth Prognosis"
For each new patient, corresponding to the overall diagnosis, a (provisional) prognosis should be established for each individual tooth:

- **Maintainable**
- **Questionable**
- **Hopeless?**

An apparently "hopeless" tooth that is functional and pain-free need not *a priori* be regarded as "hopeless." The "initial prognosis" must be constantly re-evaluated.

Periodontal Single-Tooth Prognosis

The overall evaluation of the patient and the patient's desires and possibilities, as well as the periodontal diagnosis and the evaluation of the individual single-tooth prognoses (especially that of strategically important abutment teeth) will determine whether the planned therapy will be radical or conservative, or possibly only palliative.

If the single-tooth prognoses are poor, especially in cases of rapidly progressing disease or advanced, aggressive forms of periodontitis, with simultaneous poor motivatibility of the patient, the clinician must consider during treatment planning (p. 208) whether periodontitis therapy with a questionable result is even a reasonable approach, or whether it might be more reasonable to extract remaining teeth and treat the case prosthetically.

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Prevention—Prophylaxis

Dentistry: Caries, Gingivitis, Periodontitis

Maintenance of Health and Prevention of Disease …

… are the noblest goals of modern medicine! For the patient, prevention is more comfortable, simpler to perform and, last but not least, less costly than treatment. The cost "explosion" in all disciplines of diagnostic and therapeutic medicine is enormous. It is scarcely possible to finance such costs regardless of whether socialized medicine or private insurance or direct payment by patients are invoked. Ethical, social and scientific facts demand that we move toward prevention.

Dentistry

Prevention of the ubiquitous inflammatory periodontal diseases should benefit every socioeconomic segment of society and every age group. Children, young persons and young adults in particular should be exposed to preventive measures. At the same time, they must be educated concerning the importance of their own responsibility in personal health care. Long-term preventive awareness and behavior demand insight, will and persistence from the patient (compliance). This can only be expected if the dental team, dental educators etc. continually make known the possibilities and the enormous importance of prevention, and thereby motivate the patient toward maintenance of health. This demands especially that the dentist not only possess current knowledge of new developments in periodontal diagnosis and treatment possibilities, the dentist must also recognize and keep abreast of systemic medical relationships, and communicate these to all patients.

Definition: Prevention—Prophylaxis

Prevention: Medical and dental measures to inhibit disease initiation

- *Primary prevention*: "Strengthening" health—inhibition of a disease, e.g., through vaccination; in dentistry, e.g., patient information, oral hygiene instruction, prophylactic measures
- *Secondary prevention*: Early detection and treatment of diseases, e.g., comprehensive diagnosis of the disease, and anti-infectious therapy
- *Tertiary prevention*: Stopping and preventing the recurrence of an already treated/healed disease, e.g., through regular recall (SPC—"supportive periodontal care")

Prophylaxis: Prevention of diseases (individual and collective)

- *Oral prophylaxis*: Mechanical removal of deposits, plaque and calculus
- *Antibiotic prophylaxis*

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Prevention of Gingivitis and Periodontitis

In recent years it has been demonstrated in well-documented studies that prevention of periodontitis can be successful if performed consistently and appropriately (Axelsson & Lindhe 1977, 1981a, b; Axelsson 1982, 1998, 2002).

The primary etiologic agent for gingivitis and periodontitis is the *microbial biofilm*. In the absence of biofilm, marginal inflammation will not develop. Therefore, prevention and prophylaxis involves, as always, the elimination of plaque and calculus, and motivation of the patient toward adequate oral hygiene.

From a somewhat broader prospective, prevention of periodontal disease encompasses several additional measures that are also targeted toward elimination of plaque, but indirectly. This is termed *anti-infectious therapy*, and includes the elimination of natural plaque-retentive areas (crowding, etc.) and above all the elimination of iatrogenic irritants. An overhanging restoration or a poorly adapted crown margin make oral hygiene in the interdental area impossible. Dental floss is ineffective in such cases; it tears and becomes lodged in the defective restoration. The possibility for satisfactory hygiene in each individual patient must be created by the dentist and the auxiliary dental personnel. Finally, the margins of restorations and reconstructions must be placed supragingivally whenever this is esthetically feasible in order to enhance prevention of gingival inflammation.

Total freedom from plaque is only a utopic goal. The reality is that, for each patient, an individual optimum hygiene level (plaque index) must be achieved, one which can be maintained consistently over the years, dependent upon the patient's own degree of motivation and above all the recall interval. It has been demonstrated again and again that the gingival and periodontal conditions, even in large groups of patients, do not deteriorate if an appropriate recall interval is established and maintained for the individual patient (Ramfjord et al. 1975, 1982; Rosling et al. 1976a; Axelsson & Lindhe 1981a, b; Axelsson 1982, 1998, 2002; Manser & Rateitschak 1997).

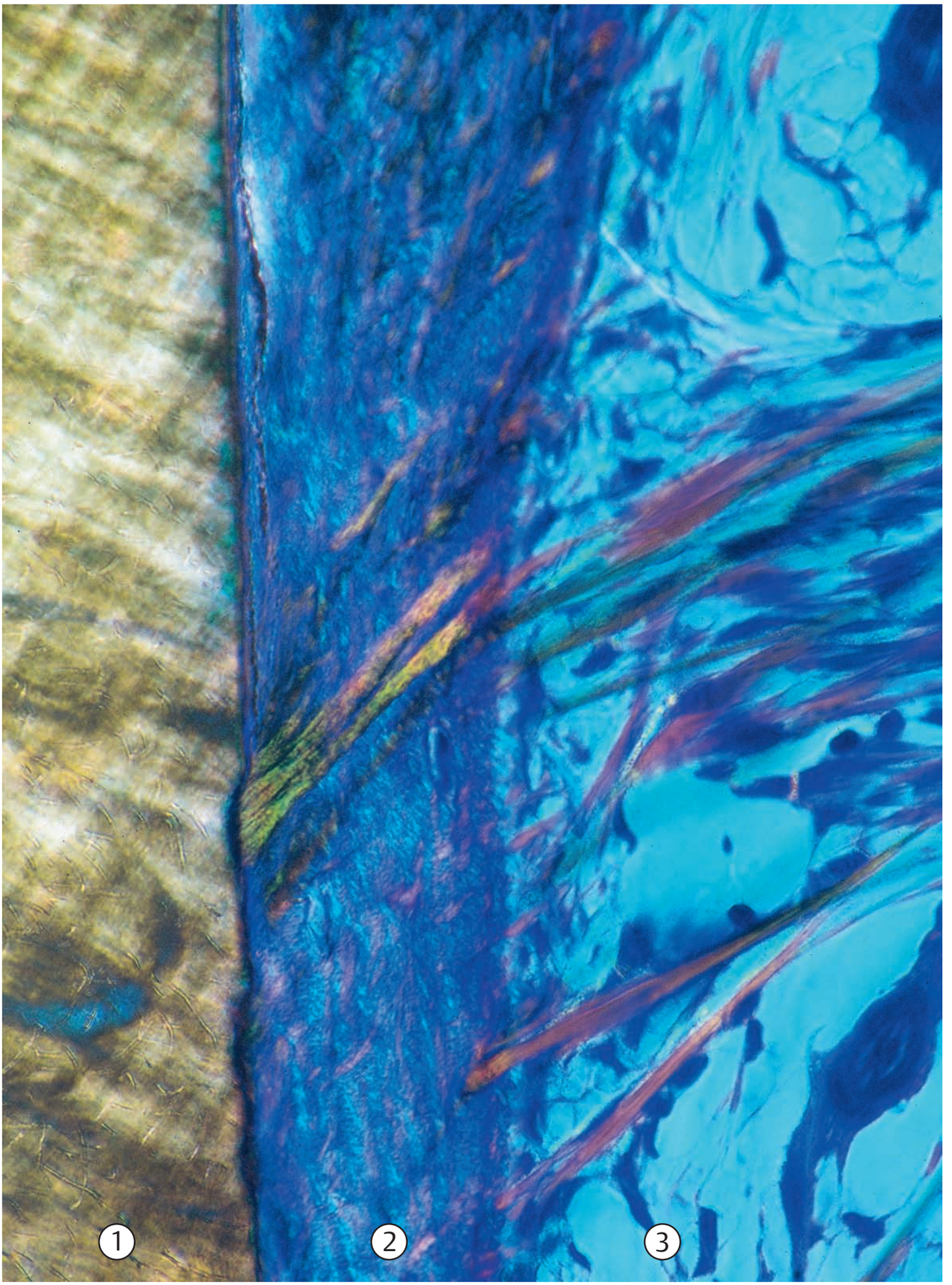
In the absence of microorganisms, there will be no gingivitis or periodontitis; on the other hand, the existence of plaque bacteria alone does not in every case lead to periodontitis. The susceptibility of the patient, her/his immune status, the existence of non-alterable (usually genetically-determined) and alterable risk factors, and the host's response to the infection are what determine the initiation and progression of periodontitis.

"Prevention," then, extends beyond elimination of the infection—as far as this is possible—and to *influencing the host* itself. Any strengthening of the immune system has, heretofore, been possible only in minute measure, and true gene manipulation stands far in the future. Most likely, the future will hold the possibility to suppress pro-inflammatory mediators or, on the other hand, to stimulate anti-inflammatory mediators. Possible already today is the effort to influence and therefore reduce *alterable risk factors*. Systemic diseases that enhance or even elicit periodontitis must be appropriately treated; e.g., diabetes must be optimally controlled. Most especially, smokers must eliminate the high risk of tobacco use. The goal is a generally healthy lifestyle, with a minimum of stress.

Dental/oral prevention cannot simply be a casual endeavor. It demands time—considerable time—from both the patient and the practitioner. This is usually underestimated, and the consequences may render treatment success problematic.

The practical performance of prevention for gingivitis and periodontitis is for the most part identical to the measures described in the chapter "Initial and Phase-1 Therapy" (pp. 211–252), and will not be further discussed in this brief treatise on "Prevention."

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Treatment of Inflammatory Periodontal Diseases

- **Gingivitis**
- **Periodontitis**

Gingivitis and periodontitis are elicited primarily by bacteria. As a consequence, the treatment must have a primarily anti-infectious nature. Reduction or elimination of the infection results for the most part from mechanical treatment of affected teeth and root surfaces as well as the gingival soft tissue. In special cases, support via topical or systemic medications may be indicated. Alterable risk factors must be eliminated as much as possible.

This section deals with the following constituents of periodontal therapy:

- Concepts of periodontal therapy: methods, goals, outcomes
- Periodontal healing, treatment planning, course of treatment

"Phase 0" Therapy — Systemic pre-treatment

- Emergency therapy

Phase-1 Therapy

- Initial therapy 1 and 2
  Anti-infectious/causal,
  non-surgical therapy
- FMT – "full mouth therapy"

- Adjunctive medicinal therapy – systemic and topical

Phase-2 Therapy — Surgical therapy
Anti-infectious and corrective therapy

- Access flap surgery
- Regenerative therapy
- Resective therapy
- Furcation treatment

- Mucogingival and plastic surgery

Phase-3 Therapy — Maintenance therapy – recall

- Additional adjunctive therapy:
  - Functional, orthodontic and splint therapy
  - Perio-prosthetics 1 and 2
  - "Alternative" therapy: Dental implants

Left side:

Histologic section in polarized light; True regeneration of the periodontium following GTR (p. 338)
M. Hürzeler et al. 1997

1 Dentin
2 *New acellular cement*, fibers
3 Periodontal fiber apparatus

Courtesy *P. Schüpbach*

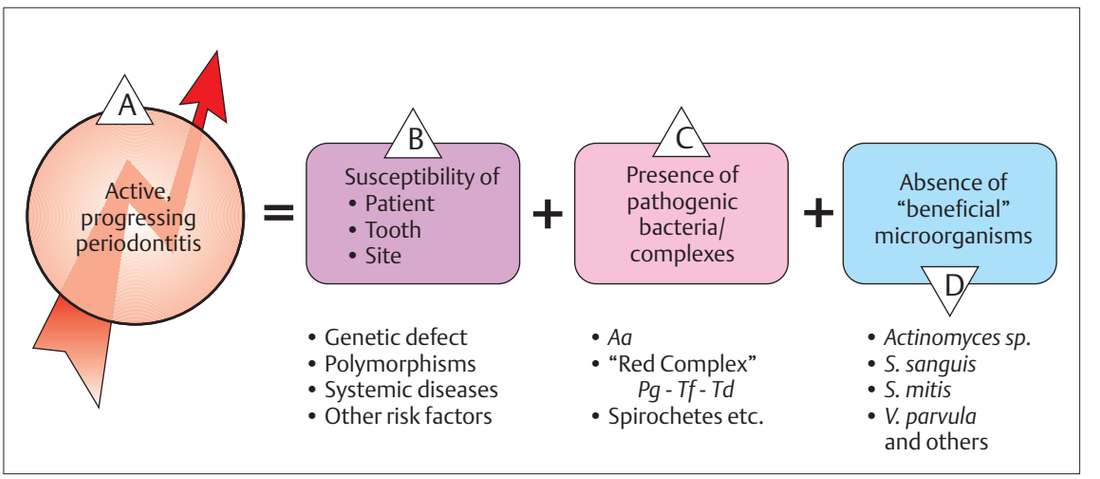
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Therapeutic Concepts and Techniques

All of the new knowledge gleaned in recent years, particularly in the areas of etiology and pathogenesis, have led to a true paradigm shift in treatment philosophy. This can be seen in many areas of practice. Following early diagnosis and early treatment, methods of and successes in prevention have become manifest. Only a few years ago, pocket elimination and freedom from plaque stood clearly in the foreground; however, today many other possibilities for combating periodontitis have entered the therapeutic larder.

Elimination/Reduction of Pathogenic Bacteria

It is impossible to achieve total freedom from plaque, either supragingivally or subgingivally. Therefore the goal of therapy is not the *elimination* of periodontopathic microorganisms, but rather "only" a significant *reduction* of the total number of microorganisms in the oral cavity. The goal is the creation of a *homeostatic balance* between resident bacteria and the host organism. Non-pathogenic microorganisms may be viewed as beneficial, because they often to maintain periodontopathic microorganisms in check (Fig. 444).



444 Etiology of Progressive Periodontitis

A The progression of periodontitis depends on various factors:
B The susceptibility of the patient (genetic defects or polymorphisms, systemic diseases, additional risk factors)
C The presence of periodontopathic bacteria and . . .
D . . . the absence of *beneficial* microorganisms.

Modified from *Socransky & Haffajee 1993*

Elimination of the Biofilm—Causal Therapy

The first step toward such homeostasis is achieved mainly by mechanical disruption of the closed community of microorganisms represented by the biofilm within the periodontal pocket, and the subsequent removal of these microbes. Disrupting the biofilm permits attack by the host defense systems and topical medicaments. This type of pocket treatment, particularly the treatment of the root surface, is performed mechanically either "closed" or "open," using ultrasonic instruments and/or conventional hand instruments.

The traditional *closed* mechanical treatment is particularly effective today when used in the "full mouth therapy" method (FMT; Quirynen et al. 1995, De Soete et al. 2001, Saxer 2002a, b). In the FMT technique, the pockets are continuously rinsed with a disinfectant solution (e.g., CHX, betadine etc.) during root planing. This combined technique improves the therapeutic results significantly, especially when all four quadrants are treated within a 24-hour period of time (p. 281).

Open (surgical) treatment also has as its *primary* goal the elimination of periodontopathic microorganisms within the biofilm. In addition, any morphologic defects of the bony pocket can be improved or corrected.

Corrective Therapy—Treating the Bony Defects

New knowledge makes it possible to predict regeneration of damaged periodontal structures:

- Filling of hard tissue defects with bone and bone replacement materials
- Using membrane-guided tissue regeneration (or combining both methods)
- Use of signal molecules such as matrix proteins, growth factors etc.

Influencing the Host

Unavoidable (non-alterable) risk factors such as genetic defects cannot be therapeutically influenced today; systemic diseases must, however, be diagnosed and treated by the physician. Thus, for example, a well controlled diabetic can be offered periodontal therapy with a good chance of success.

The patient's alterable risk factors (p. 54) must also be eliminated or severely reduced.

Protecting the "beneficial" Microorganisms

As mentioned, following periodontal therapy there should exist in the oral cavity an ecological homeostatic balance. In this regard, it is important that if the mechanical therapy is augmented by systemic medications, an antibiotic be selected that does not also eliminate the "useful or *beneficial*" microorganisms.

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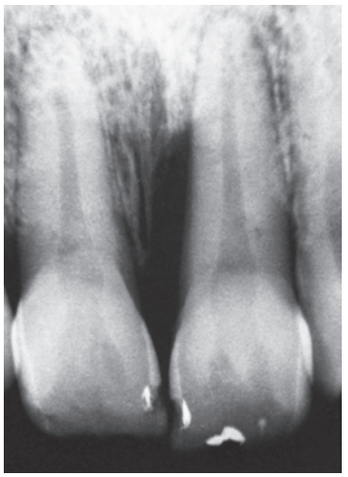
Therapy—Problems

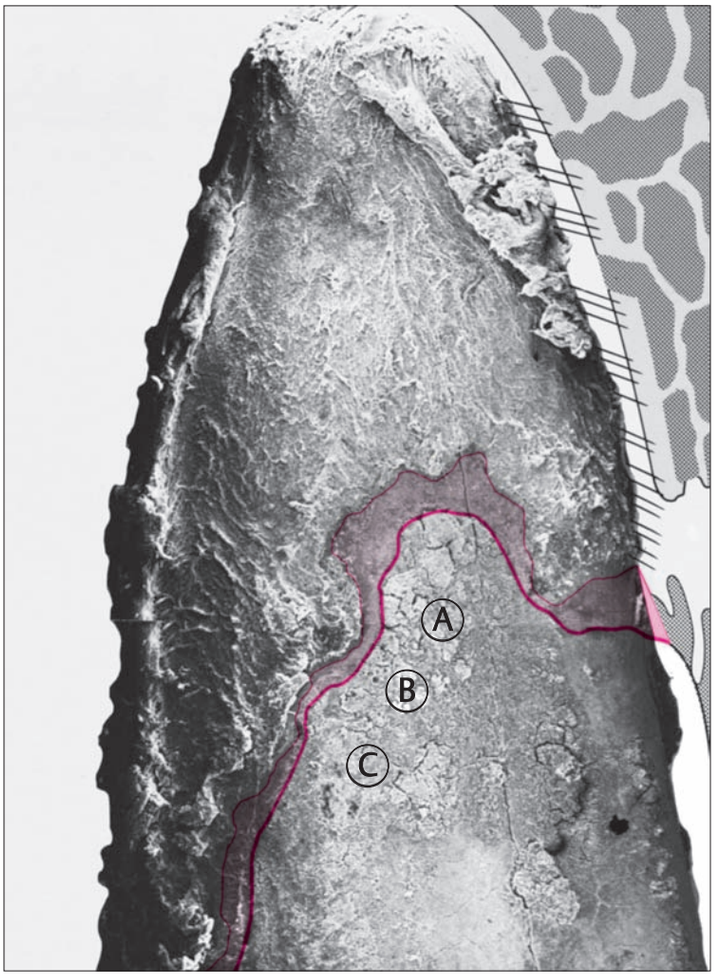
The principle of periodontitis therapy is simple: Thorough cleaning of tooth and root surfaces. However, in practice it is often associated with significant problems, among them:

- The irregular contour of the *base of the pocket and the remaining junctional epithelium*
- The *micromorphology* of the roots and furcations, especially cellular cementum, cementicles, lacunae and resorptions (Schroeder & Scherle 1987)
- The *macromorphology* of the roots, with narrow furcations, root fusions, ridges, grooves etc.

Rarely is the bottom of a periodontal pocket identical at all sites around the tooth. Usually some areas of the tooth are more severely involved than others.

The natural root surface is rough, especially in areas with cellular cementum and in furcations. It often exhibits cementicles and enamel pearls, and even in health some lacunae are observed (Schroeder & Rateitschak-Plüss 1983; Schroeder 1986; Holton et al. 1986). Cleaning of such plaque-retentive areas by means of root planing is difficult and time-consuming!





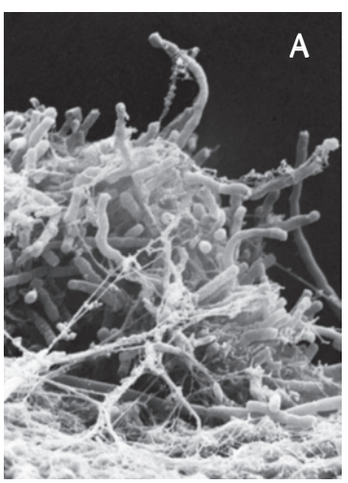
445 Irregular Base of a Periodontal Pocket in an Actual Case—Mesial View

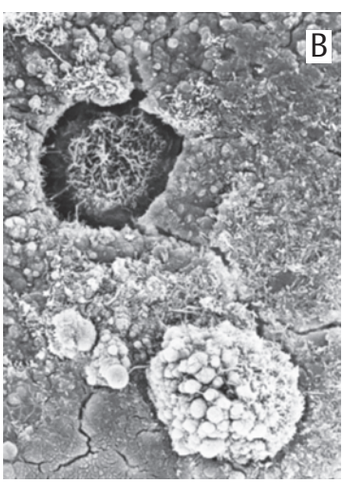
The left central incisor exhibits a very deep pocket on its mesial aspect. The junctional epithelium that persists at the base of the pocket is marked in red. Its course is irregular and at one location is even "undercut" in the apical region. Toward the palatal aspect (left in the SEM) intact periodontal structures can still be observed.

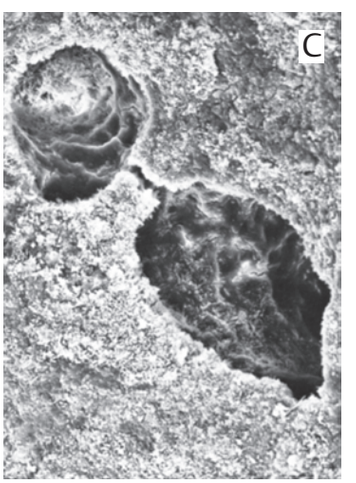
In the center of the picture, the wall of the pocket represented by the bacteria-coated root surface is visible. The difficulty for mechanical therapy becomes clear: On the one hand the bottom of the pocket must be achieved, and on the other hand any remaining soft tissue attachment must not be destroyed.

At the sites labeled **A, B** and **C**, the structures depicted below (Fig. 446) were observed.

Left: Radiograph. Tooth 21 was extracted. Its mesial root surface is depicted in the SEM (right).







446 Root Surface in the Area of the Pocket

A Adherent Plaque/Biofilm
Upon the root surface one observes a thick layer of adherent plaque.

B Lacuna Filled with Bacteria

C Empty Lacuna on the Root Surface

SEM courtesy *H. Schroeder*

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Periodontitis—Therapeutic Goals, Therapeutic Outcomes

The first goal of periodontal therapy is the complete healing of the inflammatory condition; the next goal is the regeneration of all lost periodontal structures.
Unfortunately, such total success is only seldom achieved. The enormous amount of new knowledge that has emanated from basic and clinical research, however, targets all of us in this direction. But our long-standing "gold standard" of therapy—namely closed and open tooth and root cleaning—will have to be enhanced by measures to restore defects, such as implanting bone and bone replacement materials, the GTR technique, as well as the use of matrix proteins and growth factors in the near future.

Even though the goal of complete periodontal regeneration has not yet been achieved, we can speak of partial success when applying the success criteria that are valid today. The terms "success" or "failure" and the magnitude of "partial" success following periodontitis therapy can be interpreted in many ways and depends, of course, on the initial situation, i.e., the diagnosis of periodontitis (chronic or aggressive form).

A *"healing success scale"* ranging from wishful thinking to achievable treatment outcomes today includes:

**** **Complete Regeneration**
*** **Healing of the Pockets—Repair**
** **Cessation of Attachment Loss**
* **Elimination or Reduction of Inflammation**

**** *Complete restoration/regeneration of all lost tissues ("4-star healing"):*
This type of regeneration is only achieved (today) following successful treatment of gingivitis (without attachment loss). The treatment of periodontitis generally does *not* lead to total regeneration of all diseased tissues.

*** *Pocket elimination via healing/repair*:
Within the marginal gingival soft tissues, this results in the formation of a long junctional epithelium and approximation of connective tissue to the root surface. In the apical regions, some regeneration of bone, cementum and periodontal ligament may occur. Following consolidation of the latter tissues, one can differentiate between "reattachment" and "new attachment."
- "Reattachment" refers to periodontal tissues that have been partially destroyed but which are not yet infected, and which *reattach* to the hard structures.
- "New attachment" signifies synthesis of new periodontal tissues/structures and their attachment to a previously mechanically treated root surface. Some shrinkage of the marginal gingiva occurs and this leads to even further pocket depth reduction.

This type of "*3-star healing*" following periodontitis therapy can be viewed as a very favorable result.

** *Cessation of attachment loss at its current position*:
This amounts to stopping of the progression of attachment loss in areas where pockets were in evidence, and healing by means of a long junctional epithelium. Following this type of healing, a relatively shallow and inactive residual pocket will remain. At the same time, however, the pocket depth will be reduced due to marginal gingival shrinkage, and increased tissue resiliency may lead to pocket depth reduction. In cases of advanced periodontitis, the clinician must frequently be satisfied with this type of "*2-star healing*". It requires a more frequent and regular recall interval, in order to quickly assess any new inflammatory manifestations of infection in the residual pocket. Patient compliance is especially important.

* *Elimination or reduction of clinically diagnosable inflammatory processes (bleeding*, BOP*)*:
This involves a reduction of the severity of inflammation and a certain degree of tissue shrinkage, without any periodontal tissue regeneration. Residual 4–5 mm pockets remain, but they are inactive and "dry." This "*1-star healing*" must be viewed as a partial success, but the situation can descend into a failure if the residual pocket becomes re-infected and if further attachment loss occurs.
The maintenance of this type of "partial success" is only possible over the long term if the patient's compliance is good and the recall interval is short.

In addition to these primary treatment goals of *periodontal pocket therapy*, additional improvements in the gingivoperiodontal structures are strived for:

- Improvement of gingival contour and possibly also bony architecture to render plaque control easier
- Optimization of the functional and esthetic situations via surgical procedures—gingivoplasty, covering recessions, alveolar crest remodelling etc.
- Functional therapy, selective occlusal adjustment: Improvement of function, morphology and esthetics
- Stabilization of mobile teeth (function; temporary or permanent splinting, p. 471)
- Replacement of missing teeth and restoration of morphologically defective teeth; alveolar ridge augmentation

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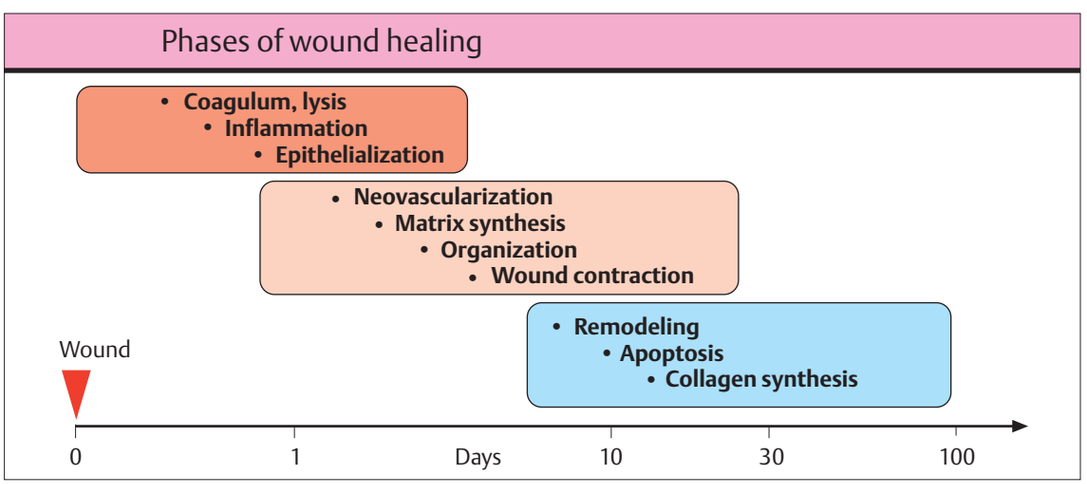
Periodontal Wound Healing

Repair → Reattachment → „New Attachment“ → Regeneration → Prevention

Periodontal wound healing follows the well-acknowledged biological principles (Clark 1996, Fig. 447), but it is also the “*most complex healing process*” in the human body (McCulloch 1993): The cells of five or more tissue types—epithelium, gingival and periodontal connective tissue, bone, root cementum—are essentially asked to create a new connection to the nonvascular and nonvital hard tissue of the root surface. Healing of the periodontal wound is also rendered more complex because it must occur in an open system, permanently contaminated and under a significant “bacterial load.” It is therefore not surprising that the healing results following all types of periodontal pocket therapy can be quite variable.

In contrast, the osseointegration of a titanium implant (p. 511) is, biologically speaking, child’s play, involving only ankylotic connection to the bone. In the case of *periodontal* healing, ankylosis represents a *failure* (root resorption)!

The most basic requirement for successful periodontal treatment is a clean, biofilm-free, decontaminated root surface. In most cases, this leads to connective tissue *repair*, a long junctional epithelium and usually residual pockets.
Ever-more successful *regenerative* treatment methods must be developed to insure optimal healing results.



447 Principal Stages of Wound Healing
The three overlapping stages will be described. Because their temporal course is influenced by a diversity of factors, each of these stages may vary considerably in length:
- Stage of inflammation
  Short duration (orange)
- Stage of proliferation
  Medium duration (beige)
- Stage of maturation
  Long duration (blue)

Adapted from *R. Clark 1996*

Regeneration of the Periodontal Defect

In addition to elimination of the tissue-destroying inflammation, true regeneration of lost tissues is one of the most important future topics in periodontology. Bony defects are filled today using autogenous or bone replacement materials; biomechanical substances (barrier membranes; GTR, p. 338) prevent the downgrowth of epithelial tissue.
Thereafter, signal molecules (differentiation factors, growth factors etc.) steer migration and differentiation of pluripotent stem cells, guided by artificial or natural structures (“tissue engineering”), matrix formation and the formation of new tissue (Lynch et al. 1999).

Great progress has been made in tissue augmentation and defect filling, soon to become a standard procedure, but the more difficult task is to achieve ”*Regeneratio ad integrum*,” namely a completely functional connection between the augmented soft tissues and especially the alveolar bone to the once infected and morphologically altered root surface (new periodontal ligament).
In all experiments to date, the formation of new cementum was only rarely identified histologically as acellular, extrinsic-fiber cementum, but more often as cellular cementum. Many authors have referred to this material as “bone-like,” which does not provide a stable connection to the root dentin.

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Wound Healing and Regeneration—Possibilities

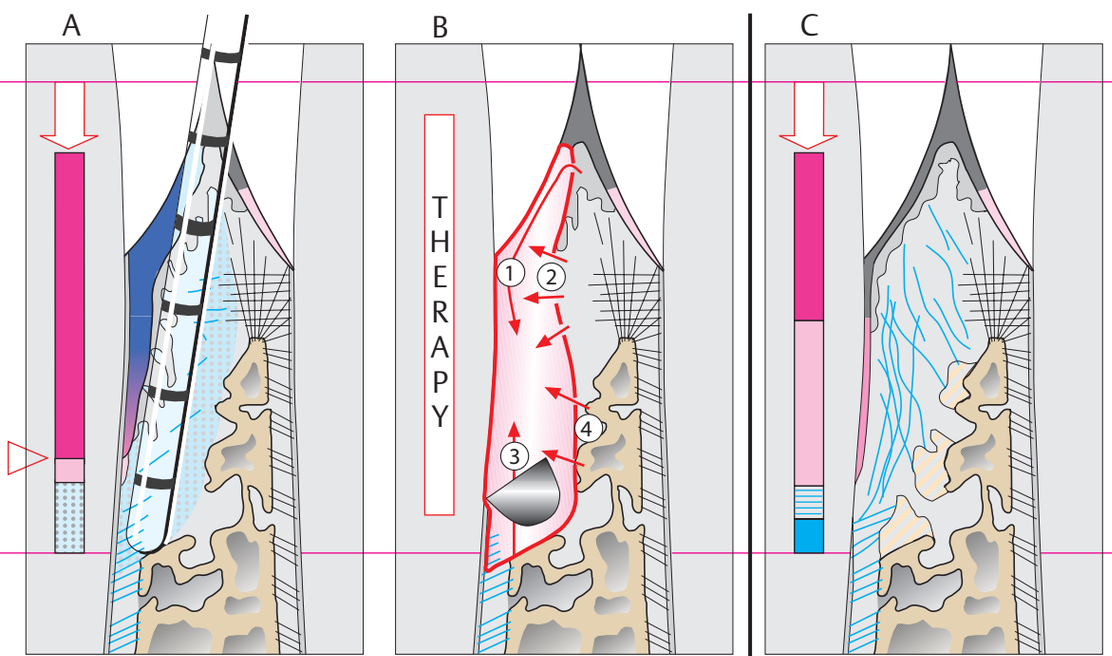
The paradigms of periodontitis therapy have changed markedly in the past two decades, due primarily to a veritable flood of new knowledge from medical specialties (McCulloch 1993, McGuire 1996, TenCate 1997, Wikesjö & Selvig 1999, Cho & Garant 2000).

New insights into the guidance and feedback mechanisms of cellular function now permit us to influence the healing processes (Bartold & Narayanan 1998, Christgau 2001, Hägewald 2002). As a result of advances in cell biology, we can better interpret the behavior of the tissues (Amar & Chung 1994, Selvig & Wikesjö 1999). For example:

- The bone-inducing capacity of demineralized bone matrix (proteins such as BMP; Jepsen 1996, Jepsen & Terheyden 2000; p. 351).
- Concepts of conditioning the nonvascular root surface (acids, Emdogain; Selvig et al. 1988, Trombelli et al. 1995, Hammarström 1997, Blomlöf et al. 2000)
- The "bioacceptability" of a formerly LPS-saturated root surface following detoxification
- The concept of tissue "compartments"; this lead to the GTR technique (p. 338), because cells colonize the surface according to the principle of "first come—first served" (Fig. 448B)
- The systemically-modulated, complex and overall active local network of growth factors and differentiation factors, signal molecules and adhesion molecules (Marx et al. 1998, Anitua 2001, Kübler & Würzler 2002)
- The existence of pluripotent stem and precursor cells within the blood stream and in the perivascular tissues.

Despite these enticing new developments and theories, periodontitis therapy today is guided by a strict anti-infectious, anti-microbial concept (Slots et al. 1999), with protocols for the individual treatment techniques. But still lacking are guidelines for the immediate postoperative period, that is during the first phase of healing, for wound care, immobilization and pharmacomechanical plaque control of the healing wound. Stabilization, especially the *stabilization of the coagulum*, is one of the most important measures. Stabilization mechanisms (adhesins etc.; Somerman et al. 1987, MacNeil & Somerman 1999, Somerman 2001) on the conditioned root surface prevent the downgrowth of epithelium and enhance the secure stabilization of the fibrin matrix. This serves as a natural guidance mechanism for the immigration of factor-guided future tissue cells during the second phase of wound healing.

448 Possibilities for Periodontal Wound Healing



Adapted from *H. Schroeder*

Depicted are diagnosis (**A**), treatment (**B**) and possible therapeutic results (**C—G**; post-therapeutic healing) for the periodontal intraalveolar defect on the tooth shown on the left side of each illustration.

Note: The periodontal tissues of the tooth on the right side of the illustration are healthy.

A Diagnosis: Interdental Bony Pocket

The periodontal probe penetrates the inflamed and infiltrated loose tissue until it contacts the bone adjacent *to* the diseased tooth, below the most coronal niveau of the interdental alveolar ridge (= "bony pocket").

B Treatment: Closed or Surgical Therapy, Root Planing

The "race among the tissues" now begins (cf. GTR, p. 338).

1 Oral gingival epithelium
2 Gingival connective tissue
3 Periodontal ligament connective tissue
4 Bone

C Healing after Debridement, via a Long Junctional Epithelium

Typical healing pattern, from apical towards coronal:

- Little reattachment (blue)
- Little new attachment (blue hatched)
- Long junctional epithelium (pink) with new collagen fibers parallel to it
- Residual pocket

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Periodontal Wound Healing—Definitions

Histologic studies of wound healing have clarified whether and to what extent healing of the gingival and periodontal attachment apparatus is possible in the form of re-attachment or regeneration (Schroeder 1983, Polson 1986, Karring 1988). One differentiates among:

• Epithelium	re-attachment?
• Epithelial regeneration	"new attachment"
• Connective tissue	re-attachment
• Connective tissue regeneration	"new attachment"

Histologic Terminology

Regeneration—*"Restitutio ad integrum"*
Complete regeneration of form and function: Gingiva with junctional epithelium and gingival connective tissue; periodontium with cementum, periodontal ligament, bone.

Repair
Restoration of the continuity in the wound or defect area, without regeneration of the originally intact tissues' form and function: e.g., long junctional epithelial attachment.

"New attachment"
New connection of connective tissue with the formerly pathologically exposed root surface, i.e., formation of new cementum with inserted periodontal ligament fibers (also, formation of new bone with Sharpey's fibers embedded).

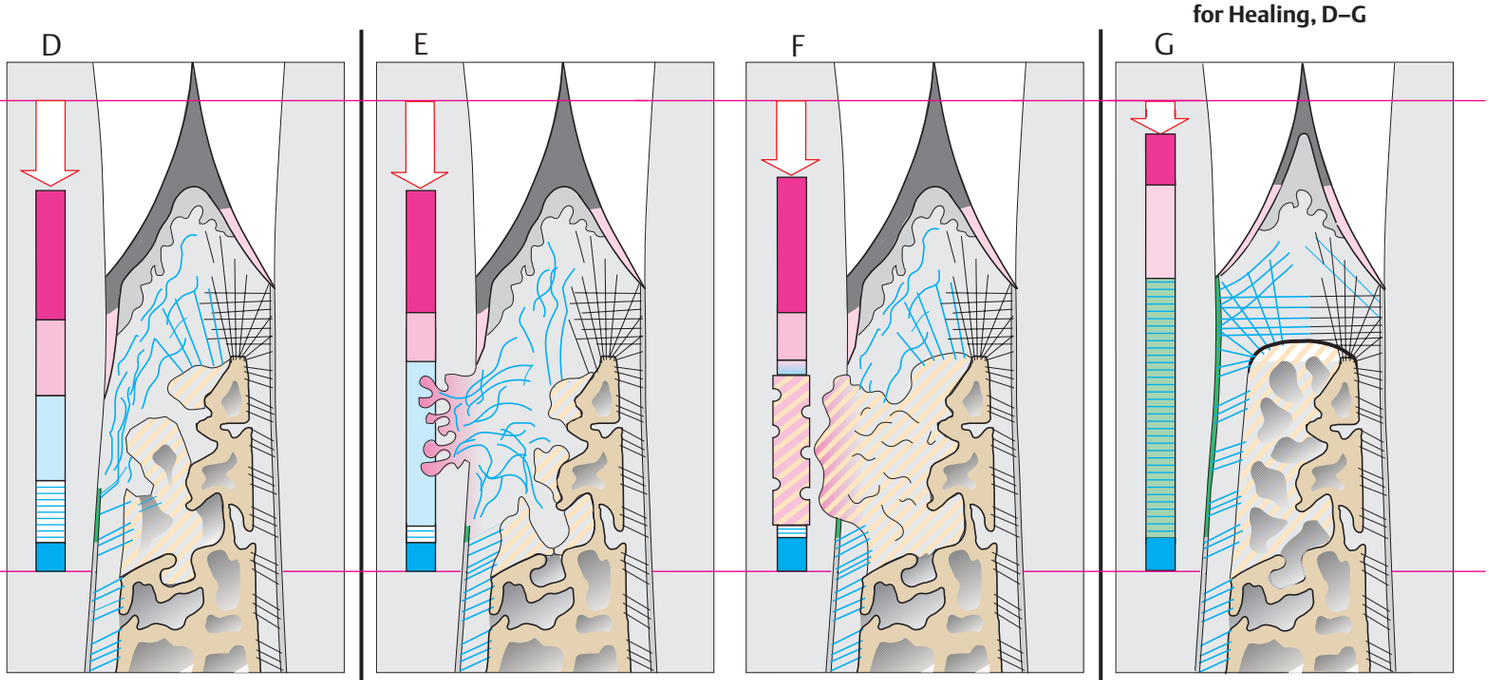
Re-attachment
Re-attachment is the re-establishment of the bond between connective tissue and the remaining vital tissue components on the root surface, e.g., cementum and remnants of the periodontal ligament (usually in the deepest areas of the pocket; "dark blue" in Fig. 448).
Note: Epithelial re-attachment does not occur. Epithelium is always established by new cells from the basal cell layer.

Alveolar Bone—"bone fill"
Filling a periodontal osseous defect does not provide evidence for complete periodontal regeneration (including newly formed cementum). This can only be demonstrated histologically (Listgarten 1986).

Clinical Terminology

For precise definitions of probing depth, clinical attachment level etc., see "Diagnosis" (p. 165).

448 Further Possibilities for Healing, D–G



D Healing after Regeneration Surgery

- Somewhat elongated junctional epithelium
- Collagen adhesion with some reattachment
- Osseous regeneration; regeneration occurs primarily in the apical regions, more coronally one finds a long junctional epithelium = repair; residual pocket = good "partial success"

E Unfavorable Healing with Root Resorption

Connective tissue repair leads to connective tissue filling the defect

Resorption of the root surface ("external granuloma")

Failure of protection by the junctional epithelium?

F Unfavorable Healing with Ankylosis

Bone regeneration with *root resorption*

Aggressively regenerating bone resorbs and fills segments of the root dentin; filled earlier by means of transplantation of illiac crest bone as a "filler."

G Total Regeneration: Future Goal? Utopia?

De novo formation of:
- Cementum (green)
- Periodontal ligament
- Bone
- Junctional epithelium

All tissues regenerate in proper amounts, in the correct temporal sequence, restoring tissue homeostasis ("*Restitutio ad integrum*")

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Treatment Planning—Sequence of Treatment

In principle, the course of periodontitis therapy is similar for all forms of the disease (p. 209), and is administered in *stages* or *phases* of varying duration, depending upon the extent and severity of the disease.
However the details of individual therapy may be dramatically different. These details are dependent upon the type of disease, the patient's own desires, patient age, financial circumstances and, not least, the preference of the individual clinician! It is well known that "many roads lead to Rome"!

Pre-phase—Systemic Health, Oral Hygiene

"Phase 0" consists primarily of identifying the patient's *systemic health* (general medical history, p. 167; readiness for treatment, p. 212), as well as a comprehensive data collection, the establishment of a provisional diagnosis and a case presentation (current status, necessary treatment). During Phase 0, any emergency treatment is also rendered.
The establishment of optimum *oral hygiene* and securing the patient's willing *compliance* are extremely important for subsequent planning and the long-term result.
Professional supragingival plaque and calculus removal, the removal of iatrogenic irritants and plaque-retentive niches, as well as patient instruction in simple yet effective plaque-control will quickly improve the intraoral situation. This will convince and motivate the cooperative patient toward further and definitive participation in the comprehensive treatment program.
It must not be left unsaid that, despite optimum information and instruction, some periodontitis patients refuse "comprehensive treatment." Such refusal must be clearly noted in the patient chart.

Phase 1—Causal, Antimicrobial, Anti-infectious

During this treatment phase, the findings, diagnosis and prognosis are verified. During the pre-phase, oral hygiene by the patient and the practitioner will have led to reductions in plaque and inflammation, therefore also to reduction of tissue swelling; it is reasonable to now establish a *definitive* diagnosis (and prognosis) based upon the important clinical findings (probing depths, attachment loss), and to formulate the definitive treatment plan.
While all of the pre-phase procedures are carried out with all patients, during Phase 1 the treatment modalities can differ. In some cases, *closed root planing*, with or without the concurrent use of medicaments (p. 287) may be performed, while in other cases the treatment moves directly into surgery (Phase 2).
In mild cases, above all with chronic periodontitis, *closed therapy* is frequently sufficient if the patient's compliance is good.
In more severe cases it may be indicated to move into *surgical-corrective therapy* immediately after an intensive pre-phase.
In most cases, however, open therapy will follow closed therapy: As a consequence, in such cases fewer sites (in fewer sextants) will have to be treated surgically. The resultant tissue loss (papillae, marginal gingiva) will be less post-operatively.
Whether the case is treated solely closed (A), solely open (C), or initially closed and subsequently open (B; p. 209) depends not only upon the pathomorphologic situation, but also upon the practice structure. If the dentist works with a competent dental hygienist, the hygienist will generally perform the entire *closed* phase of therapy (p. 454).

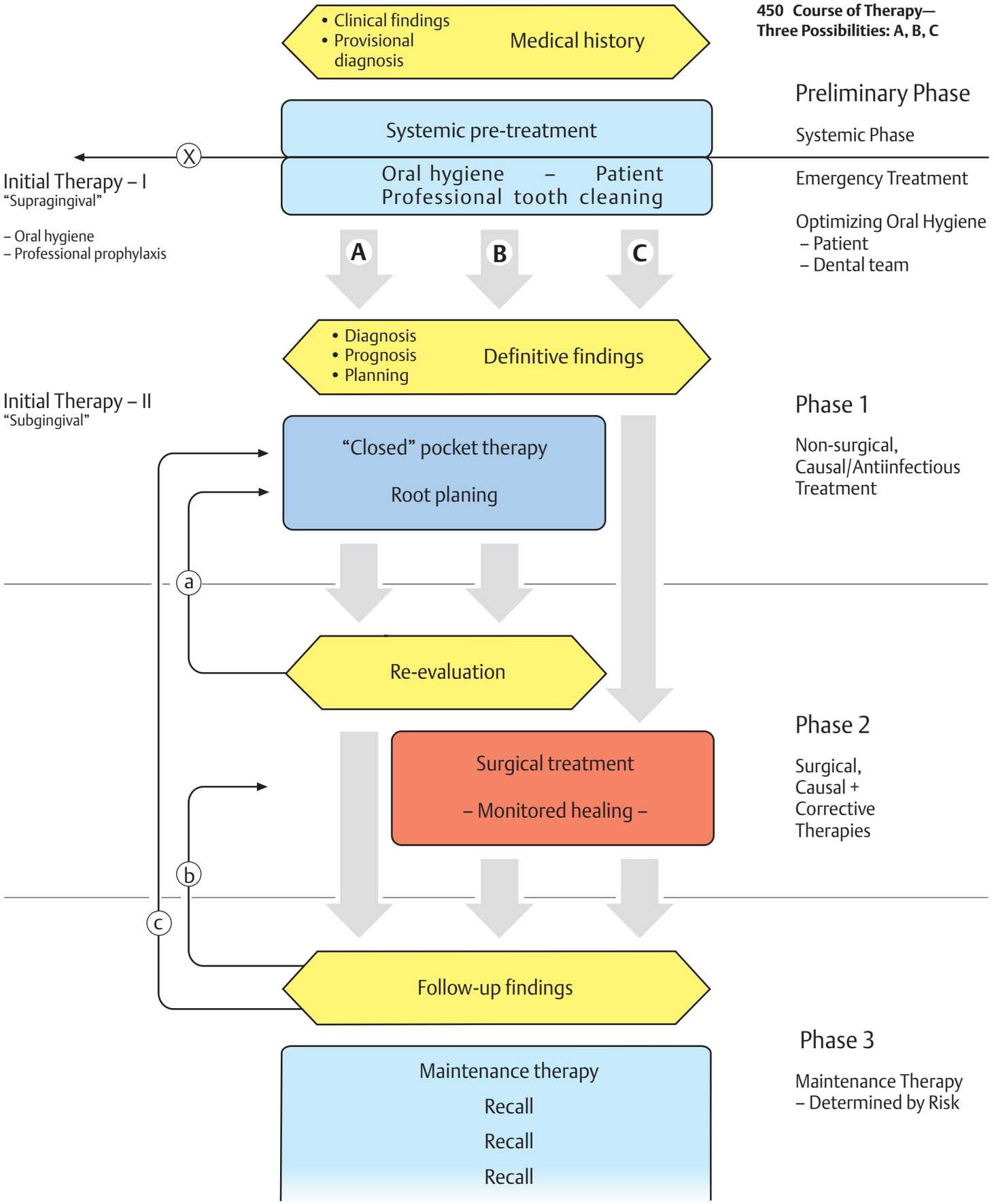
Phase 2—Surgical, Corrective

Following Phase 1 therapy that includes closed, subgingival root debridement, a *reevaluation* must always be performed. If the treatment result is not satisfactory, individual root surfaces may again be treated with closed therapy (a). If deep pockets persist and if root fusion, grooves, furcation involvement etc. are present, these must be surgically addressed.
If the goal is to *correct* periodontal defects, following the pre-phase, surgical-corrective procedures are indicated. Tissue shrinkage should be avoided at all costs so that the surgical site will be optimally covered with gingival tissue following eventual application of restorative materials and/or membranes.
Most important is that the healing after surgical therapy must be continuously and professionally *monitored*, i. e., the operated area must be maintained supragingivally plaque-free as much as possible.

Phase 3—Preventive, Anti-infectious, "Life Long"

After conclusion of active periodontitis therapy—regardless of the type of treatment—*check-up findings* are collected 2–3 months later. If the therapy is deemed to be successful, the patient should be started on a regular recall (organized maintenance therapy; p. 449).
However, if individual sites exhibit problems (residual pockets, bleeding), the sites must be re-treated either closed (c) or surgically (b).
In such situations, the use of topical slow-release medicaments may be indicated (p. 293).

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General Course of Therapy—Individual Planning

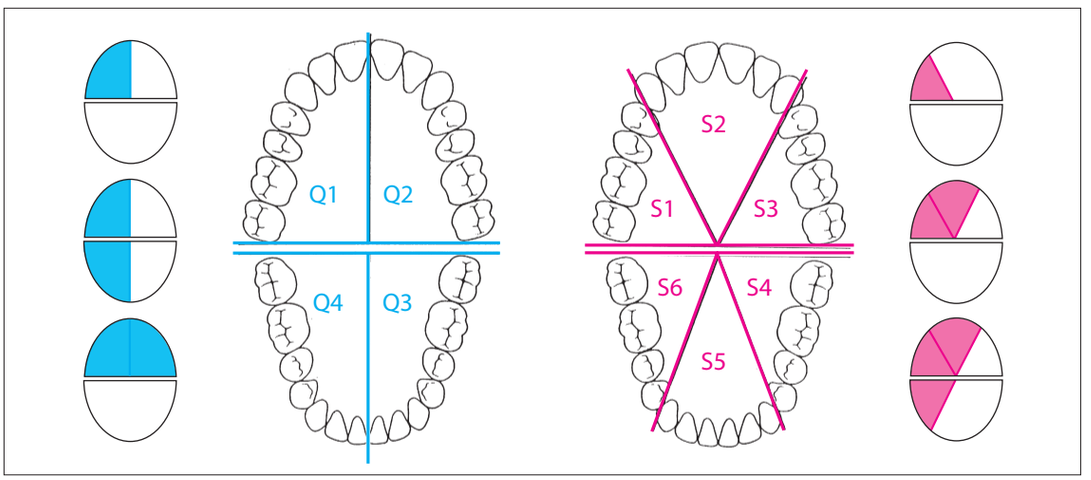
In the preceding pages the basic treatment planning and the course of therapy for patients with gingivitis and/or periodontitis were presented. In general, such treatment is performed according to the "phase" plan (phases of therapy; pp. 208–9).

Treatment, healing times and "pauses" for consideration of the patient and his/her oral hygiene will differ dramatically. Nevertheless, following each and every phase of treatment, a *re-evaluation* of the case must be performed: The treatment result and the planned further procedures must be reconsidered, and adapted as necessary to the new situation.

Causal therapy—Traditional Clinical Course

Phase 1 therapy consists of the exquisitely careful debridement of subgingival root surfaces in all periodontal pockets. For decades, this procedure has been performed *quadrant-by-quadrant* (Badersten et al. 1981, 1984; p. 280). The interval between individual appointments normally is one or two weeks. The quadrant scaling immediately follows the so-called "hygiene phase," during which the patient is educated and trained to perform adequate plaque control at home.

451 Quadrants—Sextants
Traditional mechanical/instrumental pocket treatment is usually performed at separate appointments for *quadrants* (**Q1–Q4**) or *sextants* (**S1–S6**); in mild cases, the left and right arch segments can be treated at one appointment.
The time required for each appointment will be determined by the severity of the case; appointments for such Phase 1 therapy are usually scheduled at 1–2 week intervals.



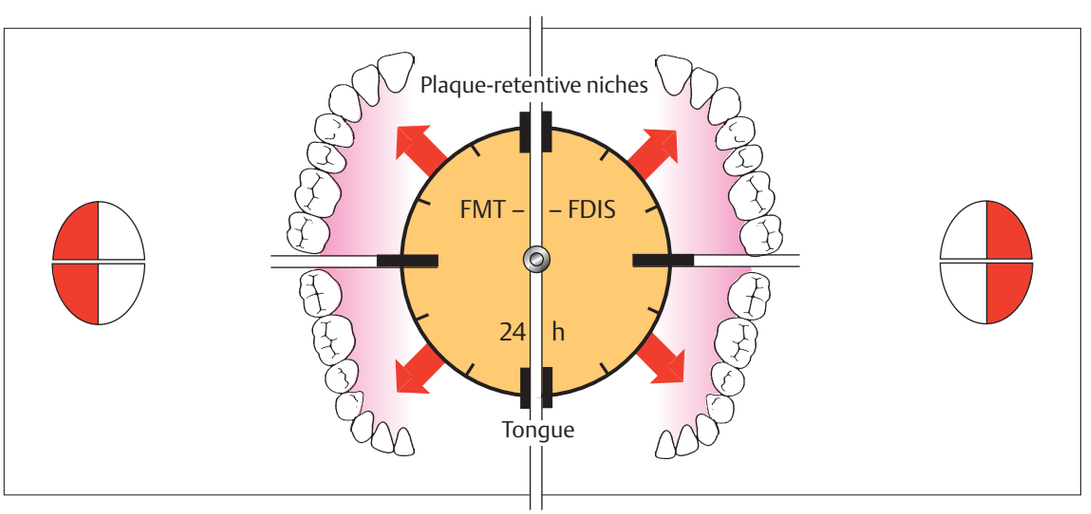
Causal Therapy—"Full mouth therapy" (FMT)

FMT represents an effort to synthesize all new knowledge from recent years. FMT is a consequential, anti-infectious approach, comprised of the following: Initial therapy includes an extended, systematic anti-microbial treatment, initially involving only supragingival pretreatment of the dentition (mechanical) and all oral niches (above all, the tongue). Only after achieving the prescribed clinical condition (p. 287), subgingival debridement (mechanically and with use of topical medicaments) is performed in the shortest possible time; in "extreme cases," within a 24-hour period. This procedure is appreciated by most patients!

During this short-term but intensive treatment, disinfection agents such as chlorhexidine (CHX; p. 235), betadine or even oxidizing NaOCl ("bleach") are used to rinse the mouth and the individual pockets. The goal is to prohibit re-colonization and re-infection of even shallow pockets (p. 256).

Because significantly better results have been achieved using this type of closed periodontitis therapy (p. 285), in contrast to the traditional "quadrant-by-quadrant" technique, it is very likely that the FMT method will become more widespread. The necessity for surgical intervention may be reduced and the comprehensive treatment of even advanced cases may become financially more feasible

452 "Full Mouth Therapy—FMT", "Full Mouth Disinfection—FDIS"
This new type of treatment using closed therapy is performed in the shortest possible time, preferably in only two appointments, e. g., for the left and the right sides.
Intensive pretreatment and optimum oral hygiene usually eliminate shallow pockets so that only the few remaining deeper pockets must be treated, using anesthesia (FMT, p. 281).



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Systemic Pre-phase

- **Systemic problems**
- **Systemic risks**

The purpose of the systemic pre-phase is to protect both the patient and the clinician by ascertaining any general systemic risks associated with the patient.

It is critically important that *infectious*, *above all viral diseases* (Herpes, Hepatitis B and C, HIV infection) be detected and/or diagnosed: Every patient may harbor such diseases! It is therefore necessary to employ the usual hygiene measures for all dental examinations and treatments, e.g., gloves, mask and protective eyewear.

Individuals with severe systemic diseases (see ASA Classification, p. 212) can seldom be treated periodontally under the rubric "comprehensive therapy." Usually these patients can only be treated with limited "emergency" measures, and with the participation of the patient's physician.

Special precautionary measures are indicated with patients who suffer from multiple maladies, and most particularly with patients who are susceptible to the *life-threatening* danger of infectious endocarditis (Reichart & Philipsen 1999).

With *non-life-threatening* diseases, dental therapy should be planned in collaboration with the physician or internist, who may prescribe appropriate medications (polypharmacy); these should be checked for possible drug interactions with other agents prescribed by the dentist, as well as the possibility of undesired adverse effects (cf., gingival hyperplasia; p. 12).

Thanks to modern medicine, many of our patients lead the normal life of a *healthy* person. The possibility of injuring such patients during dental treatment must be ruled out (allergies, anticoagulants, hypertension, hypercholesterolemia).

Genetic and hereditary risks must be assessed, and decisions made concerning the individual patient's treatability or non-treatability (uncontrolled Diabetes mellitus; smokers).

This chapter presents the following discussions:

- The patient—ASA classification
- Cardiovascular diseases—"blood thinning"
- Bacteremia—prevention of infectious endocarditis
- Endocarditis prevention—antibiotics
- Diabetes mellitus—risk factor for periodontitis
- For the smoking risk factor—information, tobacco cessation program

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Evaluation—Can the Patient be Safely Treated?

Before initiating any dental treatment, the relevant medical history provided by a "new" patient must be carefully checked, regardless of whether comprehensive therapy is anticipated or only an emergency procedure. Of particular importance are serious diseases or conditions, such as:

- Cardiovascular diseases
- Pulmonary diseases
- Renal diseases
- Endocrine diseases
- Compromised immune response
- Psychological/psychiatric conditions

Acute situations should be thoroughly discussed before treatment; these include allergies, anaphylactic reactions, but also patient fear of the treatment or even fear of the injection needle.

The dental team must be systematically prepared for emergency situations. Corresponding checklists, supplies and devices must be at hand (emergency kit, materials for cardiopulmonary resuscitation, and perhaps even a defibrillator).

The classification by the American Society of Anesthesiologists (ASA) helps to establish the physical status of a diseased patient (ASA Classes I–VI; Fig. 453)

453 ASA Classification of Patients—Health Status
Normally, only patients in Classes I and II are treated in the private dental practice, and in rare cases Class III. In the latter case, active collaboration and cooperation with the patient's treating physician is highly recommended.

ASA Class	Patient description — Classification criteria
I	Normal, *healthy* patient *without* systemic disease
II	Patient with *mild* systemic disease
III	Patient with *severe* systemic disease, which limits her/his activity but is not life-threatening
IV	Patient with a *severe* systemic disease that is constantly life-threatening
V	*Moribund* patient, who is not expected to live beyond 24 hours with or without operation
VI	*Brain-dead* patient whose organs may be harvested for transplant
E	Emergency patient — This category is re-defined, according to the clinical condition, in Grades I – IV (e. g., ASA III – E)

Medical Risk Factor—"Blood Thinning"

Patients with cardiac and circulatory diseases (post-myocardial infarction, Angina pectoris etc.) or other conditions (e. g., post-surgical condition, dialysis patients, thrombosis prophylaxis etc.) usually take *anticoagulants*:

- Short-term therapy: Heparin
- Long-term prophylaxis: Aspirin derivatives
- Long-term therapy: Cumarin derivatives (e. g., Warfarin)

In order to avoid life-threatening hemorrhage, the patient's actual "Quick-time" (Cumarin) must be assessed. A Quick value of ≥ 30% usually does not affect dental or oral surgical procedures, but values between 15 and 25 % demand consultation with the treating physician.

- The effect of blood-thinning medications is *enhanced* by non-steroidal anti-inflammatory agents such as salicylate, mefenamine acids, tetracycline, metronidazol and sulfonamides (Scully & Wolff 2002).
- Their effects will be *reduced* by barbiturates, glucocorticoids, alcohol, and foodstuffs with a high vitamin-K content.

The antidote for Cumarin is vitamin K; there is no antidote for the rapidly metabolized heparin. If necessary the patient may stop taking this medication for a short period of time.

454 Coagulation—Tests of Blood Thinning
Because the results vary greatly, the old Quick-Test will be abandoned in the near future.
It will be replaced by the INR blood thinning test ("International normalized ratio") which provides constant values and ease of use by the patient!

Therapeutic bandwidth:
INR values of **2.5–4.5**, depending upon the risk status of the patient.

Coagulation test	INR	% Quick	Seconds
Measurement boundaries	1.2	70	13.2
	1.4	50	14.2
	1.6	40	15.6
	1.9	30	16.6
	2.1	25	17.4
Therapeutic band width	2.5	20	19.0
	3.0	16	20.8
	3.5	13	22.5
	4.0	11	24.0
	4.5	10	25.5
	5.0	9	26.9
Measurement boundaries	8.0	5	34.0

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Bacteremia—Endocarditis Prophylaxis

Transient bacteremia is a natural, daily occurring situation (chewing, tooth brushing). In healthy persons, oral bacteria that enter the blood stream are efficiently eliminated by the host defense system.

Infectious endocarditis (IE) is a life-threatening disease, an infection of hemodynamically exposed defects (plaque formation on heart valves), usually elicited by oral microorganisms (streptococci).

Depending upon the virulence of the etiologic microbe and the resistance of the patient, various forms of IE can be differentiated (Müller 2001):

- *Acute, infectious forms*
  Sepsis, fever, endocardial destruction;
  death in less than 6 weeks
- *Acute/Subacute forms*
  Intermediate forms, often elicited by enterococci
- *Subacute forms*
  Slight fever; if untreated,
  death between 6 weeks and 3 months
- *Chronic forms*
  Symptoms the same as subacute;
  death in more than 3 months



Indications for endocarditis prophylaxis

Heart failure and post-operative findings

High risk for endocarditis
- Condition following biological or mechanical heart valve replacement
- Condition following infectious endocarditis, in the absence of cardiac pathology

Moderate risk for endocarditis
- Inherited or genetic heart valve insufficiency
- Inherited cardiac defects, e. g.
  - Aortic isthmus stenosis
  - Ductus Botalli apertus
  - Ventricular septal defect
  - Sub- or supra-valvular aortic stenosis
  - Cyanotic vitium
- Condition following palliative operation for genetic heart failure
- Incompletely corrected genetic cardiac failure
  Hypertrophic, obstructive cardiomyopathy (HOCM)
- Mitral valve prolapse (MVP) with systolic sounds

No elevated risk of endocarditis
- Auricular septal defect
- Condition following successful closure of an auricular or ventricular septal defect
- Condition following successful coronary bypass surgery
- Mitral valve prolapse without systolic sounds
- Physiologic, functional or otherwise harmless heart sounds
- Previous Kawasaki disease without valvular dysfunction
- Previous rheumatic fever without valvular dysfunction
- Condition following implantation of a pacemaker
- Condition following surgery for aortic isthmus stenosis

455 Heart Diseases and Cardiac Defects—Indications for Endocarditis Prophylaxis

Within the new wording of the prophylaxis program from the AHA (American Heart Association; Dajani et al. 1997) the risk structure of the heart was newly categorized in three groups:
- **High risk (red)**
- **Moderate risk (green)**
- **Non-elevated risk**

Of importance for the dentist is that the presence of a cardiac pacemaker is not an indication for antibiotic, pre-treatment prophylaxis (but care must be exercised when electronic instruments such as ultrasonic scalers are employed).

Left: Individual patient cards from the Swiss Heart Foundation, listing guidelines and dosages for endocarditis prophylaxis.

- High risk
  red → adults
  yellow → children
- *Moderate risk*
  green → adult
  blue → children

Infectious endocarditis (IE)

A wide variety of microorganisms such as bacteria, mycoplasm, fungi, rickettsia or chlamydia can elicit IE if they enter the bloodstream due to trauma or tissue manipulation. Regions of the cardiovascular system that experience slow blood circulation or a high level of turbulence are particularly susceptible to infections.

The most frequent source of microorganisms that elicit IE is the oral cavity. The primary pathogens are gram-positive streptococci (*viridans* type), especially *Streptococcus sanguis*.

In addition to *S. aureus* and *S. epidermis*, more and more often one also observes gram-negative bacteria from the oral cavity and the upper respiratory tract that elicit IE; for example, *A. actinomycetemcomitans, Hemophilus ssp., Cardiobacterium ssp., Eikenella corrodens, Kingella ssp., Capnocytophaga, Neisseria ssp.*

For the protection of IE-endangered patients, bacteriocidal antibiotics of the “penicillin type” (p. 214) are recommended. As early as 1983, J. Slots and others suggested that IE prophylaxis could also include metronidazol (see Medicaments, p. 287).

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Endocarditis Prophylaxis with Antibiotics

According to new guidelines from AHA (Dajani et al. 1997), the prophylactic dose of standard antibiotic (*Amoxicillin*) was reduced to 2 grams; in addition, no follow-up dose is now recommended; not all other Heart Associations agree.

It is comforting for dentists to know that most of the, albeit rare, cases of endocarditis do not result from invasive, surgical treatments! Nevertheless: Oral, especially periodontal surgical procedures take place in a highly contaminated area. Expansive and/or deep surgical procedures have almost always been performed with antibiotic prophylaxis. For this reason, recommendations concerning which dental procedures require endocarditis prophylaxis were welcomed (Newman & Winkelhoff 2001).

456 Endocarditis Prophylaxis
The standard antibiotic for IE prophylaxis is *Amoxicillin*.

For patients who are allergic to this bacteriocidal broad spectrum penicillin, and/or those who cannot swallow pills, some alternatives are provided.

* Maximum children's dose, depending upon body weight; do not exceed the adult dose!
** Cephalosporine and penicillin must not be used with Type 1 hypersensitivity!

Endocarditis prophylaxis – AHA (American Heart Association)

Patient	Antibiotic	Dose Adult	Dose Child*	Ingestion before dental procedure
Standard prophylaxis	Amoxicillin	2 g oral	50 mg/kg oral	1 h before
Unable to swallow pills	Ampicillin	2 g i.m./i.v.	50 mg/kg i.m./i.v.	30 min before
Penicillin allergy	Clindamycin	600 mg oral	20 mg/kg oral	1 h before
	Cephalexin** Cefradoxil**	2 g oral	50 mg/kg oral	1 h before
	Azithromycin Clarithromycin	500 mg oral	15 mg/kg oral	1 h before
Penicillin allergy *and* unable to swallow pills	Clindamycin	600 mg i.v.	20 mg/kg oral	30 min before
	Cefalozin	1 g i.m./i.v.	25 mg/kg oral	30 min before

Dental Procedures Carrying the Risk of Bacteremia

Bacteriemia will occur following all dental procedures that elicit *bleeding*. The endangered patient must be premedicated with the "one-shot" prophylaxis (AHA) before:

- Tooth extraction, surgical tooth extraction
- Root tip resection
- Suture removal, dressing change
- Calculus removal
- Intraligamentous anesthesia
- Periodontal probing (Fig. 457 left).

Bacteriemias of varying frequency and severity also occur after chewing hard foodstuffs (15–50 %), during *tooth brushing* (5–25 %), or during oral irrigation (25–40 %; according to Neu 1994).

The percentage and number *anaerobic* species were approximately twice as high in patients with poor oral hygiene and advanced periodontal diseases in comparison to patients with good oral hygiene. Bacteriemia occurs in these patients also, but patients at risk for endocarditis should not abandon mechanical oral hygiene; rather, they should rinse 30 minutes beforehand with chlorhexidine.

457 Dental Procedures Requiring Antibiotic Endocarditis Prophylaxis (E-P)
For *single* procedures the prophylaxis measures suggested above are sufficient.

If, however, an extended phase of treatment is planned, a longer-lasting (adjunctive) medication must be considered, combined with an intensive intraoral antiseptic regimen (p. 287).

Dental procedures: E-P *recommended*

Anesthesia – intraligamentous injection

Surgery – Tooth extraction, further procedures

Periodontology – Probing, scaling and root planing, surgery, recall therapy, placing subgingival medicaments

Dental implants – Seating intraosseous implants

Endodontics – Canal instrumentation or surgery beyond the apex

Professional tooth cleaning procedures if hemorrhage is anticipated

E-P: *not recommended***

Local injection anesthesia (excluding intraligamentous)

Operative and prosthetic dentistry – with or without retraction cords

Endodontics: Root canal treatment, post build-ups etc.

Various: Rubber dam placement, suture removal, impression-taking, fluoride application, radiographs, adjustment of fixed orthodontic appliances

Removal of highly mobile deciduous teeth

* E-P coverage if hemorrhage is anticipated

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Diabetes mellitus (DM)—Risk Factor for Periodontitis

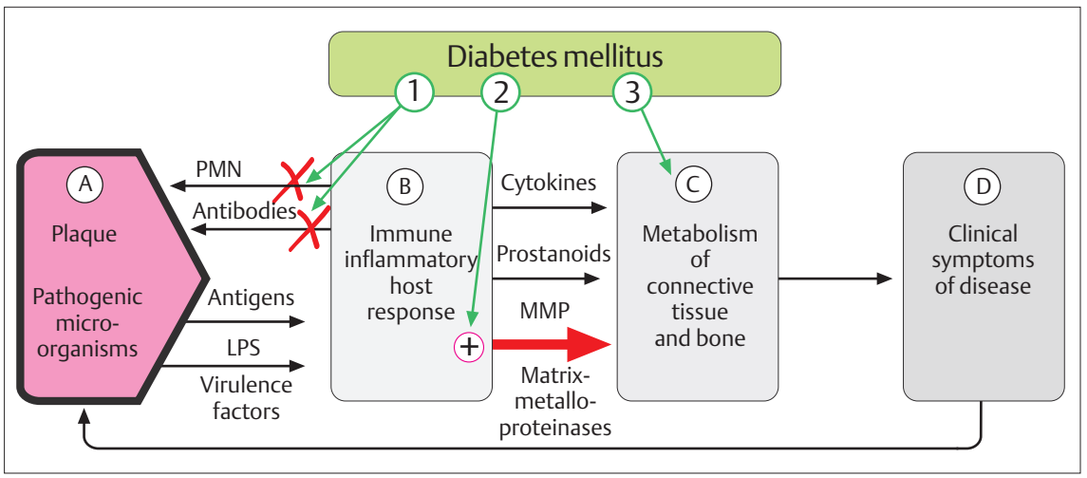
A new classification for Diabetes mellitus was presented in 1997 (WHO; American Diabetes Association):

- Diabetes mellitus Type 1 (formerly IDDM)
- Diabetes mellitus Type 2 (formerly NIDDM)
- Other diabetes with known etiologies
- Gestation Diabetes (pregnancy Diabetes)

Untreated, all forms of the disease are similar in the elevated blood sugar level and alterations in carbohydrate and lipid metabolism (DGP 2002/Risk factors).

- The less common DM Type 1 occurs due to the autoimmune-elicited destruction of insulin-producing β-cells of the pancreas. The consequence is acute insulin deficiency.
- DM Type 2 diseases are not primarily insulin-dependent (over eating, sedentary lifestyle!); Type 2 Diabetes today has achieved epidemic proportions. The number of cases is increasing rapidly, and is estimated at 150,000,000 cases worldwide.

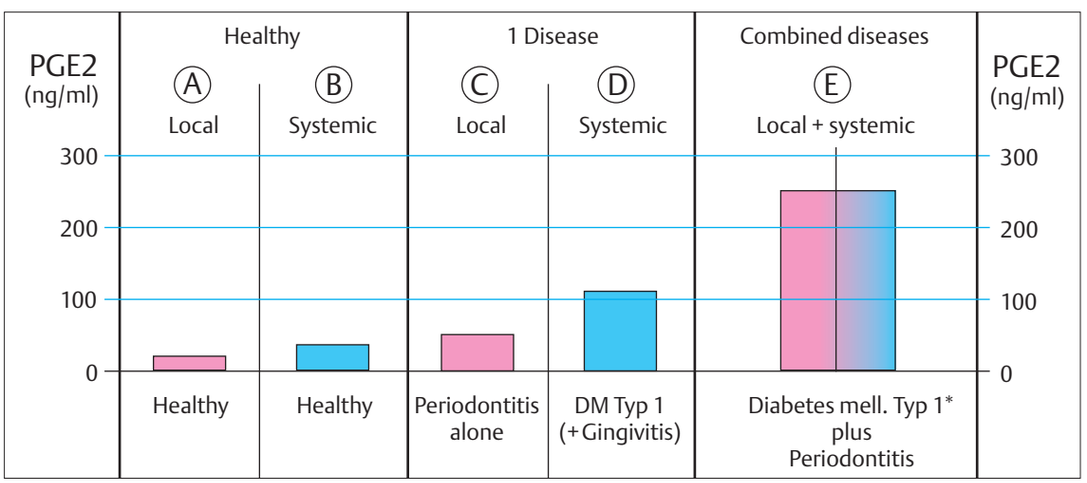
  DM today is frequently one component of the "metabolic syndrome," together with obesity, elevated blood lipid and high blood pressure ("deadly quartet"; syndrome X).



458 Risk Factor—DM
The effects of persistent hyperglycemia are many. Various target cells react inappropriately to glycosylated lipids and proteins (AGE, "advanced glycated end-products"), mediated via the specific receptor RAGE.

1 Defective PMNs and antibodies
2 Macrophages with enhanced catabolic mechanisms
3 Altered extracellular matrix

Adapted from *R. Page 1998*



459 Differences in Host Response in Health, Type 1 Diabetes and Periodontitis Patients
The levels of the strong pro-inflammatory mediator prostaglandin E2 (PGE2; p. 49) differ massively.

In patients with Diabetes and periodontitis (**E**), an extreme overproduction of PGE2 is observed, which complicates the disease picture.

Adapted from *G. Salvi et al. 1998*

Periodontitis Therapy for Diabetes Patients

An important first step is the meticulous anti-infectious initial therapy and the establishment of an optimum blood sugar level (internist). Persistence or progression of periodontitis can lead to elevated insulin resistance, and can increase the diabetic consequences:

- Retinopathy (vascular damage, p. 133; blindness)
- Nephropathy (renal high pressure as a consequence)
- Neuropathy
- Angiopathy (atherosclerosis—peripheral, heart, brain)
- Disturbances of wound healing
- Severity of periodontitis (the "sixth complication")

Periodontitis and Diabetes mellitus exhibit numerous reciprocal effects. Both can therefore be referred to as reciprocal risk factors. Chronic infection of the periodontal pocket (gram-negative microorganisms, lipopolysaccharide) elicit within the altered glycosylating cells of a diabetic an increase in excessive reactions of the defense cells, which is characterized by release of large amounts of pro-inflammatory, catabolic mediators (Grossi & Genco 1998). Diabetes and periodontitis should therefore be treated simultaneously, where necessary employing systemic antibiotic support (doxycycline) (Miller et al. 1992; Westfelt et al. 1996; Tervonen & Karjalainen 1997).

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Smoking—An Alterable Risk Factor

Because of its numerous negative consequences for general systemic health, regular heavy smoking is one of the most dangerous addictions. It is also the most significant *alterable* risk factor for periodontal diseases.

In addition to *nicotine*, tobacco smoke contains up to 4,000 toxic, cancer-causing components. Therefore, a *tumor-screening* examination should be performed on all patients who smoke, including meticulous inspection of the entire oral cavity (Reichart 2002, Reichart & Philipsen 1999).

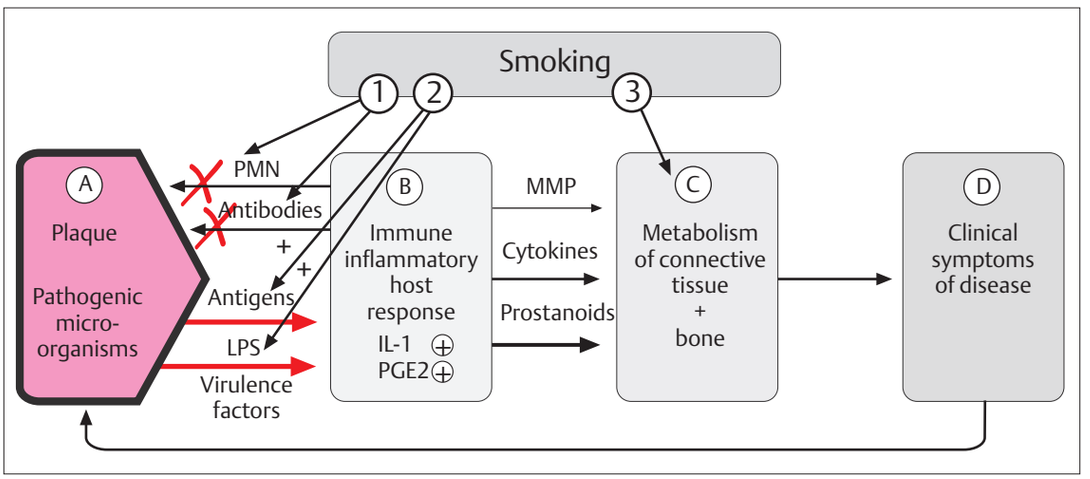
Pathogenesis: Nicotine and its by-products compromise the host response to infection, including within the periodontal tissues (Müller 2001):

- Reduced chemotaxis and phagocytosis by PMNs
- Reduced synthesis of immunoglobulins (IgG2)
- Stimulation of pro-inflammatory cytokines and additional mediators (IL-2, IL-6, PGE2)
- Elevated numbers of anaerobes in the subgingival region, including *T. forsythensis, P. gingivalis*, and also members of the "orange complex" (Haffajee & Socransky 2001)
- Damage to fibroblasts (gingival and periodontal)

460 Risk Factor—Smoking
Where and how does tobacco smoking influence the pathogenesis of periodontal diseases?

1 Activity of PMNs, reduced secretion of immunoglobulins (IgG2)
2 Enhancement of subgingival anaerobic microorganisms
3 Influence on gingival, periodontal and osseous metabolism

Adapted from *R. Page & K. Kornman 1997*



461 Smoking Cessation Using Nicotine Substitutes
"Nicorette" products; from left to right:

- Microtabs: Sublingual tablets
- Inhaler: Cartridges within a cigarette mouthpiece
- Transdermal patch: For 16 hours (5, 10 and 15 mg)
- Nicorette chewing gum

The Nicorette products are available in differing concentrations and in some instances different taste formulations.



Smoking Cessation

The dental team sees the patient in recall at least twice per year and is therefore almost predestined to accompany the tobacco cessation process. Treatment results in smokers with aggressive periodontitis and poor oral hygiene are definitively poorer, on average, than in non-smokers. Caution is the watchword before regenerative procedures, e.g., filling bony defects, whether on natural teeth or dental implants. In the face of additional risk factors (oral hygiene, Diabetes, positive IL-1 polymorphism etc.) one is wise to avoid regenerative procedures of any kind (Tonetti et al. 1995, Müller et al. 2002, Jansson et al. 2002, Machtei et al. 2003).

If the smoking cessation program is to be effective, the time factor, and uncompromising patience, will play major roles. Motivational discussions about smoking cessation often follow the "five A's": Ask, advise, assess, assist, arrange (Ramseier 2003).

Cessation programs including the various forms of nicotine replacement substances (Fig. 461) have proven effective. Without nicotine, the medicament "Zyban" (bupropion) is effective.

Emergency Treatment

Many periodontitis patients are not aware of their disease, even though it may have been progressing over many years. Only when pain and acute inflammatory symptoms appear do such patients seek out a dentist.
Such emergency cases must be treated immediately. However, to avoid life-threatening incidents, a succinct *general medical history* must be taken, with particular attention to any medicines the patient may be taking (anticoagulants!) and an assessment of the necessity for infection prophylaxis (endocarditis, HIV etc.), as well as allergies and previous significant incidents.
Next, a clinical and radiographic examination should be performed for emergency patients; despite the pain, this is absolutely necessary before any treatment.
Included in the category "periodontal emergency situations and treatments" are:

- Initial topical medicinal and mechanical treatment for acute NUG
- Treatment of acute, suppurating pockets
- Opening periodontal abscesses
- Immediate extraction of hopelessly mobile teeth that cannot be maintained
- Acute, combined endodontic-periodontal problems
- Treatment of periodontal trauma following accidents

Acute ulcerative gingivoperiodontitis (acute NUG/NUP) is painful and progresses very rapidly. Careful instrumentation and application of topical agents generally bring relief within a few hours and a reduction of the acute situation.
Caution: Ulceration may be a symptom of HIV-seropositivity (opportunistic infection).

Active suppurating pockets generally are not painful if drainage is established at the gingival margin (exception: abscess). Such pockets represent an exacerbating inflammatory process, which leads to rapid attachment loss. They must be treated immediately with application of rinsing solutions or ointments; mechanical cleansing must also be initiated.

Periodontal abscesses are usually very painful. They must be drained immediately. This can usually be accomplished via probing from the gingival sulcus.
In the case of molars with deep pockets or furcation involvement, an abscess that penetrates the bone may develop subperiosteally. These cannot always be reached via the gingival margin, and must be drained by means of an incision.

Immediate extraction should be reserved for teeth that cannot be maintained or are highly mobile or which cause the patient undue discomfort. In the case of anterior teeth, for esthetic reasons, extractions should be avoided when possible, or an immediate temporary should be prepared.

Acute, endodontic/periodontal processes have a more favorable prognosis if the primary problem is of endodontic origin. The root canal should always be treated first, subsequently the pocket (pp. 445–447).

Periodontal trauma due to accident usually requires immediate splinting (following any necessary reimplantation or repositioning of the tooth).

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462 Emergency Situation: Acute Necrotizing Ulcerative Gingivitis (NUG)
The severe pain in the acute stage permits only a very careful peripheral attempt at cleansing. Treatment of this acute condition involved gentle debridement with 3 % hydrogen peroxide, and application of a disinfecting ointment containing anti-inflammatory and analgesic ingredients. The patient was told to rinse at home with a chlorhexidine solution.



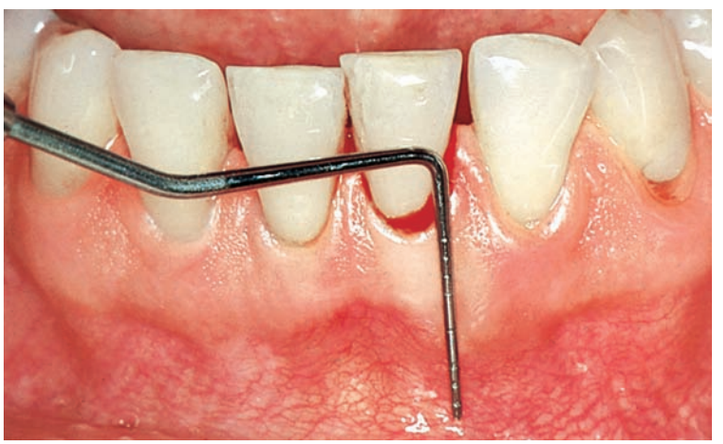
Acute “necrotizing ulcerative gingivitis” (NUG)

463 After Emergency Treatment—Subacute Stage
Several days after gentle topical application of the medicaments and careful mechanical debridement, the signs of active NUG—especially the pain –subsided. Treatment by means of systematic subgingival scaling can now proceed. A gingivoplasty may be indicated subsequently in the normal course of treatment.



464 Emergency Situation—Localized Acute Pocket
Tooth 31 is vital and should be maintained despite the 10 mm pocket. Very little pus has formed; drainage via the gingival margin appears possible. The tooth is slightly percussion sensitive. Prior to systematic mechanical treatment, the tooth is treated on an emergency basis with topical application of a medicament, and the pocket is disinfected.

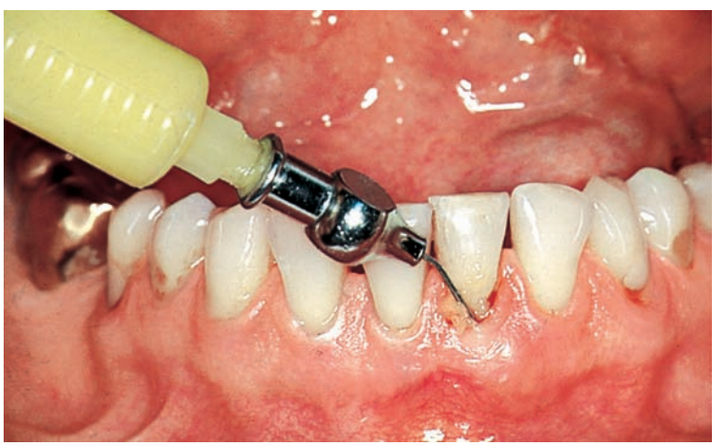
Right: Note the deep defect on the distal of tooth 31.





“Acute pocket”—acute pocket inflammation

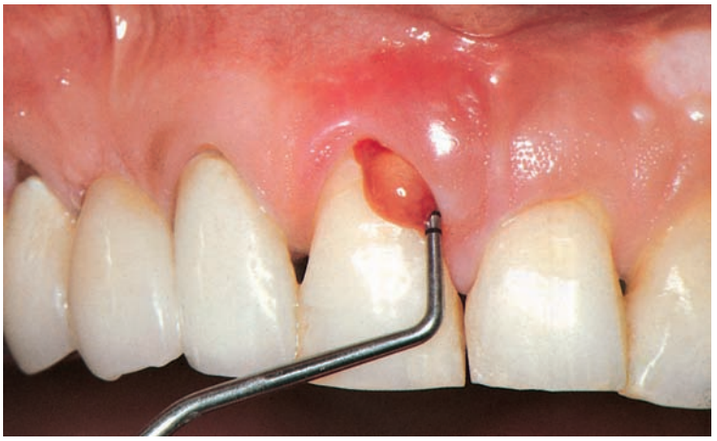
465 Emergency Treatment Using Local Medicament, and Follow-up
As an emergency measure the pocket was first rinsed thoroughly with chlorhexidine solution and then filled with achromycin ointment (3 %). Once the acute symptoms subside, a thorough root planing can be performed.
Right: Eight weeks after the emergency therapy, the gingiva has regained its resiliency and has shrunk somewhat. Probing depth is now only ca. 3 mm.





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466 Emergency Situation: Pocket Abscess—Drainage after Probing From the Gingival Margin
Originating from a deep pocket mesial to tooth 11, a periodontal abscess has formed. Copious pus exudes when the pocket is probed.

Left: The radiograph depicts the periodontal probe inserted to the base of the osseous defect.

Pocket abscess





467 Emergency Treatment Using Topical Medicament—Radiographic Follow-up
The abscess has opened via the gingival margin. The pocket is first thoroughly rinsed, then filled with an antibiotic-containing ointment. Once the acute symptoms have subsided, definitive therapy can be undertaken.

Left: Radiograph 6 months after definitive therapy: New bone formation is apparent.



468 Emergency Situation: Periodontal Abscess Anticipates Drainage Through the Gingiva
Originating from the deep, one-wall infrabony pocket mesial to the tipped but vital tooth 47, an abscess has developed. The buccal gingiva is distended as the abscess is about to penetrate through the mucosa.
Tooth 47 is an abutment for a removable partial denture that is ill-fitting, but the patient wants to retain the partial denture.

Periodontal abscess





469 Abscess—Drainage
As soon as the mucosa was touched the abscess opened and copious pus exuded.

Left: In the radiograph one observes the deep mesial periodontal pocket with a hoe scaler *in situ*. Since the furcation appears not to be involved, it is possible to consider maintaining this tooth. The ensuing treatment included mechanical debridement and topical application of medicaments.

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470 Emergency Situation in the Posterior Segment: Hopeless Molar (37)
Pus exudes spontaneously from the deep distal pocket and the buccal furcation of tooth 37.

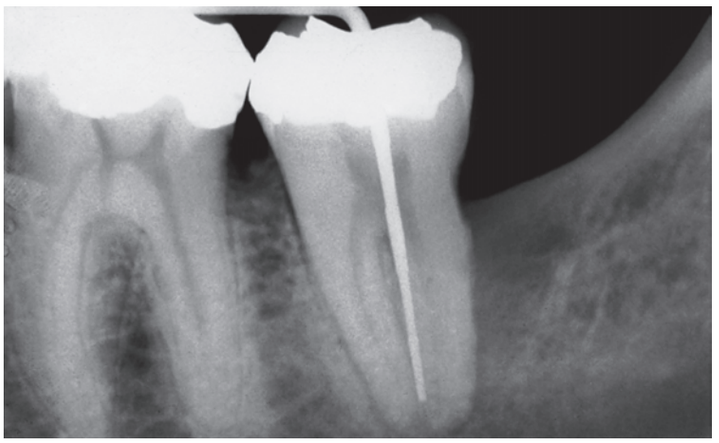
The tooth is vital, highly mobile and painful to the slightest touch.



Abscess—distal pocket/furcation

471 Radiograph of 37 Before Immediate Extraction
The periodontal probe can be inserted almost to the root apex in the deep buccal pocket. Without clinical probing, such a defect at this location would be almost impossible to detect. The shape of the furcation is very unfavorable in terms of treatment; the two roots appear to fuse apically.

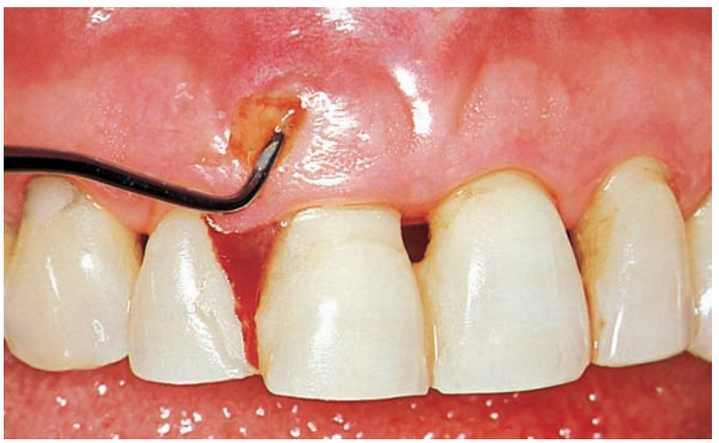
Right: Highly infiltrated granulation tissue remains attached to the root after extraction.

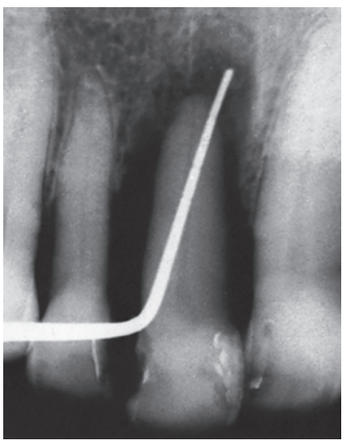




472 Emergency Situation in the Anterior Area: Painful Tooth 11 is Hopeless—Fistula
A fistula emanating from the deep pocket has developed. The non-vital tooth is highly mobile and sensitive to percussion. This tooth cannot be saved; it was extracted immediately.

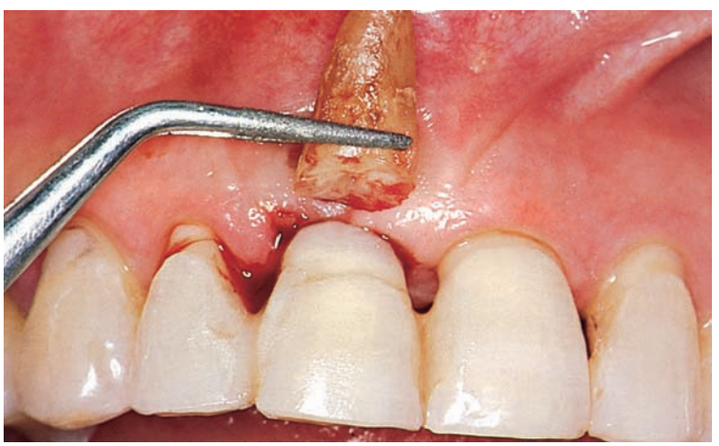
Right: The radiograph reveals that a probe can be carefully inserted far beyond the apex (endo-perio problem, p. 445).

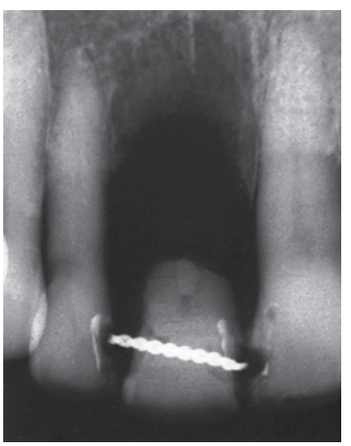




Acute perio-endo problem—fistula

473 Immediate Extraction—Immediate Temporary
An immediate temporary is necessary for esthetics. Following extraction the root was severed and the crown was used as a temporary. A wire and acid-etched resin secured the crown to the adjacent teeth. This type of temporary can usually be maintained until definitive reconstruction.
Right: Radiographic view of the temporary replacement consisting of the patient's own tooth crown.





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Phase 1 Therapy

**Causal, Antimicrobial,
Non-surgical Therapy**

Primary prevention—the *early* prevention of disease—is initially the responsibility of parents. Using simple, non-traumatic tooth brushing methods, the small child is gently guided down the path of personal hygiene and especially oral hygiene, hopefully with the support of his/her role models: Mother, father and perhaps older siblings. It should become a habit that the results of a child's own efforts in oral hygiene be observed, lauded and, when necessary, modified.
In *healthy* individuals, prophylactic measures are usually short, painless and require little time. Nevertheless, they prevent dental caries and gingival diseases!

If disease occurs over the course of time, e. g., initial gingivitis in the specialty of periodontology, a brief description of the cause (bacterial biofilm), and its professional removal, as well as renewed instruction in plaque control by the patient (tooth brushing, oral hygiene), healthy conditions can be again established—*secondary prevention*.

If true pockets have formed and attachment has been lost, it is necessary to intervene as early as possible for such cases of early *periodontitis*. Using the simple measures of closed, causal therapy (debridement, root planing) such cases can be *cured*. Only a few years ago, predictable treatment success was only possible in pockets of 4–6 mm; but today it is possible to achieve predictable success even in pockets of 8 mm and more with the help of new and improved procedures ("After Five" curettes, disinfectants, modified timing, and other equipment innovations).

Nevertheless, anatomical relationships such as furcation involvement, grooves, narrow bony craters, and the regeneration of tissue defects oftentimes make it necessary still today to employ a multifaceted, often complicated corrective (surgical) technique with open therapy (Phase 2 therapy/surgery; p. 295) following Phase 1 (closed, subgingival) treatments.

This chapter, "Phase 1 Therapy," will unfold as follows:

- Case presentation – Motivation toward self-help
- Initial treatment 1 – Oral hygiene by the patient
  – Creation of hygienic relationships by the dental team
- Initial treatment 2 – Traditional closed pocket treatment
  – FMT—"full mouth therapy"

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Case presentation—Motivation—Information

The maintenance or reinstatement of periodontal health (freedom from inflammation, full function) is possible even in terms of esthetics assuming certain prerequisites. But this can only be accomplished through the *cooperative* interaction between the patient and the dentist: The patient must be interested in maintaining the health of the oral cavity, and must be *interested* in a proposed treatment, and must be *motivated* to participate (compliance).

The patient should first be informed about the causal relationships that led to the disease process. The dental team has numerous possibilities to demonstrate to the patient soft tissue alterations elicited by inflammation, and the responsible etiologic factors (Roulet & Zimmer 2003).



474 Motivation and Information During the Case Presentation
During the very first appointment, during the discussion of medical history and the collection of diagnostic data (here by the dental hygienist), the patient will want to be informed as much as possible about his/her oral condition.
This is an opportunity to motivate the patient and solicit compliance, because "without compliance, a good result will not be achieved."
A primary goal of the case presentation is to convince the patient of the many possibilities for bringing the treatment to a successful conclusion.

Oral Hygiene Instruction Aids

The clinician must consider and take seriously the fact that the patient's own case is what interests the patient most. Panoramic radiograph on the light box, easily visible clinical findings such as recession, plaque-retentive areas, bleeding on probing, gingival erythema and swelling etc. are easy to demonstrate and explain to the patient who holds a mirror.

At subsequent appointments, the interested patient can again be informed and motivated, for example through use of disclosing agents (p. 224) to visualize plaque at the gingival margin or in the interdental spaces. Dental plaque microbial "vitality" can also be used for patient motivation by means of microscopic enlargement (Plakoskope, p. 180); an inexpensive TV camera and monitor can dramatically depict plaque vitality for the patient.

Additionally, other instructional materials such as brochures, tooth models, and high-tech instruments such as digital intraoral cameras can be used to transmit to the patient the necessary information about health and disease.

It has been reported and demonstrated many times that a flood of new information often overloads or exceeds the patient's available memory, and the patient is soon no longer capable remembering all of the details. It has therefore proven to be helpful to give the patient small but informative take-home brochures or pamphlets, to be read and studied as follow-up to the information the patient heard as he/she sat in the treatment chair.

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Initial Treatment 1— Oral Hygiene by the Patient

The patient's oral hygiene (plaque control) remains today the primary supportive pillar of periodontal *prophylaxis*. It also supports *treatment*, and has great significance for *maintenance* of the treatment results.

Without continuous compliance by the patient, periodontal treatment by the dentist and the dental team will be less successful and the success will be of shorter duration. Oral hygiene by the patient means, above all, reduction of the amount of plaque and pathogenic microorganisms in the oral cavity. Gingival massage with the toothbrush is of secondary importance, with perhaps some "psychological" effect.

In special indications, mechanical plaque control can be enhanced or supported for a limited period of time by topical medicaments (disinfection agents such as chlorhexidine).

This chapter will describe:

- Plaque disclosing agents, revealing the plaque
- Manual toothbrushes
- Toothbrushing techniques, systems
- Electric toothbrushes
- Interdental hygiene—interdental hygiene aids
- Dentifrices
- Chemical plaque control—CHX, additional products
- Irrigators, of value?
- Halitosis, bad breath—oral hygiene
- Possibilities, successes and limitations of oral hygiene

Toothbrushes of all kinds are important aids for mechanical plaque removal. They reach, however, only the *facial, oral* and *occlusal* tooth surfaces.

The initial lesions of gingivitis and periodontitis, as well as dental caries, usually occur in the *interdental region*. Therefore, the toothbrush must be enhanced by additional hygiene aids that can ensure cleansing of the interdental area.

There is no single oral hygiene method that is right for every patient. The type and severity of the periodontal disease, the morphological situation (crowding, spacing, gingival phenotype etc.) as well as the patient's own manual dexterity determine the required hygiene aids and the cleaning techniques. During the course of periodontitis therapy, the techniques may have to be changed or adapted to the new morphological situation (longer teeth, open interdental spaces, exposed dentin).

The patient must be informed about his/her daily oral hygiene, its frequency, time spent and amount of force to be applied. In most cases, *once per day* is sufficient for a thorough and systematic plaque removal (disruption of the developing biofilm; Lang et al. 1973).

In the final analysis, though, it is not the hygiene aids, the technique or the time spent that is the determining factor, rather the result: *Freedom from plaque*. This parameter as well as the health of the of the gingiva (BOP) must be checked at regular intervals.

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Motivation—Gingival Bleeding

Since 1980, the clinical symptom “bleeding on probing” has assumed the foreground in patient motivation, replacing plaque disclosure. The profession realized that it is not the amount or expanse of plaque or its depiction in a microscope that was most meaningful to patients, rather it was the reaction of the patient’s own tissues to the microbial irritation that held the highest motivational value.

Each person exhibits very *different individual reactions* to the biofilm, its constituents and especially the microbial metabolites. Thus, even with identical amounts of plaque, quite different levels of pathogenic danger may be present.

Using the PBI (Saxer & Mühlemann 1975, Mühlemann 1978) or BOP (Ainamo & Bay 1975, p. 69) the severity of gingival inflammation can be numerically portrayed. If the gingival bleeding index decreases during initial treatment (1), as depicted by repeated clinical recording of the index, this provides visible evidence of success while simultaneously giving further motivation to the patient.

Bleeding on Probing as a Motivating Factor

475 Initial Condition: Moderate Periodontitis
The patient can clearly see the severe hemorrhage as the clinician performs the bleeding index (PBI or BOP).

Right: As a second step, *disclosing the plaque* reveals the cause of the disease. The next steps include initial oral hygiene instruction and professional, supragingival prophylaxis.





476 Clinical Situation: Two Weeks Later
Following professional prophylaxis and repeated OHI, the patient can readily see the return to health as indicated by the minimal bleeding during recording of the PBI data.
The patient is motivated by this success to further intensive cooperation and compliance.



477 Clinical View at 4 Weeks
The virtual absence of bleeding (inflammation) and the dramatic plaque reduction convinces the patient definitively of the logic of this treatment.

Right: The minimum plaque accumulations demonstrate the correlation: Less plaque = less gingivitis.
Additional oral hygiene instruction is now targeted toward those sites that are not completely plaque-free, e. g., interdental areas.





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Plaque Disclosing Agents

Frequently during the case presentation, when motivation is being emphasized using the bleeding index, the patient will pose questions concerning the *cause* of periodontal disease. Now—right now!—is the prime time for demonstration of microbial plaque, the most important etiologic factor in gingivitis and periodontitis.

Using non-toxic food coloring agents, the adherent plaque on tooth surfaces and gingiva can be selectively stained. The patient watches in a mirror as the clearly visible plaque is revealed and then scraped off using a probe.

Patients are further impressed to hear that only 0.001 grams of plaque contain ca. 300,000,000 bacteria. The necessity and possibility for plaque removal via oral hygiene measures becomes visible to the patient, and the initial toothbrushing instruction session falls on fertile soil.

One disadvantage of plaque disclosing agents that remain in the mouth for some time can be avoided by using the Plaklite system (Fig. 480). A solution that is virtually invisible in daylight clearly reveals accumulated plaque bacteria when illuminated with blue or UV light.







478 Red and Violet Disclosing Agents
Left: Classic red staining by erythrosin. This procedure is still permitted by the FDA.

Middle: Plaque disclosing agents for the patient (tablets) and for the clinician (solutions, pellets).

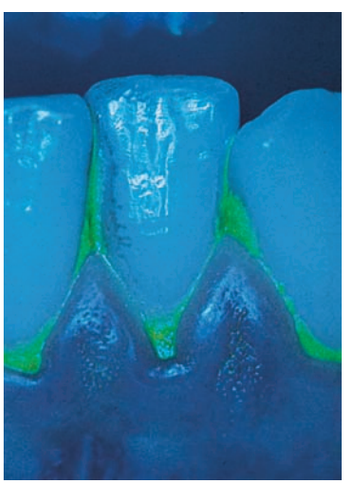
Right: Differential disclosing agents that stain "fresh" plaque *light violet*, and old, "mature," plaque a *darker violet* color.

Erythrosin	Patent Blue	Phloxin B	Na^+-Fluorescein
Tetraiodofluorescein sodium C. I. Acid Red 51 CI 45430 E 127 Do not use if patient is iodine-sensitive!	CI 42090 FD + C Blue No. 1 Brilliant blue E 133	CI 45410 C. I. Acid Red 92 Tetrachloro-tetrabromo-fluorescein	Fluorescein disodium salt Soluble fluorescein

479 Details of Four Plaque Disclosing Agents (adapted from Roulet & Zimmer 2003)
Older, deeply-staining agents such as basic fuschsin, malachite green and other "histologic stains," which are depicted in some illustrations in this *Atlas*, are no longer used in clinical practice because of their potentially injurious side effects or toxicity.







480 Fluorescent Disclosing Agents, Blue or UV Light
In normal room lighting, plaque disclosed with 0.75 % Na-fluorescein solution appears light yellow (left), but glows intensively yellow-green under blue light (right).

The disclosing products are available from Vivadent, Lactona, Clairol and Intern. Pharmaceutical Co. (IPC) and others.

A disadvantage of this technique is the requirement of a special light source or a filter mirror.

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Toothbrushes

For centuries, the toothbrush has served to remove of food debris and plaque from all *facial, oral* and *occlusal* tooth surfaces. Today the toothbrush remains indispensable, but it does not provide adequate interdental hygiene. In addition, when used with excessive force it has the potential to injure even healthy gingiva.

There is no ideal toothbrush (shape, size, handle) but in periodontics more and more brushes with softer, flexible bristles have found acceptance. Rounded bristle tips are the standard today.

Worthy of consideration also is the fact that toothbrushes are always used with toothpastes (p. 234). It seems only reasonable that these two components should be "synchronized" for each individual patient (König 2002) and this must be accomplished by the dental team. This should replace the often wildly extravagant commercial claims with *facts*, and permit targeted recommendations for each individual patient.

481 "The Best Brush"
Patients always ask: "What's the best toothbrush"? Who wins gold, silver or nothing?
Fact: There is no "best" brush!

For the dental team it is important to know the commercially available brushes, but more important to know the needs of the individual patient, in order to properly advise.



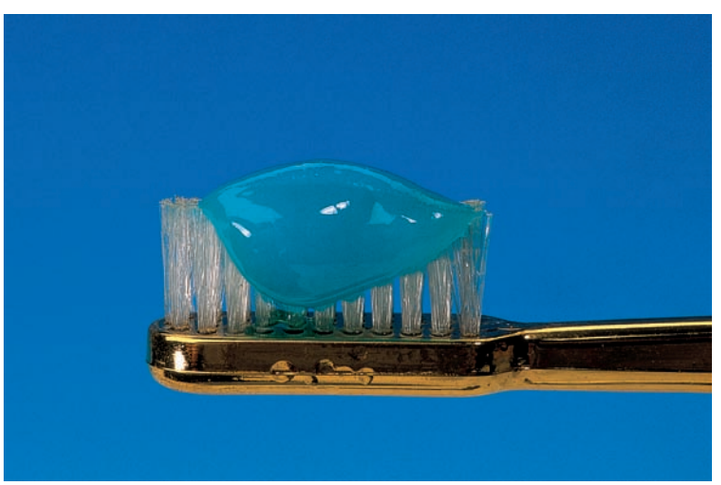
482 ADA Type and New Trends
New brushes and their efficacy are standardized according to ADA guidelines after *in vitro* testing. Clinical tests with human subjects are more costly and provide limited data. The ADA "norm" (left) is a four-row brush, multi-tufted. At right today's trend, the Oral-B "Cross Action."

Right: Rounded bristle tips are today's standard.





483 Toothpaste and Toothbrush: One Goal
The role of the toothbrush is generally overvalued! Only in combination with a dentifrice is it effective; the toothbrush is really only a carrier. Its positive effects (gingival massage?) are insignificant. Its negative effects, especially with hard bristles, can be grave: Damage or injury to gingiva and mucosa can lead to gingival recession or aphthous ulcers.



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Oral hygiene devices have become a huge worldwide market. The industry has done and will continue to do everything in its power to persuade consumers of the efficacy of its product, using brilliant colors and bizarre shapes! Using the latest generation of high-tech machines, it has been possible to create exceptional types of bristle arrangements—parallel or crossed, variously colored bristles, flat plane or irregular, straight or round brush heads etc.
The question that remains, however, is whether any of this is actually *useful* for patients!

It is up to dentists and dental hygienists not to *react* but rather to *act*! Guidelines for good toothbrushes need to be defined, for example, for periodontitis patients with thin gingiva, recession, large interdental spaces etc. Worthy of thought: A motivated patient brushes daily for 60, 70 or 80 years! Long-term freedom from injury is more important than momentary efficiency.

It seems that a start has been made: Superfine bristles—they clean just as well as hard bristles—and unconventional 3-headed brushes are being widely discussed.



484 Modern Toothbrushes—Frontal View
How is one to evaluate the enormous differences in shape, bristle array, handle etc.?

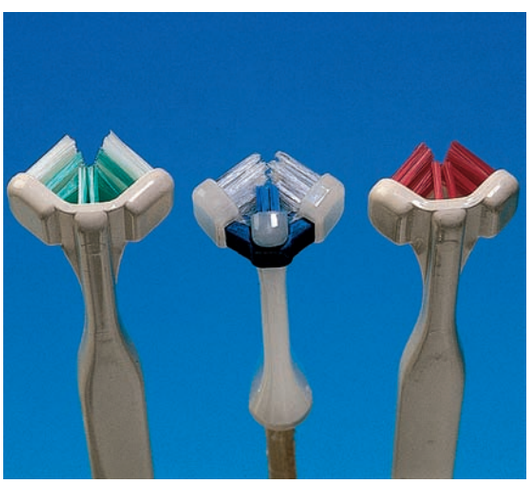
The best that a toothbrush can offer is its ability to effectively clean the teeth, while preventing damage or injury to gingiva or teeth, and help reduce bad breath.



485 Modern Toothbrushes—Lateral View
This figure corresponds to Fig. 484. From left to right. . .

- **Elmex** Supersoft
- **Paro** Future
- **Elmex** Inter X medium
- **Trisa** FlexHead soft
- **Dr. Best** X-aktiv Flex
- **Oral-B** Cross Action
- **Colgate** Navigator medium
- **Mentadent** Insider soft-medium
- **Superbrush** Junior





486 Meridol and Superbrush—New Developments; Progress?
Left: The Meridol brush has superfine, flexible bristles, designed to absorb excessive force.

Right: The three brush heads simultaneously clean vestibular, occlusal and oral tooth surfaces. Using a light vibratory movement, teeth are cleaned one-by-one. This design was superior to other brushes in clinical trials (middle: head of the Nais electric toothbrush. cf. p. 230).

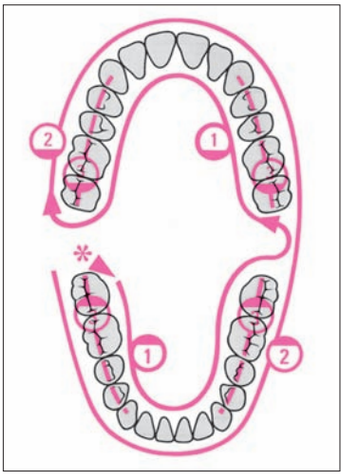
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Toothbrushing Technique

Innumerable toothbrush movements have been recommended over time, and then abandoned: Rolling, vibrating, circular, vertical and horizontal (Jepsen 1998). More important than the technique is the *efficiency* of cleaning, a *systematic* procedure and that *no damage* is caused.

Dentists and dental hygienists have recognized again and again that most patients, despite instruction, seem to be satisfied with an apparently "genetically determined" *horizontal scrubbing technique*.

The most frequently recommended "modified Bass technique" (Bass 1954) is depicted below.



487 Systematic Toothbrushing
The sequence depicted has been shown to be effective (start: right, posterior*)

1 **Oral Surfaces** mandible/maxilla, and all distal surfaces at the end of each arch.
2 **Facial Surfaces** mandible/maxilla
3 **Occlusal Surfaces** mandible/maxilla

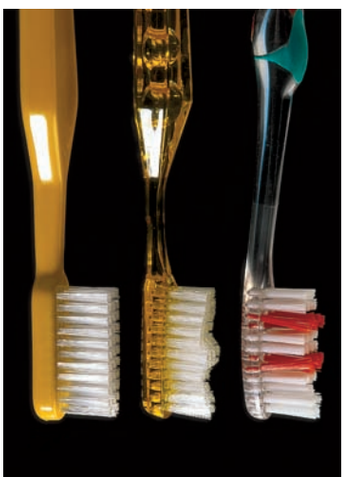
4 **Interdental Spaces,** using special hygiene aids

Modified Bass Technique

488 Placement of the Toothbrush
Toothbrush bristles positioned perpendicular to the tooth long axis will not effectively clean the interdental spaces.

Right: Instead of the "original" Bass technique, which used a two-row brush, today the "sulcus cleansing technique" is performed with the more common three- or four-row brushes. This combination should provide improved cleaning.





489 45° Angle of the Bristles—Occlusal View
When the brush is applied at a 45° angle to the teeth and then rotated toward the occlusal plane, the bristles slip easily into the interdental areas and gingiva sulci *without* excessive force. With the brush in this position, *small rotatory* or *vibratory* movements will effectively remove plaque.

Problem zone: Angular arch form in the canine regions.



490 45 Degree Angle—Distal Surfaces
Viewed from the distal, the position of the bristles in the Bass technique becomes obvious.

Right: Distal surfaces will not be effectively cleaned by hard bristle brushes; extremely flexible toothbrush bristles may provide effective distal cleaning.
Dental floss is not recommended for such surfaces, which often exhibit concavities and/or furcations. Single-tuft brushes are more effective (right).





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The Solo Technique—A Different Way to Brush Your Teeth

In 2000, Jiri Sedelmayer described the unreasonableness of the usual toothbrushing methods of many individuals: "Among other things, areas that scarcely need cleaning, such as prominent tooth surfaces and gingiva, are often injured. Notorious plaque-retentive areas, above all the interdental spaces, gingival sulcus and oral-distal tooth surfaces are regularly cleaned by only a disappearing few."
Sedelmayer was in fact correct, and in the same breath suggested a new technique, an alternative to the usual scrubbing, at least for individuals who are prepared to devote adequate time for their oral health.

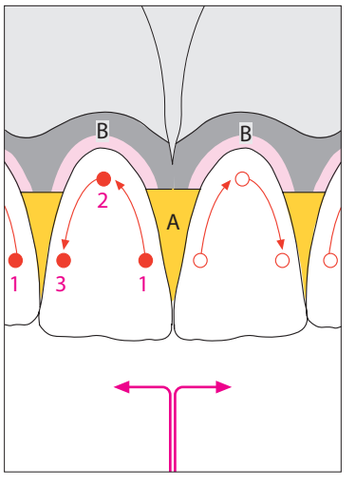
The problem with classical toothbrushes is that when they are used with the recommended non-traumatic slight pressure, the niches are not achieved; with heavier force, however, prominent tooth surfaces and gingiva are so abused that true long-term damage is the consequence (recession, wedge-shaped defects etc; p. 456; Lussi et al 1993).
Using a soft, round, single-tuft brush with light force, the technique cleans tooth-by-tooth, above all the lingual aspects perfectly, including marginally and deep into the interdental space. However, other special hygiene aids are also necessary.





491 "Solo" Technique—Begin at Mesial of Tooth 11
The round, single-tuft brush is applied to the tooth surface with light force, and the bristles splay out. The mesial sulcus of tooth 11 is cleaned using minimal circular movements.

Left: An example of "solo" brushes, beginning (left) with the ages-old chewed twig!
Round "solo" brushes are commercially available from TePe, Curaden, Tandex etc.





492 Continuing Along the Gingival Margin...
The gingival margin and gingival sulcus are effectively de-plaqued, using the patient's own sense of feel.

Left: Simple schematic—initiate tooth cleansing "from the middle toward the side" (e. g., tooth 11):
1 Mesial
2 Marginal
3 Distal

A Papilla
B Gingival Margin





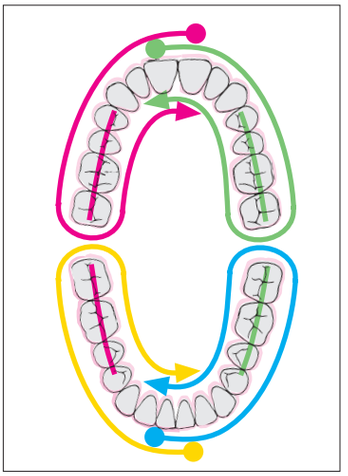
493 ... and Continuing Distally
The brush is guided distally and angled so that it achieves maximum contact with distal tooth surfaces, sulci and the papillary region.
This procedure is repeated for each tooth.

Left: What the classical brushes almost never accomplish: Perfect cleaning of the ligual marginal surfaces and interdental areas in the posterior segment of the mandible.

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Electric Toothbrushes

Comparative clinical studies have shown that the efficiency in plaque removal of the newest electric toothbrushes is at least as good as that achieved with manual toothbrushes. Various products offer primarily advantages for persons with reduced manual dexterity, or the handicapped, but also offer an alternative to the hand toothbrush for highly motivated individuals. Preferred today are electric brushes with round heads, and sonically active toothbrushes, the latter because of their additional hydrodynamic activity (van de Weijden et al. 1996, Zimmer et al. 2000, Warren et al. 2001).



494 Effective Cleaning with Electric Toothbrushes
One can use the same system described for manual toothbrushes (Fig. 487) for the "quadrant system" built into some electric brushes. This provides 30 seconds of brushing per quadrant (see below; Q1-Q4).

Q1 Quadrant 1
Q2 Quadrant 2
Q3 Quadrant 3
O4 Quadrant 4

495 A "collection" of some commercially available electric toothbrushes
(from left to right)

- **Interplak**
- **RotaDent**
- **Philips Sonicare**
- **Braun Oral-B**
- **Waterpik**
- **Ultra sonex**
- **Roventa**
- **Nais**
- **Oralgiene**



496 Brush heads
The choice of a brush head is as important as the choice of the electric brush itself. With the high frequency brushes, there is a potential danger of gingival trauma. Brushes whose bristle movement stops when excessive force is applied should be recommended (e. g., Sonicare).

Each patient should be informed and instructed in the proper use of the electric toothbrush.



Sonic brushes



Three-head brush



Round brush heads

497 Current trends ...
... include primarily sonic or combined sonic-ultrasonic toothbrushes, e. g., the Ultrasonex, Sonicare and Waterpik (*left*), the Nais electric toothbrush (*middle*; with its "normal" and three-unit head). The hydrodynamic effects of these brushes removes plaque even where the bristles cannot physically reach.
Brushes with round, rotating or oscillating heads are today mature products of high efficiency (Braun Oral-B, 5th generation and Trisa, *right*).







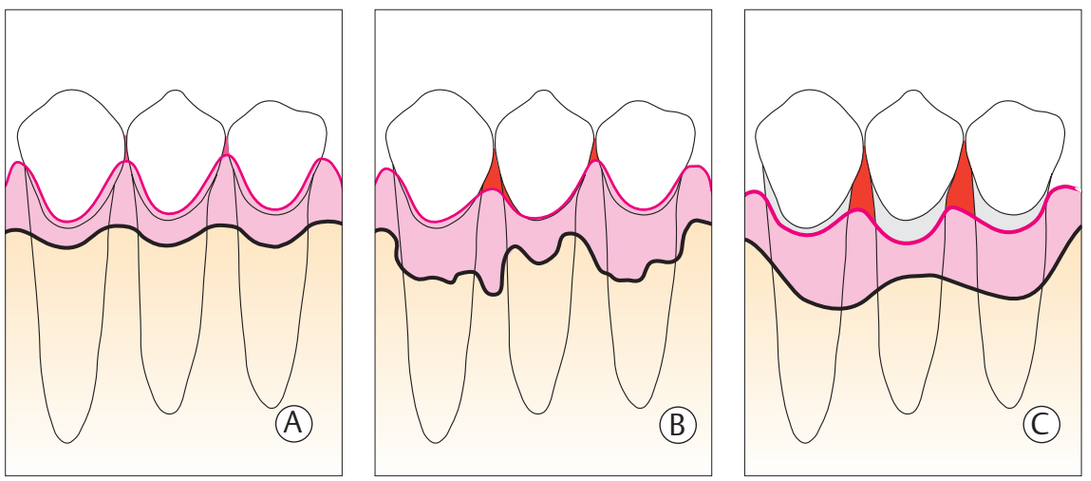
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Interdental Hygiene

Gingivitis and periodontitis are usually more pronounced in the interdental area than on oral or facial aspects. Dental caries also occurs more frequently in the interdental region than on oral or facial smooth surfaces. Therefore, interdental hygiene, *which cannot be achieved with the toothbrush*, is of critical importance for periodontitis patients. The most appropriate interdental hygiene aids must be selected for each individual patient. The selection from the numerous commercially available devices depends for the most part on the morphological situation of the interdental spaces.

Dental Floss

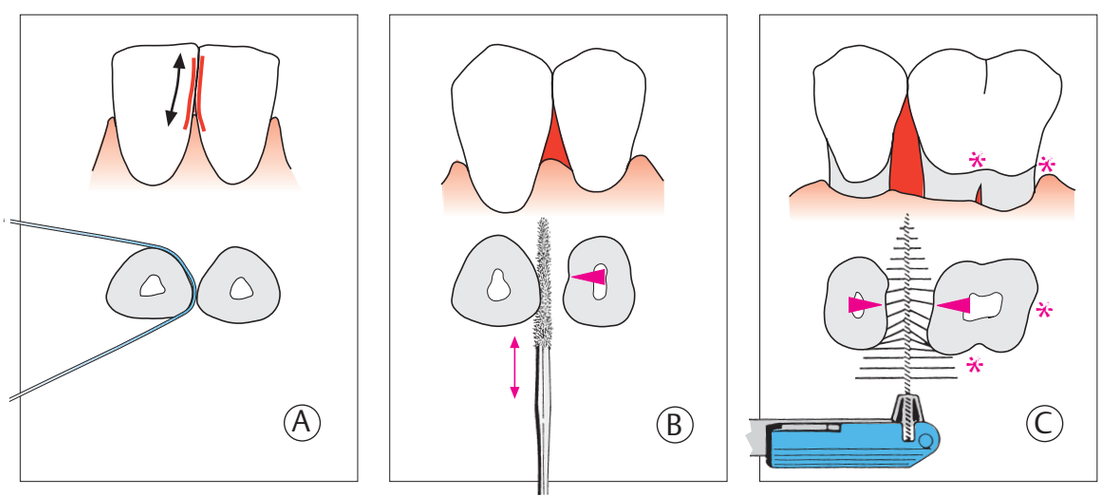
The use of dental floss is indicated for healthy patients, and for gingivitis and mild periodontitis cases, as well as for patients with crowded teeth. It is well known, however, that the acceptance of dental floss (floss, tape, super floss) is quite low for most patients, especially males! Alternative interdental hygiene aids should be recommended even if these are less efficient than floss if patients will at least use them daily.



498 Morphology (schematic) of the Periodontium

A Healthy
B Periodontitis
C Treated, Healed Periodontitis

These three situations require differing techniques and various hygiene aids for interdental plaque control.
The diagrams portray the course of the alveolar bone, the gingival margin and the expanse of the interdental spaces (red).



499 Size of the Interdental Space and Oral Hygiene Aids
The choice of a hygiene aid for interdental plaque control depends primarily upon the size of the interdental space.

A Dental floss
for narrow interdental spaces
B "Tooth sticks"
for slightly open interdental spaces
C Interdental brushes
for widely open interdental spaces, root concavities and grooves

Toothpicks, Interdental Brushes

Toothpicks or *interdental brushes* are indicated for plaque removal if the interdental spaces are open, e.g., following completion of periodontitis therapy, as well as for patients who accept dental floss but seldom actually use it.
The newest "toothpicks" are no longer simply wooden throw-away articles, rather they are adorned with tiny hair-like bristles, they are elastic, multi-use, fine plastic files ("Brush Sticks"). Some patients become passionate about these devices!

With expansive areas of exposed root surface, especially in the molar regions, one often observes more or less severe grooves which can only be cleaned using *interdental brushes*.
These devices should be used without dentifrice except in special cases and then only short-term. The abrasive in dentifrice would rapidly abrade exposed dentin in the interdental space.
These devices can also be regularly used to apply fluoride or chlorhexidine gel into the interdental space to prevent caries or the recolonization of residual pockets.

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Dental floss

500 Choices: Floss, Tape, Ultra- and Superfloss
Today's dental floss, with filaments of nylon, kevlar etc., is strong enough to traverse even the tightest contact points. For splinted teeth, bridges etc., various threading devices are available (e. g., Eez Thru, Butler). Both waxed and unwaxed dental floss can be recommended.

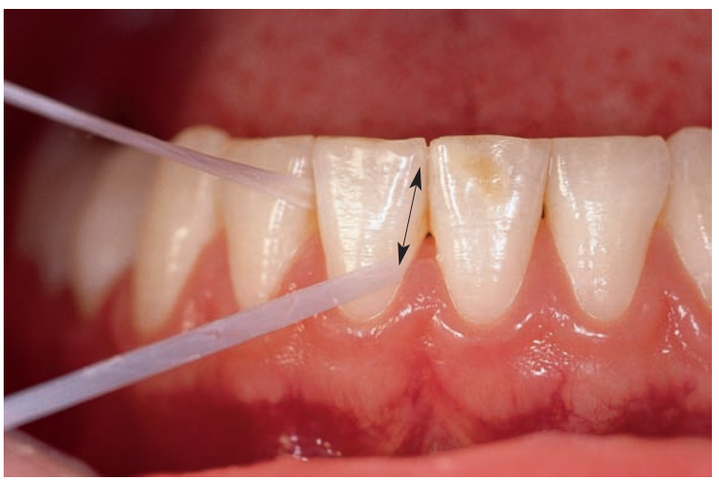
Right: **Dental tape** (Colgate)





501 Use of dental floss
To avoid injury to the papillae, the dental tape is carefully maneuvered through the contact point using a sawing motion. Cleaning of both proximal surfaces is accomplished with up and down movements (double arrow) into the sulcus with the floss stretched tightly.

When the floss "sings" the tooth surface is clean.



Other Devices for Interdental Cleaning

502 Selection
The traditional wood interdental cleaners exhibit a triangular profile. The various manufactures use either hard or soft wood (birch, balsa, linden). Some products are impregnated with various substances (fluoride, CHX, mint, nicotine!).

Right: The new **"Brush Stick"** (Esro) cleans very effectively.



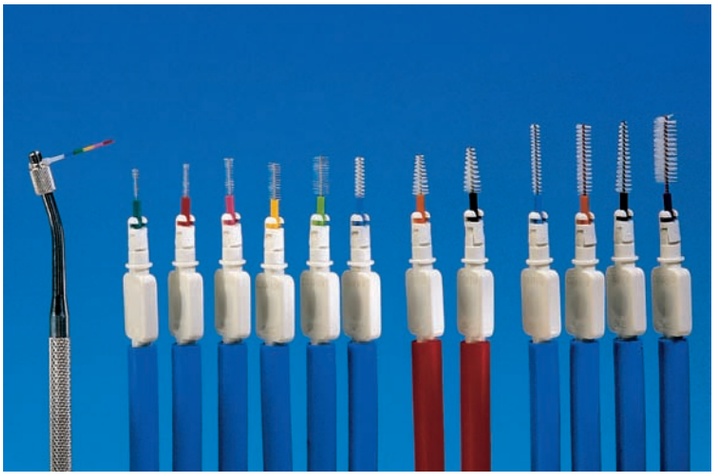


503 Clinical Use of "Brush Sticks"
The red, felt-like tip is guided into the interdental space at a slight angle. Plaque removal is accomplished by horizontal back and forth movements (double arrow). If the interdental spaces are large, the brush stick is first pressed against the proximal surface of one tooth, and then the adjacent tooth. Concavities can be cleaned very well using the blunt end of the Brush Stick!



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Interdental brushes

504 Selection

Numerous companies offer interdental brushes. Excellent products include various bristle lengths, strength and diameter, with handpieces or separate holders (pictured is the assortment from *Curaprox*; at left, the coded measurement probe for the choice of brush type).

Left: **Oral-B** (with handpiece), **Top Caredent, TePe, Oral Prevent, Paro (*Esro*)**.

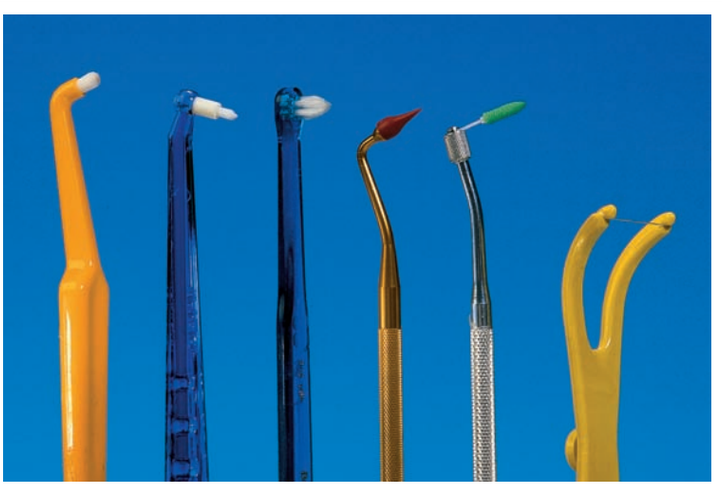


504 Use of a Large Interdental Brush

Appropriate interdental brushes are available today for the smallest to the largest interdental spaces. They represent the ideal cleaning tool, especially for periodontitis patients. The brush is inserted obliquely into the interdental space, from apical. Cleaning is performed with a back and forth motion (double arrow).

The trend today is toward long, flexible (soft) bristles.



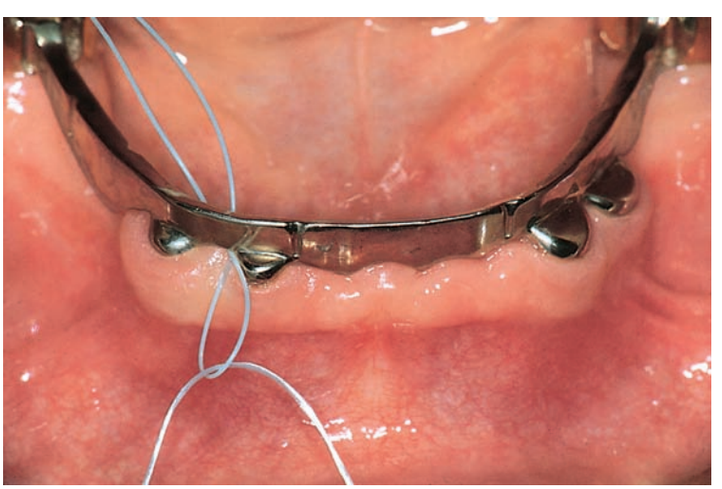


Additional Hygiene Aids

506 Hygiene Aids for Special Problems

- **Round solo brush** (p. 229)
- **Pointed, single-tuft brush,** e. g., for the furcation entrance
- **Stimulators** Massage in the area of the interdental papillae
- **Soft-foam attachment** (Oral-B) Cleaning for titanium implants
- **"Fork"** for dental floss

Left: **Threader** for floss and tape.



507 Threading Dental Tape

With fixed splints, bridges and bar constructions, the dental floss cannot be applied from the occlusal, but must be inserted. In such cases this can be accomplished using a threading sling (e. g., Butler).

The threader is particularly helpful for patients with limited manual dexterity, and also for interdental spaces for which the superfloss threader is too "soft."

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Dentifrice

A dentifrice is an integral component of daily home care to enhance oral hygiene. Dentifrices effectively double the efficiency of mechanical *plaque removal* and therefore help to prevent oral diseases such as caries and gingival inflammation (*active principle of prevention*).

The most important component of any dentifrice is the abrasive substance. Manufacturers use many different abrasives, which vary not only with regard to their chemical composition (phosphates, carbonates, silica, alumina etc.), but also in particle size and shape (rounded, angular). These differences determine the polishing effect of the product, as wells as the abrasiveness of the dentifrice on dentin (measured *in vitro* as the RDA value: "Radioactive/relative dentin abrasion").

The non-mechanical components of dentifrices include their chemical ingredients (*passive principle of prevention*): These ingredients prevent caries (fluorides), treat hypersensitive exposed dentin (K- and Sr-salts; fluoride; p. 458), possess disinfecting properties (Triclosan), and serve to whiten stained teeth (H_2O_2; carbamide): All in one tube!

508 Dentifrices
From the enormous number of products on the market, the dental team must select only a few, and must know the advantages and disadvantages of each in order to provide proper information during oral hygiene instruction.

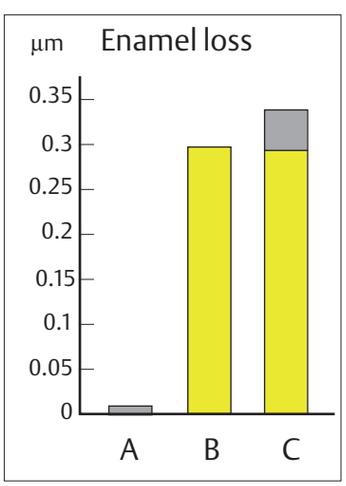


509 Damaging Toothpaste? Damaging Technique?
Left: Healthy dentition. How will it look in 40 years?

Middle: Loss of enamel is minimal after a single toothbrushing (**A**), but this is not the case with fruit acids and chelators from foodstuffs (grapefruit; **B**/yellow). Toothbrushing after consumption of acidic foods is yet worse (**C**).

Right: This patient brushed with a scrubbing technique for 50 years, while also consuming acidic foods!







510 Types of Dentifrices

- **"Whitening" Dentifrices** (left)
- **Normal Fluoride Dentifrices** (middle)
- **Dentifrices for Tooth Neck Sensitivity** (right)

Does long-term use of whitening dentifrices cause tooth damage? Must exposed dentin be cleaned using mildly abrasive dentifrice?







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Chemical Plaque Control—“Soft Chemo” Prevention

Purely mechanical plaque control is effective up to a certain point, but excessively robust toothbrushing can injure the gingiva and damage tooth structure. Optimum personal oral hygiene can only be achieved through the use of certain supportive antimicrobial agents, which may be included as ingredients in dentifrices or mouthwash. These chemical ingredients must not interact with other substances within a dentifrice and should exhibit high substantivity (AAP 1994, Brecx et al. 1997, Cummins 1997). The substantivity of plaque-inhibitory substances depends upon:

- Pharmacokinetics
- Concentration and dose
- Effectiveness over time
- Site of application

A therapeutically effective “soft chemo” substance should affect at least an 80 % plaque reduction. Until today, this level of effectiveness has been achieved only by the bisbiguanide chlorhexidine (CHX). For this reason, CHX in all of its commercially available forms remains today the substance of choice.



511 CHX Digluconate Products
In Europe and elsewhere, but not in the USA, various CHX mouthwashes, gels, sprays etc. are available for patients, in concentrations of
- **0.06–0.12 %**
- **0.1–0.2 %**
- **10 %** (concentrate; PlakOut)

For clinical use, concentrations up to **20 %** CHX are available, as well as **CHX-HCl** in powder form. Diluted or dissolved, this provides a cost-effective disinfectant, and can also be used as the cooling solution for ultrasonic instruments.

Chemical plaque control – Disinfection agents, “soft chemo”

Chemical classification	Examples	Effect	Products
Bisbiguanides	• Chlorhexidine (CHX)	• Antimicrobial	Mouthwashes, gels, dentifrices, throat spray
Quaternary ammonium substances	• Cetylpyridium chloride • Benzalconium chloride	• Antimicrobial	Mouthwashes
Phenols and essential oils	• Thymol, menthol, eucalyptus oil • Triclosan	• Antimicrobial • Antimicrobial, anti-inflammatory	Mouthwashes, dentifrices
Metal ions	• Tin, zinc • Strontium, calcium	• Antimicrobial, desensitizing	Mouthwashes, dentifrices
Halogens – Fluorides – Iodine	• Sodium fluoride, sodium monofluorophosphate • Stannous fluoride • Amine fluoride • Providone (PVP) iodine	• Caries inhibitory (antimicrobial), desensitizing • antimicrobial	Dentifrices, gels, mouthwashes, varnish Rinsing solutions
Amine alcohols	• Delmopinol	• Inhibits biofilm formation	No product yet available
Oxygen-splitting ligands	• Hydrogen peroxide • Sodium perborate • Sodium percarbonate	• Antimicrobial	Mouthwashes
Plant products	• Sanguinarin	• Antimicrobial	Mouthwashes, dentifrices
Enzymes	• Glucose oxidase • Amylglucose oxidase	• Antimicrobial	Dentifrice

512 Agents for Chemical Plaque Control—“Soft Chemo”
CHX in all its available forms is the most potent agent for *supragingival* plaque control.

Most of the additional disinfection products listed in this table work as *antimicrobials*, and in appropriate concentrations are *bacteriocidal*.

While CHX represents the second generation of effective plaque inhibitors, the aminoalcohols (e. g., delmopinol) represent the third generation. These are not bacteriocidal, rather they inhibit the formation of biofilm. This new group evolved from the newest research on biofilm formation (p. 24).

Note: Plaque reduction is not synonymous with inflammation reduction. Therefore, in this table no specific *inflammation-reducing* products are listed.

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Irrigators

The supragingival efficacy of mouthwashes containing caries-preventive (fluorides) or antiseptic ingredients (e.g., chlorhexidine) is well acknowledged. Mouthwashes and also the use of intraoral irrigators represent only adjuncts to mechanical oral hygiene.

The pulsating, hydrodynamic forces produced by irrigators can rinse away food debris from interdental spaces and plaque-retentive areas, but do not remove plaque biofilm (Hugoson 1978). Irrigation solutions have always contained aromatics, and later also disinfective ingredients. The use of chlorhexidine in sub-optimum concentrations (e.g., 0.06 %) led to improved plaque inhibition and an anti-inflammatory effect (Lang and Räber 1981, Flemmig et al. 1990).

In contrast, the success of pulsating irrigators with the common tips is limited in the subgingival area, and in periodontal pockets (Mazza et al. 1981, Wennström et al. 1987). With special tips, the pulsating stream penetrates more deeply, but biofilm is not removed (Flemmig 1993, reported up to 90 % penetration during pocket rinsing).

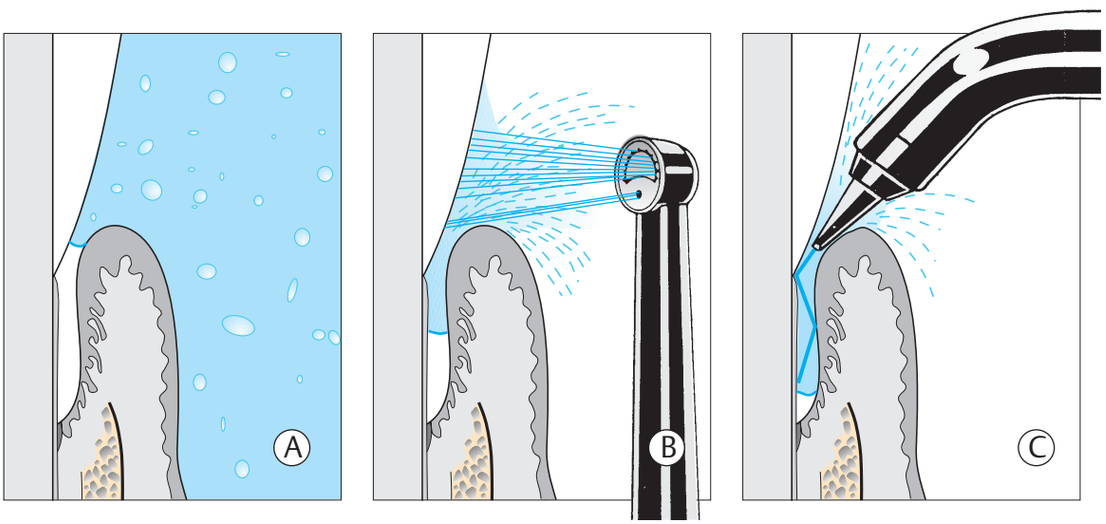
513 Mouthrinsing—Irrigators

A Mouthrinsing—no solution enters the pocket (< 5 %).

B Single or multiple sprays—about half of the pocket depth is penetrated (ca. 50 %).

C Special tips (PikPocket)—the solution is forced deeply into the pocket (ca. 90 %).

Modified from *Flemmig 1993*



514 Oral Irrigator—Waterpik
The "mother of all oral irrigators."

Waterpik represents the first technically simple oral irrigator. Contemporary models feature an off/on button on the handle, as well as pressure regulation. In this case, CHX was added to the rinsing solution; the toothbrush in the foreground reminds that the irrigator should be used *after* toothbrushing.

Right: Normal tip (left) and special tip (PikPocket).





515 "Professional Care Center"—Braun Oral-B
The well-known *Braun electric toothbrush* offers a large assortment of brushes, including the "Interspace" brush, and the special *oral irrigator* (Oral-B Oxyjet) in a combined unit.

Right: In combination with a special attachment, this device produces fine *micro air bubbles* in the water stream. It is claimed that this effectively attacks plaque bacteria and removes food deposits from between the teeth.





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Oral Hygiene for Halitosis

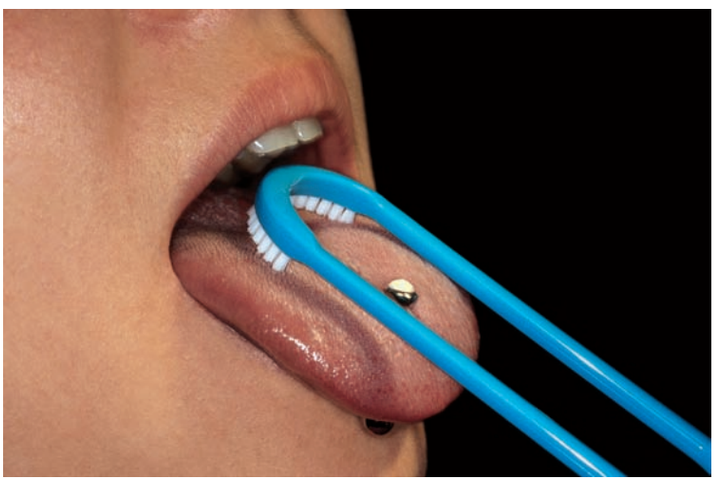
It has been estimated that half of all people suffer from episodic or permanent halitosis. A myriad of factors could be responsible for this pathologic *Foetor ex ore*—systemic, oronasal and oral, associated with a large and diverse number of volatile molecules.

In the absence of systemic diseases, 9 out of 10 cases of halitosis originate from the tonsils and the tongue, particularly the posterior dorsal tongue surface (Stassinakis 2002).

Gram-negative anaerobic microorganisms from the periodontal pocket may also contribute to halitosis, primarily by way of their metabolites, not only their lipopolysaccharides but also short-chain fatty acids such as butyric acid and propionic acid. Breakdown products of the host defense response may also play a role in halitosis. Clinically, it is relatively easy to measure volatile sulfur compounds such as hydrogen sulfide (H_2S) and thiol (mercaptans) (v. Steenberghe and Rosenberg 1996, Loesche and Kazor 2002).

The most important step to reduce halitosis appears to be improved oral hygiene, especially cleansing of the tongue (Saxer 2000, 2002; Seemann et al. 2001).





516 Tongue Cleansing
Special scrapers are available for cleaning the tongue. Patients should be informed that it is most important to clean the *posterior* portion of the tongue dorsum. An initial gag reflex usually subsides quickly.
Note: This procedure not only reduces halitosis, it also removes a large *reservoir* of periodontopathic bacteria.

Left: Combo for oral hygiene: Toothbrush, interdental brush, and the tongue brush.





517 Tongue Scrapers
In addition to the normal toothbrush, which can also be used for tongue cleaning, a variety of tongue scrapers is commercially available: Scraper, tongue brush and combinations.

Left: In addition to mechanical cleaning, antiseptics may also be used; e. g., 1 % chlorhexidine gel. The toothbrush is loaded with gel.





518 Additional Options on the Road Toward "Full Mouth" Cleansing
Chewing *sugar-free* gum increases saliva flow (enamel remineralization), massages the gingiva and may remove some bacteria that are then swallowed.

Left: Articles for oral hygiene—mechanical: toothbrush, tongue scraper; chemical: antimicrobial sprays, gels and mouthwashes (e. g., retarDEX, Esro).

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Possibilities, Successes and Limitations of Oral Hygiene

Oral Hygiene and Prophylaxis

There is no longer any doubt that effective mechanical oral hygiene (toothbrush, interdental hygiene) is the best periodontal prophylaxis (reviewed by Axelsson 2002). Mechanical oral hygiene can be optimized by the short-term use of chemotherapeutics (above all chlorhexidine). With optimum oral hygiene, oral health can be *maintained* (primary prevention).

Oral Hygiene and Gingivitis

Beyond pure prevention, optimum supragingival plaque control is also effective *gingivitis therapy* (secondary prevention; Löe et al. 1965). Because the patient cannot remove calcified plaque (calculus), professional tooth cleaning must be performed at regular intervals.

Oral Hygiene and Periodontitis

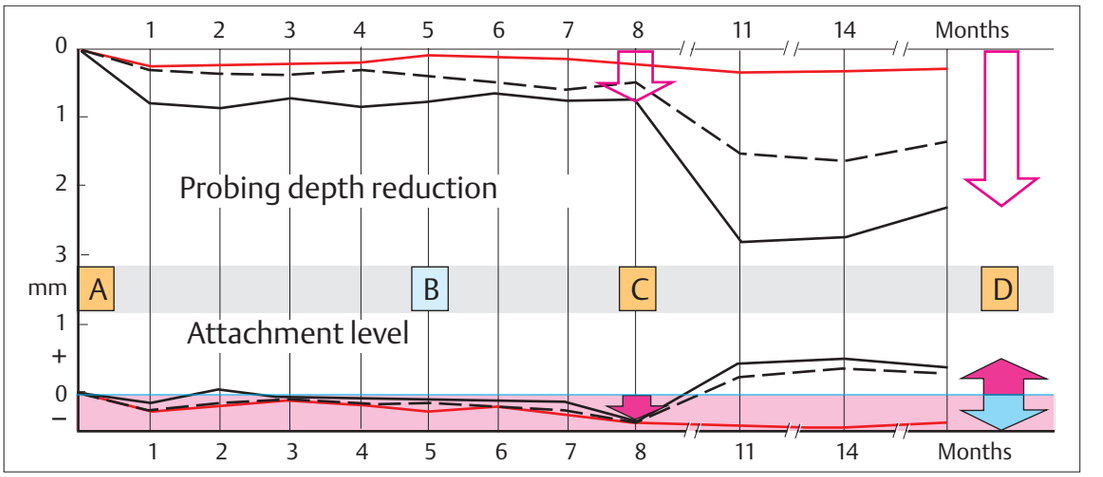
As meaningful as oral hygiene is for disease prevention, gingivitis treatment and oral health maintenance, it is relatively ineffective when used *alone* for treatment of periodontitis. Even with a highly motivated and dextrous patient, there exist clear limitations: The best efforts in home care cannot reach the deep subgingival plaque. Calculus and endotoxin-containing cementum can never be removed by the patient.

An example of these relationships was published in 1983 by Dr. Egelberg's research group (Cerecek et al., Fig. 519): Distributed according to probing depths, periodontitis patients were treated in three phases: Phase A—normal oral hygiene; Phase B—additional use of a perio-aid (blunt toothpick) supragingivally; Phase C—professional *supra-* and *subgingival* debridement (Cavitron) with anesthesia. Follow-up examinations (D) were then performed over a 9-month period following termination of the experimental phase. The results clearly demonstrated that oral hygiene alone significantly reduced the plaque and bleeding indices, but not the probing depths, which were virtually unaffected. Only following professional subgingival scaling (C) did a significant reduction of probing depths and true attachment gain occur (compare *results* following traditional subgingival debridement, p. 280).

519 Oral Hygiene vs. Subgingival Scaling (Cercek et al. 1983)

A Oral hygiene by the patient: Toothbrush and interdental hygiene aid
B Oral hygiene as in A—additional use of the PerioAid
C Professional subgingival scaling (ultrasonic scaler)
D Results after 17 months

Initial pocket probing depths:
0–3.5 mm
4–5.5 mm
> 6 mm



Oral Hygiene Following Periodontal Treatment

Oral hygiene by the patient is the most important factor for maintenance of the results of periodontal therapy. Only with optimum plaque control by the patient, enhanced by the dentist or dental hygienist at recall appointments (maintenance therapy) can recurrence of disease or re-infection of inactive residual pockets be prevented (Rosling et al. 1976b, Nyman et al. 1977, Knowles et al. 1979, Axelsson 2002).
Where are the contradictions to the findings presented on this page?
How is it possible that with good oral hygiene alone—which apparently is ineffective subgingivally—that a treated residual pocket of, e.g., 4 mm or more, can *remain* inactive for months or even years, when it is known today that such pockets will be re-colonized in a very short time (Petersilka et al. 2002)?

The answer may come from the Socransky group (Haffajee 2001a, b; 2003). They reported that a permanent excellent plaque control regimen can *slowly* but *surely alter* the microbial composition of the pocket flora. In particular, the pocket flora contains a lower percentage of periodontopathic bacteria! The prevention of migration of pathogenic microorganisms from the supragingival to the subgingival region appears also to play a certain role.

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Initial Therapy 1— Creating Conditions that Enhance Oral Hygiene

- **Tooth Cleaning—Supragingival Scaling**
- **Creation of Hygiene Capability**
- **Gingivitis Treatment**

Active professional treatment by the dentist or dental hygienist should begin even as oral hygiene instruction, patient motivation and monitoring of home care are ongoing. The patient cannot be expected to improve her/his oral hygiene if the preconditions for optimum home care are not simultaneously created (creation of hygiene capability). Professional prophylaxis is particularly important in this regard, as well as elimination of any plaque-retentive areas (niches) that represent harbors for bacterial accumulation.

The procedures described below are part of the *first phase of initial therapy*. Together with oral hygiene by the patient, these procedures comprise the *only* treatment necessary for gingivitis, and are important prerequisites in periodontitis therapy as well.

The following pages provide details concerning:

- Instruments and materials, and their uses
- Supragingival tooth cleaning and calculus removal
- Removal of iatrogenic irritants (niches)
- Reduction of naturally-occurring plaque-retentive areas
- Subgingival plaque and calculus removal from pseudopockets and shallow periodontal pockets

The various treatments performed during the first phase of initial therapy cannot be strictly separated from each other, either in the presentations that follow in this book, or in the practice of dentistry. At a single appointment, for example, calculus removal, elimination of amalgam overhangs, minor odontoplasty and occlusal equilibration may all be accomplished.

The subgingival treatment of root surfaces (second phase of initial therapy) may also intersect with the first phase. In clinical situations that represent the indistinct transition from gingivitis to incipient periodontitis, i.e., when pockets are shallow, supra- and subgingival scaling often can be performed simultaneously.

On the other hand, scaling and definitive root planing in deep pockets and eventual soft tissue curettage must be relegated to the *second phase* of initial therapy. These procedures are often categorized as actual surgical therapy.

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Supragingival tooth cleaning—Power-driven Instruments ...

The removal of all stains, deposits and concrements comprises the first phase initial therapy. It is also an important preventive measure in the healthy periodontium, and the most significant post-operative measure following completion of periodontitis therapy. Thorough tooth cleaning is performed during each recall appointment (maintenance phase, p. 447).

The prevention/treatment/maintenance therapy trio "without end" demands the use of auxillary personnel such as the dental hygienist or the trained dental assistant. It also demands rationalization, standardization and work simplification, as well innovation in the development of new instruments (ultrasonic devices, Air-Scaler etc.).

Difficult-to-remove stains resulting from medicaments (e.g., chlorhexidine), tobacco, beverages (tea, wine) and foodstuffs as well as dental plaque can be removed using instruments that provide a water-powder spray (e.g., Cavitron-Jet). The powder that is used in the water spray must be minimally abrasive for dentin and restorative materials (Iselin et al. 1989). Furthermore, the spray should never be

520 Powder-water Spray Device (Cavitron-Jet)
The powdered abrasive consists primarily of sodium bicarbonate ($NaHCO_3$) which can remove tough deposits and stains when used with a water spray.
The water-powder spray requires use of a high-speed evacuator.

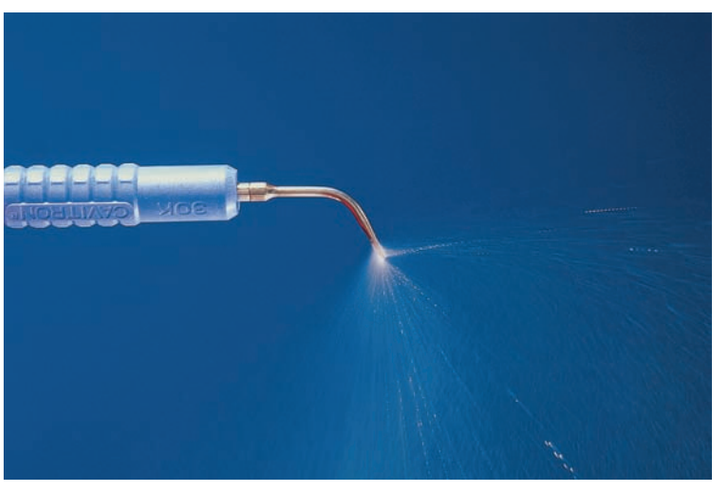
Right: **"Jet Shield"**
This "mini-evacuator" is affixed directly to the working end of the Cavitron-Jet.





521 Stabilized Power System (SPS) with Ultrasonic Scaler Tips (Cavitron Thru Flow inserts—TFI)
In modern instruments, the water coolant is directed through the instrument tip in a groove on the instrument head. Ultrasonic scalers work at between 25,000–50,000 cycles per second, with very small amplitudes.

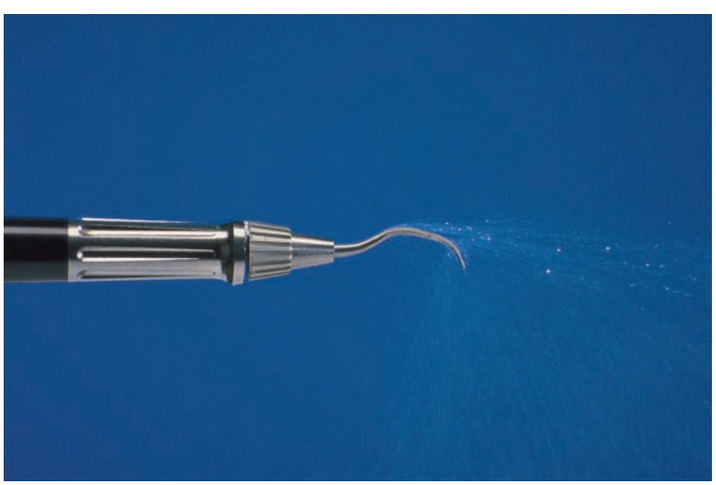
Right: Various ultrasonic scaler tips; from left to right:
TFI-1000, TFI-9, TFI-1, TFI-7.





522 Air-Scaler (Titan-S, "Sonic Scaler")
The air-scaler has a regulable frequency of maximum 6,000 Hz and thus is considerably slower than an ultrasonic instrument. The motion of the tip of the instrument is between 0.08–0.20 mm; relatively slow.

Right: Three tips for the Titan-S device.
Additional manufacturers:
- **KaVo**
- **Satelec, and others**





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... and their Use

directed perpendicular to the tooth surfaces, and should usually be used only on enamel, with constant movement of the tip. Such devices do not guarantee perfect cleaning in interdental spaces or niches. The spray with normal abrasive powder should not be directed into pockets. With the new, "mild," minimally abrasive agents and fine tips, effective cleansing can be achieved, in certain circumstances, even subgingivally (e.g., glycine powder from Espe, with the EMS Airflow Handy 2; p. 282; Petersilka et al. 2002).

After the removal of soft deposits, calculus becomes visible. Calculus is an excellent substrate for plaque accumulation and must be completely removed. Numerous power-driven instruments are available: Ultrasonic apparatus (e.g., Cavitron) as well as Air-Scaler that can be attached to the air-water supply of the dental unit (e.g., Titan-S, Satelec; Sonicflex KaVo etc.; Hermann et al. 1995).

However, the most important and most precise means for removal of concrements remains: hand instruments (p. 242).



523 Removal of Soft Deposits and Stains

Tough deposits, plaque and stains from tobacco, tea, wine or chlorhexidine can be removed from accessible enamel surfaces using the powder-water device. Cleaning in interdental areas, however, is insufficient. The stream should be directed onto the tooth surface at an angle of 45°. A high speed evacuator is used to retrieve the reflected solution.

Caution: Highly abrasive on cementum, dentin and restorations!



524 Removal of Hard, Supragingival Concrements with an Ultrasonic Device

Following removal of soft debris and plaque, remaining calculus is completely removed using the ultrasonic device. In narrow, poorly accessible sites and niches, fine ultrasonic tips or hand instruments must be used afterwards.

Caution: Overheating, cracks in enamel and porcelain!



525 Removal of Hard Supragingival Concrements Using the Air-scaler

This instrument, which attaches to the turbine handpiece air-water orifice, permits removal of concrements in a manner similar to the ultrasonic instrument; however, the sensitivity is improved and the frequency can be regulated. Less pressure is necessary, and rinsing is continuous. This simplifies therapy, improves visibility and permits more efficient performance.

Pictured is the Titan-S scaler.

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Supragingival Tooth Cleaning—Hand Instruments, Prophy Pastes ...

In addition to ultrasonic devices, hand scalers and curettes remain the most important instruments for periodontal therapy and prophylaxis. For the removal of soft deposits and stains, hand instruments are enhanced by the use of brushes, rubber cups and polishing strips along with cleaning and polishing pastes.

It is not the manufacturer that is critical for successful treatment, rather the shape of the instrument, especially its degree of sharpness, and above all the manual dexterity of the clinician (scaling technique).

For the removal of supragingival deposits, *chisels*, straight and angled *scalers* and also *lingual scalers* are effective. In premolar and molar segments, also on difficult-to-reach areas, grooves and depressions on the crown, as well as exposed root surfaces, the removal of supragingival concrements may require *curettes* in addition to scalers, usually without anesthesia.

526 Scalers
For supragingival calculus removal and for concrements that are located only a few millimeters below the gingival margin, sharp-edged, pointed scalers in various shapes are indicated:

- **Zerfing Chisel** ZI 10 (white)
- **Zbinden Scaler** ZI 11, 11 R+L (blue), straight and paired
- **Lingual Scaler** ZI 12 (black)

Right: Working end of the Zerfing chisel (45° sharpening angle!) and the lingual scaler.

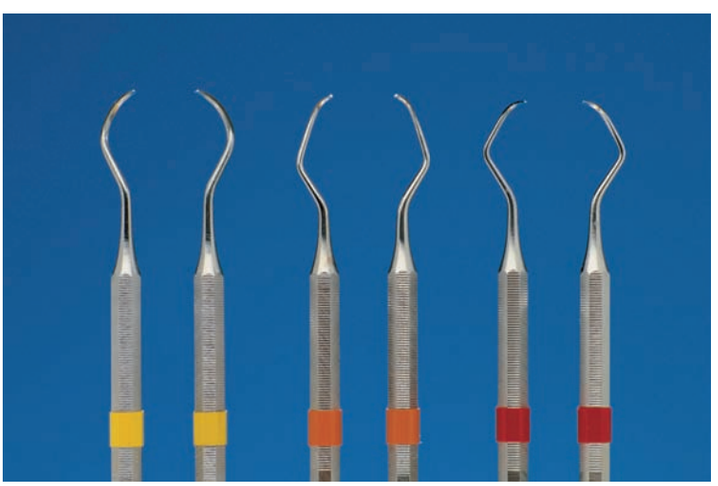




527 Curettes
For difficult-to-reach areas and for subgingival accretions, the scaler armamentarium must be enhanced by curettes:

- **Universal Curettes** ZI 15 (yellow) 1.2 mm wide
- **Anterior Curettes** GX 4 (orange), Deppeler
- **Posterior Curettes** M 23 A (red); both are ca. 0.95 mm wide; Deppeler

Right: Working ends of a pair of universal curettes.





528 Standardized Prophy Pastes—RDA
Prophy pastes are available according to abrasiveness. The standardization is achieved on the basis of dentin abrasion, measured by radioactivity. All are fluoride-containing:

RDA Value	Abrasiveness	Color
• 40	mild	yellow
• 120	normal	red
• 170	moderate	green
• 250	heavy	blue

Right: Finger cups with color-coded prophy pastes.





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... and their Use

For the first phase of initial therapy, the classical universal curettes are indicated. The slender *Gracey curettes*, which are sharpened on only one edge, are used almost exclusively for subgingival scaling and root planing in periodontitis patients (p. 259). Today, ultrasonic and sonic devices are being used more and more often, in addition to hand instruments.

If supragingival calculus is covered with thick soft deposits, these should be removed with brushes and coarse prophy paste before mechanical debridement.

Whenever calculus is removed, the teeth should be polished afterwards with a rubber cup and polishing paste. This polish of the teeth and any exposed root surfaces is performed with fluoride-containing prophy pastes, which are classified according to their *dentin abrasiveness* (radioactive dentin abrasion = RDA; p. 234).

Contact points and the interdental areas can be cleaned using fine polishing strips (see p. 244).



529 Supragingival Calculus Removal
The Zerfing chisel is the only instrument that is used in the anterior segment with a *pushing* motion.
Straight and angled scalers and/or ultrasonic devices are then used for removal of any remaining calculus.
The lingual scaler (Fig. 526, right) smoothes the narrow lingual surface of mandibular anterior teeth.



530 Subgingival Calculus Removal
The largest masses of subgingival accretions are located only a few mm apical to the gingival margin. These should be removed during gross debridement, without anesthesia, using scalers and curettes or ultrasonic devices, as necessary.

Gingival bleeding will occur even during very careful scaling, as the ulcerated pocket epithelium is injured.



531 Polishing with Rubber Cup and Prophy Paste
Each time scaling is preformed, the teeth must be polished, otherwise rough surfaces will enhance re-accumulation of plaque bacteria.
Rubber cups and polishing paste are ideal for this procedure (RCP technique, “rubber cup and paste”), because they are kinder to the gingival margin than are rotating brushes. The rubber cup can be used near shallow pockets to achieve polishing 1–2 mm beneath the gingival margin.

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Creating of Conditions that Enhance Oral Hygiene—Removal of Iatrogenic Irritants

Concurrent with mechanical removal of plaque and calculus, all imperfect dental restorations are corrected with the goal of creating smooth supra- and subgingival tooth surfaces as well as impeccable transitions areas between natural tooth surfaces and the margins of restorations and crowns. Only when this is done will it be possible for the patient to maintain efficient plaque control: *Creation of hygiene capability*.

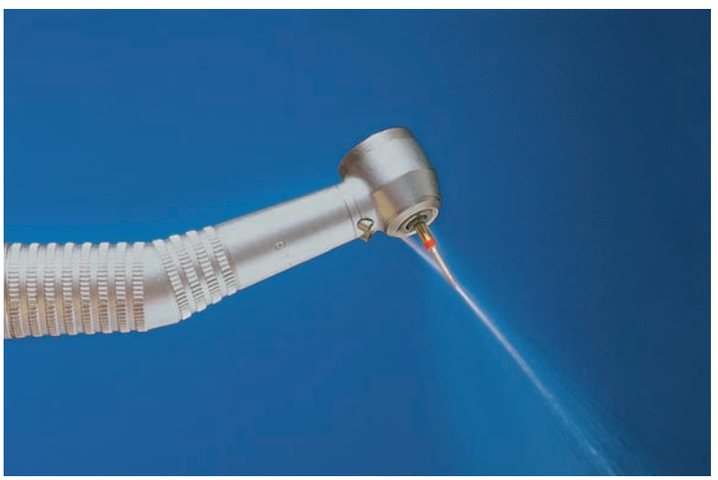
The most important iatrogenic irritants, which must be removed or corrected, include:

- Rough, poorly contoured restorations
- Overhangs of restoration margins
- Open crown margins located subgingivally
- Improperly contoured bridge pontics
- Depressible clasps, prosthesis saddles etc., which can injure the periodontium directly.

532 Instruments for Recontouring and Polishing Old Restorations
High-speed handpiece with variously-shaped, fine, flame-shaped diamond burs.

Right:

- Round stone
- Round bur
- Flame-shaped, fine diamond bur
- Rubber tip polisher (Shofu “brownie”)



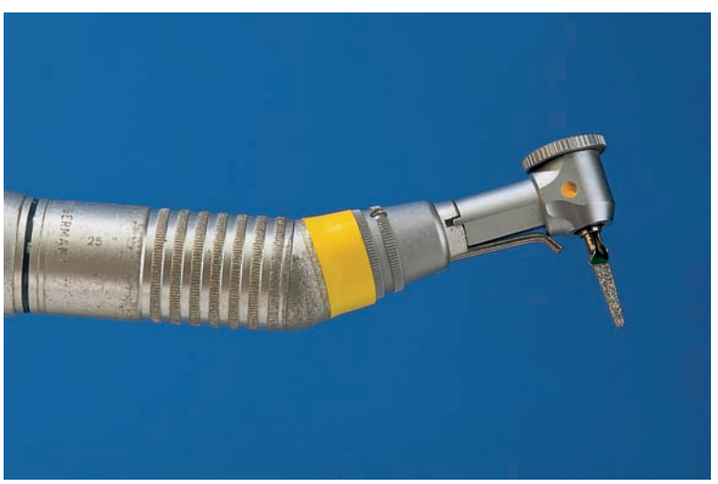


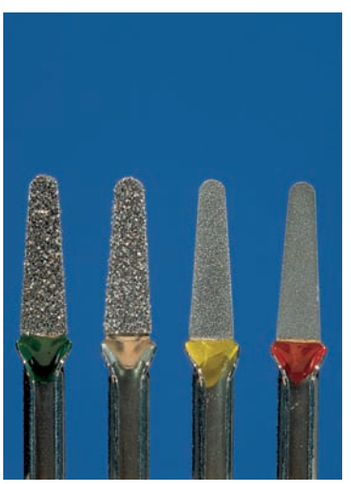
533 Mechanical Files
Handpiece with prophylaxis head (EVA system; KaVo).
0.4–1.5 mm length. Regulable speed, up to 10,000 rpm.

Right:

- **Proxoshape Set** (Intensiv) Diamond coating coarseness: 75 µm, 40 µm (yellow), 15 µm (red)

Possibility to remove restoration overhangs even in relatively narrow interdental spaces.





534 Manual Filing
A strip holder simplifies the removal of restoration overhangs and polishing in areas of narrow interdental spaces, and also protects the cheeks, tongue and lips. Pictured:

- **LM Holder; Steel Strips** (Horico)

Right: **MEBA separator**
Extremely narrow contact points are not always accessible even for the finest strips. The separator can provide access.





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More important than direct irritation, however, is the fact that even minor imperfections of dental restorative work represent plaque-retentive areas. The result at such locations is gingival inflammation and, over the long term, possibly periodontal destruction (Lange et al. 1983, Iselin et al. 1985).

The finishing and polishing of all restoration surfaces can be performed with fine diamonds (water cooling), round burs and finishing disks.

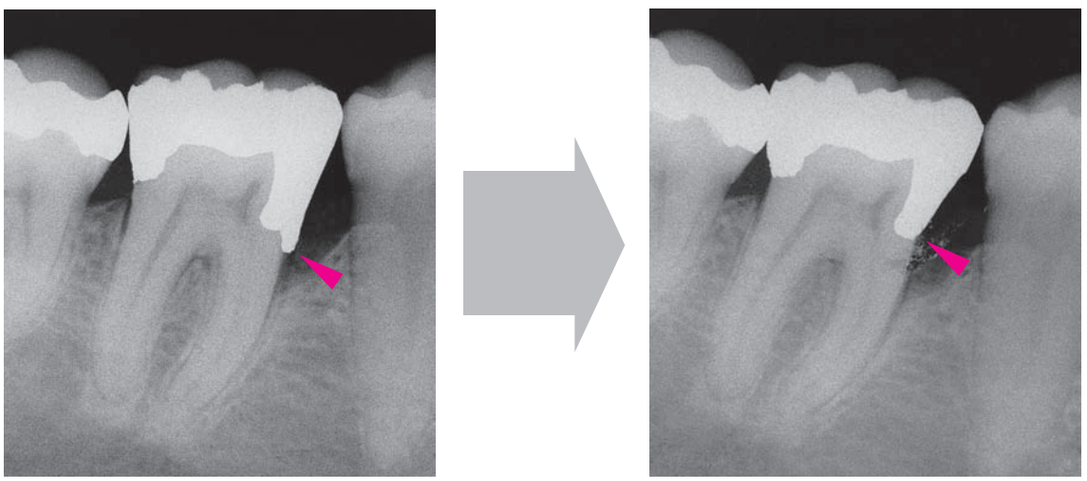
Interdental amalgam overhangs can be removed with flame-shaped diamonds and periodontal files or with the Proxoshape files (EVA system).

Metal and plastic strips, either hand-held or in an appropriate holder, are also useful in the marginal/proximal regions for smoothing restoration margins. Old restorations exhibiting overhangs or cracks *should be replaced*, because secondary caries is common beneath defective restorations!



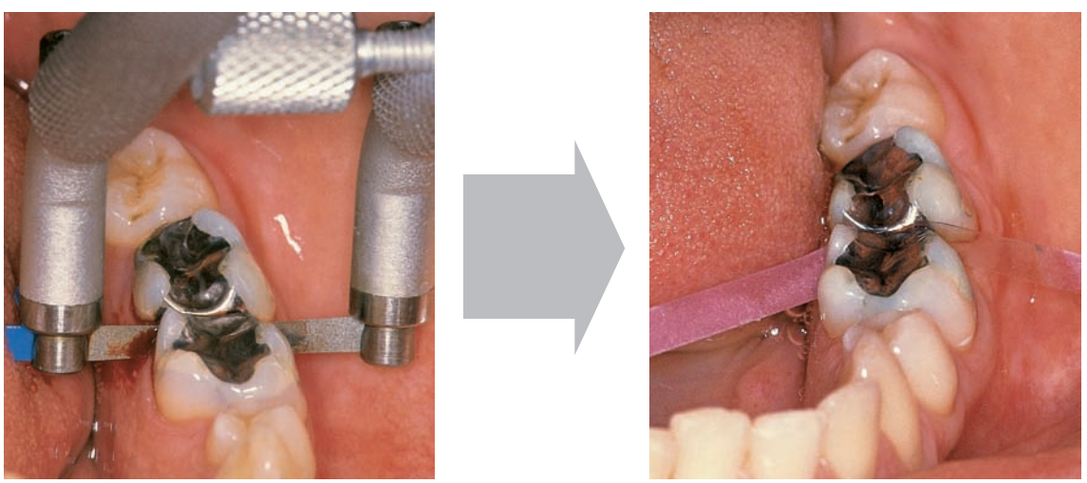
535 Old amalgam restorations before and after recontouring
Left: The rough, discolored surface of old restorations enhances plaque accumulation. Even though the occlusal surfaces do not have any direct contact with the marginal periodontal tissues, polishing the restorations leads to a reduction in the bacterial load in the oral cavity. Functional contacts in centric occlusion and lateral occlusion must be maintained.

Right: Contoured and polished old amalgam restorations.



536 Amalgam overhang before and after removal
Left: Tooth 46 exhibits a pronounced marginal overhang on the mesial aspect (massive plaque accumulation, arrow). A deep bony pocket is also located near this iatrogenic niche.

Right: The proximal overhang was removed and the restoration was polished. The margin is now perfect, discouraging any further plaque accumulation. If there is suspicion of caries, the restoration should be replaced.



537 Smoothing the proximal restoration surface using strips
Left: LM strip holder with diamond-coated steel strip during smoothing of the distal surface of tooth 36. The contact area must be protected.

Right: The interdental surface of the restoration is finally polished using fine, very mildly abrasive linen strips, which can also be used in the contact area.
Goal: Smooth proximal surfaces make possible perfect interdental hygiene with dental floss.

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Correction of Iatrogenic Irritants—Bridge Pontics

For esthetic reasons, bridge pontics in the *anterior region* should contact the attached gingiva. An ovoid pontic (p. 502) that presses into the gingiva can enhance esthetics by stimulating papilla formation around the abutment, but may render hygiene more difficult.

In *posterior areas*, where esthetics is not a consideration, it is often beneficial to leave 1–2 mm of space beneath pontics to facilitate use of hygiene aids for the removal of plaque *under* the pontic.

A facetted pontic ("modified ridge lap," p. 502) makes it easier to use interdental brushes, stimudents or superfloss to clean the abutment teeth, because of the guidance that is absent when the pontic is far removed from the gingiva, or when a bar-like pontic is used.

Improperly constructed bridge pontics may be corrected on an emergency basis using flame-shaped diamonds. The undersurface of pontics treated in this manner can be polished almost to laboratory smoothness using Proxoshape files (15 µm abrasiveness).

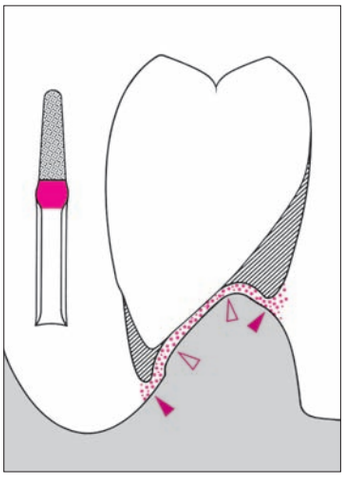
538 Improper Bridge Pontic Form
This pontic is too long and its saddle shape contacts both attached gingival and *mobile* oral mucosa on the facial aspect (type "ridge-lap," p. 502). It is also too wide, contacts the mesial abutment over a broad expanse, encroaches on the mesial gingival papilla and precludes any effective interdental hygiene. The planned shortening and contouring is indicated by the dashed line.



539 Correction of the Pontic
The fixed bridge is functional. Therefore, the poorly contoured pontic is recontoured on its marginal and proximal surfaces using flame-shaped diamonds.

Right: Massive plaque-retention area beneath the concave, saddle-shaped pontic (solid red arrows). Contouring renders the pontic's undersurface flat to convex, and plaque retention is therefore reduced (empty arrows).





540 Pontic Area Accessible for Hygiene
A periodontal dressing (Coe-Pak) remained *in situ* for 10 days. After healing of the gingival wound, optimum oral hygiene is possible in the proximal surfaces of the abutment teeth and beneath the pontic, e. g., using superfloss or "Brush and Floss".

The yellowish fiber-optic light used from the lingual aspect reveals healthy, keratinized gingiva.



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Removal of Natural Plaque-retentive Areas—Odontoplasty of Grooves, Depressions, Irregularities

The natural morphology of a tooth manifests grooves, irregularities, depressions etc. on both the crown and the root. In a healthy dentition, these can usually be cleaned satisfactorily with the toothbrush and interdental hygiene aids.

However, morphologic anomalies that represent plaque-retentive niches may be found at the cervical aspect of some roots. Frequently encountered are narrow *grooves* that extend apically from the lingual pit of an incisor far down the root surface.

Furthermore, fused roots of multirooted teeth may exhibit an irregular profile, with large grooves that may extend deeply into the subjacent dentin. Following initial periodontal pocket formation, such grooves are exposed and become plaque-retentive areas that are difficult to ascertain clinically. They may assume an etiologic role in localized, progressive destruction of periodontal supporting structures.
To a certain degree, such niches can be opened by means of mild odontoplasty, and thus rendered more accessible for cleaning.





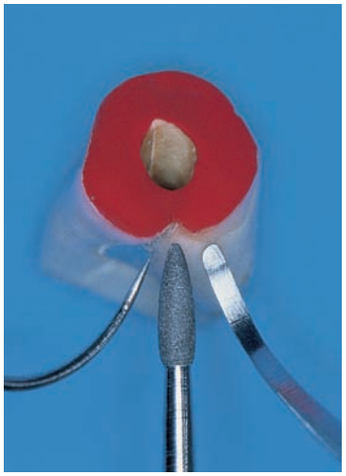
541 Diamonds for Contouring and Polishing—the Perio-Set ...
... consists of slender, conical and flame-shaped diamonds for subgingival debridement, odontoplasty and final polishing of ground tooth or root surfaces; two shank lengths are available.
Left: Flame-shaped Perio-Set diamonds in three degrees of abrasiveness (SEM):

Perio-Set (Intensiv Co.)
- 75 µm (blue)
- 40 µm (yellow)
- 15 µm (red)



542 Recontouring (opening) a Depression on Tooth 22
Apical to the lingual pit amalgam on this lateral incisor, a fine groove that could be detected clinically with an explorer extended 5 mm apically into the periodontal pocket.

The groove was so narrow that its depth could not be probed with either a scaler or a curette. An odontoplastic modification was indicated, which included widening, rounding and polishing with a 15 µm diamond.





543 Tooth 22 after Odontoplasty
The narrow groove was opened up using diamond burs. The depth of the groove can now be reached with fine hand instruments (e. g., an 0.8 mm wide curette), making adequate professional debridement possible. After odontoplasty, tooth surfaces should be repeatedly treated with topical fluoride.

Left: Section through a similar anterior tooth, at the level of the gingival margin.

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Reduction of Natural Plaque-retentive Areas—Crowding: Morphologic Odontoplasty

Crowding is one of the few tooth position anomalies that may play an indirect role in the initiation and progression of gingivitis/periodontitis. In such cases, occlusal/functional factors play no role. Crowded teeth create niches for plaque accumulation, and simultaneously render the patient's oral hygiene more difficult.

Extensive orthodontic treatment (p. 463) involving selective tooth extraction, is often not possible due to the large technical, temporal and financial considerations, especially in adults.

Careful *morphologic odontoplasty* may represent an alternative to orthodontics in cases of tooth crowding; the procedure can also improve the esthetics. Odontoplasty is performed using fine diamonds, *exclusively in enamel*. Following the procedure, tooth surfaces must be polished and treated with fluoride.

544 Crowding—Plaque-retention Factor
In this case of severe anterior crowding in the mandible, one observes severe gingivitis and plaque accumulation on those tooth surfaces that do not benefit from *self-cleansing* by the lip and tongue. This situation can be significantly improved by minor odontoplasty.

Right: View from the occlusal aspect. Tooth 31, in its severely labioverted position, is never touched by the tongue.





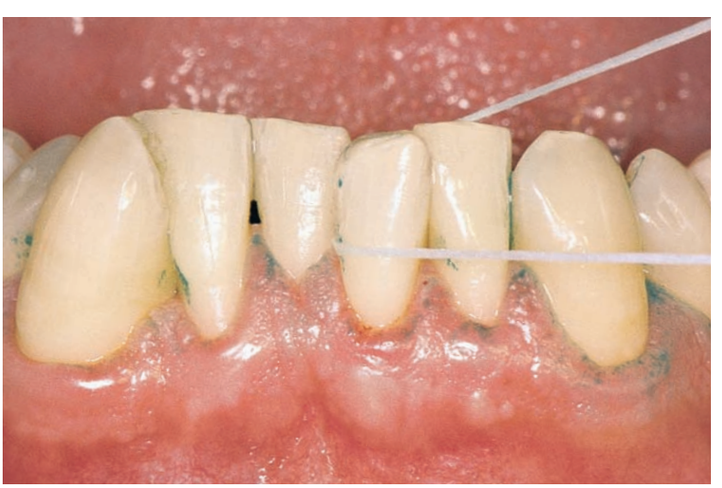
545 Odontoplasty—Morphologic Grinding
Minor esthetic odontoplasty was performed on the incisal edges, while retaining all occlusal contacts in centric (red). The black surfaces will be selectively and morphologically smoothed, to decrease the niches.

The extremely narrow contact surfaces are polished using abrasive strips so that the interdental areas become accessible for dental floss.



546 Clinical View Following Odontoplasty and Prophylaxis
The plaque-retentive areas are no longer so expansive. Disclosing agent reveals only minor plaque accumulation. Interdental hygiene with floss is now possible.
This minor odontoplastic treatment and optimization of oral hygiene by the patient led to significant reduction of gingival inflammation.

Right: Even in the lingual area, the clinical situation is improved. Mild marginal inflammation persists.





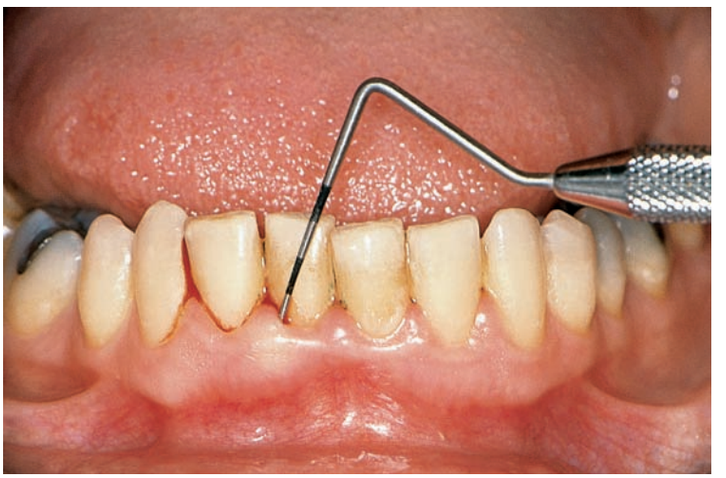
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Treatment of Plaque-elicited Gingivitis

Clinical Procedure, Step-by-Step

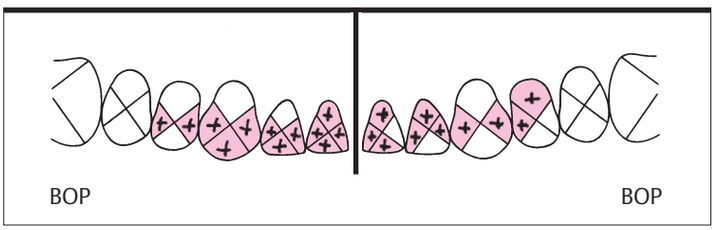
The treatment for gingivitis ("gingival disease," classified as Type I A, p. 520) is identical to the "initial treatment 1" (hygiene phase) performed collaboratively between the patient and the clinician. It includes motivation, information, oral hygiene instruction and check-up, as well as professional supragingival plaque and calculus removal (*debridement*).
When performed throughout the entire dentition, this treatment can be extremely time-consuming. It will be demonstrated here in the mandibular anterior segment. The 30-year-old female exhibited a moderate, plaque-induced gingivitis. There was *no* attachment loss, but pseudopockets to 4 mm were noted. The patient had never received comprehensive oral hygiene instruction. OHI was therefore provided simultaneously with *professional tooth cleaning* procedures.

Initial findings (mandibular anterior):
PI: 72% BOP: 69% TB: 0–1 (p. 174)
The clinical appearance and radiographs are depicted below.



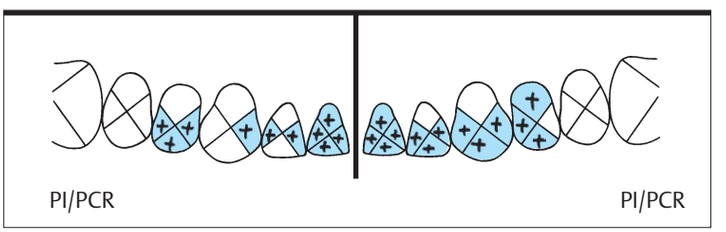
547 Initial Findings—Moderate Gingivitis
The gingivae are swollen by edema, especially in the papillary area. Profuse hemorrhage occurs immediately after gentle probing, particularly in interdental areas. The patient's generally mediocre oral hygiene is enhanced in the mandibular anterior area by mild crowding, which favors plaque accumulation.
The width of the attached gingival is normal.





548 Bleeding Index (BOP) and Plaque Index (PI or PCR)

BOP: On 22 of the 32 measured sites (mesial, facial, distal, oral) bleeding occurs after gentle periodontal probing (indices, p. 68).



PI/PCR: Plaque was detectable on 23 of the 32 examined tooth surfaces (8 teeth: 34–44).

Left: Radiographic findings. There is no radiographic evidence of bone loss.



549 Stained Plaque
On almost all teeth, there is more or less pronounced plaque accumulation, particularly in the marginal and interdental regions.

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Plaque and Calculus Removal in the Mandibular Anterior Area; Lingual View

550 Initial Condition
A plaque-induced gingivitis with supragingival calculus is shown from the lingual aspect.
Oral hygiene instruction (toothbrush) was given at the first appointment.
Interdental hygiene (using dental floss) was demonstrated at the next appointment.

Right: Gingivitis, facial view.





551 Debridement Using the Ultrasonic Scaler
Power-driven devices are particularly indicated for the initial removal of soft and hard supragingival deposits (gross debridement).

Right: Historical (left) and modern ultrasonic instruments with internal cooling (middle; Dentsply Cavitron) or sonic scaler (e. g., KaVo Sonicflex, right).

Caution: These create aerosols; barriers are indicated.





552 Scaling—Tooth Cleaning Supragingivally Using Hand Instruments
After use of power scalers, it is imperative that hand instruments (scalers and curettes) be systematically employed for fine debridement.
Less important than the particular instrument used is the result:
A clean tooth.



553 Checking with a Pointed Explorer
The smoothness of the tooth and root surface is checked with a very fine explorer.

Right: Fine, pointed explorers:

- **EXD5**
- **EXD3CH**
- **EXS3A**
  All instruments from Hu-Friedy Co.





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554 Subgingival Cleaning with Universal Curettes
Even in gingivitis therapy, fine curettes are applicable. Their rounded ends injure gingiva only slightly when they are used subgingivally in pseudopockets with a horizontal stroke.

Left: Instruments:

- **Scaler M23**
- **Curette M23A**
  (Deppeler Co.)



555 Plaque Removal And Preliminary Polishing in the Interdental Area—Strip with Mild Abrasiveness
Following mechanical therapy of the oral and facial tooth surfaces, the proximal surfaces and the contact areas are cleaned with strips incorporating fine abrasives.

For polishing, dental tape or smooth, uncoated strips may be used with a fluoride-containing prophy paste (p. 232).





556 Polishing with a Rubber Cup and Paste—RCP Method
Using the RCP method, tooth surfaces are carefully treated. Soft deposits (plaque!) should be removed exclusively with RCP, because repeated *mechanical* scaling (e. g., during recall visits) could enhance the formation of recession and wedge-shaped defects.

Left: Depending upon the tenacity of the deposits (stains), rubber cups or soft-hard brushes can be employed.





557 Mandibular Anterior Area Immediately after Treatment
Plaque and calculus have been removed. The time for these procedures was ca. 15 min. Even with very careful mechanical treatment of the tooth surfaces, minute trauma to the gingiva cannot be avoided. Such minor injuries heal within a few days.

Left: View from the facial, immediately following plaque and calculus removal.

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Gingivitis Treatment

Summary

In this 30-year-old female, restorations were placed from time to time over many years. At these infrequent dental appointments, however, oral hygiene instruction was never provided. The patient reported that a very brief calculus removal was performed at such appointments.

During gingivitis therapy in all sextants by the dental hygienist, the patient was readily motivated. She learned the demonstrated plaque control methods using toothbrush and dental floss without any difficulty. Her plaque index (PI or PCR) fell to 12 %. Upon probing the gingival sulci, only minimal bleeding occurred (BOP 9 %, p. 69).

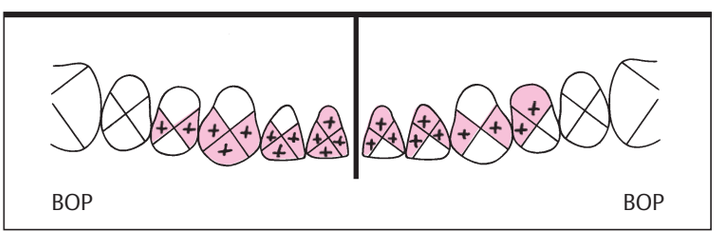
The patient's continued excellent cooperation was expected; therefore, 1–2 short follow-up appointments were scheduled. Subsequently, a recall interval of ca. 6 months was deemed sufficient to maintain the successful therapeutic result: Freedom from clinical inflammation, and periodontal health. The recall interval should also make it possible to detect early on any problems or failures with home care.

559 Initial Condition—Mandibular Anterior Segment
Inflamed and slightly swollen gingiva, pseudopockets, bleeding on probing. Generalized marginal plaque accumulation is barely visible.



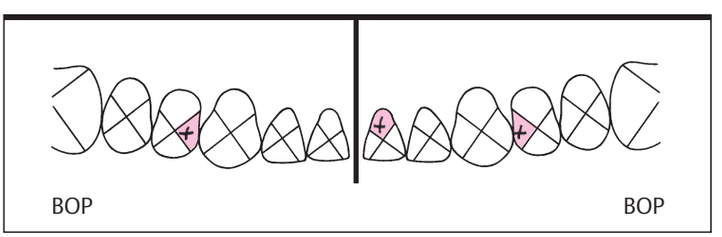
560 Bleeding Index (BOP)

Before Initiation of Therapy
Over two-thirds of all marginal and interdental areas of the 8 teeth examined (34–44) bled after probing (BOP 69 %).



Before

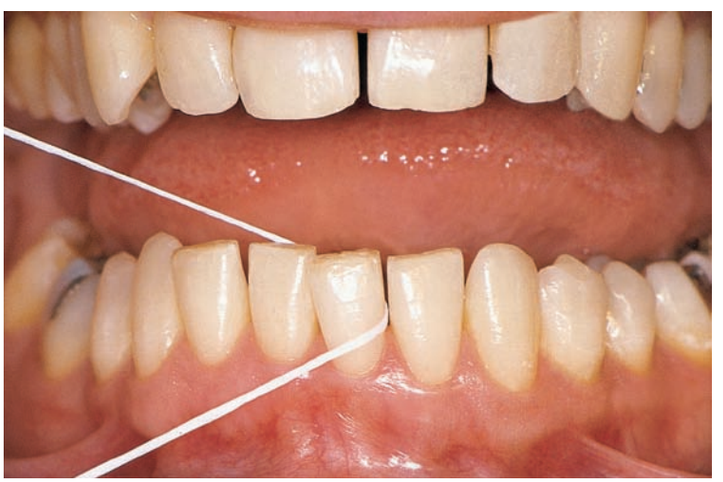
Following Treatment
The number of bleeding sites was reduced from 69 % to 9 % by means of local therapy alone. In the depicted region, there are only three sites that exhibit mild, point bleeding upon probing.



After

561 Six Months later
Gingivitis has been eliminated. The gingiva exhibits normal form, color and contour. It is coral pink, keratinized and mildly stippled. Plaque is virtually absent.

This patient brushes the teeth using a hand toothbrush and the modified Bass technique, and cleans the interdental areas with dental floss. The patient now understands the cause of her gingivitis, and therefore can prevent recurrence (*tertiary prevention*).



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Phase 1 Therapy

- **Initial Therapy 2—Causal, Antimicrobial Therapy**
- **Traditional, Non-Surgical Pocket Treatment**
- **FMT—Pharmacologically Supported "Full Mouth Therapy"**

Actual Phase 1 therapy—the second component of initial treatment—includes closed debridement of periodontal pockets. This is also referred to as *conservative* therapy, in contrast to periodontal surgery (p. 295), and also as "non-surgical" therapy. The following procedures comprise this aspect of treatment:

- Debriding the root surfaces of plaque and calculus
- Removing endotoxin-containing cementum layers (?)
- Root planing
- Possible soft tissue curettage
- "Full mouth therapy"

Definitions

Nomenclature for these procedures is not internationally uniform. Presented here is a recapitulation of definitions (AAP "Glossary of Periodontal Terms," 2001):

Debridement—Plaque and Calculus Removal (Scaling)
Removal of non-adherent and adherent biofilm as well as calcified plaque (calculus) from gingival or bony pockets, without modifying the root surface.

Detoxification of the Root Surface
Mechanical or chemical detoxification of the root surfaces. Removal of any remaining endotoxin-containing layers of cementum. This procedure is not necessarily differentiable from subgingival calculus removal on the one hand, or root planing on the other.

Root Planing
Smoothing the root surfaces using curettes and possibly also diamond burs. This procedure is also not a distinct entity vis-à-vis subgingival cleaning and planing of root surfaces.

Gingival Curettage
Removal of the pocket epithelium and infiltrated subepithelial connective tissue (often classified as surgical therapy).

Conservative Therapy—"Closed Curettage"
All of the above-defined procedures are performed without reflecting the soft tissue, i. e., without direct vision into the pocket or onto the root surface (as of today; cf. p. 282).

"Open Curettage"
After creating a gingival flap (modified Widman procedure, p. 309), the gingiva is reflected to such an extent that root planing can be performed with direct vision. This is classified as a surgical procedure.

"Full Mouth Therapy"—FMT
Definitive antimicrobial, closed, non-surgical, *mechano-pharmacologic* pocket therapy of the entire oral cavity (FDIS—"full mouth disinfection") within 24 hr, to avoid re-infection and microbial transmission.

The differences between the treatment measures in Initial Therapy 1 (*supragingival* plaque and calculus removal) and Initial Therapy 2 (*sub*gingival) are not sharply defined.

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Non-Surgical, Anti-Infectious Therapy—Goals of Treatment

The goal of traditional, non-surgical therapy is the elimination of the microorganisms responsible for periodontal destruction, from the pocket and surrounding tissues. The creation of a clean tooth and a clean, biologically compatible root surface that is as smooth as possible, and the removal of diseased or infected tissues are essential to therapy (Frank 1980, Saglie et al. 1982, Allenspach-Petrsilka & Guggenheim 1983, O'Leary 1986, Adriaens et al. 1988, Petersilka et al. 2002).

Removal of the pocket epithelium and portions of the infected connective tissues was a matter of controversy until recently; current research results clearly demonstrate the possibility of bacterial colonization of pocket epithelial cells (intracellular) and of connective tissue components. The most frequently encountered colonizers are *A. actinomycetemcomitans, P. gingivalis, T. denticola* and, in addition to the acknowledged pathogens, also *Streptococcus constellatus* (Socransky & Haffajee 2002).

Today's question? Should the pocket epithelium be removed in addition to removal of biofilm from the soft tissue pocket wall?

562 Principles of Conservative Pocket Therapy

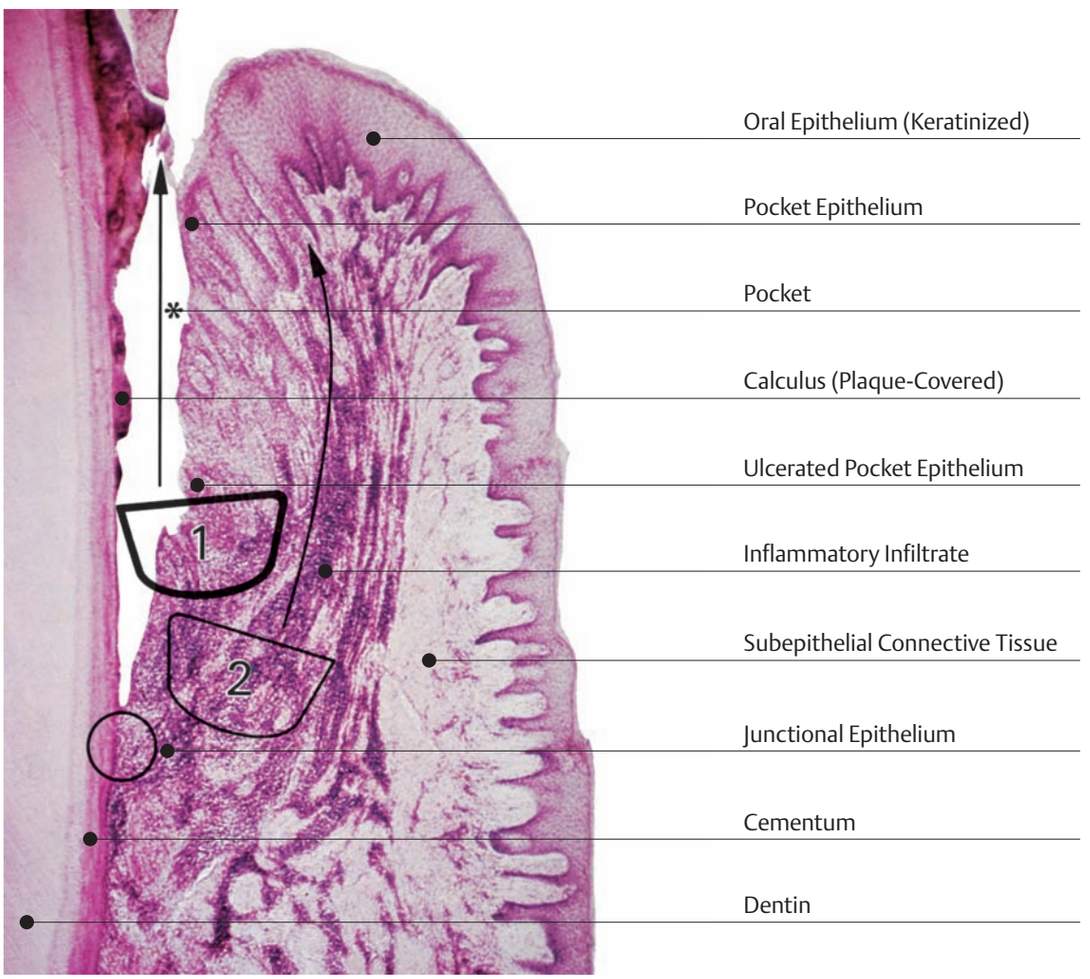
This stained histologic section through a gingival pocket (HE, 20x) will be used to illustrate *root planing* (*debridement*) and *soft tissue curettage*.

Soft tissue curettage (**2**) is never used alone. True "causal" therapy includes the elimination of microorganisms wherever they have become organized as a biofilm.

It is imperative that the root surface be thoroughly debrided (**1**).

1 Scaling and Root Planing
The unilaterally sharp Gracey curette (**1**) removes plaque, calculus, endotoxin-containing cementum and sometimes dentin from the root surface. The arrow indicates the direction toward which the curette tip is pulled.

2 Gingival Curettage
A Gracey curette sharpened on its other edge (**2**) is used to remove pocket epithelium and remaining (apical) junctional epithelium, as well as the infiltrated connective tissue.



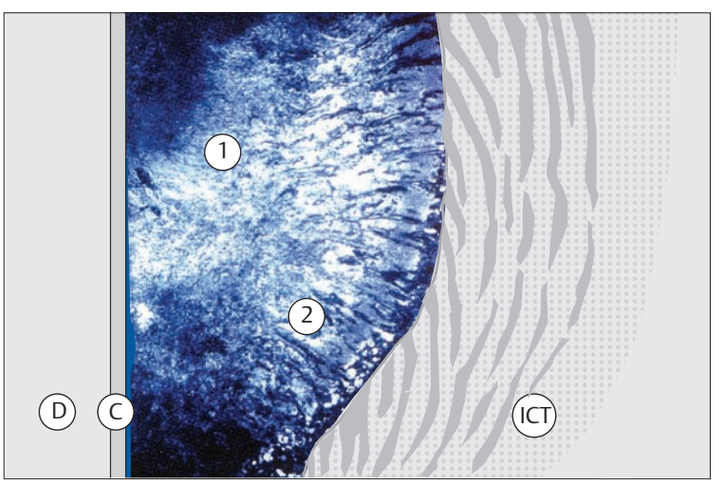
563 Subgingival Biofilm at the Base of a Pocket

Histologic section, plaque stained with toluidin blue and methylene blue, modified.

Of special interest is the biofilm formation on the soft tissue wall.

1 *Biofilm*/plaque on the root surface
2 *Biofilm* attached to the soft tissue wall!
D Dentin
C Cementum
ICT Infiltrated connective tissue

Courtesy *M. Listgarten*



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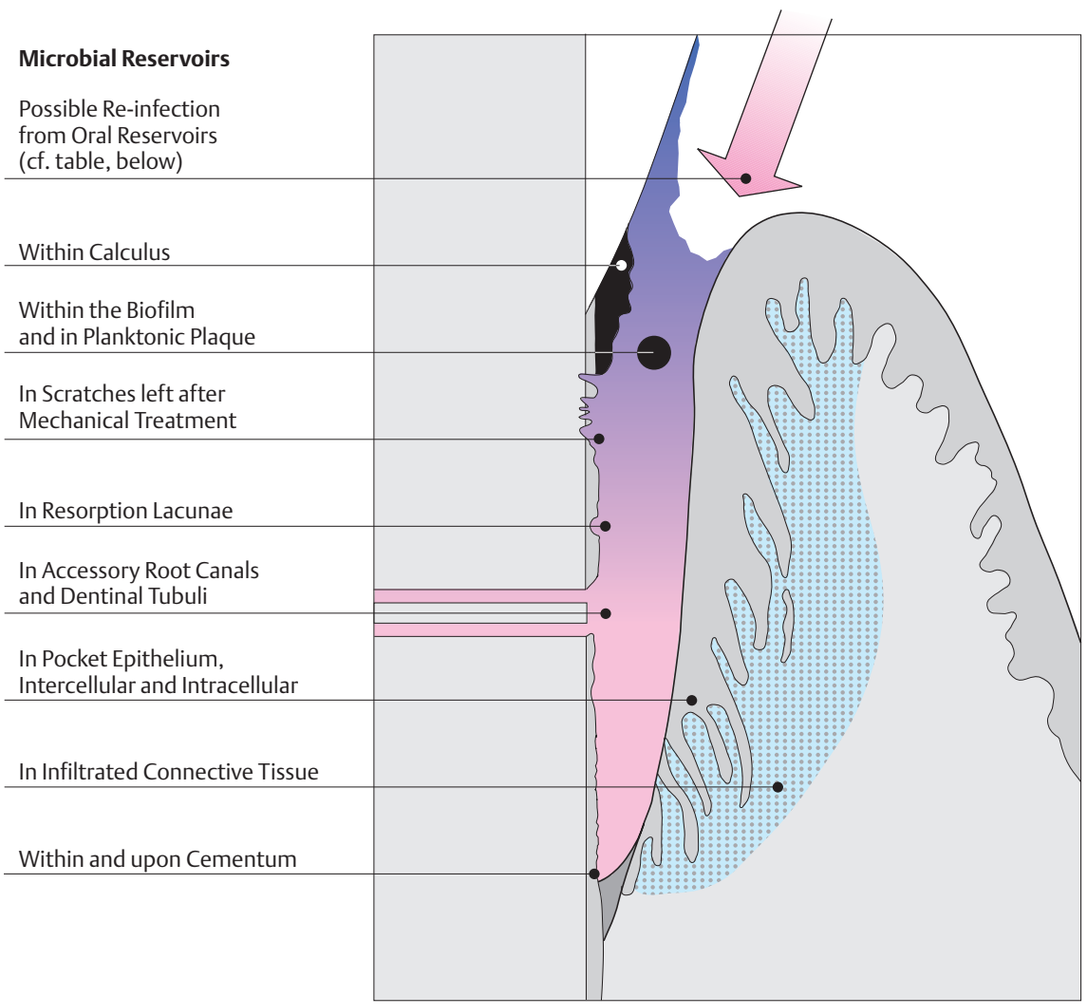
Antimicrobial Therapy—Combating the Reservoir

The answer to the question posed on the previous page would be: *Yes*. However, the AAP (2002) in its "Academy Statement Regarding Gingival Curettage," concluded that soft tissue curettage has *no* additional effect beyond scaling and root planing.

In any case, it is most important that purely mechanical therapy as a means to achieve the ultimate goal—periodontal healing—should be enhanced by the use of all antimicrobially effective measures.

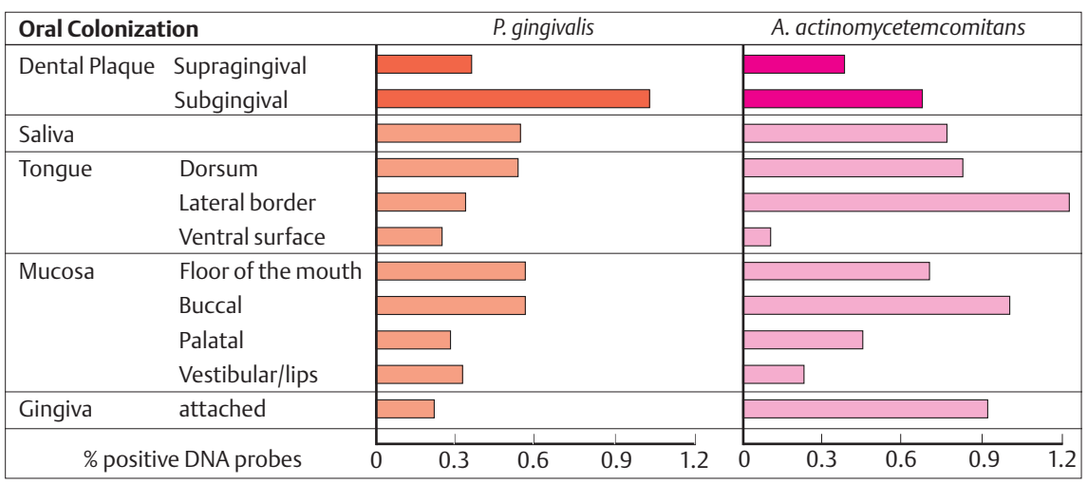
The initial question concerns the bacterial reservoirs in the ecosystem represented by the oral cavity. The diagram below (Fig. 564) demonstrates the possible niches, the plaque-retentive areas in which periodontopathic microorganisms may be harbored.

Such microorganisms can rapidly contaminate (re-colonize) a freshly-treated pocket, and thus compromise the treatment results. Therefore, such bacterial reservoirs must also be "treated," especially in highly susceptible patients.



564 Oral Microbial Reservoirs
Increases in plaque accumulation occur wherever microorganisms find a secure and undisturbed niche with good nutritive sources. Clusters of microbial colonies are always found in the small scratches that remain following root surface debridement, as well as the deep crypts in tonsils and on the dorsal surface of the tongue; many such clusters harbor the well-acknowledged periodontopathic anaerobes. Even a 0.5 mm thick biofilm provides oxygen-free (anaerobic) conditions for these microbes.

Modified from *P. Adriaens 1995*



565 *P. gingivalis* and *A. actinomycetemcomitans* in the Oral Reservoirs
Occurrence of the two species in 24 patients.
The presence and relative concentrations support the thorough cleaning of the tongue surface (pp. 237 and 281), and not only for patients with halitosis.

Modified from *Socransky et al. 1999*

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Root Planing—With or without Curettage?

The primary goals in pocket treatment are removal of the biofilm and thorough debridement of the root surfaces.

Following elimination of non-adherent and adherent plaque, all subgingival calculus is removed. The superficial layers of the root cementum contain endotoxin. This lipopolysaccharide (LPS) from gram-negative bacteria can inhibit connective tissue regeneration and reestablishment of the periodontal ligament to the root surfaces. For this reason, root planing should be performed into "healthy" (hard) cementum or dentin layers.

After the root surface is thoroughly planed, the "peeling out" of the pocket epithelium and infiltrated connective tissue can be accomplished. If the curettes used for root planing are sharp on both edges (universal curettes), some soft tissue curettage will be accomplished inadvertently while the hard tooth structure is being planed.

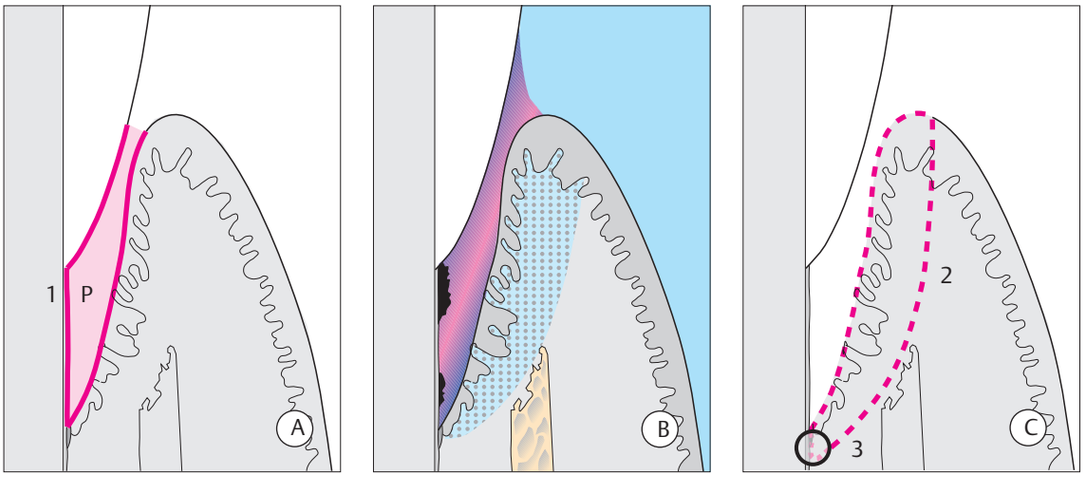
The goals of these procedures include elimination of infection within the pocket and the pocket epithelium, and the ultimate healing of the periodontal lesion.

566 Root Planing and Curettage—Principles

A Cleaning the Root and the Pocket:
The root surface is treated (**1**), and the plaque (**P**) is removed from the pocket.

B Original Pocket:
With calculus, adherent plaque and non-adherent microorganisms.

C Soft Tissue Curettage:
In addition, but never as the sole therapy, pocket epithelium/infiltrate (**2**) and junctional epithelium (**3**) are removed.



Effects of Subgingival Debridement—Pocket Healing or Recolonization?

The goal of closed anti-infectious therapy is the complete healing of all periodontal pockets, but this goal is seldom achieved. Access and vision are severely limited during closed instrumentation. Almost always, here and there, residual pockets of varying depths persist (Badersten 1984; p. 280). The so-called "critical depth" of residual pockets is 4–5 mm. Such pockets offer anaerobic conditions, which provide the well-known pathogenic, gram-negative anaerobic microorganisms a favorable environment. Remaining deeper pockets can serve as a bacterial reservoir for the recolonization of residual pockets. Patients who harbor residual pockets should be maintained in a strict recall schedule to control or eliminate such pockets.

Subgingival instrumentation (debridement) normally removes about 90 % of the bacteria from a pocket, including both "favorable" and pathogenic flora: The processes of healing and recolonization are in competition with each other, and residual pockets usually persist.

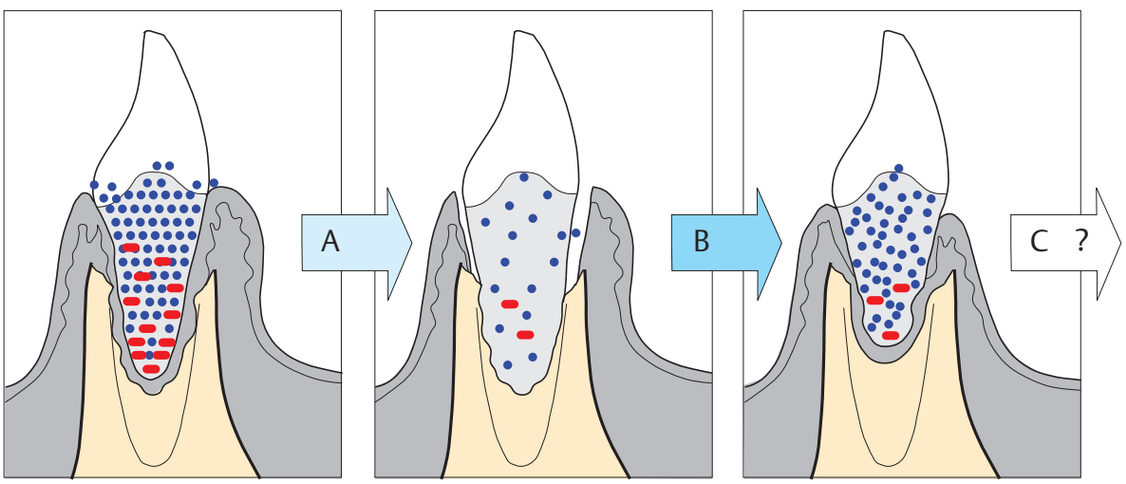
The favorable effect of closed pocket treatment is that the non-pathogenic flora recolonizes the pocket faster than the pathogenic microorganisms (Fig. 567; Petersilka et al. 2002).

567 Pocket Instrumentation—Schematic of Residual Pockets

A Untreated pocket:
Non-pathogenic (blue) and pathogenic (red) pocket flora.

B Following instrumentation:
The pocket flora is dramatically reduced.

C Recolonization:
The percentage composition of the "blue" (non-pathogenic) has increased; these or the host response have kept the "red" (pathogens) in check.



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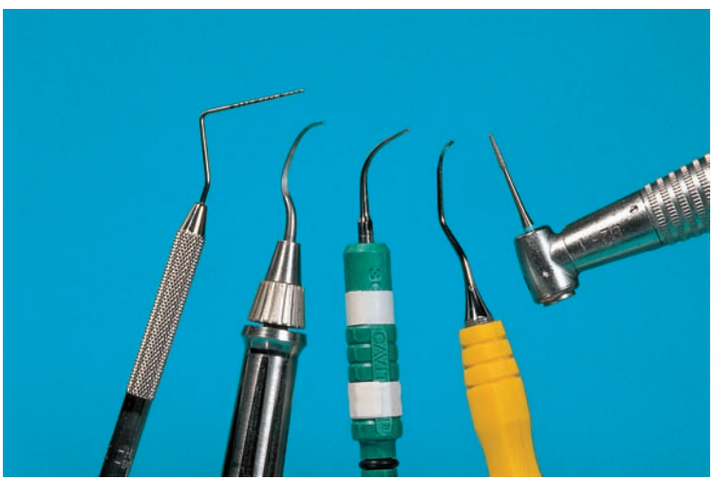
Closed Therapy—Indication, Instrumentation

It has been stated many times: Periodontal diseases should be prevented, or, failing prevention, should be diagnosed and treated early on.

Standard methods for the treatment of mild and moderate periodontitis include closed, non-surgical, anti-infectious pocket treatment (scaling and root planing). This approach is effective, tissue-friendly (minimal recession), less hemorrhagic, and routinely results in favorable treatment results. Perhaps even more important today, it is *affordable* for the (informed) patient.

For the dentist and her/his therapeutically active dental hygienist, closed therapy is the *definitive* therapy for uncomplicated cases, and represents the initial therapeutic approach for complex, advanced cases.

Contraindications for this approach are rare (patients on anticoagulant medications, risks for focal infection, systemic diseases).



568 Instrumentarium for Mechanical/Anti-Infectious "closed" Therapy
These types of instruments are required for all patient treatments:

- **Periodontal probe**
- **Sonic scaler**—supragingival
- **Ultrasonic device**—pockets—subgingival
- **Curettes**—scaling, polishing
- **Motor-driven special instruments**—furcations, grooves, etc.



569 Chemical/Pharmacologic Adjunctive Therapy
The paradigm shift toward anti-infectious/antimicrobial therapy includes disinfectants and antibiotics more and more as co-therapeutic agents, above all for the treatment of aggressive cases.

- **Disinfectant agents**
  FMT (pp. 235, 283)
- **Local CRD**
  "Controlled release drugs" (p. 292)
- **Systemic antibiotics**
  (p. 288)

Conclusions

The goals of subgingival scaling are simple:
- Complete removal of biofilm and calculus
- Root planing (to reduce new plaque formation)
- Creation of a bioacceptable root surface (chemical conditioning with various substances, following mechanical treatment).

Subgingival scaling is a technically difficult endeavor, because it is performed without direct vision. Even the experienced clinician will not always effectively treat all root surfaces, nor completely remove all plaque and calculus from all surfaces.

Today, the question is: How can we *improve* the "gold standard" of closed causal therapy and subgingival scaling?
Most recently, in addition to the classical, mechanical instrumentation, topical antimicrobial agents have been successfully employed (disinfectants, antibiotics); however, such adjunctive measures will only be helpful when used in combination with thorough scaling and root planing! Systemic antibiotics may often be indicated in severe, aggressive cases (p. 287).
A new television technique may bring "light and vision" into the periodontal pocket! The Dental View device can dramatically reduce the amount of "missed" subgingival calculus.

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Hand Instruments for Scaling and Root Planing—Curettes

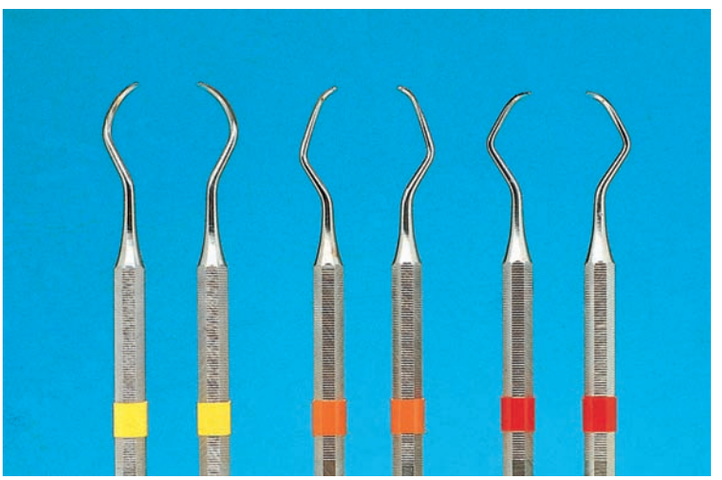
For the removal of large subgingival calculus deposits, curettes are indicated, in addition to sonic and ultrasonic devices (p. 259). For root planing and soft tissue curettage, curettes are the instruments of choice.

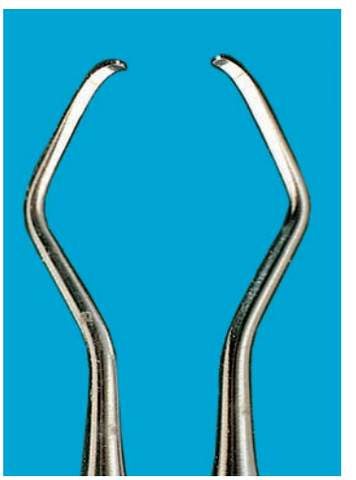
Numerous manufacturers offer a myriad of hand instruments, which may vary with regard to quality (e. g., steel) and design. In this *Atlas*, we do not make recommendations concerning specific manufacturers or instrument sets, because it is acknowledged that every "school" as well as each and every dentist has its/her/his favorite instruments. Most important is that a set of curettes must provide effective approaches to all root surfaces, while providing the proper angle of application of the blade to the root surface (ca. 80°). Curettes must be sharpened before each use (p. 268).

It is important to note the difference between universal curettes, which have two cutting edges, and Gracey curettes, which have only one cutting edge. Gracey curettes are primarily indicated for debridement and root planing, and less often for soft tissue curettage.

570 Curettes
(Deppeler Co.)

- **Universal curettes, ZI 15**
  Yellow; for initial gross debridement
- **Anterior curettes, GX 4**
  Orange; for anterior teeth and canines, and sometimes premolars
- **Posterior curettes, M 23 A**
  Red; for premolars and molars



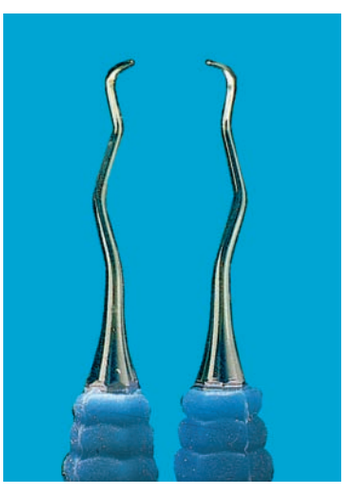


Right: Working end of the paired posterior curettes, M 23A.

571 Minimum Gracey Set
In most clinical cases, a set consisting of four double-ended Gracey instruments is sufficient. Depicted here are color-coded curettes with soft grips (ADEC, Deppeler).

- **Gracey 5/6** yellow
- **Gracey 7/8** gray
- **Gracey 11/12** red
- **Gracey 13/14** blue

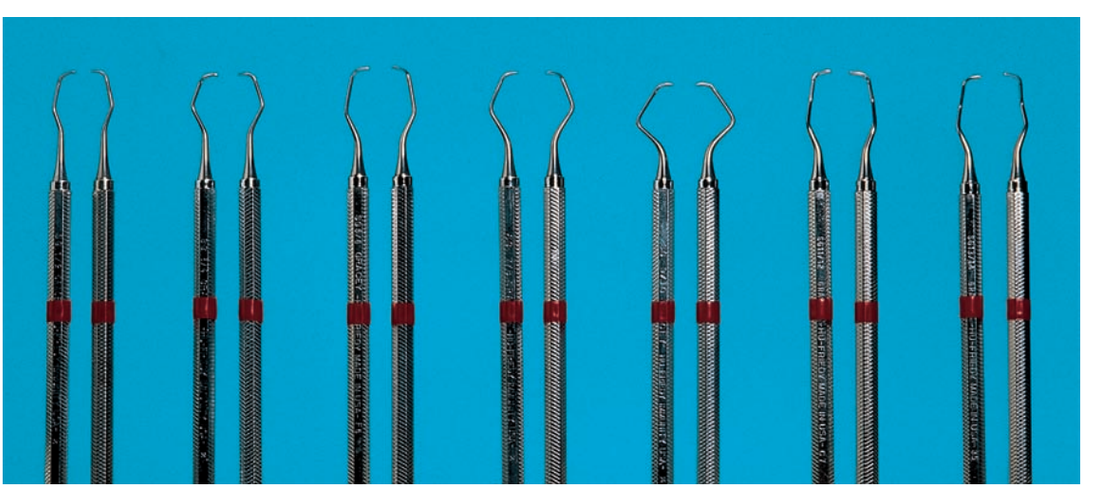




Right: Double-bend shank and working end of the paired Gracey curettes 13/14.

572 Complete Set of Gracey Curettes: 7 Double-Ended Instruments
(Hu-Friedy Co.)

- **Gracey 1/2**
- **Gracey 3/4**
- **Gracey 5/6***
- **Gracey 7/8***
- **Gracey 9/10**
- **Gracey 11/12***
- **Gracey 13/14***



* These four instruments comprise the minimum set (Fig. 571).

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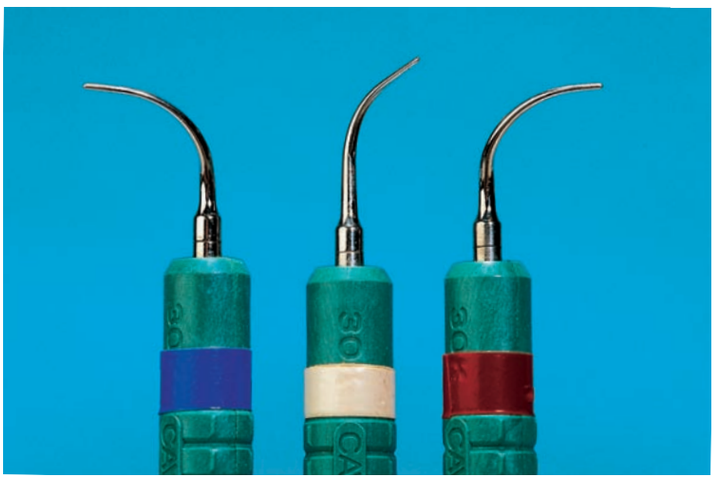
Powered Instruments for Debridement

In addition to the classical hand instruments, powered devices are finding increasing use in practice today for supra- and subgingival periodontal debridement. Included are:

- Ultrasonic devices (20–50,000 Hz)
- Sonic devices (up to 6,000 Hz)
- Motor-driven devices incorporating diamond-coated tips.

The goal, with both hand instruments and powered devices, is to create biologically acceptable root surfaces. Calculus must be completely eliminated, but without creating root surface roughness; rough root surfaces are more quickly colonized by bacteria than smooth surfaces. If used properly, curettes and ultrasonic devices can achieve relatively smooth surfaces; on the other hand, sonic and motor-driven devices more often elicit roughness (Römhild 1986; Schwarz et al. 1989; Ritz et al 1991; Axelsson 1993; Kocher & Plagmann 1997). Rough but clean tooth surfaces, especially in the region of the gingival margin, should always be given the "finishing touch" with curettes to ensure smoothness, and to inhibit or delay re-infection of the pocket.

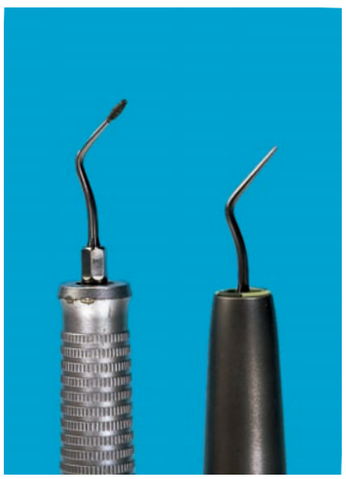


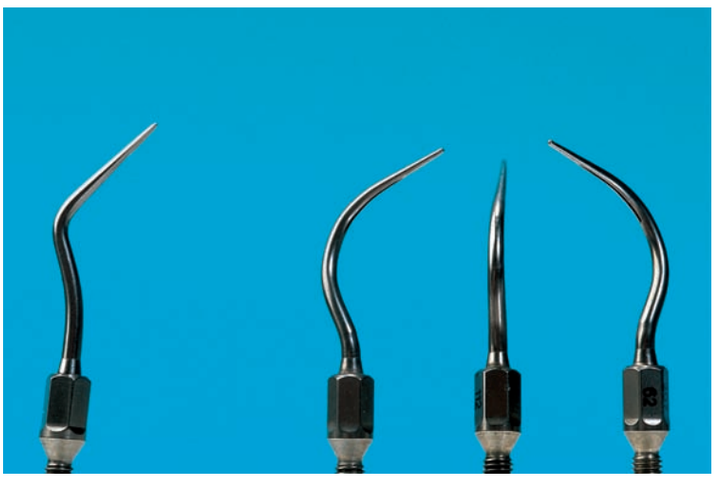


573 Ultrasonic Device
Ultrasonic devices function in direct contact with the tooth surface according to the principles of cavitation and acoustic microstreaming. Examples:

- **Dentsply**
  Pictured: Slimline attachments
- **EMS**
- **Satelec**

Left: **Dual Select** (Dentsply). With this attachment, one can select water or one of two antimicrobial cooling solutions.

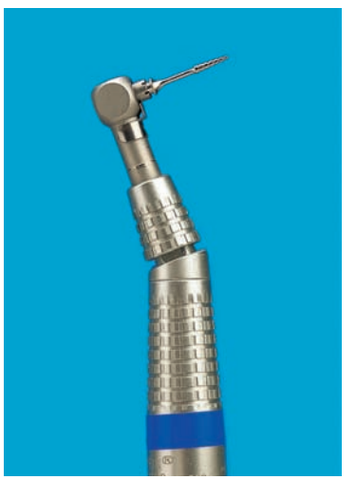


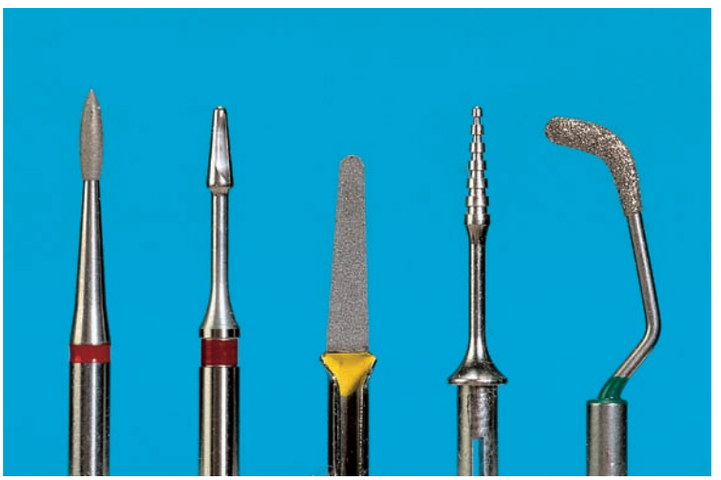


574 Sonic Devices—KaVo Sonicscaler—Insert No. 8 with Elongated Tip
No. 60, 61 and 62 are special tips for subgingival application.

Examples of sonic devices:
- **Sonicscaler, KaVo**
- Airscaler, Titan-S
- **Siroson, Siemens**

Left: Working tips—overview. Diamond-coated tips for working in furcations.





575 Additional Motor-Driven Instruments for Subgingival Application

- **Perioset—diamonds** (Intensive SA)
- **Scalex tips** (Rotex)
- **EVA-System—Proxoshape** (Instensiv SA)
- **Peri-O-Tor** (Dentatus)
- **Perioplaner, Periopolisher** (Mikrona)

Left: Handpiece with the Peri-O-Tor attachment.

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Gracey Curettes—Areas of Use

Complete Set

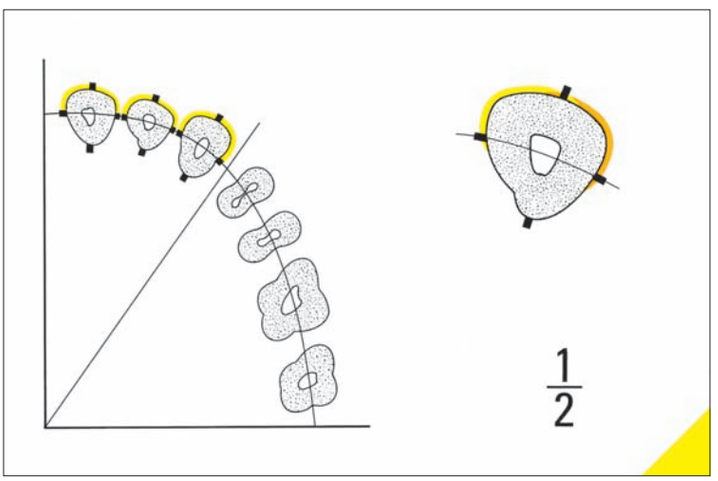
For closed ("blind") subgingival scaling and root planing, special instruments are indicated that are adaptable to the most varied root shapes. As early as the 1930's, Dr. C.H. Gracey, a dentist, together with an instrument maker by the name of Hugo Friedman (Hu-Friedy!), conceptualized a set of instruments that "…gives every dentist the possibility to treat even the deepest and least accessible periodontal pockets simply and without traumatic stretching of the gingiva. In addition, these curettes make it possible to completely remove all subgingival calculus, and to perfectly clean and plane every root surface, which will enhance subsequent tissue adaptation and re-attachment."

Numerous modifications of the instruments led finally to the Gracey curettes of today. The use of these instruments has been described in detail (Pattison & Pattison 1979, Hellwege 2002).

Anterior Teeth to the Premolars
Figs. 576–582 demonstrate the systematic use of the original Gracey curettes (GRA) in quadrant 2 (maxillary left).

576 Gracey 1/2—Incisors, Canines
The primary area of use for this paired instrument is the *facial* root surfaces of incisors and canines.

Right: **GRA 1/2**
Medium length shank, mild angulation

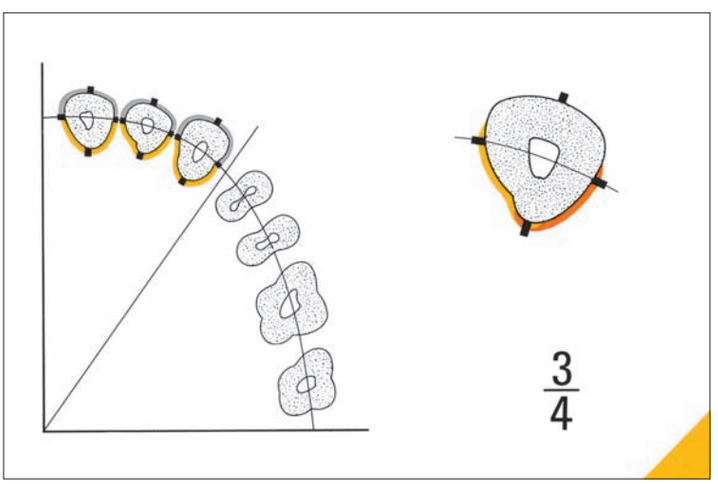




577 Gracey 3/4—Incisors, Canines
Primary use is the same as with GRA 1/2, but because of the more severe angulation, the 3/4 is particularly indicated for *palatal* and *lingual* surfaces.

Right: **GRA 3/4**
Shorter shank, sharper angulation.

The cutting edge of these paired instruments is the "outer" edge, along the *convex* curvature of the working tip.



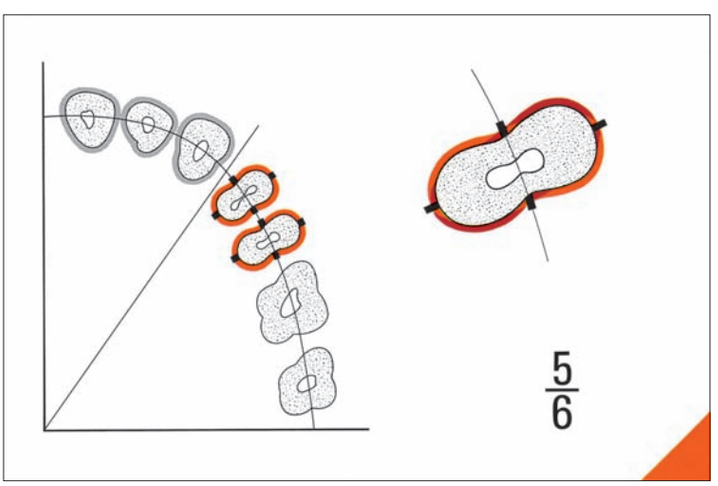


578 Gracey 5/6*—Anterior Teeth and Premolars
The area of use corresponds for the most part to that of an anterior universal curette.
This Gracey curette with its long, straight shank can be used in virtually all areas of the dentition where deep pockets exist.

Right: **GRA 5/6**

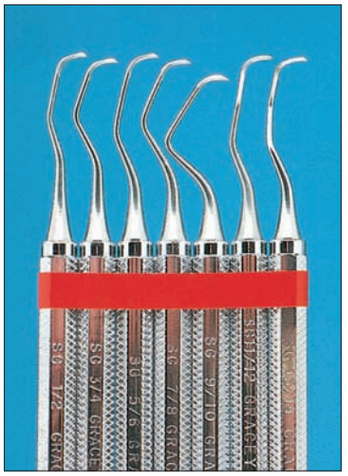
Longer shank, slight angulation.

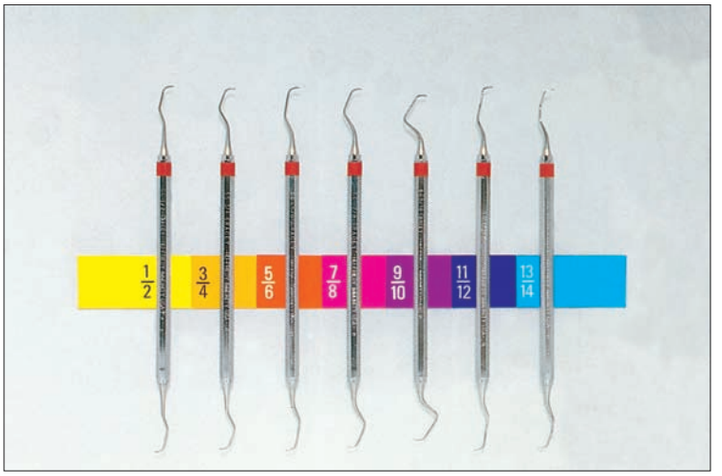
* *The four double-ended instruments marked with an asterisk comprise the minimum Gracey set (cf. Figs. 571 and 586).*





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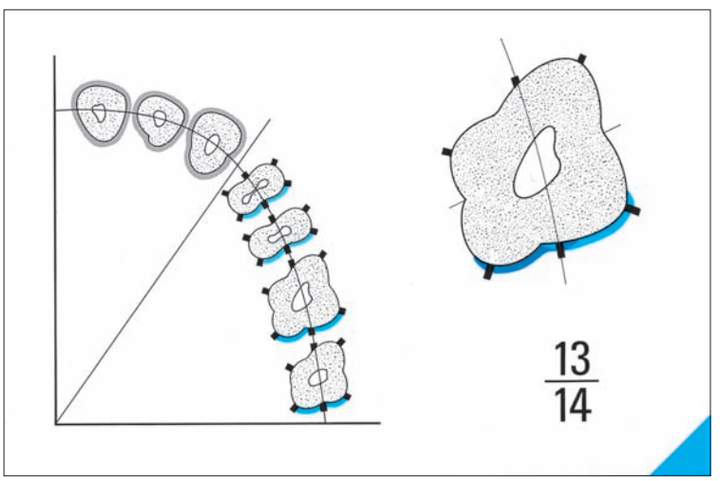
579 Complete Gracey Curette Set
(original Hu-Friedy Co.)

GRA	**1/2**	**yellow**
GRA	**3/4**	**orange**
GRA	**5/6***	**red**
GRA	**7/8***	**magenta**
GRA	**9/10**	**purple**
GRA	**11/12***	**violet**
GRA	**13/14***	**blue**

(Color-coding valid for this double page.)

Left: The seven different working ends of the complete Gracey set.

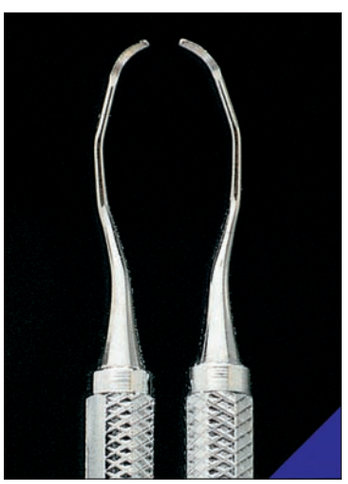


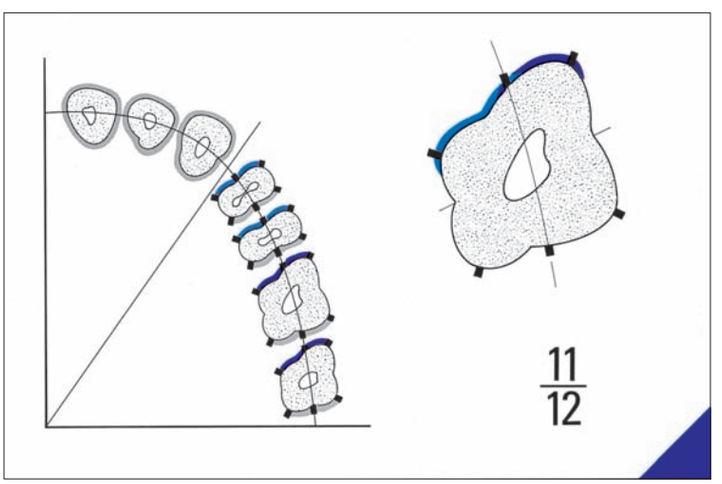


Posterior Area

580 Gracey 13/14*—Molars, Premolars—Distal
First, for example, all *distal surfaces* are cleaned, from the facial (buccal) aspect and then from the oral (palatal/lingual) aspect. The *line angles* are indicated by short bars. This is the transition from one tooth (root) surface to another, where the use of individual instruments changes.

Left: **GRA 13/14***
Triple-bend shank.

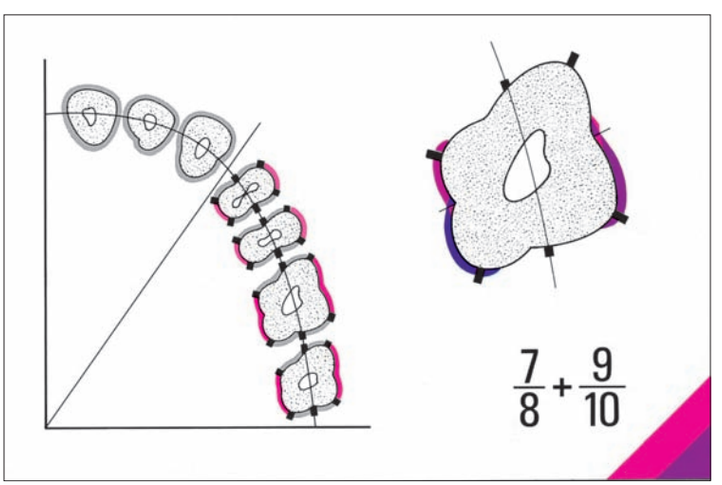




581 Gracey 11/12*—Molars, Premolars—Mesial
This instrument, with its sharp convex cutting edge, is indicated for treatment of all *mesial surfaces* from the buccal aspect, and the other end for mesial surfaces from the oral aspect.
Like the GRA 13/14, the GRA 11/12 is particularly well suited for multirooted premolars as well as in furcations and depressions.

Left: **GRA 11/12***
Longer shaft with several mild angulations.





582 Gracey 7/8* and 9/10—Molars and Premolars—Facial and Oral Surfaces
Due to the rather severe angulation of the longer shank, both instruments, in addition to their primary indication on buccal and oral surfaces in the posterior area, are also useful in deep depressions and furcations. The angulation permits axial, oblique and horizontal strokes.

Left: **GRA 7/8***
Medium length, severely angled shaft.

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Hand Instruments for Special Problems—Curettes

Closed debridement, whether as definitive treatment for mild periodontitis or as initial therapy prior to surgical intervention, is a demanding task that often includes scaling and root planing of poorly-accessible, irregular root surfaces in deep pockets and involved furcations, even in patients with minimal mouth opening capacity. These difficulties will also be encountered when mechanical debridement is performed in tandem with antimicrobial irrigation ("full mouth therapy," p. 218).

Problems and problem areas include:

- Deep, narrow pockets, e. g., on anterior teeth
- Distal pockets
- Patients with minimum mouth opening
- Substantial furcation involvement (p. 381)
- Dental implants in periodontitis-susceptible patients

Caution: Manufacturers offer hundreds of different hand instruments! The clinician is wise to limit the number of instruments to only those that are necessary for successful treatment!

583 Special Gracey Curettes
Several special variations of the classic Gracey curettes are available for use in demanding situations (exemple: 11/12; Hu-Friedy):

- **Type SGR** "rigid": Incorporating a wider and more rigid shaft and working end
- **Type SG** "normal": With an elastic shaft
- **Type SRPG** "after 5": Elongated shaft for depths up to 8 mm
- **Type SAS**: Longer, finer working end; anterior teeth

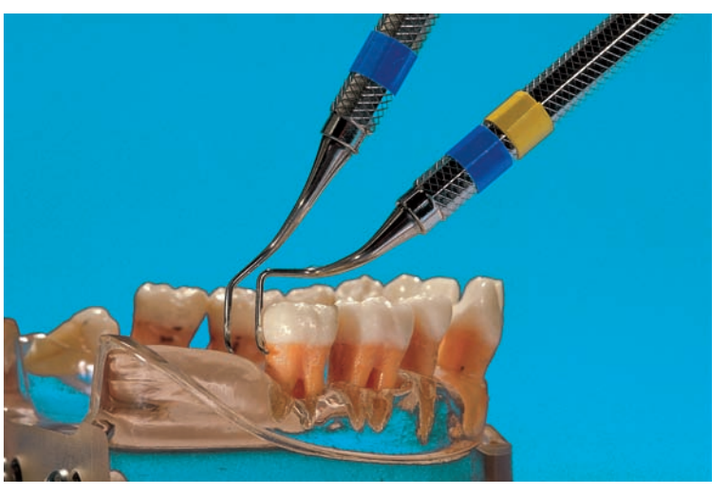
Right: Comparative view.





584 Enhanced Angulation
Gracey curettes 11/12 (mesial) and 13/14 (distal) require that the patient open wider for treatment of second and third molars. Depicted is the 13/14 (blue collar) with normal angulation of the shaft; the new number 17/18 (blue/yellow) can be used even with minimum mouth opening.

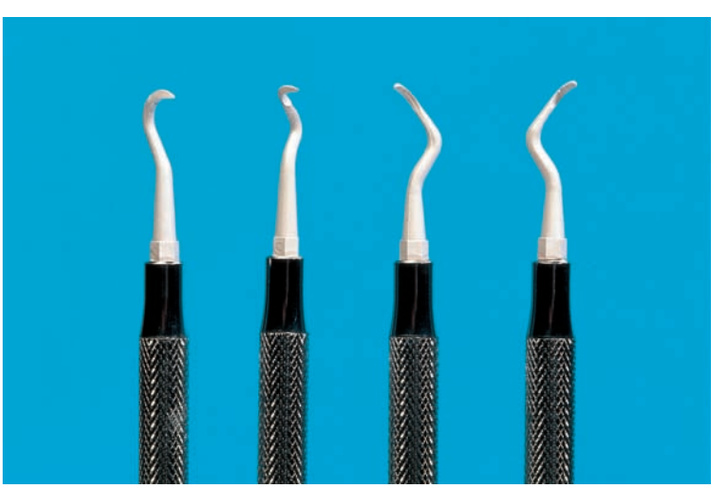
Right: Different angulation of the working end.





585 Plastic Instruments
For the treatment of the surfaces of titanium dental implants, the clinician must employ instruments that will not damage the fixture surfaces. Especially in cases of *mucositis* and also *peri-implantitis*, the accessible titanium surfaces should be cleaned using only *plastic* scalers.

Left: Curette tips (Hu-Friedy); *right*, probe (Deppeler) and carbon fiber curette (HaWe).





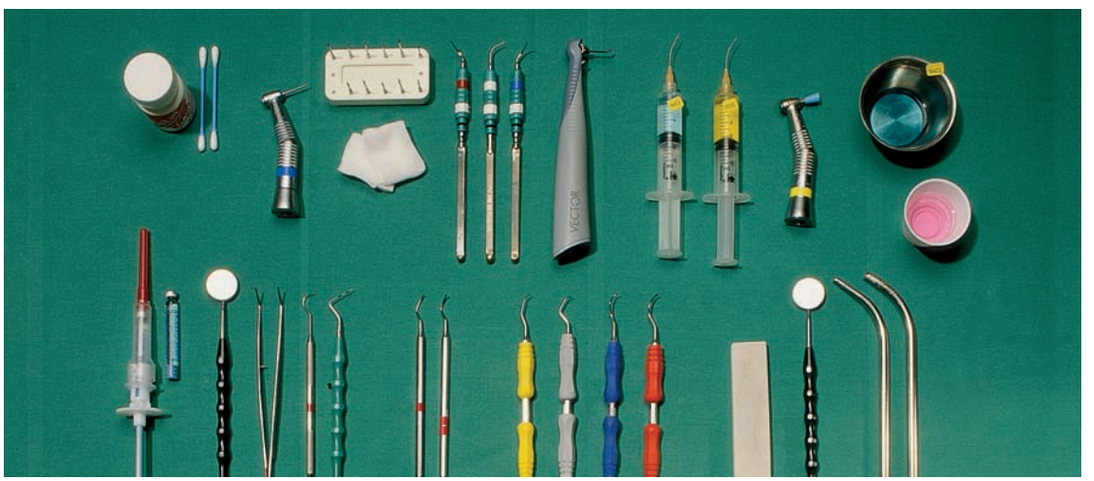
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Practical Scaling Technique with Gracey Curettes—Systematic Approach

Minimum Gracey Set

An introduction to the scaling technique using Gracey curettes is simplified by limiting the instrument set to four double-ended instruments (p. 258, Fig. 571). Color-coded handles simplify their assignment to certain tooth surfaces. In addition to an adequate armamentarium, certain prerequisites must be fulfilled in order to optimally perform the technically difficult scaling procedures, such as patient position, operatory arrangement, and operator position (Wolf 1987):

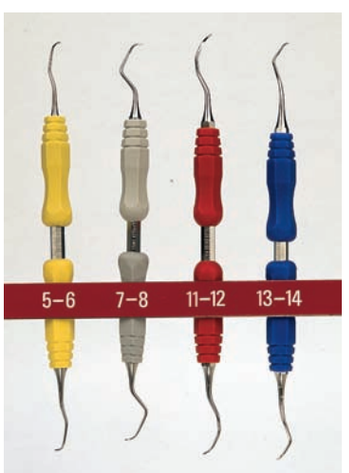
- Positioning of the patient and the clinician
- Operatory lighting
- Sharp instruments and selecting proper cutting edge
- Secure stroke (modified pen grasp and fulcrum)
- Precise knowledge of all probing depths
- Systematic procedure, step-by-step
- Detecting roughness

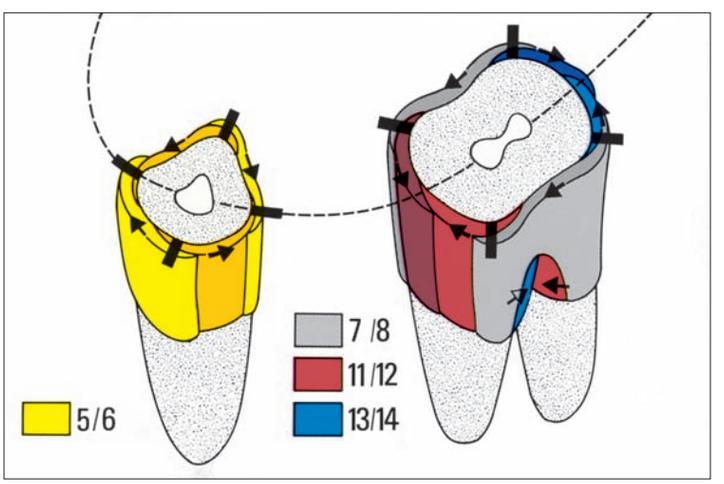


586 Basic Instrumentarium for Scaling—Complete

Above:
- Ultrasonic tips
- PerioSet diamonds
- Rinsing solution

Below:
- Anesthesia
- Mirror, forceps, pointed probe
- Periodontal probe
- Scaler, universal curettes
- **Minimum Gracey set**
- Sterile sharpening stone etc.





587 Minimum Gracey set: Areas of Use, Color Codes

Gracey 5/6 anteriors/canines	yellow
Gracey 7/8 premolars and molars, buccal and oral	gray
Gracey 11/12 molars and premolars, mesial, furcations	red
Gracey Gracey 13/14 molars and premolars, distal, furcations	blue

Left: Minimum color-coded set.

Checklist—Scaling Technique

Operatory:	Light, power, water, air, instrumentarium
Clinician:	Eye protection, mask, gloves
Treatment technique:	Operator position, instrument selection, fulcrum, direct or indirect vision (mirror). Patient's periodontal data, including probing depths and radiographs.

The following pages, 264–267, demonstrate the systematic use of the minimum Gracey set in the maxillary left quadrant.

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5–6

Anterior Region, Facial

588 Scaling on Distobuccal Surfaces—Tooth 22
Patient's head: Reclined position, head inclined to the right

Dentist: ca. 9 o'clock position
Rest position/fulcrum: Intraoral, indirect, upon the thumb of the left hand
Vision of the operator: Direct

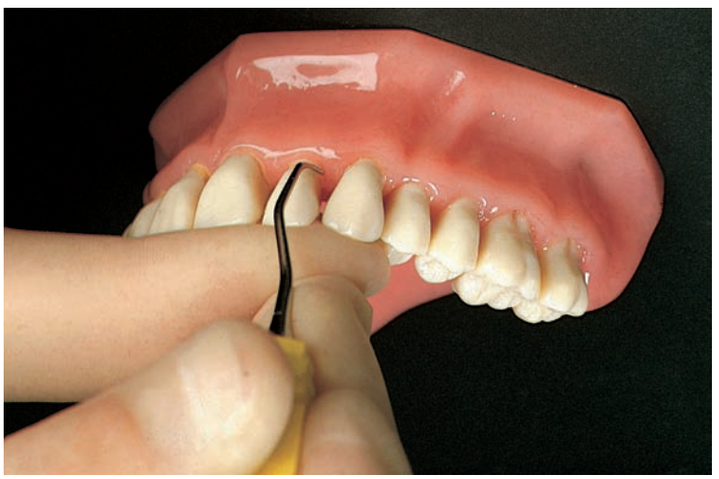
Right: GRA 5/6, working ends.





589 Model Situation
This indirect, intraoral rest position permits the fulcrum point of the working hand (4th finger) to be placed nearest to the root surface being treated.

Right: The cutting edge of the instrument cleans all distobuccal root surfaces, then the mesiobuccal surfaces are treated with the other blade of the double-ended Gracey 5/6 curette. Instrument changes are made at the indicated markings.



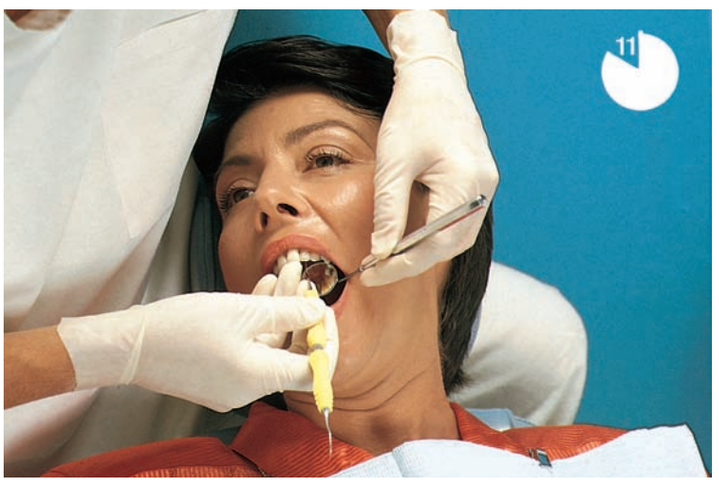


Anterior Region, Palatal

590 Scaling on Distopalatal Surfaces—Tooth 22
Patient's head: Extended to the right and dorsally

Dentist: ca. 11 o'clock position
Rest position/fulcrum: Intraoral, directly on tooth 21
Vision: Indirect (mirror)

Right: Parallel, 3–4 mm working strokes from the palatal toward the interdental area.





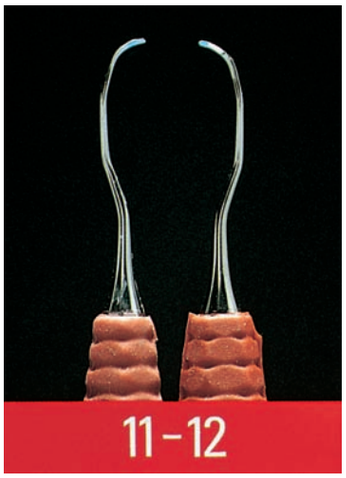
591 Model Situation
The mouth mirror permits indirect vision and insures proper lighting of the working field. Observe the modified pen grasp.

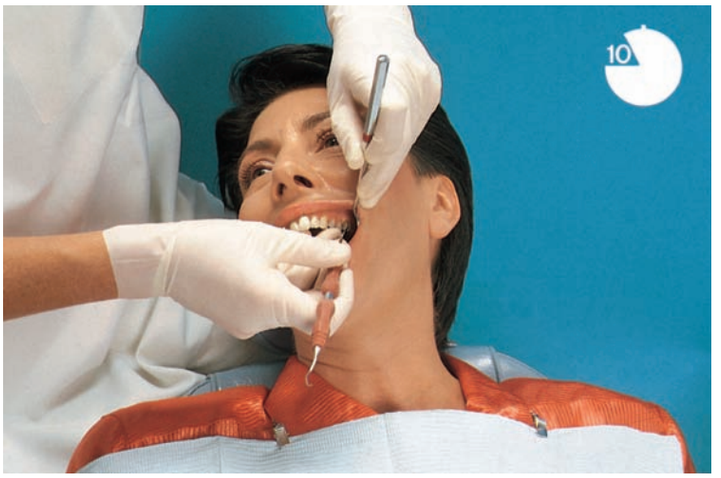
Right: Mesiopalatal and distopalatal root surfaces are approached using alternate ends of the 5/6 Gracey curette.





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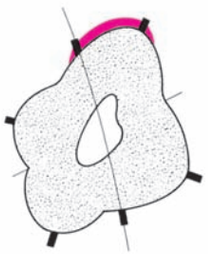
11–12

Posterior Segment—Mesial

592 Scaling on Mesial Surfaces, from the Buccal—Tooth 26
Patient's head: Inclined slightly to the right

Dentist: ca. 10 o'clock position
Rest position: Intraoral, directly on the adjacent tooth
Vision: Direct

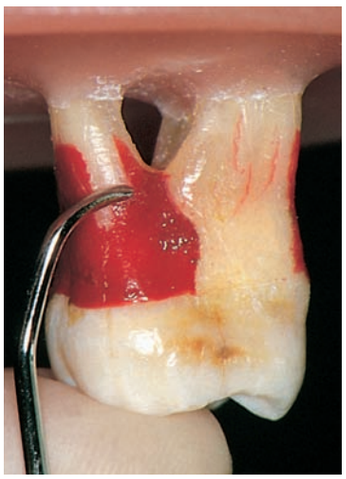
Left: Two working ends of the double-ended GRA 11/12 curette.





593 Model Situation
The ring finger establishes a fulcrum for the working hand, as near as possible to the mesial surface of tooth 26. The working strokes for subgingival scaling are initiated by a rotating movement of the *forearm* around the fulcrum.

Left: Section through tooth 26. GRA 11/12 is used from the buccal approach to scale the mesial surfaces from the mesiobuccal line angle, under the contact area, toward the palatal, including the mesial furcation.



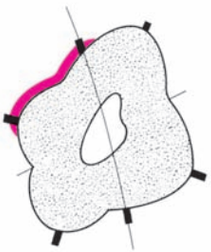


Posterior Segment—Mesial

594 Scaling on Mesial Surfaces from the Palatal—Tooth 26
Patient's head: Inclined to the left and back

Dentist: ca. 8 o'clock position
Rest position: Extraoral on the mandible or intraoral on the opposing arch. Guidance by the left thumb.
Vision: Direct

Left: Entrance to the mesial furcation can only be achieved from the palatal approach.





595 Model Situation
The left thumb guides and stabilizes the curette. Only light pressure is necessary for scaling the root surface if the instrument is properly sharpened.

Left: Section through tooth 26. The working area for the Gracey curette 11/12 is from the palatal approach.

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13–14

Posterior Segment—Distal

596 Scaling on Distal Surfaces from the Buccal—Tooth 26
Patient's head: Inclined far to the right

Dentist: ca. 10 o'clock position
Rest position: Intraoral, directly upon the adjacent tooth
Vision: Direct; mirror retracts the cheek.

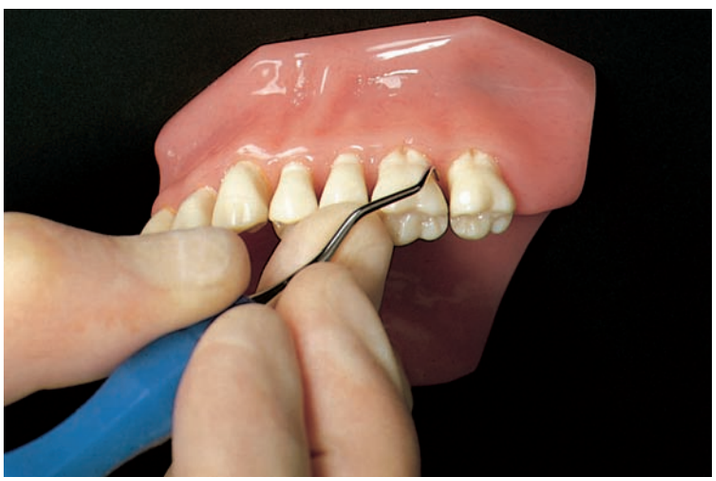
Right: GRA 13/14 working ends.

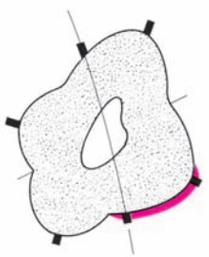




597 Model Situation
The rest position is with the ring finger on tooth 25, very near the working (distal) area of tooth 26. The shank portion immediately adjacent to the curette blade must be parallel to this tooth surface.

Right: Section through tooth 26. Contact points and the four line angles are depicted. From the buccal approach, the GRA 13/14 is indicated for the distal surface, from the distobuccal line angle to the contact area.





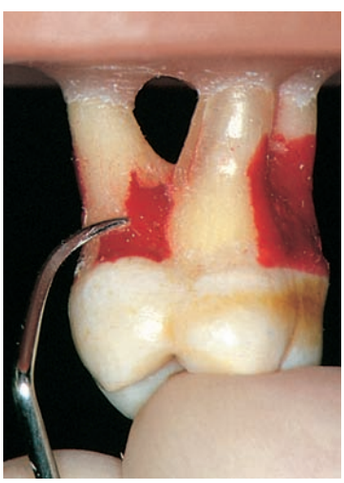
Posterior Segment—Distal

598 Scaling on Distal Surfaces from the Palatal—Tooth 26
Patient's head: Inclined to the left

Dentist: ca. 9 o'clock position
Rest position: Intraoral, indirect, on the back of the first finger of the left hand. This finger also serves to guide the instrument and apply pressure to it.
Vision: Direct

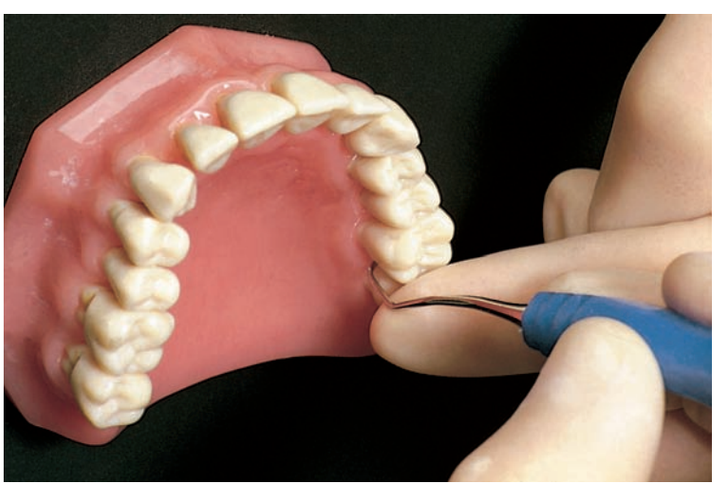
Right: The palatal root is treated from the palatal toward the contact area and the distal furcation.

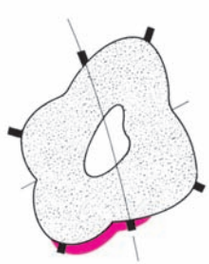




599 Model Situation
The first finger of the left hand serves two functions:
- Fulcrum for the working hand
- Guidance and lateral pressure for the curette

Right: Beginning on the palatal surface, the GRA 13/14 curette scales the distal surface of tooth 26 from the line angle, across and into the furcation region, then under the contact area.





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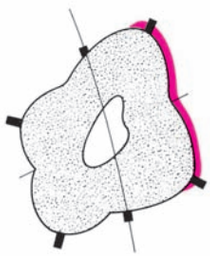
7–8

Posterior Area—Buccal

600 Scaling the Buccal Surfaces—Tooth 26
Patient's head: Tilted slightly toward the operator

Dentist: ca. 10 o'clock position
Rest position: Intraoral, directly on the adjacent tooth
Vision: Direct

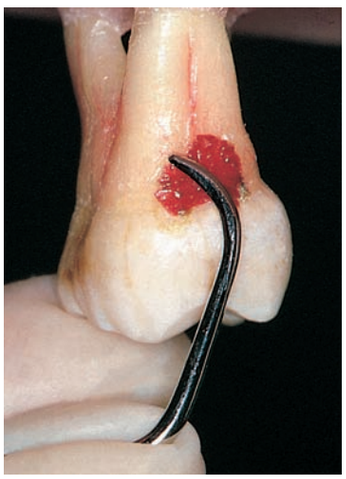
Left: Working ends of the GRA 7/8 curette





601 Model Situation
In addition to axial strokes, the buccal molar surfaces are often treated using oblique or horizontal strokes. The modified pen grasp is obvious, with the middle finger in the first curve of the instrument shank.

Left: The indentation represents the entrance to the buccal furcation. *Note*: Mesial and distal sections of the exposed roots near the furcation are treated using the GRA 11/12 (mesial) and 13/14 (distal).



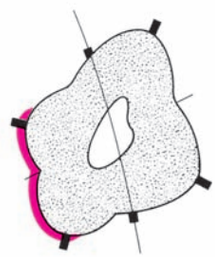
Posterior Area—Palatal



602 Scaling the Palatal Surfaces—Tooth 26
Patient's head: Tilted toward the left, away from the operator

Dentist: ca. 8 o'clock position
Rest position: Intraoral, directly upon the occlusal surfaces
Vision: Direct

Left: The palatal root segments are basically round in shape, however, shallow grooves do occur and these may present difficulties during scaling.





603 Model Situation
Rest position (fulcrum) directly on the occlusal surface of tooth 26.

Left: GRA 7/8 curette is used to scale the palatal surfaces from the distopalatal to the mesiopalatal line angles. In this area, there are no furcations, but often grooves.

After scaling this final (palatal) posterior tooth surface, systematic treatment of the maxillary left quadrant is complete. The pockets are then rinsed and any bleeding is staunched.

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Instrument Sharpening

The curette is a universal instrument that serves both non-surgical as well as surgical periodontitis therapy. The curette is used for scaling, subgingival debridement, root planing, and curettage of the gingival soft tissues.

Knowledge of its characteristics and maintenance of its functions are therefore of great significance. Instruments that have become dull must be re-sharpened; no degree of manual dexterity or force can compensate for the disadvantage of a dull instrument. Dull instruments lack "bite"; calculus is burnished rather than removed.

The systematic sharpening of curettes may be accomplished before, during or after patient treatment. Especially the small, slender curettes quickly become dull during scaling because of contact with enamel or metal restorations. Such instruments must be re-sharpened during the treatment appointment, using a sterile, mildly abrasive sharpening stone (Fig. 606).

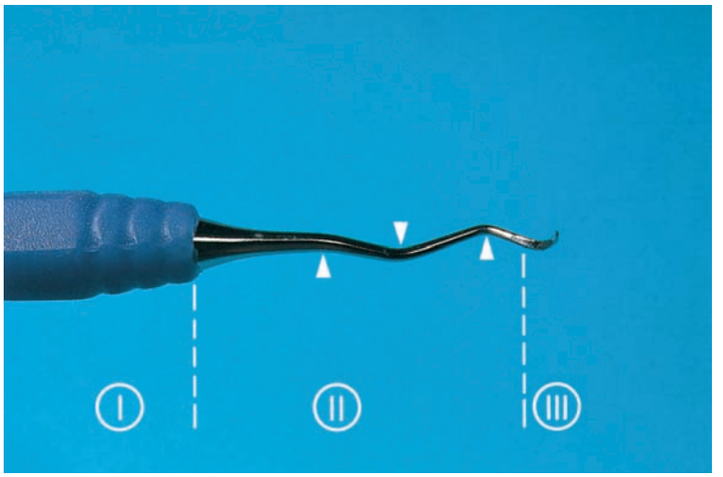
A curette that has been sharpened 10–15 times becomes thin and may break; it must be replaced.

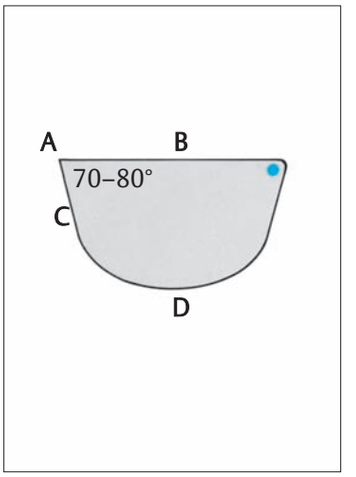
604 Curette—Nomenclature (Example: Gracey Curette 13/14)

I Handle
II Shank
III Working end with cutting edge (blade)

Right: Section through the working end (Gracey curette).

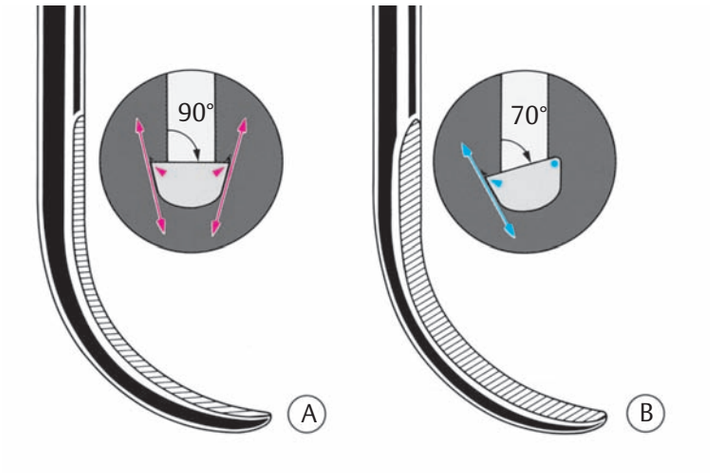
A Cutting edge
The convex edge is sharp; the other edge is blunt (blue dot).
B Face
C Side
D Back





605 Differences between Universal (A) and Gracey (B) Curettes

1 The face (B; Fig. 604) of the working end forms a 90° (universal) or 70° (Gracey) angle to the shank.
2 Both edges of the working end (universal curette) are sharpened; but only the convex (lower) edge of the Gracey curette.
3 Only in Gracey curettes is the working end arched over the surface as well as over the edge.



606 Sharpening Stones—Sharpening Oil

C "Carborundum"
SiC/Silicium carbide; artificial; gross, highly abrasive
I "India"
Al_2O_3, aluminum oxide, artificial, course grained, abrasive
A "Arkansas"
Al_2O_3, aluminum oxide, natural, average-to-fine grained, mildly abrasive

Right: Acid-free mineral oil (Hu-Friedy).





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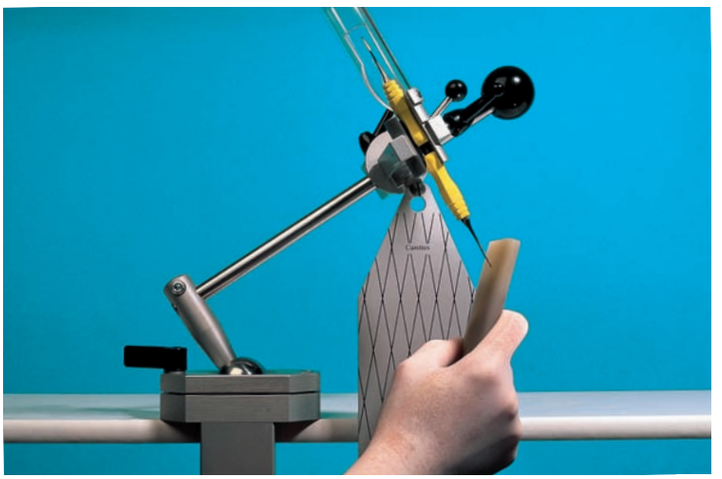
Manual Sharpening of Hand Instruments

Even though powered devices (sonic and ultrasonic) are widely used today, scalers and curettes still command a prominent role for all treatment techniques including both closed and open therapy, surgical procedures, and for prophylaxis (professional tooth cleaning), alone or in combinations.

Efficient and thorough scaling and planing, particularly on infected root surfaces, can *de facto* only be optimally performed with sharp instruments (Bengel 1998, Christan 2002).

Years of experience have clearly demonstrated that "free-hand" instrument sharpening is only rarely successful in providing perfectly shaped and sharp hand instruments. Thus, for manual sharpening and re-sharpening, sharpening *aids* are necessary. On the basis of years of experience, dental hygienist C.M. Kramer (1989, 1999) developed the two sharpening stations depicted below. The basic principles include precise positioning of the instrument (the "horizontal, facial side") and the predetermined sharpening angles on the jig (template), with separate settings for scalers and curettes.

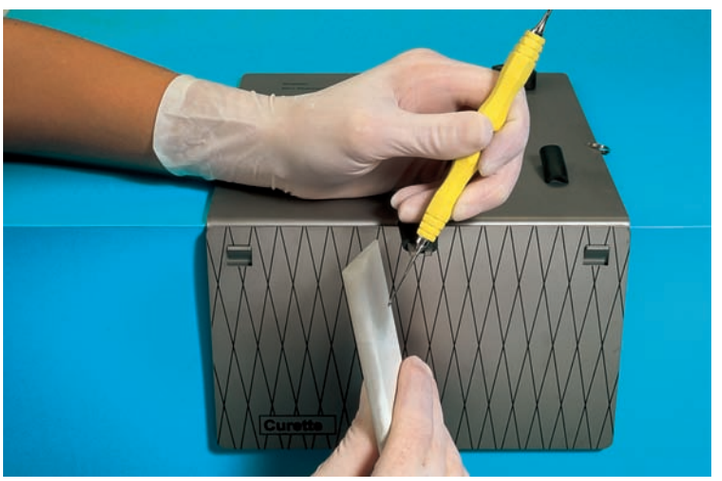




607 Instrument Sharpening with the Kramer Sharpening Station
The vise-like holder with the large ball joint permits positioning of the instrument in any conceivable position.

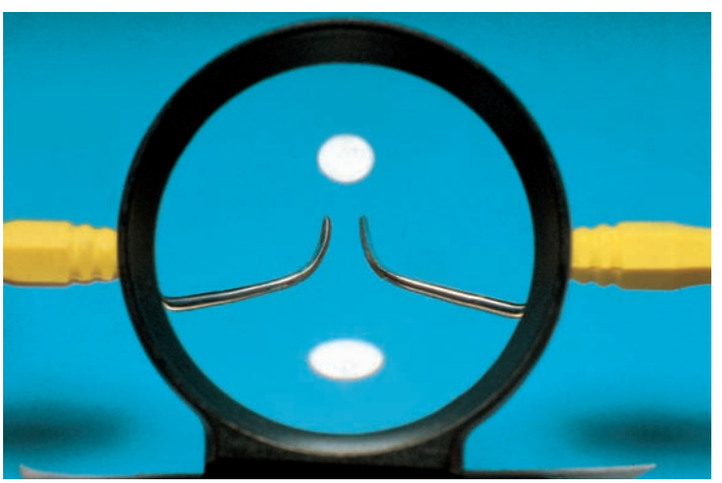
Left: Initially, the special clip is used to grasp the working end. The instrument face must be always positioned *horizontally*, and then the sharpening procedure can begin. The sharpening stone is held parallel to the guiding lines on the front of the station.





608 A Simplified Version—Kramer "Mini" Sharpening Holder
The above sharpening station is ideal for individuals with little experience, and is optimal for sharpening numerous instruments.
The advantage of the mini-sharpening station is that dull instruments can be sharpened at chairside.
The diamond-shaped lines on the vertical portion of the station insure that the scaler/curette is presented to the stone at the proper angle.





609 Tests for Sharpness: Light Reflection Test and Scratch Test
Manual sharpening (cf. Fig. 608, left) is performed with an oiled Arkansas stone.
The metal removal is clearly visible; it remains suspended in the oil and can be easily removed.
The final test for sharpness of the cutting edge: Dull edges do not reflect light, but sharp edges do.

Left: Acrylic rod (PST, Hu-Friedy) tests the sharpness of the instrument, which removes acrylic chips.

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Automated Sharpening

The biggest problem during free-hand sharpening is the difficulty of maintaining the angle of the sharpening stone to the instrument tip, and maintaining this angle during the entire sharpening procedure. It demands manual dexterity, knowledge of the individual instrument's characteristics and … practice (Römhild & Renggli 1990, Pöschke 1990).

The primary goal of mechanical instrument sharpening includes not only simplification of the sharpening procedure, but also the elimination of the above-mentioned difficulties. This is possible using the "PerioStar" (Mikrona Co., 1990).

The main advantages of the PerioStar 2000 include:

- The device precisely holds the shank of the instrument, and permits fixation in any position.
- A special clip permits the horizontal positioning of the facial surface (the only "approximation").
- The adjustable sharpening module with its mounted sharpening wheel; when properly positioned, sharpening is performed with a secured angle of 72° for scalers and 78° for curettes.

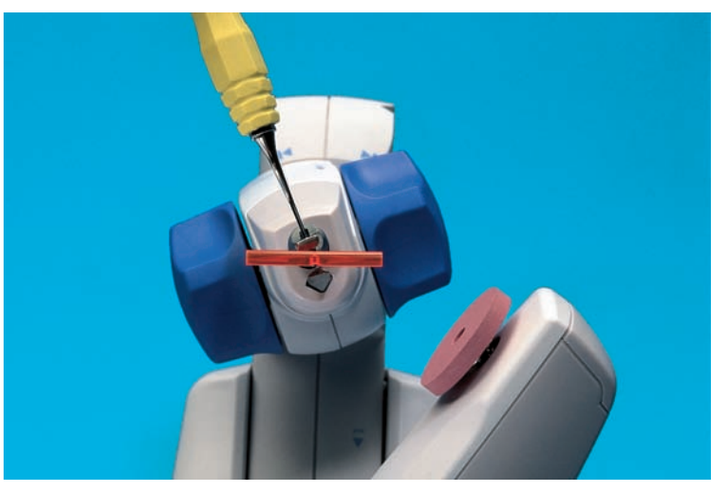
Commercially available today is a simplified variant, the PerioStar 3000 (HaWe Neos; see below).

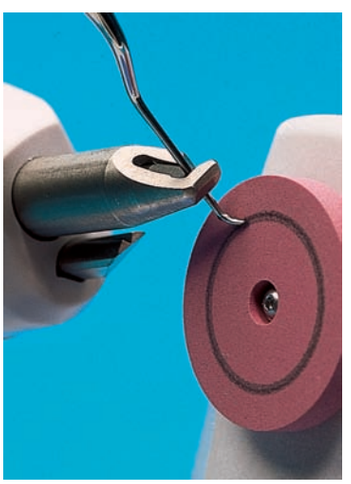




610 PerioStar 2000 (Left) and PerioStar 3000 (Right)
These commercially available devices are provided with various sharpening stones, sharpening pastes, as well as an acrylic rod for testing sharpness.

Right: Sharpening stones and the diamond-coated block for removing grooves and scratches from the sharpening stone.
Sharpening stones in fine, medium and coarse abrasiveness are available (coded with white, red and blue dots).





611 Adjusting and Sharpening—Gracey Curette 5/6 and PerioStar 3000
The mildly fluorescing rod of the chip—it sits upon the facial surface of the curette blade—is oriented horizontally with the instrument. The abrasive element is then brought to bear upon the instrument tip.

Right: The lubricated sharpening disk in action. Clearly visible is the precision holding device.





612 Overview—Adjusting and Sharpening with the PerioStar 2000
The sharpening disk can be adjusted with the dark *rotating table* (below) appropriately onto the curette tip for sharpening: The sharpening angle relative to the "face" is automatically established and reduced to 55°!

Right: Sharpening in progress—Note the precise holding mechanism to insure that the instrument is maintained at the most proximal portion of the shank.

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Subgingival Debridement—Closed Root Cleaning

Step-by-Step Clinical Procedure

Indications and prerequisites for closed therapy were described previously (p. 253).

The procedure is performed without direct vision. Treatment of a single tooth with moderately deep pockets may require 4–10 min, because of working "blind," and dependent upon the individual root anatomy/morphology. Ultrasonic instruments and hand curettes can be used in combination.

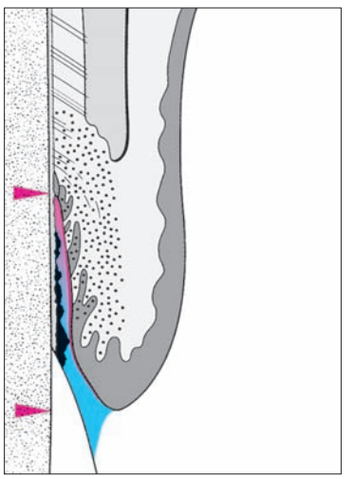
In the case described here, the maxillary right quadrant is treated using Gracey curettes and a closed technique. In the other quadrants, following closed therapy, several sites required "open" therapy (p. 277).

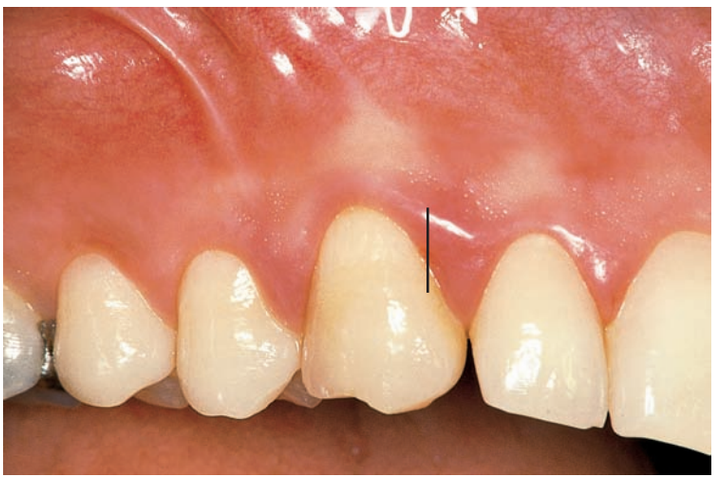
The 29-year-old patient suffered from aggressive periodontitis (Type III), with localized deep pockets and pronounced gingivitis. A microbiologic examination revealed high levels of *Aa* and *Pi*.

Initial findings (values for Quadrant 1):

PI: 61% BOP: 86% TM: 0–2 (p. 174)

The clinical picture, probing depths and radiographic findings are shown in Figs. 613–615.

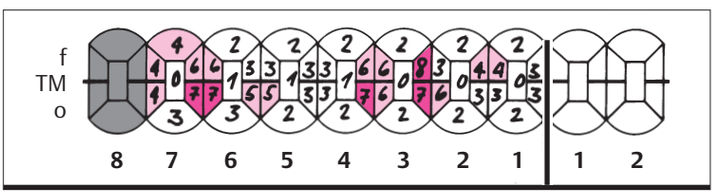


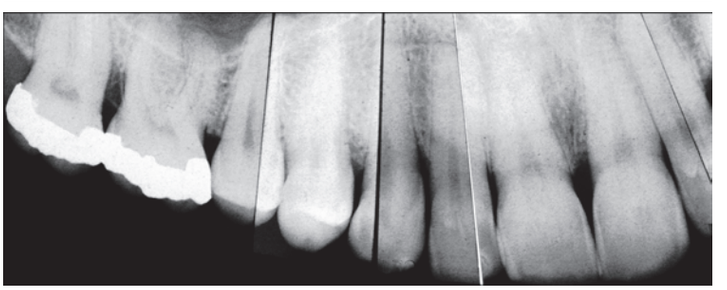


613 Initial Condition—Before Creation of Hygienic Relationships

Especially in the papillary areas, the gingiva is edematous and erythematous. Virtually no stippling.

Left: The schematic depicts the histologic situation on the mesiofacial aspect of tooth 13. In the cervical area, calculus and adherent plaque (blue) cover the root surface, and apically elevated levels of gram-negative microorganisms (red). The gingival connective tissue is infiltrated.





614 Clinical and Radiographic Findings
Probing Depths and Tooth Mobility (TM)

Probing depths up to 8 mm were measured. The very deep pocket on the mesiofacial of tooth 13 resulted from both attachment loss and swelling of the papilla.

Radiographic Findings

The radiograph clearly reveals the massive bone loss that was anticipated by the clinical pocket measurements.





615 Hemorrhage and Plaque

After probing the pockets around teeth 13 and 14, copious hemorrhage resulted. This is a clinical indication of the severity of the inflammatory reaction in this case of periodontitis ("gingivitis").

Left: Stained supragingival plaque. Plaque accumulation on the mesial surface of tooth 14 is minimal, and does not correlate with the copious hemorrhage in this region. The etiology is likely a strong reaction to the subgingival periodontopathic bacteria.

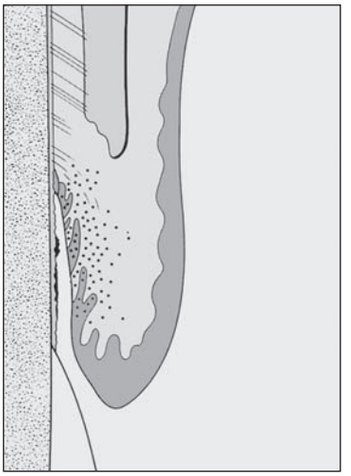
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After the Hygiene Phase

616 Buccal View
Two weeks after supragingival plaque and calculus removal, initial motivation and OHI, the gingivae are already less erythematous and swollen. The enamel defect on the distoincisal of tooth 13 was attributed to bruxism.

Right: *Sub*gingival concrements and plaque persist.





617 Palatal View
Following the first phase of initial therapy—oral hygiene and professional supragingival debridement—the gingivae are still slightly edematous.

Of note in this photograph is the pronounced wear facet on tooth 13. The patient must be made aware of the stress-elicited bruxing habit, and be encouraged to reduce this parafunction.



Subgingival Debridement

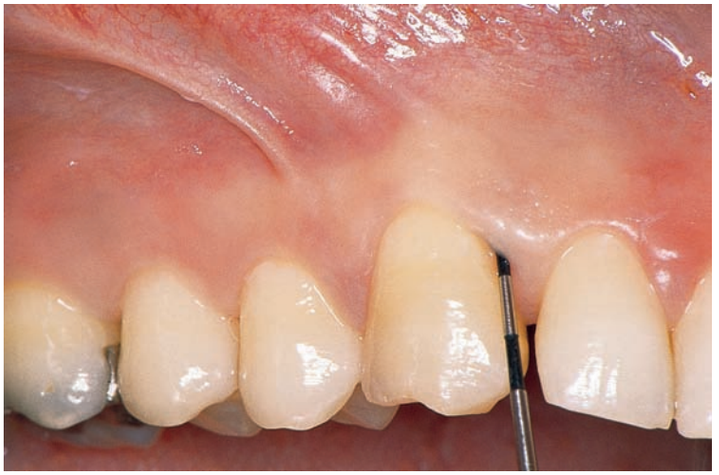
618 Anesthesia
Subgingival debridement is performed using hand instruments, generally with infiltration or nerve block anesthesia.

With the use of slim ultrasonic instruments ("Slimline," Cavitron) it is often possible to treat shallow pockets without any need for infiltration anesthesia.



619 Probing the Depth of the Pocket
Before any mechanical treatment of the root surface, the depth of the pocket is ascertained circumferentially under anesthesia. The floor of the pocket is usually irregular. Usually only isolated sites on the root surface exhibit pronounced attachment loss.

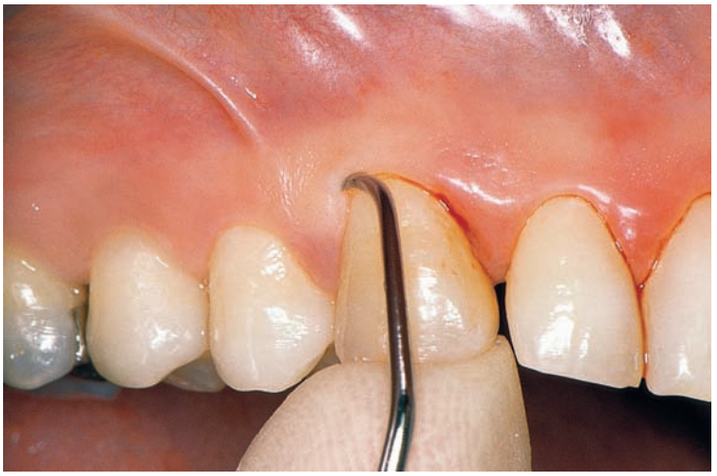
Right: The radiograph reveals that the periodontal probe, using a force of 0.25 N, almost reaches the interseptal bone.





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Double-Ended Curette (Gracey 5–6, yellow) Used in the Anterior Segment

In the anterior and canine regions, the double-ended curette is applied to treat root surfaces on two adjacent surfaces.

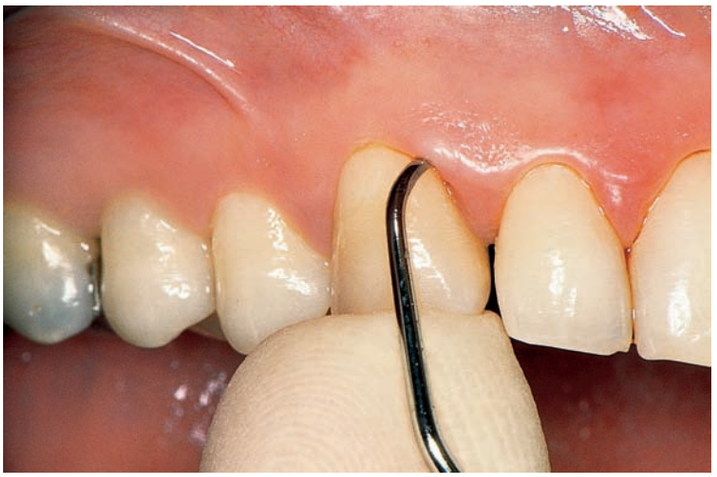
620 Gracey 5—Distobuccal
Using this end of the curette, with overlapping and primarily vertical strokes, plaque and calculus are removed.



621 Gracey 5—Mesiopalatal
The same working end of the curette that treated the distobuccal surface, can also clean the diagonally opposed mesiopalatal root surface.

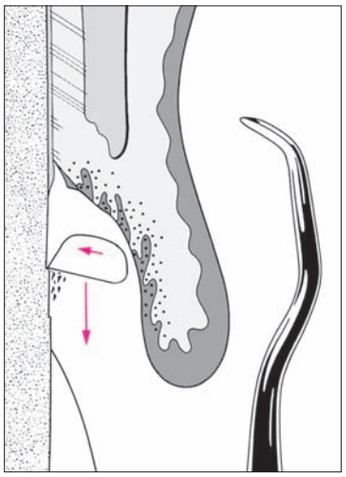
Note: Photographic limitations do not permit depiction of the fulcrum.





622 Gracey 6—Mesiobuccal
This end of the curette functions mesiobuccally and distopalatally. If the first “long” shaft of the curette is parallel to the root surface, the effective angle of the “face” is the desired value of ca. 80°.

Left: The Gracey curette does not achieve the fundus of the pocket, in contrast to the more elegant curettes (e. g., “minifive”) with their shorter and narrower working ends.





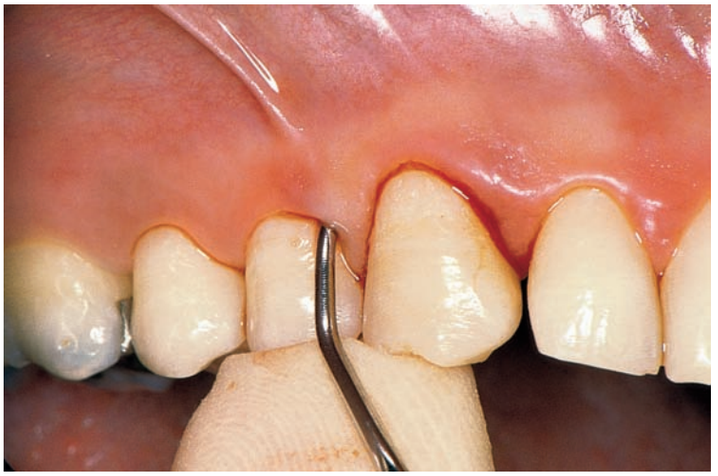
623 Gracey 6—Distopalatal
The same working end of the curette (above, Fig. 622) also serves for treatment of the distopalatal root surface.

Left: The angle of the curette blade to the root surface should be ca. 80°.

The working end of all Gracey curettes is sharpened on only one surface!

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Scaling with Gracey Curettes in the Posterior Area
Double-Ended Curette, GRA 11/12 (red), for Use on Mesial Surfaces





624 Gracey 12—Mesiobuccal
The mesial root surfaces of premolars and molars are best approached with the two paired working ends of the double-angled Gracey 11/12 curettes; shown here in the maxillary right quadrant, with GR 12 from the buccal, and GR 11 from the palatal.



625 Gracey 11—Mesiopalatal
This blade of the double-ended 11/12 cleans the mesial surfaces of the premolar from the palatal aspect.
The mesial surfaces treated with the double-ended 11/12 curette must also be treated in the interdental area with overlapping strokes.

(In the maxillary left quadrant, the Gracey 11 is applied buccally and the Gracey 12 from the palatal approach.)

Double-ended Curettes, Gracey 13/14 (blue), for Treating Distal Surfaces



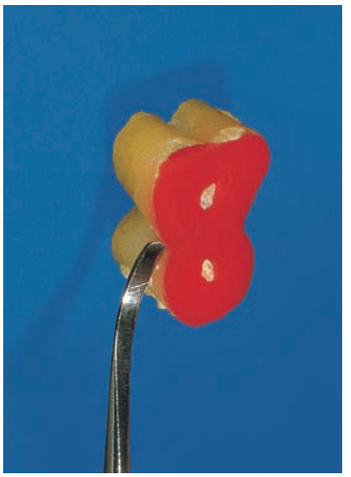


626 Gracey 13—Distobuccal
The distal aspect of the premolar (tooth 14) is treated from the buccal approach.

(On the left side of the maxilla, Gracey 14 would be used.)

Right: The working ends of the double-ended Gracey curette 13/14.





627 Gracey 14—Distopalatal
Note that the terminal shank of the instrument is parallel to the root surface.

Right: Remarkable is the "hourglass" profile of the root surface, exhibiting furcation entrances that must be probed and as far as possible effectively cleaned.

The morphology of the roots and use of the wrong instruments establish the limits of subgingival scaling even more than the absolute pocket depth.

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Double-Ended Curette, Gracey 7/8 (Gray), Indicated for Root Planing on Buccal and Oral Surfaces in Posterior Segments

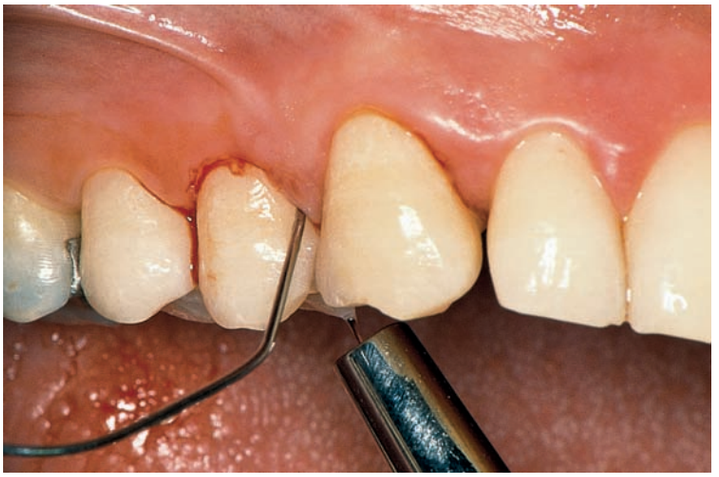
628 Gracey 7—Facial
Usually, subgingival debridement is performed from the *buccal* and *oral* aspects; depicted here in the maxillary right quadrant, Gracey curette 7 is used from the buccal approach up to the line angle and into the mesial interdental space.

Left: Working ends of the double-ended Gracey 7/8 curette.



629 Gracey 8—Oral
In most cases, during the scaling of mesial (Gracey 11/12) and distal (Gracey 13/14) root surfaces (p. 274), the usually shallower buccal and oral pockets and the corresponding line angles are also effectively treated. Thus, using the Gracey 8, the palatal treatment of tooth 14 can be quickly completed.





630 Pocket Rinsing
Following mechanical/instrumental treatment, the pocket should be thoroughly rinsed with NaCl 0.9 % and/or H_2O_2 3 % in order to remove from the pocket any remaining microbial plaque, calculus or cementum. This final phase of closed therapy will enhance the anti-infectious component of treatment.

Left: NaCl (0.9 %) and H_2O_2 (3 %) solutions. The canula must be blunt (e. g. Max-I-Probe, Hawe Co.)



631 Root Surface Evaluation
Using a fine, pointed probe (depicted: EXS3A; Hu-Friedy) the surface of the root is evaluated with regard to its smoothness. This simple clinical test reveals whether the treatment procedure using curettes has effectively reached all segments of the root and removed all deposits.

But the most important aspect is not the planing and smoothing of the root surface, rather the removal or the destruction of the subgingival bacterial biofilm.

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Closed Therapy in Quadrant 1 ...

Summary

Initially, the 29-year-old patient with aggressive periodontitis exhibited only moderate compliance with oral hygiene instructions. Following initial supragingival prophylaxis and repeated OHI, the patient's compliance improved. The visible gingival inflammation was reduced; however, BOP persisted.

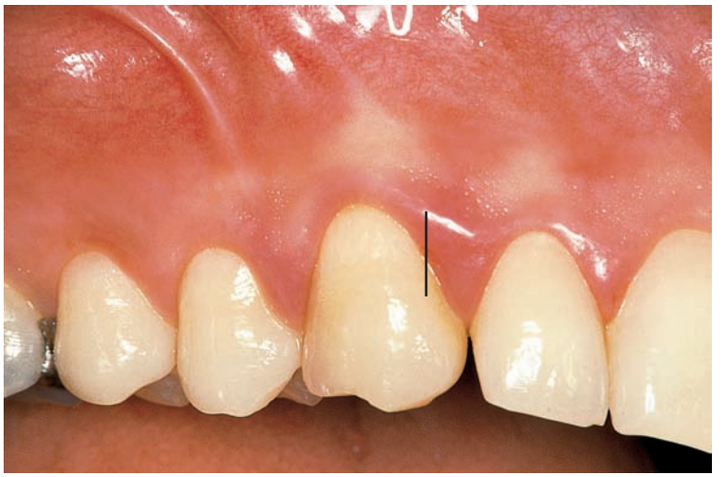
Closed root planing was performed in the entire dentition, as depicted for the maxillary right quadrant. Teeth 17 through 11 did *not* require surgical intervention following the closed scaling therapy (cf. probing depths, lack of furcation involvement), but in several other quadrants, especially in the molar regions, flap surgery was indicated.

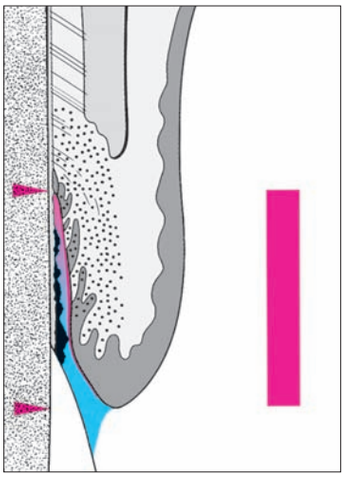
This case illustrates again that periodontitis is seldom generalized. It is the rule that each tooth, even each surface of each tooth, will exhibit differing severities of periodontal defects (single-tooth diagnosis, p. 196).

Initial Findings Before Initiation of Treatment

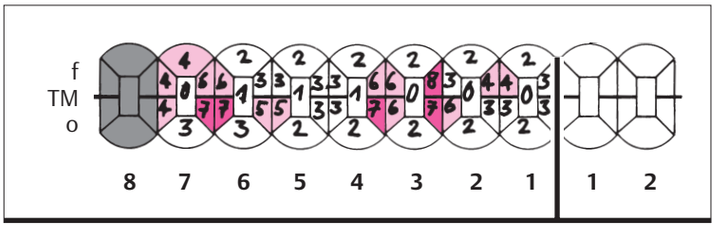
632 Clinical View
Gingivitis and generalized mild, but localized severe periodontitis. The gingiva bled copiously upon probing. The patient had never been adequately informed about oral hygiene.

Right: Pocket "filled" with plaque and calculus (red bar = pocket depth), pocket epithelium and infiltrated subepithelial connective tissue.



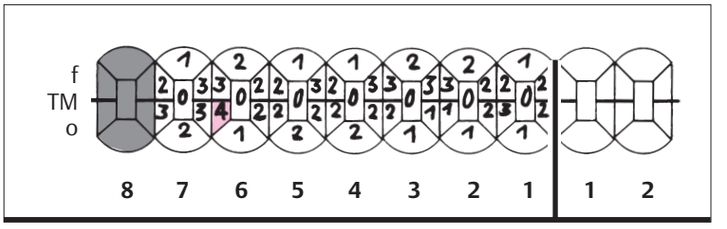


633 Probing Depths—Initial Findings (Quadrant 1)
Because of the gingival swelling, the *attachment loss* must be somewhat less valued than the measured probing depths (p. 169).



Before

Three Years after Subgingival Debridement
Physiologic probing depths are in evidence, resulting from the gingival shrinkage and connective tissue repair. Only one site shows a probing depth beyond 3 mm.

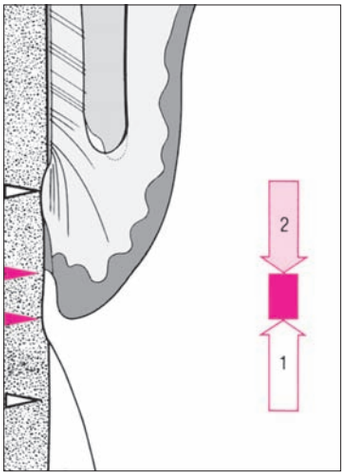


After

634 Clinical View, Three Years after Treatment
Professional treatment and the patient's oral hygiene endeavors led to mild recession yet fully healthy gingiva, but without pronounced stippling.

Right: An active pocket responded to the treatment and the result is a shallow, inactive "residual pocket" (red bar).





1 Shrinkage
2 Attachment gain (repair)

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... and in the Rest of the Dentition?

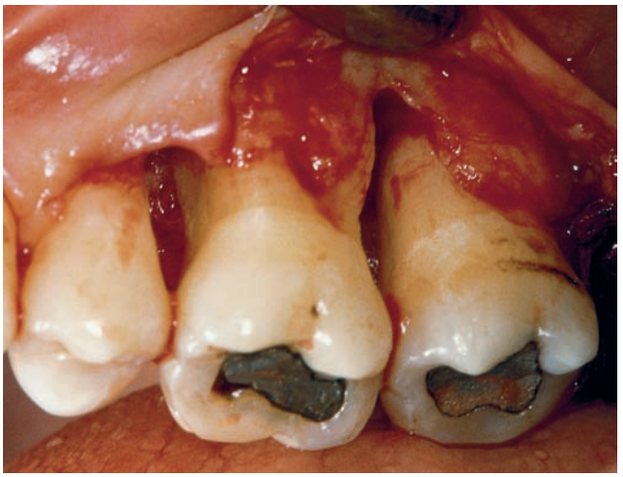
Summary of the Entire Case

Following closed subgingival root planing in the entire dentition, not unexpectedly some sites exhibited persistent deep and active pockets in quadrants 2, 3 and 4.

Therefore, on teeth 25 (distal), 26 (distal), 36 (mesial) and 46 (mesial) paper points were used to harvest microbiologic samples for the preparation of *cultures* (Fig. 636, left). The presence of *Aa* was, at that time (1991), only qualitative ("yes/no"). On the other hand, the black-pigmenting Bacteroides types (BPB) and the sum of all anaerobic flora were quantitatively determined ("colony forming units," CFU).

Flap surgical procedures were performed on all of the remaining deep pockets, combined with systemic administration of amoxicillin and metronidazol (van Winkelhoff et al. 1989). The patient remained on a 3-month recall schedule. Two years later, physiologic probing depths were in evidence, with only a few exceptions.

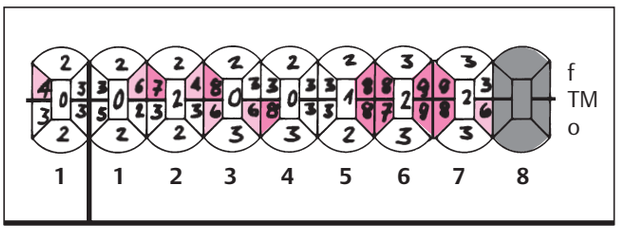




635 Intra-Operation View of Teeth 25, 26, 27
Deep bony pockets, but the furcations are closed. Even with direct vision, root planing was difficult and time-consuming.

Left: Initial anterior view. The gingiva, especially in the maxilla, is erythematous, swollen and not stippled. But this clinical view does not provide any clues as to the severity of periodontitis.

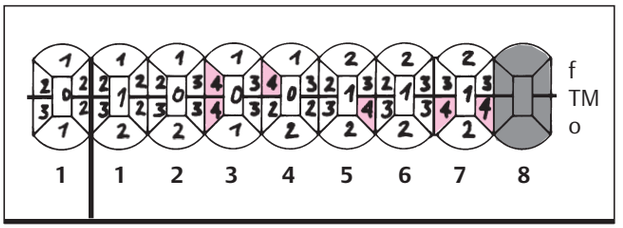
Microorganisms	Tooth sites			
	16d	26d	36m	46m
Aa	–	–	+	+
BPB	10^5	10^7	10^5	10^7
Σ Anaerobes	10^7	$>10^8$	10^7	10^8



636 Probing Depths and Tooth Mobility—Initial Findings
Periodontal pockets up to 9 mm.

Left: Bacterial culture—findings from active residual pockets following Phase 1 therapy.

Microorganisms	Tooth sites			
	16d	26d	36m	46m
Aa	–	–	–	–
BPB	$>10^2$	10^5	10^3	10^4
Σ Anaerobes	10^4	10^5	10^4	10^5



Probing Depths and Tooth Mobility—Two Years later

Left: Bacterial culture—significantly reduced findings six months after completion of treatment.





637 Teeth 25, 26, 27—Findings after Two Years
Irregular course of the gingival margin (shrinkage, cf. bony configuration in Fig. 635). Reduction of probing depths: 2–4 mm.

Left: Final view, anterior segment. The gingiva is inflammation-free and very thin, and therefore does not exhibit stippling.

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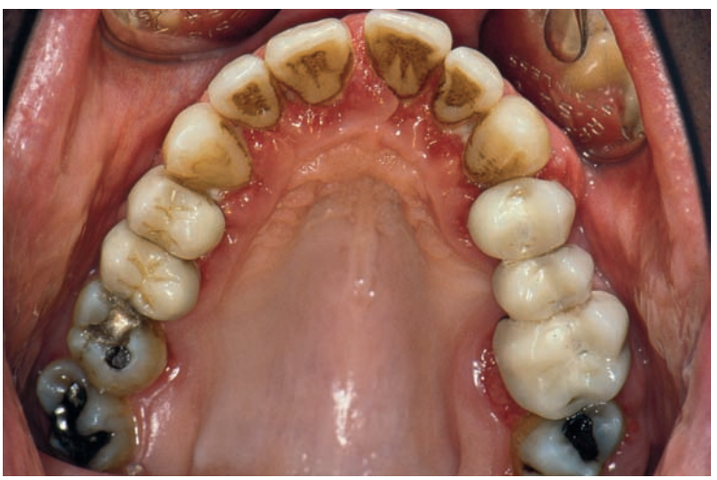
Limitations of Closed Therapy

The boundaries between closed and open (surgical) root treatment are dependent upon a myriad of criteria, and therefore not always easy to clearly define:

- Closed *and* open treatment may be necessary, and the latter always follows the former. Frequently, following optimum debridement, a few areas will require surgical therapy, e.g., in poorly accessible regions (molars and furcations) and with expansively progressive diseases.
- *Wound healing* occurs more rapidly following closed procedures, and often with fewer pain complications.
- The treatment procedures are also dependent upon the *philosophy* of the individual practice: If the dentist collaborates routinely with a dental hygienist, in most cases the DH will initially perform the closed therapy. In mild/slight cases, this may be the only therapy required, but in more severe situations, surgical treatment may be necessary subsequently.

638 Initial View—Occlusal, Maxilla
Periodontitis accompanied by severe gingivitis. The gingivae are severely erythematous and hyperplastic. BOP is ubiquitous. The patient practiced *no* oral hygiene. The teeth were severely stained. The initial supragingival debridement and OHI had to be careful and supportive.

Right: Pre-treatment view of teeth 13, 12; 21.

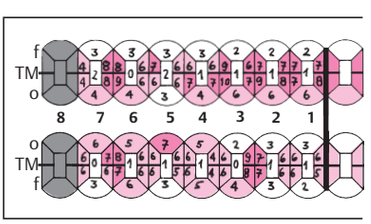




639 Initial Findings, Anterior Segment
Initial ulcerations were obvious on several papillae. The diastema between teeth 11 and 12 had remained unchanged over the past two years. Note gingival recession on teeth 34, 33 and 43, 44.

Right: Initial probing depths and tooth mobility.
The severity of this case made it difficult to believe that treatment consisting solely of closed debridement would bring a successful result.





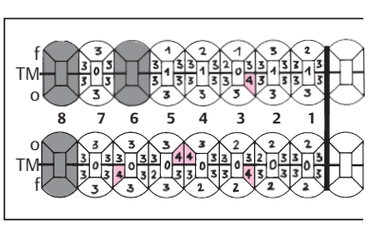
Before

After

640 Radiographic Findings
The radiographs confirmed the clinical diagnosis of severe, aggressive periodontitis. The mandibular molars also exhibit furcation involvement (F1), confirming the clinical findings. Tooth 16 was extracted.

Right: Probing depths and tooth mobility *after* treatment. At almost all sites, physiologic probing depths are in evidence. *How to explain such success?*





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- The *dentist's own philosophy* also plays a role:
  Many dentists prefer an optimum, precise and relatively hemorrhage-free conservative approach; others prefer a surgical approach and are quicker to move to the scalpel. Worthy of note, also, is that even surgical interventions may vary from extremely conservative toward relatively radical (see Surgery, p. 295).

- Last but not least, the patient's own wishes must be considered. The patient may also have an opinion regarding conservative treatment versus surgical intervention.

In the case depicted here, advanced periodontitis was treated using closed therapy, with a successful result.

The 34-year-old patient suffered from generalized, aggressive periodontitis with simultaneously pronounced gingivitis, and exhibited ulcerations in some areas. Until recently, he had been a heavy smoker.

Initial findings:

PI: 70% BOP: 78% TM: 0–2

The figures below depict the clinical situation, probing depths and radiographic findings.





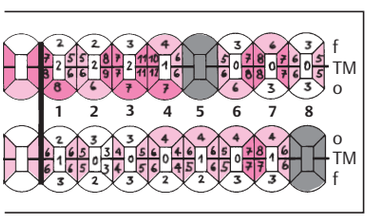
641 Clinical Findings Two Years after Closed Treatment—Occlusal View
Healthy gingival and periodontal conditions. Note the spontaneous closure of the diastema between teeth 11 and 21. In Quadrant 1, a bridge was fabricated from teeth 14, 15 to 17. The old, overcontoured bridge in Quadrant 2 was left in place because of financial considerations.

Left: Clinical view of teeth 13, 12 and 11 after therapy and optimum oral hygiene.



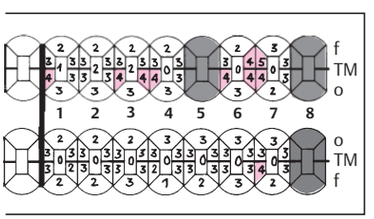
642 Final Result, Two Years after Closed Therapy—Anterior View
As depicted in this clinical photograph, purely closed treatment led to relatively favorable results, including some shrinkage of the gingival tissues; however, the degree of gingival recession had increased. The clinical, esthetic result necessitated free gingival grafting procedures above the canines.

Left: Initial probing depths (left side).



Before

After





643 Radiographic Findings Two Years after Treatment
The intraoral conditions have "stabilized." Several teeth exhibit new bone apposition, e. g., mesial of tooth 43 and in the mandibular furcations.

Left: Probing depths and tooth mobility two years after therapy (cf. Fig. 640, right).

Courtesy *L. Ritz*

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Possibilities and Limitations of Closed Therapy ...

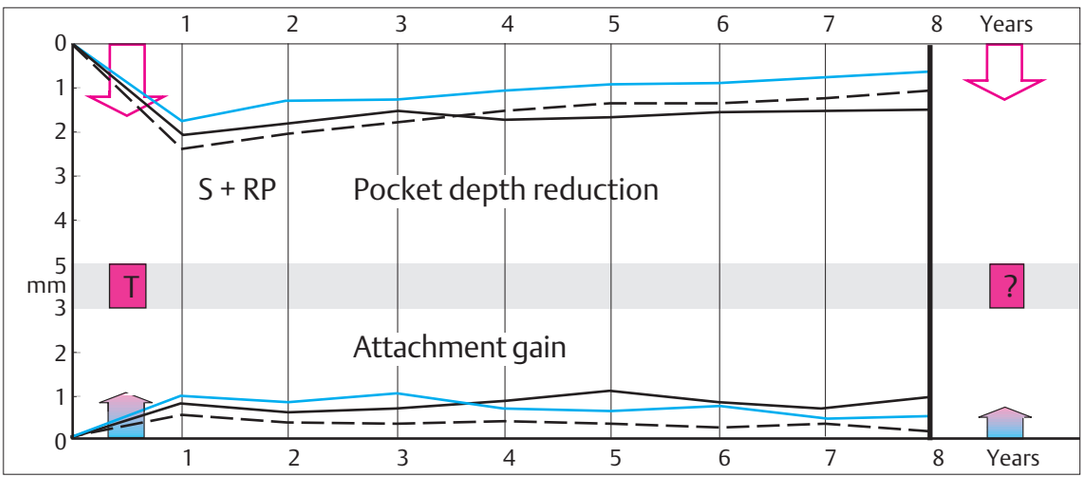
Phase 1 therapy (I: plaque control, *supragingival* debridement; II: *subgingival* scaling, root planing and eventual curettage) is certainly the most important and fundamental measure for comprehensive treatment of periodontal disease. Phase 1 therapy is truly causal therapy, the *gold standard*.

The often necessary adjunct to Phase 1 therapy is periodontal surgery. This permits treatment of the root surfaces with direct vision, and is therefore also "causal" therapy. In addition, surgical approaches can eliminate or correct symptoms or consequences (defects) of the disease process.

644 Pocket Depth Reduction and Gain of Attachment after Various Treatment Methods in 4–6 mm Pockets (Knowles et al. 1979)

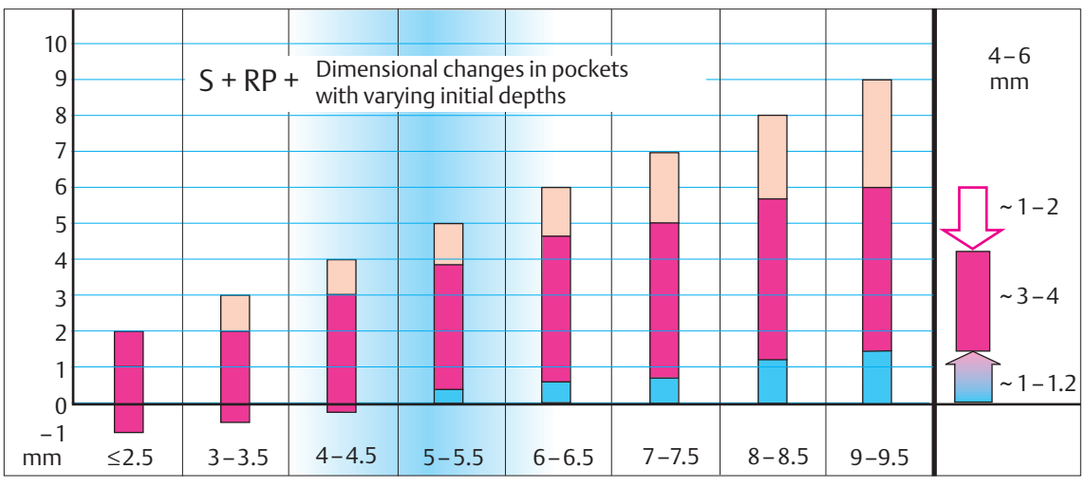
Root planing and curettage
Modified Widman procedure
Surgical pocket elimination

Compare also Fig. 727: Pocket reduction and attachment gain in *deeper* (7–12 mm) pockets.



645 Changes in Pocket Depths Following Closed, Non-surgical Therapy

Badersten et al. (1984) demonstrated early on the advantages and shortcomings of closed treatment. Ideal areas are those with probing depths (PD) up to ca. 6 mm (blue region). Attachment loss is present with PD of 3 mm and less, to deep "residual pockets" with PD greater than 7 mm. In such cases, other techniques (surgery) or the use of medications is more promising (p. 287).



... in Mild Periodontitis

In such cases, especially on single-rooted teeth, Phase 1 therapy alone usually leads to good results (Badersten 1984; Badersten et al. 1984a, b; cf. Fig. 644).

... in Moderate and Advanced Periodontitis

In such cases, success after only Phase 1 therapy is rare. As already noted, subgingival plaque and calculus removal becomes more difficult as pocket depth increases (Waerhaug 1978).

... in Gingivitis

In most cases of gingivitis, initial therapy (Phase I) is the only treatment necessary. One exception is fibrous gingival hyperplasia, which may persist even after elimination of the inflammation. This is often the case following severe gingivitis, with mouth breathing, as well as chronic ingestion of certain medications that have gingival overgrowth as a side effect (phenytoin, dihydropyridine, cyclosporine-A; p. 121). In such cases, Phase 1 therapy must be followed by surgery (gingivectomy/gingivoplasty).

Root irregularities, grooves, furcations and fusions cannot be perfectly cleaned; scaling and planing of the root surface can be performed only incompletely at best. The consequences include a total lack of new attachment (regeneration) and re-infection of the residual pockets.

It is therefore absolutely necessary to re-evaluate the patient's periodontal condition ca. 8 weeks after completion of Phase 1 therapy ("Phase 1 evaluation"). At this time, a decision can be made regarding the necessity for surgical intervention.

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FMT—“Full Mouth Therapy”

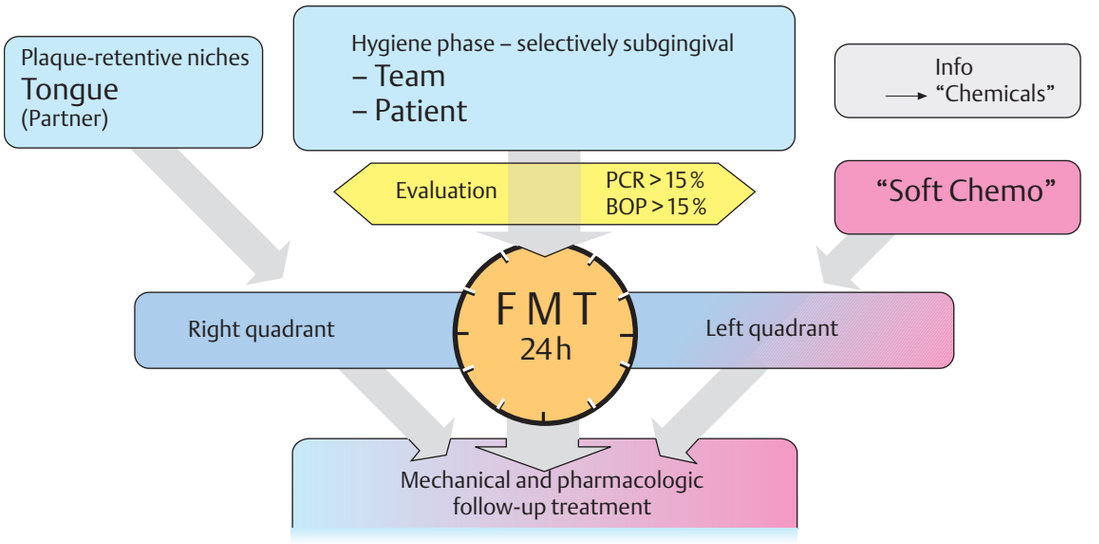
- **Non-Surgical Therapy, plus ...**
- **FDIS—“Full Mouth Disinfection”**

“Full Mouth Disinfection”—FDIS

It is becoming more and more clear that purely mechanical, non-surgical periodontitis therapy can be greatly improved if all of the currently available pharmacologic possibilities are utilized. Such possibilities improve the success rates for pocket reduction and clinical attachment gain to the levels achieved by surgical methods (Drisko 2002; Quirynen et al. 2002).

The therapeutic terms *antimicrobial* and *anti-infectious* do not imply only that the root surfaces in the area of pockets are scraped clean; rather, that the microbial colonizers of the pocket must be reduced, and those microbes with pathogenic potential must be eliminated. This principle also holds true for the entire oral cavity with all of its plaque retentive niches, and the recolonization of residual pockets; the oral cavity must therefore also be thoroughly disinfected (FDIS). And finally, the oral cavity of the patient’s *partner* must be examined and treated as necessary.

The principal procedure for “full mouth therapy” including “full mouth disinfection” is portrayed in the figure below (Fig. 646).



646 FMT—“Full Mouth Therapy”—Schematic
Important: The *intensive anti-infectious* treatment begins with an extended *phase of purely mechanical hygiene*, until the patient’s PI and BOP are reduced to 15 % by home care procedures.

The decision is then made whether FMT will be performed quadrant-by-quadrant or (better) *within 24 hours*. Now also begins the intensive use of antiseptic agents in the pockets and in the oral cavity, before, during and after the active FM therapy.

Practical Procedure for FMT

The procedure is simple and includes the following steps (Quirynen et al. 2001; Saxer 2001):

- An extended hygiene phase; goal: PI and BOP ≤ 15 %
- Actual closed pocket therapy—FMT, pharmacomechanical, within a short period of time
- Supervised follow-up care (mouth, tongue, teeth)

During the *hygiene phase* the patient is motivated in toothbrushing technique, tongue cleansing with a brush or scraper, and the periodontal pockets are *purely mechanically* debrided, deeper and deeper at each appointment.

After achieving the established hygiene goals, the actual “full mouth therapy,” now *pharmacomechanical*, is instituted within a 24-hour period of time (p. 210):

- Oral rinses (CHX 0.1–0.2 %) 1–2 days before initiating therapy (reduction of the “bacterial load” in the oral cavity)
- *Mechanical pocket therapy*, including: Use of antiseptics during the FMT; repeated pocket rinsing (CHX 0.2 %; H_2O_2 3 % plus betadine 0.5 %), and “filling” the pocket with CHX gel after treatment
- Supervised follow-up care (tongue cleansing with CHX).

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FMT—Instrumental/Mechanical and ...

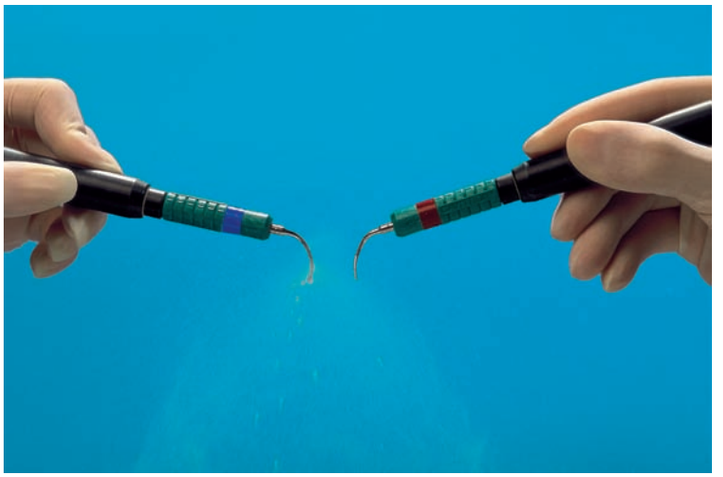
Is “full mouth therapy” the *wonder concept* of the future? Here remains the controversy! Where praise is heard, criticism is not far behind. The loudest outcry against FMT: The “brutal” hours-long *stress situation* for the patients during the 24-hour treatment, and the occasionally occurring *bout of fever* following therapy!

It has long been known that fever, even septic shock, may be the reaction after massive antibiotic administration and the subsequent massive death of bacteria; this also leads to an equally large release of bacterial metabolic by-products, e.g., LPS (also PGE2, IL-1, IL-6 etc.). The host response may be overcome.

The proper use of FMT prevents this type of stress and fever: During the *extended hygiene phase*, the bacterial mass in the oral cavity and within pockets is reduced successively from appointment to appointment using careful mechanical instrumentation and without local anesthesia. Thus, at the end of the hygiene phase, most shallow pockets are already “healed” and only a few deep and active pockets remain to be treated by the FMT method.

647 Fine Tips for the Ultrasonic Device—Cavitron
The use of thin, fine tips with internal cooling (pictured, the paired Slimline tips) permits effective subgingival debridement in areas where curettes are difficult to maneuver, and usually without anesthesia (caution: contaminated spray aerosol!).

Right: Using the thin, curved ultrasonic tips, even the narrowest furcations can be debrided with a “closed” approach.





648 Linear Vibrations of the Vector Ultrasonic Device without Spray Aerosol
The Vector device is only mildly abrasive and therefore not indicated for the removal of hard calculus. However, for the destruction and removal of *subgingival biofilm* it is ideal and also gentle on exposed root surfaces. It is therefore well indicated for follow-up care postoperatively and during recall.

Right: Unusual characteristic—the Vector without any spray aerosol.





649 The Vector in Use—Delicate!
Two powder suspensions provide the mild abrasiveness for polishing capability of the device. The purely linear vibrations reduce pain sensation. Only very few patients require local anesthesia.

Right: **EMS—Handy 2**
This powder-water spray device, with its special fine tip, can also be used subgingivally, thanks to the minimally abrasive powder (amino acid glycine; Petersilka et al. 2003).





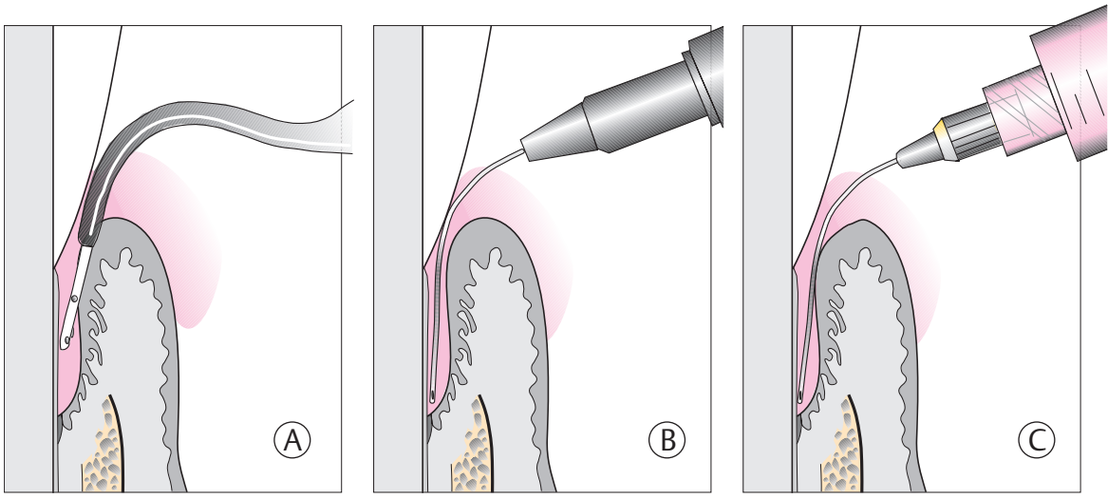
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... Pharmacologic Therapy

If this more "patient" procedure is employed, neither fever nor particular stress will occur. And, 9 out of 10 patients questioned reported that they would decide on this form of therapy again. The overriding question is, what does the patient consider to be less burdensome: Long-term results and financial concerns? The choice of "getting it all over with" (FMT in 24 hours), or the more traditional approach to periodontal surgery, sextant by sextant? These questions must be answered by a thoroughly informed patient.

The authors who have enthusiastically endorsed closed, anti-infectious treatment methods (FMT) hope that their efforts will bring us closer to the goal stated by Dr. Jörgen Slots (2002): "*Effective, safe and affordable periodontal therapy*" (Bollen & Quirynen 1996; Vandekerckhove et al. 1996; Bollen et al. 1998; Quirynen et al. 1998; Mongardini et al. 1999; DeSoete et al. 2001, and others).

The antiseptic agents described below, and the various possibilities for their uses represent cost-effective pharmacologic agents for FMT.



650 Pocket Rinsing with Antiseptics—Subgingival
Using the depicted blunt and therefore non-traumatic cannulae, the fundus of the pocket can be achieved in most cases.

A Perioflex (Oral-B)
B Periodontal Pik (WaterPik)
C Max-I-Probe (HaWe)
on a 10 ml syringe

A single rinsing has only a short-term effect. Prolonged effects can only be achieved with *repeated* irrigation using relatively high concentration disinfectants.





651 Disinfection Agents for Subgingival Application
The depicted agents can be used alone or in combination, together with the mechanical treatment:

- **NaCl 0.9%**
- **CHX 0.2%**
- **H_2O_2 3%**
- **Betadine 0.5%**
- **NaOCl 0.5%**

Left: Two CHX modalities

- **CHX rinsing solution 0.2%**
- **CHX gel 2%** (larger cannula)





652 At-home Oral Hygiene, Brought to the Highest Level
In addition to toothbrushing and interdental cleaning, the "FMT patient" optimizes oral hygiene by regular use of a tongue cleansing device (cf. halitosis, p. 237). The regular brushing of the dorsum of the tongue with chlorhexidine gel helps to decimate the enormous bacterial reservoir.

Left: **Syrette** (Oral-B).
Using this device, the dextrous and instructed patient can rinse residual active pockets.

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FMT—Radiographic Results

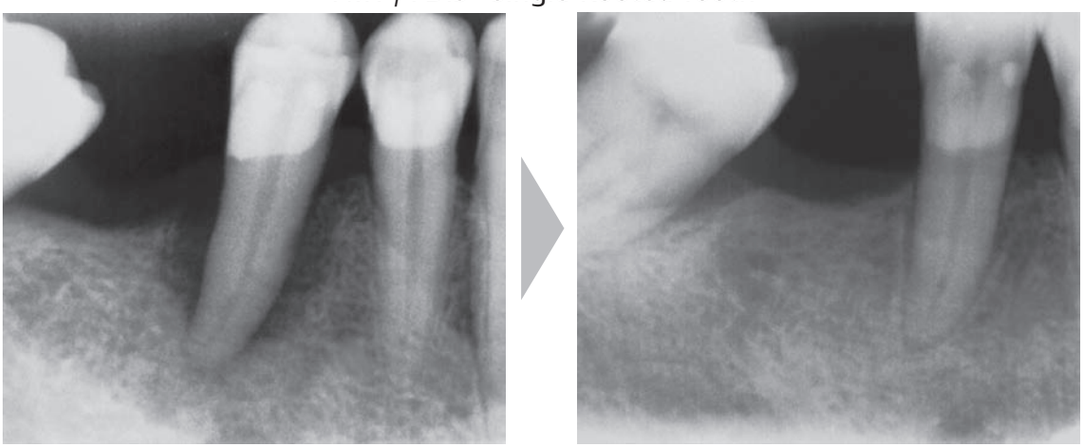
The biggest practical difficulties in any type of periodontitis therapy include, of course, deep pockets, roots with unusual morphology, limited physical access, poor visibility or none whatever, etc.
The most frequently encountered and most difficult problems, especially with closed treatment, include:

- Deep and *narrow* bony pockets
- Furcation involvement in multirooted teeth
- The distal pocket following third molar extraction.

The potential of "full mouth therapy" was tested in precisely these difficult situations. In the radiographs below, "bone fill" is demonstrated that is seldom achieved with conventional closed therapy, or even with "access surgery" (p. 309).

Bone fill does not, of course, reveal anything about true, effective *histologic* healing. On the other hand, bone fill never occurs in the vicinity of an active pocket! Perhaps particularly astounding is that the cases depicted below were treated by students in a dental hygiene school.

FMT / FDIS—Single-Rooted Tooth

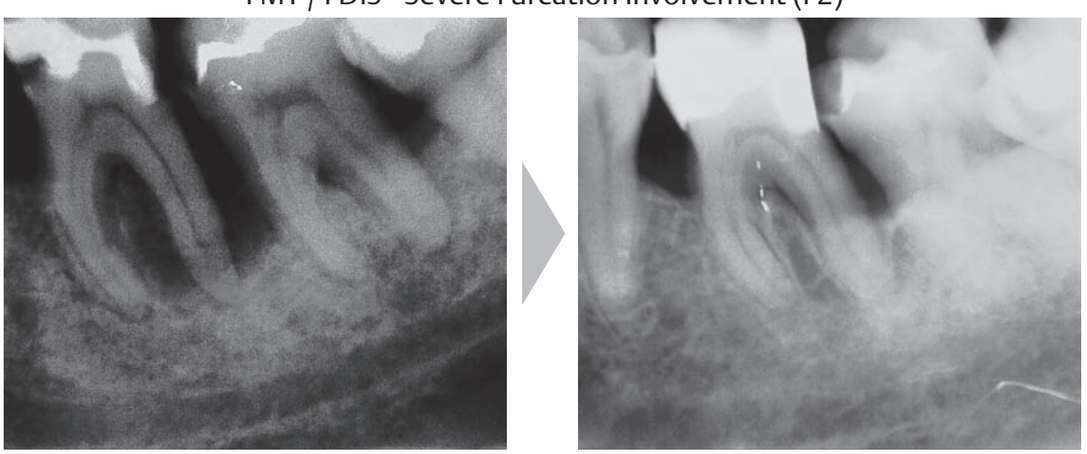


653 Single-Rooted, Vital Premolar with Large Vertical Osseous Defects—Initial Situation (Left)

Right: Five months after initiation of treatment (hygiene phase) and subsequent FMT with "full mouth disinfection" the osseous defect has regenerated almost completely.

Antiseptics used during the active FMT: Combined irrigations with H_2O_2 mixed 1:1 with betadine.

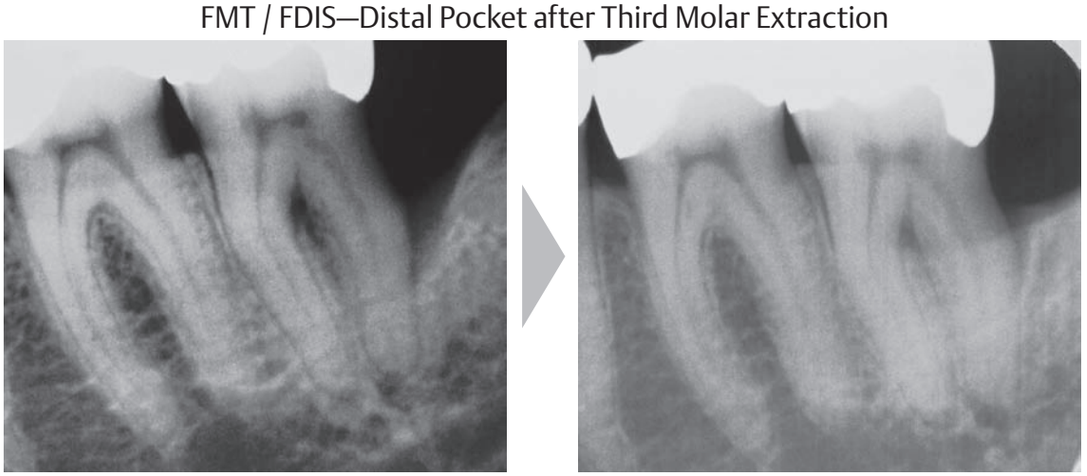
FMT / FDIS—Severe Furcation Involvement (F2)



654 Molar 36 with a Distal Bony Defect and Severe Furcation Involvement—Initial Situation (Left)

Right: Radiographic view two years later. The hygiene phase included removal of restoration overhangs, polishing as well as closed mechanical therapy/FMT. Note the almost complete osseous regeneration in the furcation.

FMT / FDIS—Distal Pocket after Third Molar Extraction



655 Distal Pocket Following Third Molar Extraction—Initial Situation (Left)
Massive bone loss and an unfavorable open area for any future bone regeneration (cf. anatomic defects, p. 324).

Right: Radiographic view two years later. The bone has regenerated, leaving only a small residual defect. The probing depth at this site was 4 mm.

Courtesy *U. Saxer*

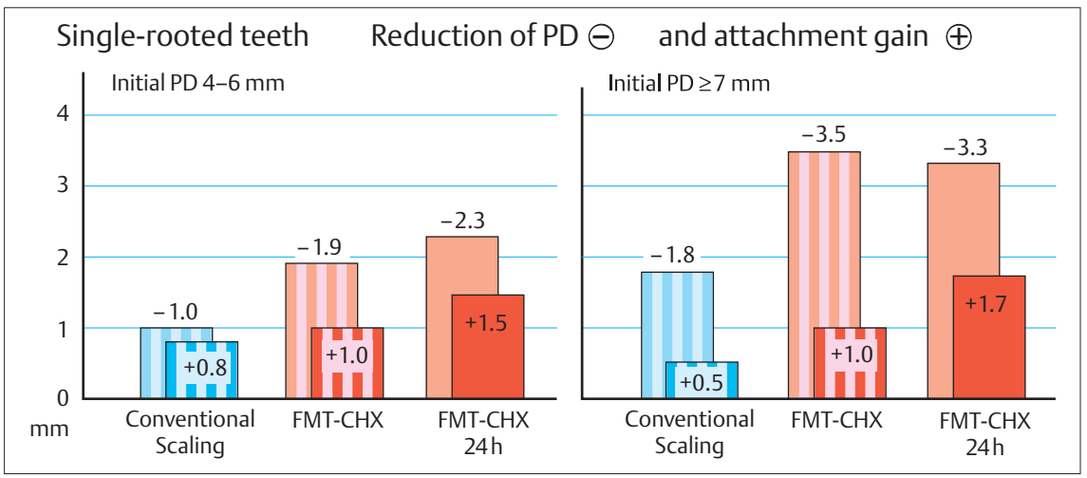
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FMT—Statistical/Numerical Results

During the 1980's the Loma Linda group (Badersten et al. 1981, Badersten 1984, Badersten et al. 1984, Nordland et al. 1987) reported their imposing results using closed, *mechanical*, non-surgical therapy, and described the successful results and the presumed limitations of that gold standard.

New knowledge acquired during the 1990s—the characteristics of plaque "biofilm," the colonization of niches as well as epithelial cells throughout the entire oral cavity, even by periodontopathic microorganisms, their transfer from site to site and even from person to person—led to a true *paradigm shift* and to re-thinking of the then-contemporary treatment strategies.

The results of this re-thinking about periodontitis was a combined *pharmacologic-mechanical* treatment concept that also included niches outside the actual periodontal pocket: The "full mouth therapy" (FMT) including its important component "full mouth disinfection" (FDIS). The results of FMT are, indeed, promising. Figures 656 and 657 (below) summarize a clinical study by the Leuwen group (Quirynen et al. 2000).

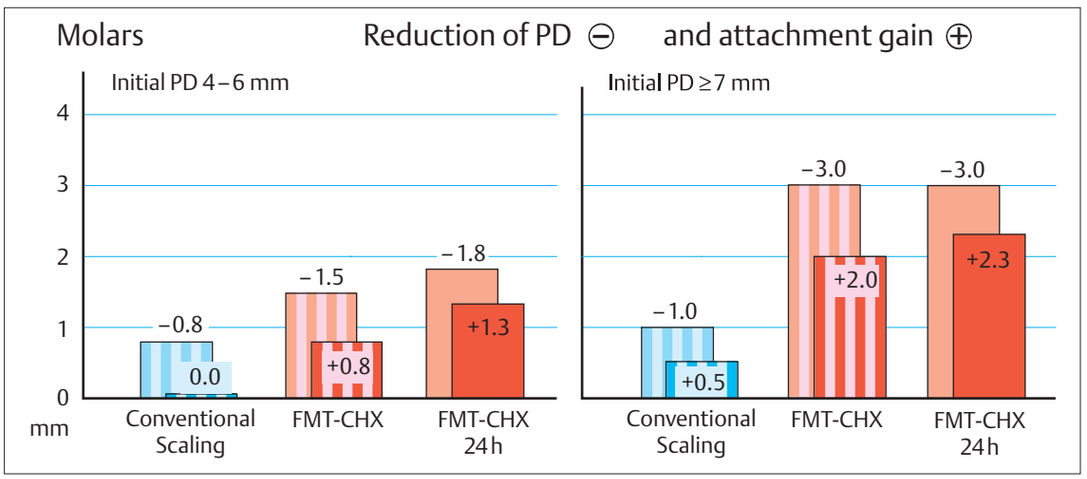


Comparison of Three Types of Closed Therapy

656 Treatment of Single-Rooted Teeth

Legends, see Fig. 657

Negative values: Reduction of PD
Positive values: Attachment gain



657 Treatment of Molars

- Left:
  Initial probing depths 4–6 mm
- Right:
  Initial probing depths ≥ 7 mm

Columns: Type of treatment

Left:	Conventional scaling, 1 quadrant/1–2 weeks
Middle:	FMT—scaling as above, with addition of FDIS using CHX
Right:	FMT 24—as with FMT, but treatment of all four quadrants within 24 hours

Results: Possibilities and Limitations

Using the FMT treatment concept, clinical healing rates are possible that far exceed results obtained using conventional closed therapy, and even those of surgical access therapy (p. 309). The successes of FMT are causing major changes in the philosophy of periodontal practice:

- Periodontitis is an infection that must be treated strictly anti-microbially ("pharmacomechanical").
- Any re-infection of a treated pocket must be prevented by optimum oral hygiene and rapid integration of subgingival treatment procedures (debridement, supported by antiseptics).
- Oral niches must be thoroughly and regularly cleaned both before and subsequent to treatment.

Some of the disadvantages of traditional therapy (scaling and root planing) also limit FMT, e. g., lack of direct vision in the field of operation and the still insufficient effect of antiseptic agents in the pocket.

New agents are being sought for "vision control," e. g., the pocket television (DV-TV). More highly concentrated antiseptics, eventually even topical antibiotics (p. 292) and root conditioning agents such as Emdogain (p. 352) are even today being developed and clinically evaluated.

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Medicaments

- **Anti-Microbial, Antibiotic, Anti-Infective: Pharmacological Co-Treatment**
- **Host Factor Modulating Substances**

Anti-Infectious Supportive Therapy—Antibiotics in Periodontitis Therapy

The accumulation of bacteria upon the teeth represents the primary cause of gingivitis and periodontitis. The regular mechanical removal of plaque biofilm from all non-desquamating surfaces is therefore essential, and it is also the primary measure for prevention or inhibition of the progression of periodontitis (Mombelli 2003).

Through systematic, careful debridement of teeth and affected root surfaces, periodontitis can in many cases be successfully treated. Two disadvantages of this *non-specific mechanica*l treatment, which is repeated regularly at recalls, are the irreversible and ever-increasing damage to tooth hard structure, especially roots within periodontal pockets, as well as gingival recession. In addition, it is virtually impossible to mechanically remove dental plaque from narrow grooves and scratches, narrow furcations and other bacterial reservoirs within the pocket area (p. 255).

Thus it is appropriate to combine mechanical plaque suppression with a medicinal, anti-infectious parallel therapy. Since only a few bacterial species are potentially periodontopathic, it is reasonable to eliminate these groups *specifically* (Mombelli, Slots, van Winkelhoff). These groups contain bacteria that can colonize cells of the pocket epithelium and thus escape both the host response and mechanical cleaning efforts (*A. actinomycetemcomitans, P. gingivalis, S. constellatus*; Herrera et al. 2002). This situation can be effectively combated using systemic antibiotics or topically-applied medicaments.

In addition to antimicrobial agents, mainly antibiotics with their well-known side effects and the ever-increasing emergence of resistant microbial stains, more and more new substances are being offered for use in periodontal therapy, especially agents that modulate the host response.

This chapter on “Medicaments” is arranged as follows:

- Decision-making criteria—When to use antibiotics?
- Systemic antibiotics for periodontitis therapy
- Antibiotics—Bacterial sensitivity and resistance
- Systemic versus topical antimicrobial treatment
- Topical antimicrobial treatment—Controlled release drugs
- Host response—modulating substances

Which pill is the correct one?

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Decision-Making Criteria—When to Use Antibiotics?

Remember: Periodontitis is an *infectious disease*, caused by periodontopathic, usually opportunistic microorganisms that are organized within a protective biofilm.

Both non-pathogenic and pathogenic species live everywhere in the oral cavity, above all in niches of every sort. They construct a biofilm characterized by close community interrelationships, and exchange metabolic by-products, virulence factors, resistance factors etc. The biofilm protects against the host response as well as against antimicrobial pharmacologic agents (Haffajee et al. 2003).

Even though purely mechanical/instrumental treatment—surgical or non-surgical—will usually very much improve the clinical parameters in most cases, in certain situations an *antimicrobial supportive therapy*, applied systemically or topically, can improve the treatment outcome (Hung & Douglass 2002, Mombelli 2003).

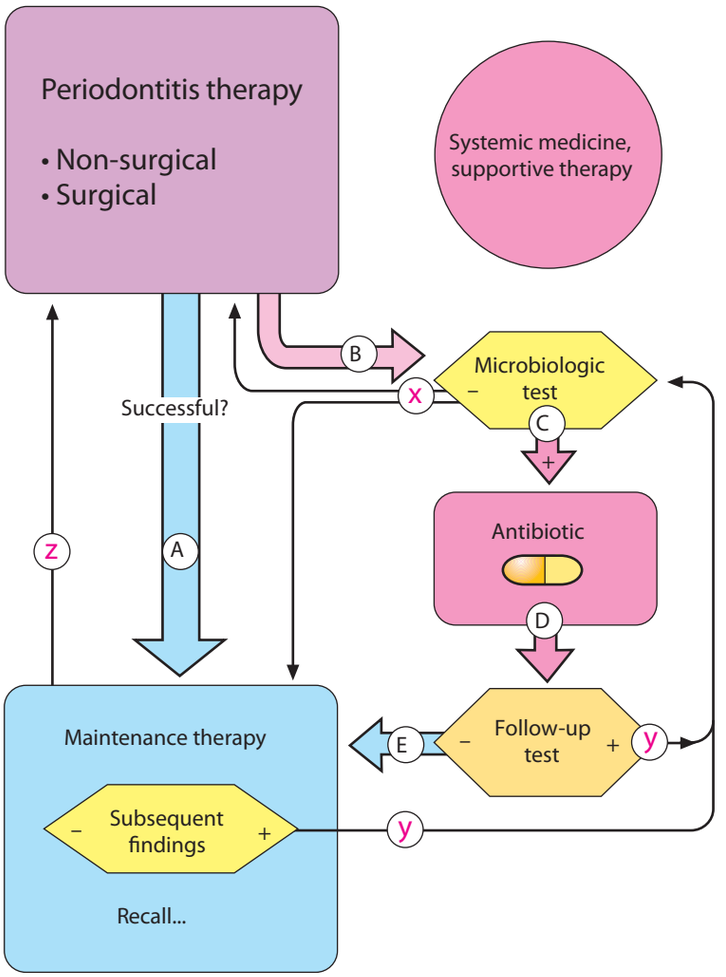
Antibiotics help to subdue the infection; they do not affect healing. Only the host organism can do this. The sensitivity and the nature of the colonization will determine the choice of an antibiotic.

658 Medicinal Supportive Therapy—Yes or No?

Severely progressive cases of chronic periodontitis/Type II, particularly periodontitis cases associated with compromised host response (aggressive periodontitis/Type III, and periodontitis with systemic diseases/Type IV etc.) require systemic supportive therapy in addition to mechanical/instrumental treatment. It is imperative that treatment failure not be caused by the patient's own poor oral hygiene!

The flow-chart depicted here shows a reasonable "decision tree."

The question always arises, whether and when are microbiologic tests indicated. Such tests provide better information about the necessity to employ systemic antibiotics against specific microbial pathogens. Additional tests after therapy can reveal whether or not the targeted microbial species have been eliminated.

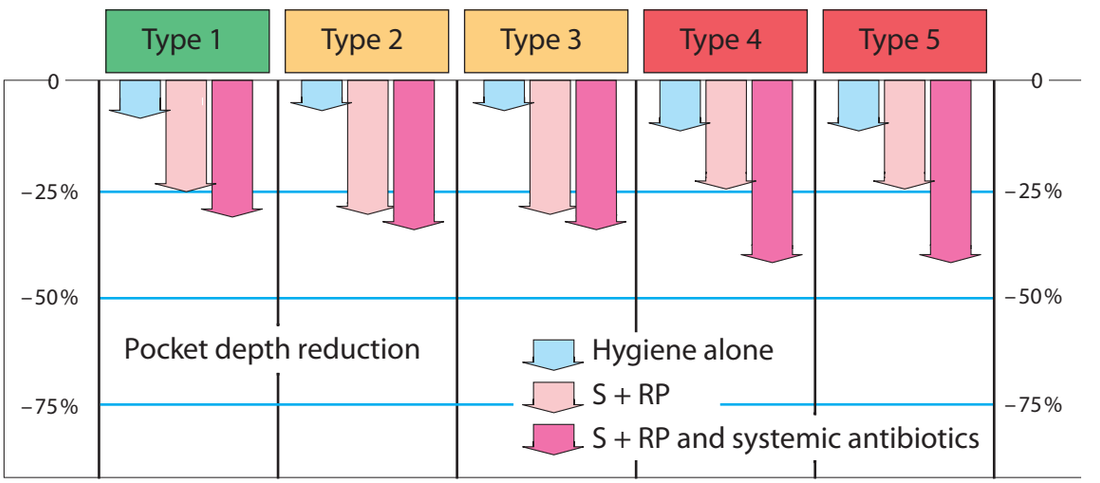


A Successful therapy → maintenance therapy
B Failure → microbial test
C Positive test result (+): → antibiotic targeted toward *pathogenic flora*
D Follow-up, four weeks after antibiotic regimen
E Normal healing → maintenance therapy/recall
x Negative test is "positive" for the patient → maintenance therapy
y Check-up result with a positive test: → pharmacomechanical treatment repeated
z* Recurrence/recolonization: → repeated instrumental/mechanical therapy
* For remaining active sites, possible topical medication using a "controlled release drug" (CRD)

Modified from *van Winkelhoff et al. 1996*

659 Test Results—Example of the IAI PadoTest 4.5

On the basis of its affinity for pathogenic species *Aa*, *Tf*, *Pg*, and *Td*, this test (p. 185) defines five pocket types (clusters), and provides decision-making information about the use of antibiotics. Beyond the reduction of probing depths that can be achieved by improved oral hygiene alone, better clinical results usually follow scaling and root planing (S + RP), especially S + RP combined with a systemic antibiotic in types 4 and 5.



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Microbiologic Testing

- Tests *before* treatment define the pathogenic species, above all the presence of *Aa* and/or *Pg*, and provide a basis for selecting an antibiotic.
- Tests *after* treatment show whether or not the maker bacteria have been eliminated.

Pooled Findings or Individual Findings?

With the use of systemic antibiotics, pooled findings are sufficient. However, if individual active residual pockets remain after treatment, the pooled findings provide no information about the initial status.

When Indicated: Which Antibiotic to Prescribe?

The spectrum of efficacy of antibiotics, their important side effects and the compliance of the patient must be understood in advance: Oral ingestion (tablets) over an extended period of time demands discipline from an informed patient (Newman & van Winkelhoff 2001).

In general, broad spectrum antibiotics (e.g., tetracyclines) are used only for special indications. While the commensal flora consists primarily of gram-positive aerobes, the periodontopathic organisms are mainly gram-negative and anaerobic.

Systemic Antibiotics – Adjunctive Periodontal Therapy (Choices)

Class	Antibiotic	Adult dose (mg/day)	Duration Days
Tetracyclines* bacteriostatic (-static)	• **Tetracycline-HCl**	4 x 250	14 – 21
	• **Doxycycline-HCl**	1 x 100 (1 per day x 100)	14 – 21
	• **Minocycline-HCl**	1 x 200	14 – 21
Penicillins* (-cidal)	• **Amoxicillin** plus 125 mg **Clavulanic acid:** → **Augmentin****	3 x 500	7 – 10
Nitroimidazoles (-cidal)	• **Metronidazole**	3 x 500	7 – 10
	• **Ornidazole**	2 x 500	7 – 10
Macrolides (-cidal)	• **Azithromycin**	2 x 250	3
	• **Spiramycin** combined with **Metronizadole:** → **Rodogyl*****	3 x 33 M.I.E	>4
Lincosamines (-static/-cidal)	• **Clindamycin**	4 x 300	7 – 10
Quinolones (-cidal)	• **Ciprofloxacin**	2 x 500	7 – 10
	• **Ofloxacin**	2 x 200	5
Combinations: (-cidal)	*„parallel“*		
	• **Augmentin**** see above	3 x 625	7 – 10
	• **Rodogyl***** see above	2 x 2	7 – 10
	• **Amoxicillin 375 + Metronidazole 250**	3 x tid 1	7 – 10
	• **Metronidazole 500 + Ciprofloxacin 500**	2 x tid 1	7 – 10
Combinations	“*Serial*” use */*		
	• Bacteriocidal, then bacteriostatic		

660 Established and Frequently Employed Systemic Antibiotics for Supportive Periodontitis Therapy

Medication class, average dosage and duration of medication are shown.

In order to prevent the emergence of resistant bacterial strains, medication should never be prescribed with excessively low doses or short regimens!

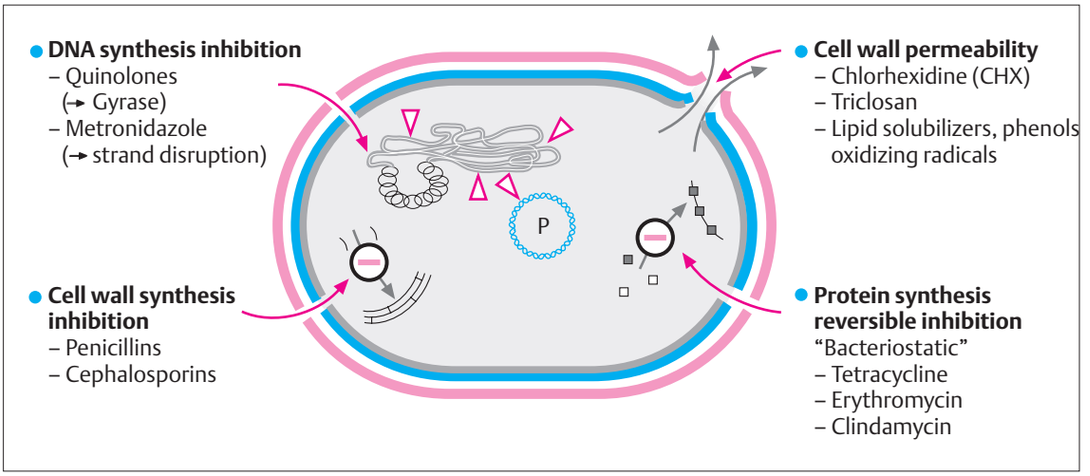
Bacteriocidal antibiotics (AB; “-cidal”) exert their effects much quicker than bacteriostatic (“-static”) agents. Bacteriocidal agents should *never* be given simultaneously with bacteriostatic antibiotics.

On the other hand, the serial use of antibiotics (one-after-the-other, see combinations) may provide optimal effects (cf. varying treatment of HIV, p. 149):

* Successive drug use: Penicillin → tetracycline

** Augmentin: Combination of amoxicillin (AB) and the penicillinase inhibitor clavulanic acid

*** Rodogyl: Combination of metronidazole and spiramycin



661 Effect of Various Antibiotics and Antiseptics on the Target Organisms

Bacteriocidal agents effect the ...

- Cell wall integrity
- Cell wall synthesis
- DNA synthesis and packaging

Bacteriostatic agents inhibit ...

- Protein synthesis

Modified from *J. Goodson 1994*

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Antibiotics—Bacterial Sensitivity and Resistance

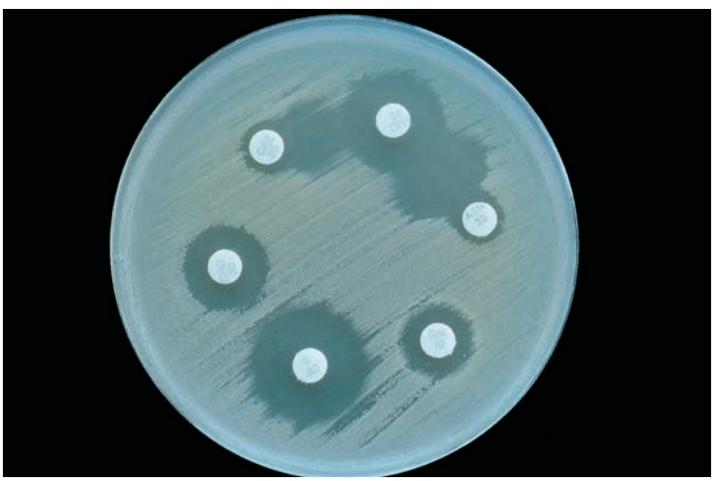
One of the greatest, world-wide medical problems of the next decades will certainly be bacterial resistance: Antibiotics that have up until now effectively eliminated sensitive bacterial species will no longer have any effect. The spectrum of activity of the well-known antibiotics will become ever smaller, and new antibiotics have not been developed for a very long time. The reason for the increase in bacterial resistance can be found in the careless and widespread use of antibiotics by physicians and in hospitals, but also in the almost grotesque use of antibiotics as growth enhancers in the food industry in certain countries; more than half of all antibiotic production, world-wide, is dedicated to this arena. In addition to *naturally* occurring resistance, resistance of previously sensitive bacterial species can occur in several ways:

- Transfer via plasmids (virulence transfer, p. 35)
- Point mutations
- Selection via genetic "survival of the fittest"

The latter frequently occurs due to the overuse of antibiotic agents: If the dosage is too low or massively high (only the *most* pathogenic microbes survive), or if the antibiotic regimen is too short or too long.

662 Bacterial Culture—Sensitive Species
In the *sensitivity test* depicted, the microorganisms were sensitive to all of the test substances (six antibiotics); the antibiotics would be effective.
Note: Both antibiotics (upper left and upper middle) exhibit *synergism*; their inhibitory effects are mutually supportive.

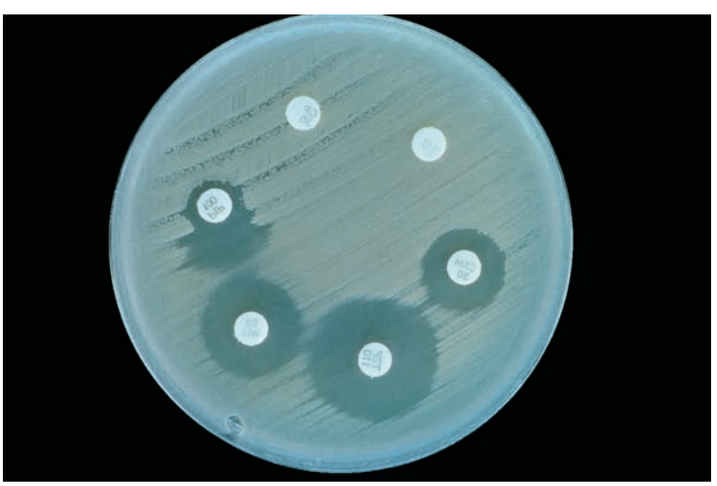
Right: Synergistic antibiotics, e. g., *Augmentin* (above) and *metronidazole* (below); the so-called "Winkelhoff cocktail."





663 Bacterial Culture—Resistance Against Two Antibiotics
It is becoming more and more often the case, especially in hospitals, that numerous bacteria are resistant to antibiotics (antibiotic pellets without any inhibitory effects; above, left and right).

There exist varying resistance parameters in the population and in hospitals; see below!



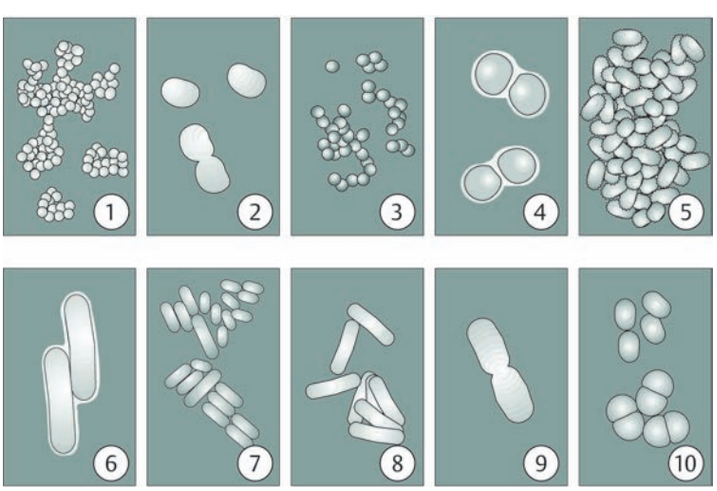
Figs. 662 and 663:
Courtesy of *A. Mombelli*

664 Bacteriologic "Rogue's Gallery"
The ten most feared bacterial species, resistant to almost all antibiotics, and their most frequent "location."

P Population
H Hospitals, institutions
U Ubiquitous

Periodontologically relevant species are, fortunately, not (yet) found in this list.

Modified from *S. Levy 1998*



List of Resistant Strains
(as of 1998)

1	*Staphylococcus aureus*	U
2	*Acinetobacter*	H
3	*Enterococcus faecalis*	H
4	*Neisseria gonorrhoeae*	U
5	*Haemophilus influenzae*	U
6	*Mycobacterium tuberculosis*	U
7	*Escherichia coli*	U
8	*Pseudomonas aeruginosa*	U
9	*Shigella dysenteria*	P
10	*Streptococcus pneumoniae* ("Pneumococci")	U

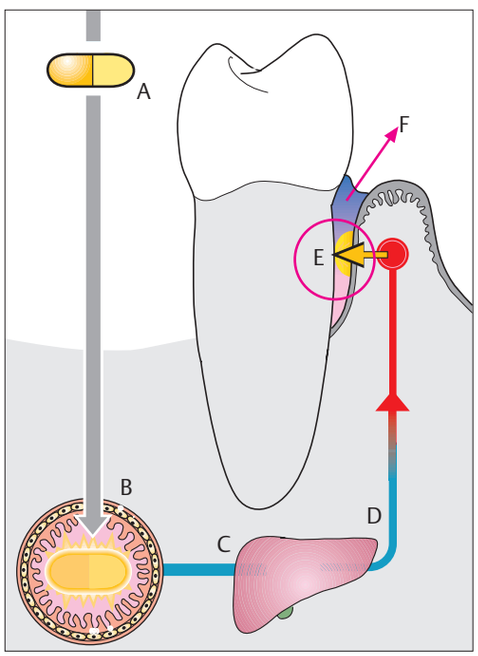
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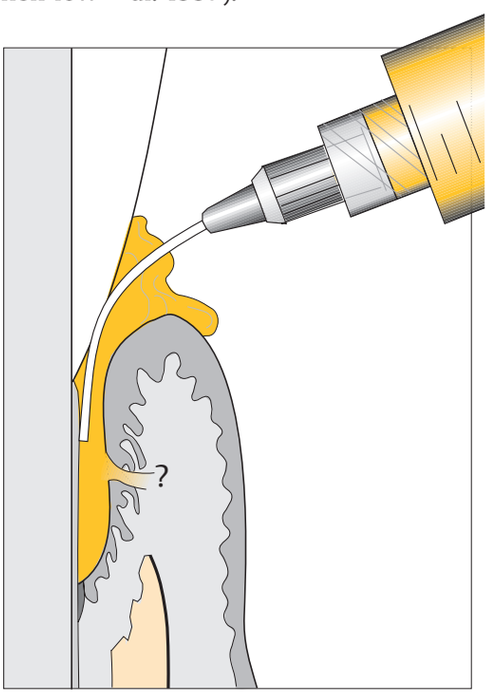
Systemic Versus Local (Topical) Antimicrobial Therapy

Antibiotics—systemic or topical—should only be prescribed under the most strict indications. Antibiotics can *not* compensate for poor mechanical/instrumental treatment; mechanical instrumentation is of primary importance, because in each patient, the etiology of the inflammation and destruction—the bacterial *biofilm*—must be removed or at least so reduced that the medicaments can work effectively and directly upon the microbiota.

Systemically ingested medicaments have the advantage that all vascularized areas of the body are reached, including also the periodontal tissues. The disadvantages include their low concentration within the tissues, the necessity that the patient take the medication for the appropriate length of time, and the possible elimination of non-pathogenic, "beneficial" bacteria, as well as systemic adverse effects.

Locally (topically) applied medicaments can be extremely effective if high concentrations are permitted to work at the affected site for a sufficient length of time. A disadvantage, however, is the extremely limited radius of action, i. e., diseased neighboring periodontally involved areas may be quickly re-infected, leading to poorer healing (Mombelli et al. 1997).

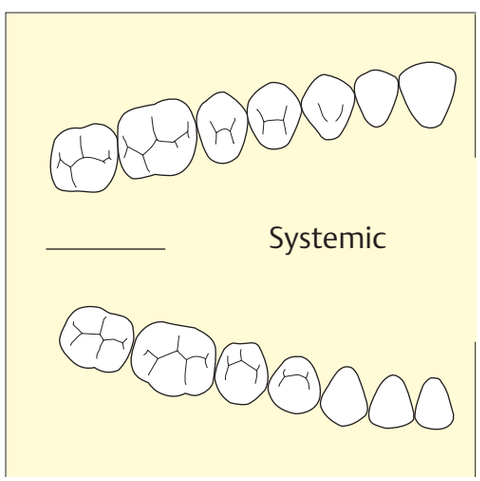


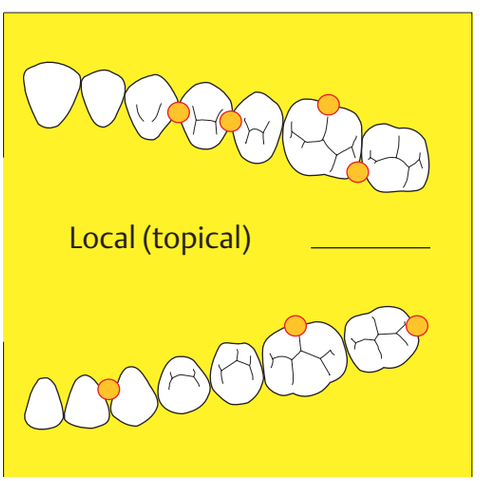


665 Systemic Versus Topical Application
Left: A tablet (**A**) begins its lengthy pathway to its goal, the "periodontium." Absorbed in the intestine (**B**), and modified in the liver (**C**), it is disseminated within the vascular system (**D**) and finally arrives, very diluted, in the periodontal tissues (**E**); the antibiotic then reaches the connective tissue compartment of the periodontal pocket, and finally the biofilm itself before it "evaporates" in the oral cavity (**F**).

Right: The highly concentrated medicament and its carrier are deposited directly into the periodontal pocket.
Additional differences?

Systemic Administration	Topical Application	Systemic/Local—Results
• Broad spectrum of activity	• Narrow, limited spectrum of efficacy	← Distribution
• Low local concentration	• High local concentration	← Local concentration
• Also achieves distant reservoirs of pathogenic microorganisms	• Better effect against biofilms	← Advantages
• Systemic side effects	• Possible re-infection of non-treated sites	← Disadvantages
• Choice of antibiotic, determined by the pathogen		





666 Systemic Administration Versus Topical Application
Left: Systemically applied medicaments reach the periodontal tissues of the entire dentition and are therefore good for new, yet-untreated cases. Because the antibiotic concentrations are low, all periodontal pockets must be previously completely debrided.
Right: During recall appointments, and before regenerative procedures (osseous craters) isolated active residual pockets can be treated by topical application of medicaments.

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Local (Topical) Antimicrobial Therapy—"Controlled Release Drugs" (CRD)

It has been routine in dentistry for decades that local, acute processes such as abscesses or NUG lesions are treated with topical medicaments for so long a time as necessary to stop the acute symptoms. Oral rinsing and irrigation have only limited effects if subgingival debridement of the bacterial colonization is not performed simultaneously. All of these types of applications are associated with the disadvantage that the medicament applied is effective for only a very short period of time because it is rapidly rinsed out of the pocket by sulcus fluid secretions.

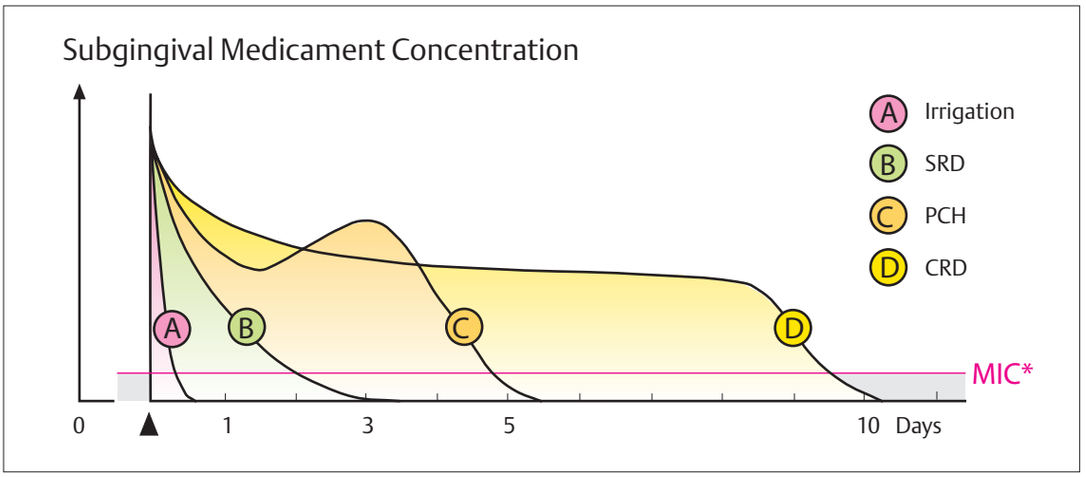
Only with the development of targeted carrier systems did it become possible to apply topical medicaments with effective, high doses and controlled release at the appropriate site.

The problem, however, was the same as with systemic antibiotics, namely, what are the *indications* for use of topically applied antimicrobials? Research studies have not yet provided adequate information concerning how long mechanical therapy should be continued and at what point the use of "controlled release drugs" should begin. To date, this decision remains in the hands and mind of the treating dentist.

667 Effective Subgingival Concentration Following Four Methods of Application
With all systems, the initial concentration far exceeds the minimal inhibitory concentration (**MIC**), but these concentrations decrease quite variably.

A Irrigation (cf. p. 283)
B SRD
"sustained release devices"
C PCH CHX-PerioChip
D CRD "controlled release drug"

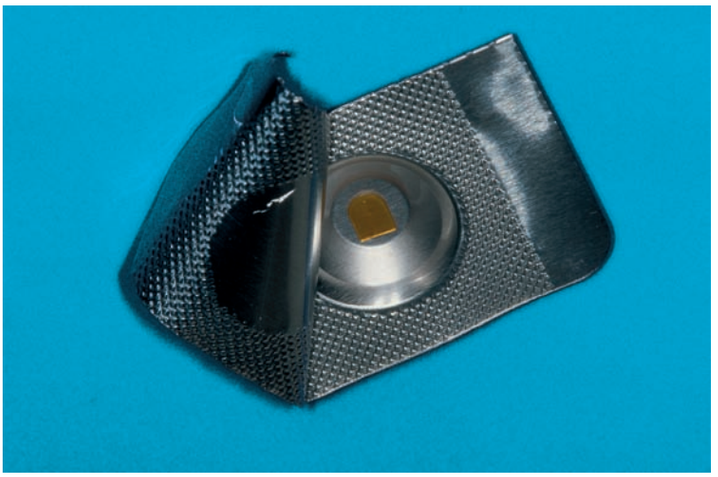
Modified from *M. Tonetti 1997*



668 PerioChip
Active ingredient: 2.5 mg chlorhexidine digluconate
Vehicle: Resorbable gelatin

The vehicle was temperature sensitive and for this reason the sterile packaging had to be maintained in a refrigerator.
This problem is now solved.

Right: Close view of the PerioChip, ca. 3 x 4 mm in size.





669 Inserting the PerioChip into a Periodontal Pocket
The chip is removed from the refrigerator immediately before its insertion into the periodontal pocket; this is necessary to insure that the chip retains the necessary stiffness. Using a forceps, the PerioChip is slowly inserted, at least 5 mm into the periodontal pocket ...

Right: ... and forced to the depth of the pocket, using the forceps. The chip should be completely subgingival.





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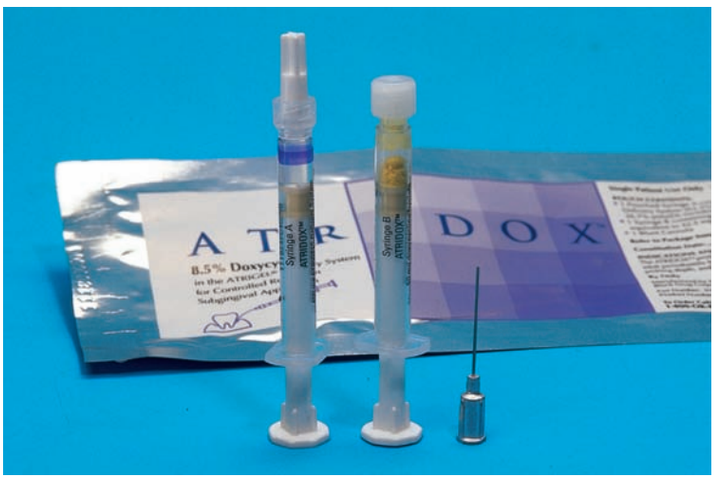




670 Elyzol (Colgate)
Active agent: 25 % Metronidazole benzoate
Vehicle: Mixture of glycerol-monooleate/sesame oil.
Effective time: 24–36 hours.
Application of the prepared contents of the carpule, via the special cannula, directly into the pocket. The gel solidifies upon contact with fluid. Application should be repeated seven days later.

Left: Detailed view of the cannula and the gel.





Atridox (Block Drug)

671 The Product . . .
Active ingredient: 8.5 % doxycycline hyclate
Vehicle: Atrigel technology; must be refrigerated.
The active ingredient and vehicle are provided separately as a two-syringe mixing system (ready for use after coupling of the two syringes and 50 back-and-forth movements).

Left: Atridox in comparison to a 2 ml ointment syringe.





672 ... and the Final Mixing and Application
After final mixing, the syringes are separated and the cannula is affixed.

Left: The cannula is inserted into the 6 mm pocket. The pocket is filled from its apical aspect, rapidly, because the gel quickly begins to solidify.
Following deposition, the cannula is quickly removed from the pocket.





673 Arestin (Ora Pharma)
Active ingredient: 1 mg minocycline-HCl per dose
Vehicle: Microcapsules, powder-form

Arestin is the newest development on the market (2001). Pre-prepared Arestin portions are provided within a disposable tip. The powder is “blown” via air pressure within the metal tip into the pocket.

Left: Arestin portions. Each pocket receives an individual portion.

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Host Response/Reaction—Modulating Substances

None of the *antimicrobial* therapies available today can eliminate periodontal pathogens over the long term.

- Mechanical/instrumental *debridement* is essential, because it directly removes the biofilm, which is the most important entity that provides perpetuity for the periodontitis-associated bacteria.
- Adjunctively employed *antimicrobial pharmaceuticals* (Fig. 674, 1) reduce the absolute number and the percentage composition of pathogenic species in the subgingival plaque in "aggressive" cases or cases that are "refractory" to conventional therapy.

In the final analysis, a stable, long-term periodontal success (control of pathogenic species) will only be achieved if bacterial habitat surroundings can be changed: Minimal supragingival plaque (no re-colonization), reduced inflammation (less nutrition), and reduced pocket depths (reduced microbial reservoir), as well as cessation of *damaging* habits (smoking etc.).

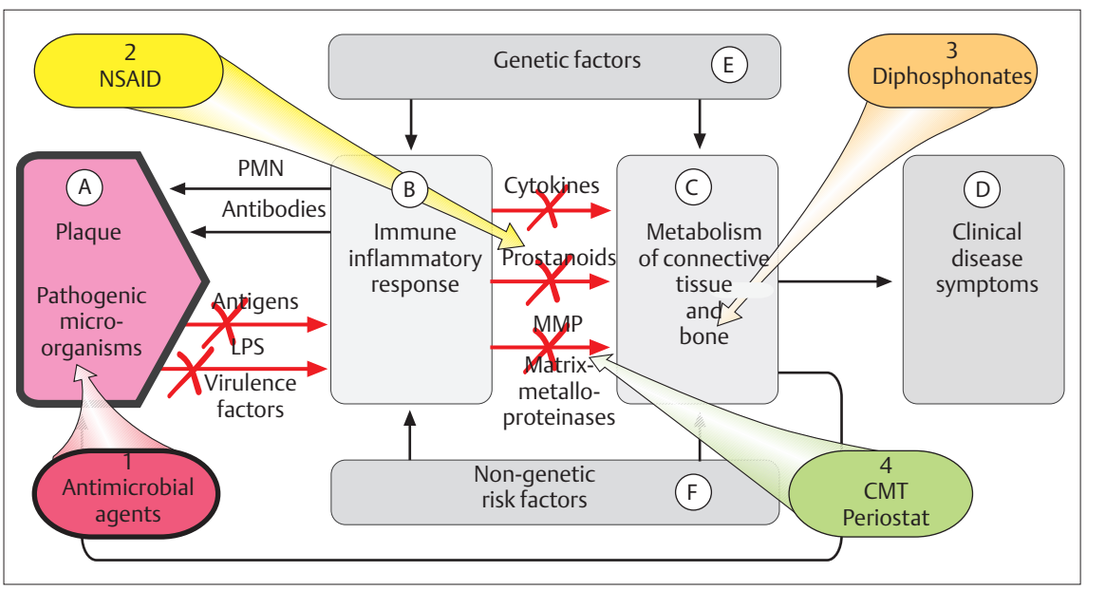
If these proven modalities do not lead to stabilization of the periodontal status (a tolerable proportion of pathogenic species), additional therapeutic measures must be considered (Haffajee et al. 2003).

674 Possible Aids for Mechanical Periodontitis Therapy—Mechanisms of Action

1 Antimicrobial agents: Antibiotics, antiseptics
2 NSAID—non-steroidal anti-inflammatory drugs
3 Diphosphonates (inhibit bone resorption)
4 CMT—chemically modified tetracyclines against MMP

1 works *against* bacteria.
2, 3, 4 *enhance* the host defense against the various mechanisms (see text).

Modified from *R. Page 1998*



Prevention of Unfavorable Host Responses

The primary problem in periodontitis is the suppression or elimination of host enzymes that elicit inflammation and tissue loss. In addition to anti-bacterial therapy (**1**, see above), the following reactions must be modulated:

1 Pathogenic bacteria → endotoxin/LPS → various host responses (tissue loss)
2 Macrophages → prostaglandin E2 → inflammation, destruction of alveolar bone
3 Osteoclasts → lysozomal enzymes → destruction of alveolar bone
4 Macrophages, PMNs etc. → metalloproteinases → periodontal tissue loss

2 Non-Steroidal Anti-Inflammatory Agents

NSAID—"non-steroidal anti-inflammatory drugs"

These pain-reducing and anti-inflammatory substances represent diverse chemical groups, all of which reduce prostaglandin (PGE2) synthesis by blocking cyclooxygenases 1 and 2 (COX-1 and COX-2; p. 49). Because of the possibility of significant adverse effects (blood clotting, stomach lining irritation), only pure COX-2 inhibitors should be used over the long term and in higher doses (Salvi et al. 1997, Morton et al. 2001, Holzhausen et al. 2002).

3 Diphosphonates

Diphosphonates represent an additional category of pharmaceuticals that can be employed for regenerative periodontal procedures and dental implants, as well as the above-mentioned types of periodontitis, for modulation of the host response to bacterial metabolites (lipopolysaccharide; Paquette et al. 2000). Diphosphonates are routinely used for the treatment of systemic bone diseases (Paget's disease, osteoporosis) because of their ability to inhibit bone resorption. Diphosphonates (e. g., Alendronate) have a high affinity for bone, render hydroxyapatite less soluble, and inhibit osteoclasts while enhancing osteoblast differentiation (see summary by Tenenbaum et al 2002).

4 CMT—Low-Dose Chemically Modified Tetracyclines

At reduced dosage, CMT (e. g., doxycycline) is no longer antibacterial (thus, no elicitation of resistant strains), but it works only to reduce Zn^{2+} or Ca^{2+}-dependent enzymes such as matrix metalloproteinases (MMP from resident and infiltrate cells; p. 50; Golub et al. 1985, Vernillo et al. 1994, Ryan et al. 1996, Ramamurthy et al. 2002).

The drug "Periostat" is taken for 90 days (20 mg/d) systemically; it is effective against soft tissue loss and bone loss.

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Phase 2 Therapy
Periodontal Surgery—Corrective Phase

Periodontal surgical therapy is only *one component* of complete periodontal treatment. If surgery is necessary, it is usually performed as a second phase (corrective), following a thorough *evaluation* of the clinical results of Phase 1 therapy. The patient must be motivated, and must exhibit adequate plaque control. The first phase of *initial therapy*, professional supragingival tooth cleaning, must be complete. In addition, clinical studies have demonstrated that the debridement of subgingival calculus and biofilm must be performed *before* surgical intervention. Following such extensive pre-treatment, surgery is less often necessary, the surgical procedures are associated with less hemorrhage, and the morphological results are better, with less tissue loss ("longer teeth" are less of a problem).

The *primary* goal of periodontal surgery is the elimination of infected pockets that have not responded to conservative treatment and/or medicinal adjuncts. Surgical intervention is therefore indicated with deep pockets, intraosseous defects and furcation involvements.

The *secondary* goal is the correction of defects in cases of unphysiologic gingival and osseous architecture, with special regard to creation of conditions that simplify or guarantee efficient plaque control, especially in the interdental areas.

Pocket depth reduction and/or pocket elimination maintain their importance in contemporary periodontal therapy (Slots 2002, Petersilka et al. 2002, Socransky & Haffajee 2002; cf. p. 358).

Toward these goals, a vast array of various surgical techniques is available; they will be described in this chapter.

The following important aspects will be considered:

- Purposes and goals of periodontal surgery
- Patient selection and defect factors
- Influences on the treatment results
- Methods and indications
- Preparations, instruments
- Incisions, sutures, knots
- Flap surgery:
  - "access flap" (open therapy, access flap)
  - regenerative methods
  - resective therapy
- Gingivectomy—gingivoplasty
- Furcation treatment

Mucogingival, plastic surgical methods, their goals and indications will be discussed subsequently in a separate chapter (beginning on p. 397).

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Purposes and Goals of Periodontal Surgery

Periodontal surgery, its goals and purposes, can only be considered in conjunction with complete periodontal treatment. For example, initial therapy and surgery are two entities with identical goals, but which use different methods to achieve these goals (closed root planing versus root planing with direct vision). Furthermore, initial therapy may be the *only* therapy required for mild periodontitis, whereas in severe cases it may represent only a preparatory presurgical phase.

The *goals* often cannot be achieved using a *single* specific surgical procedure; rather, often combinations of various surgical methods are required, either simultaneously or one following upon the other:

- Root cleaning/debridement with direct vision
- Reduction or elimination of plaque-retentive areas that promote infection, especially periodontal pockets
- Elimination of inflammation
- Enhancing the regeneration of periodontal tissues
- Elimination of diseased tissues—resective therapy
- Creation of physiologic morphology/architecture of the marginal periodontium
- Correction of mucogingival defects, restoration of esthetics in the aveolodental tissues

Root Cleaning with Direct Vision (Access Flap)

The root surfaces are exposed to clinical view either by reflecting a gingival flap or, less often, following excision of the gingiva (gingivectomy, p. 367). Plaque and calculus can then be removed from all root surfaces, including furcations, irregularities etc., with *direct vision*.

Removal of Infection-Enhancing Niches

The most important niche for subgingival microbial flora are *periodontal pockets* themselves. Also of importance are open furcations, root irregularities, fusions, grooves and other oral structures.

Periodontal pockets can be eliminated by flap surgery or gingivectomy (*resective* therapy). One may also attempt to *heal* the defects, especially bony pockets, through *regenerative* surgical procedures (p. 323).

The above-mentioned root irregularities, fusions and grooves can be reduced by means of careful odontoplasty, usually after flap reflection.

Elimination of Inflammation

The clinical procedures described above (root debridement or planing and reduction of niches) lead to the *elimination of the causes* of periodontal inflammation. Clinical symptoms of activity such as exudation, bleeding and suppuration are eliminated. Freedom from inflammation *always* leads to consolidation of the gingival tissues, shrinkage or gingival recession; this alone results in a more or less pronounced pocket depth reduction.

Enhancing Regeneration of Periodontal Tissues

The hoped for results of surgical procedures include not only cessation of the disease process, but also “healing” of the pocket via regeneration of periodontal tissues.

This goal has been approached with some successes during the past two decades. The implantation of *bone* and *bone replacement materials* into the intraalveolar pockets, the *GTR technique*, the use of *matrix proteins* and in the future, use of growth factors are extremely promising. Unfortunately, the predictability of success using these methods is, today, neither understood nor reliable (see also patient factors, p. 324).

Elimination of Diseased Tissues—Resective Therapy

As mentioned above, the results of regenerative surgical procedures cannot always be predicted with certainty. But the practitioner strives for a “pocket free” periodontium whenever possible, especially when restorative/reconstructive procedures are planned upon those teeth that were previously periodontally involved.

Investigations by the Slots group (2000, p. 358) have also demonstrated that following *resective procedures* (*osseous surgery*), in comparison to simple “access flap” procedures, the depth of the residual pockets is less, and also the colonization by periodontopathic microorganisms (anaerobic!) is more significantly reduced following use of more radical surgical procedures.

Creation of Physiologic Morphology of the Marginal Periodontium

During the course of periodontitis within any given patient, gingival swelling or on the other hand gingival shrinkage may occur. Therefore, the goal of surgical intervention is to create a harmonic course of the gingival margin, which is achieved through the choice of incision (sulcular, paramarginal), the recontouring of the alveolar bone and the type of flap repositioning, usually at a somewhat more apical niveau. In addition to improved esthetics, plaque control should also be simplified for the patient.

Correction of Mucogingival Defects—Re-Creation of Esthetics

The purposes and goals of *mucogingival surgery* include the widening of the attached gingiva, which is usually associated with deepening the oral vestibulum. Another major goal is soft tissue coverage in areas of gingival recession (p. 413) and correction of alveolar ridge defects, which anticipate additional treatment in the areas of prosthetics and dental implantology.

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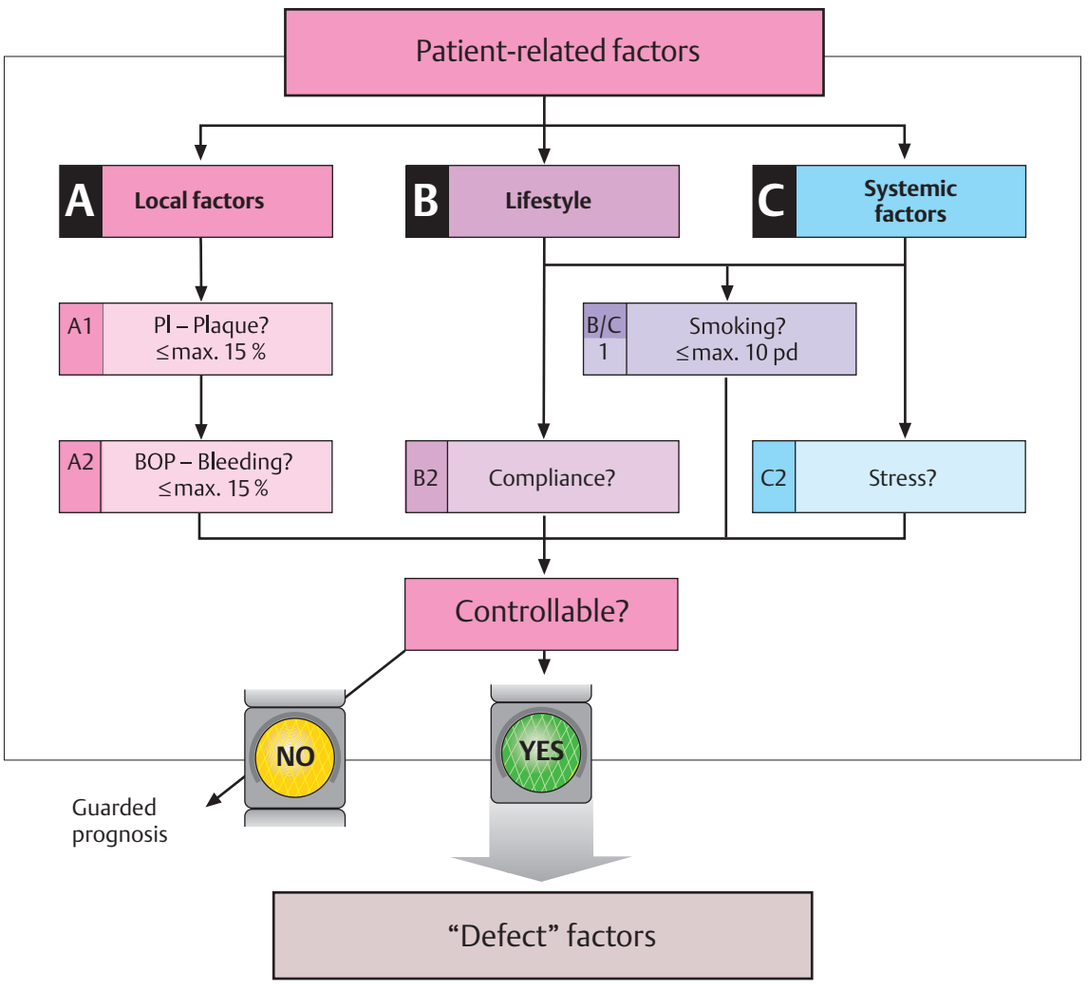
Patient Selection

In most industrialized nations today, every patient has the "right" to medical/dental treatment.

The term "selection" therefore does not imply ruling out any type of therapy; treatment recommendations and the definitive treatment plan may vary considerably for each individual patient. The general medical history, local and systemic risk factors, "behaviors," understanding of the individual oral situation and the interdependent consideration of the patient's compliance are critical.

Perhaps most important, even beyond the active treatment itself, are follow-up examinations (and treatments) during recall appointments. The long-term success of periodontitis treatment must be secured *life-long*, and this is a demand that not every patient can fulfill.

All of these factors lead to the obvious conclusion that the therapeutic possibilities and the type of therapy may be quite different for each individual patient. These possibilities may range from a purely palliative treatment with a severely reduced prognosis, to systematic and synoptic periodontitis therapy including surgical procedures, functional therapy, comprehensive reconstructions and even dental implants.



675 Patient Factors: Considerations that Render Patient Treatment Controllable

A *Local factors*:
What form of periodontitis is present? Chronic or aggressive?
What is the extent and severity of the disease?
What is the magnitude of plaque accumulation (PI score), and what is the level of patient compliance?
What is the level of expression of the disease with regard to symptoms of activity, bleeding (BOP)?

B *Behavior* and compliance (see also the extensive discussion in the chapter "Etiology and Pathogenesis"). Are alterable risk factors present (smoking: cigarettes per day?, packs per year?, nutrition, awareness of health status)?

C Systemic, *general medical diseases*, genetically-elicited syndromes, but also stress, psychic consequences and non-alterable risk factors

Modified from *P. Cortellini & G. Bowers 1995*

Defect Factors

In addition to the listed *patient factors*, there are, of course, also *defect factors* such as *morphologic defects*, that are of importance for the prognosis of any therapeutic procedure. Included here are the width and thickness of the gingiva (phenotype), course of the horizontal bone loss and the depth of supra-alveolar pockets. Most importantly:

- *Intra-alveolar* defects are classified as 1-, 2- or 3-wall bony pockets, with consideration of the depth and the width of the "opening angle" of the defect, acknowledging that most of these defects represent "combined" bony pockets: In the apical region, three walls are in evidence, in the "middle," two walls, and coronally only one wall!
- *Furcation* involvement, and its severity
- In patients with gingival *recession*, the depth and width of the gingival recession (Miller classification, p. 162) is of importance for predicting the therapeutic success.

Both *patient factors* and local *defect factors*—as well as the "aptitude" of the clinician, are of determining significance for the planning, type of surgical procedure and the ultimate results (see next page).

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Factors that Influence the Treatment Result

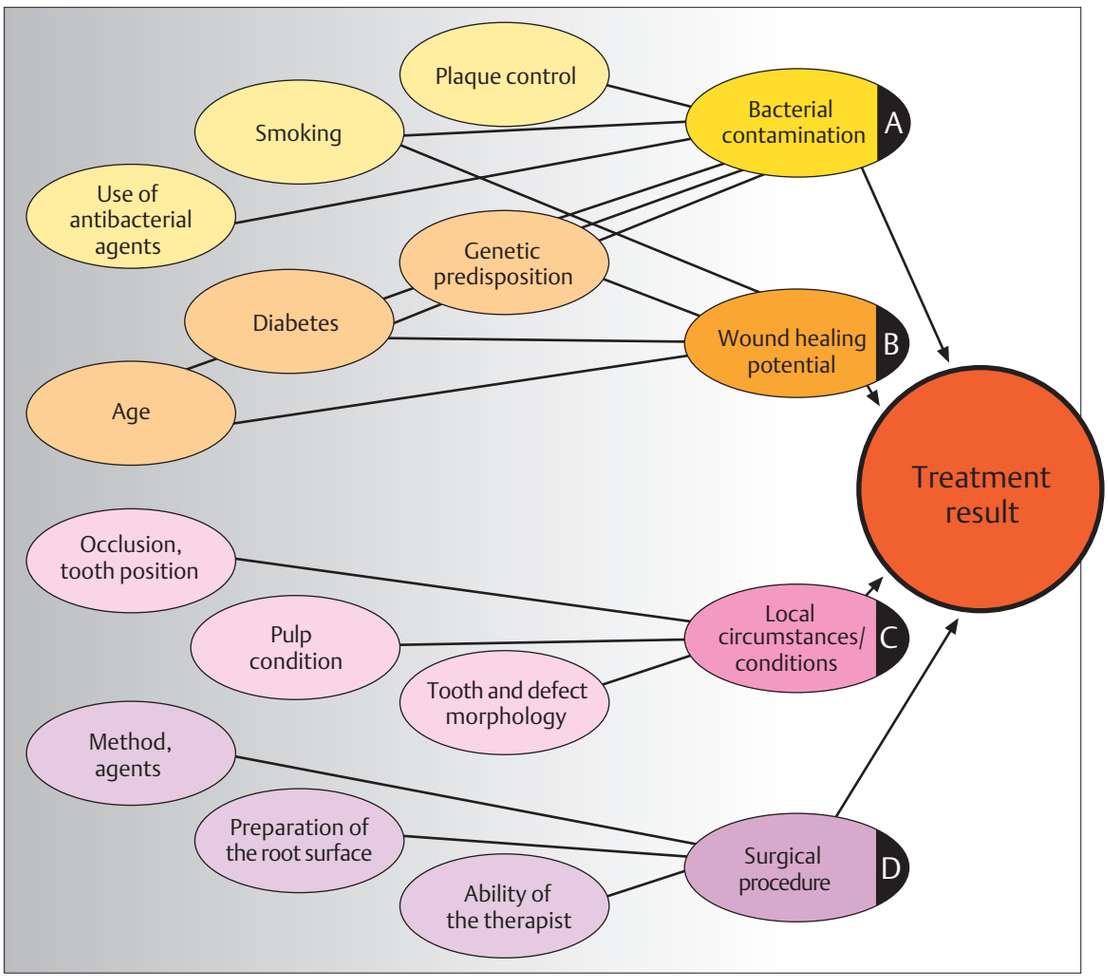
On the previous pages, we discussed factors about patient selection and periodontal defect morphology, which are of importance for the selection of an effective surgical procedure. The term *success* (a *positive treatment result*) must be defined, and especially must differentiate between *short-term* and *long-term* success.

In the *short-term*, these goals could be achieved by means of the classical—decades-long acknowledged—methods of open or closed root scaling and planing, if adequate plaque control is subsequently insured.

The goal must, however, include *long-term* measures after active therapy, over years and decades, to insure a healthy periodontium and therewith the patient's oral integrity.

The most important criteria of success include:

- Regeneration of the alveolar bone
- Attachment gain or at least maintenance of the attachment level, reduction of pocket depth and/or pocket elimination
- Elimination of symptoms of disease activity (bleeding)
- Stabilization of tooth mobility
- Maintenance therapy!



676 Factors that Influence the Treatment "Outcome"
An optimum, *long-term* treatment result depends not only on precise local treatment, but is also "steered" by a myriad of influences and factors.

Purely mechanical plaque control remains the cornerstone of treatment, supported in cases of aggressive periodontitis by medicinal adjunctive therapies.
In addition, however, efforts should also be targeted toward influencing co-factors within the overall etiology. Systemic diseases can usually be successfully treated; tooth position, tooth and alveolar defective morphologies as well as occlusal disharmonies can be corrected; endo-perio problems can usually be solved. On the other hand, correction of the genetic constellation of a patient is not possible, the aging process cannot be halted, and a patient addicted to smoking is extremely difficult to manage.

Modified from *K. Kornman & P. Robertson 2000*

The long-term success is dependent upon many factors:

A Without question, the "bacterial load" plays a determining role, but in addition to the microbiologic load, the pathogenicity and specificity of the microorganisms are critical (Etiology and Pathogenesis, p. 21; Diagnosis, p. 165).

B In addition, the wound healing potential plays an important role, and this can vary considerably from patient to patient through genetic determination, systemic disorders or even heavy tobacco use.

C Oral, local and morphologic situations may be of significance.

D In addition, the type of therapy, i.e., the surgical procedure itself, plays an important role in determining the long-term success.

These four primary parameters are based upon a myriad of other influences (Fig. 676). In the final analysis, long-term success is determined not only by the proper choice of a treatment methodology and its flawless performance, but also by the expertise of the surgeon. But the most important variable remains optimum plaque control (patient; recall!).

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Methods of Periodontal Surgery and their Indications

Which periodontal surgical procedure to select? The choice depends primarily upon the disease *type* (AAP classification, 1999, p. 519), as well as the *severity* and the *expanse* of the disease.

If the patient presents with *aggressive periodontitis* (AAP Type III), and if the etiology includes not only periodontopathic bacteria but also significant co-factors (see p. 298), a more radical surgical procedure should be considered, also possibly incorporating systemic or topical medicaments, and perhaps even extraction of severely involved teeth. On the other hand, if the patient's cooperation and compliance (as assessed during Phase 1 therapy) are not optimum, one must consider whether simple palliative treatment (debridement) should be performed, because the long-term results of extensive surgical procedures would be compromised from the onset.

The *degree of severity* of the disease, i. e., the present state of progression, Class III (severe form) demands pharmacologic support following radical or intensive surgical procedures, much more than the therapy of less severe cases (Class I or Class II).

In addition to the type and severity of disease, the *pathomorphology* of the diseased periodontium will also determine the nature of surgical intervention. Are the gingiva and/or the alveolar bone thin, or thick? Is the periodontal bone loss horizontal in nature, or are vertical (angular) osseous defects present? Does the patient present with a complete dentition, or are teeth missing? Even the morphology of the individual teeth and their position in the arch can influence the choice of a surgical procedure.

This myriad of factors will determine, in an individual patient, or even in a given segment of the dentition, which of the various surgical methods must be employed adjacent to each other or combined in a surgical procedure.

Periodontal Surgical Procedures

- *"Access flap surgery,"* open flap debridement (OFD), e. g., modified Widman flap (MWF), p. 309
- *Wedge excisions* on lone-standing or individual teeth (p. 319)
- *Regenerative methods*, pocket implantation with bone or bone replacement materials, "guided tissue regeneration" (GTR), use of matrix proteins or growth factors (p. 323)
- *Resective procedures* (osseous surgery, pocket elimination, crown lengthening; pp. 355, 493)
- *Gingivectomy (GV)/gingivoplasty (GP)*: These procedures are included in the resective or modeling methods (p. 367)
- *Surgical furcation therapy* (p. 381)
- *Mucogingival plastic surgery* (p. 397)

"Access flap" (p. 309) connotes the creation of visual access to periodontally involved root surfaces. The methods comprise for the most part the techniques formerly referred to as "modified Widman procedures." In principle, even in the more complicated flap procedures the goal is to create access. The guiding principles of the modified Widman flap procedure—incisions, creation of the flap, tight adaptation during flap repositioning—must also be adhered to.

Wedge excisions may be indicated when, distally, an end-standing or a lone-standing tooth exhibits bony pockets.

Regenerative methods are becoming more and more popular in periodontitis therapy. In contrast to the more traditional resective methods, these attempt to rebuild lost tissues, and the regeneration of periodontal defects is surely a desirable goal. Unfortunately, today, the long-term results cannot yet be predicted with certainty. Regenerative treatment techniques are indicated primarily in cases of vertical osseous defects, in furcation involvement (F1 or F2), and for covering areas of gingival recession (Class I and Class II).

Resective procedures retain, even today, their position as the most predictable of success, even long-term. Resective procedures are indicated with irregular alveolar bone loss, and when the osseous morphology requires osteoplasty or ostectomy. There are significant disadvantages to resective therapy, which temper their indication.

Gingivectomy (GV) and *gingivoplasty (GP)* represent surgical techniques within the rubric of resective soft tissue treatment methods. As a treatment modality for periodontitis, GV is only rarely employed today. On the other hand, GP remains the treatment of choice for contouring hyperplastic gingiva.

Surgical furcation therapy is indicated in Class F3 furcation involvement, and often also F2 involvement. Such treatment may be resective or maintenance. Amputation of individual tooth roots, hemi- and tri-section with maintenance of individual or all roots can be recommended for maintenance of the masticatory efficiency of a dental quadrant or if a tooth must be maintained as a component of a total arch reconstruction. The alternative may involve the use of osseointegrated, root-form dental implants.

Mucoplastic Procedures

Mucogingival surgery is not truly a method for the treatment of periodontitis. It is indicated primarily for the treatment of progressive gingival recession and its consequences, as well as in the treatment of alveolar ridge defects. It is truly a form of plastic surgery, which helps to eliminate esthetic problems.

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Principles of Various Treatment Modalities—Advantages and Disadvantages

The *principles* of the most important—*non-surgical* and *surgical*—treatment methods are depicted schematically below. These must often be modified, expanded, or combined with other adjunctive measures such as supportive medicinal therapies. Each technique is described, including its advantages and disadvantages.

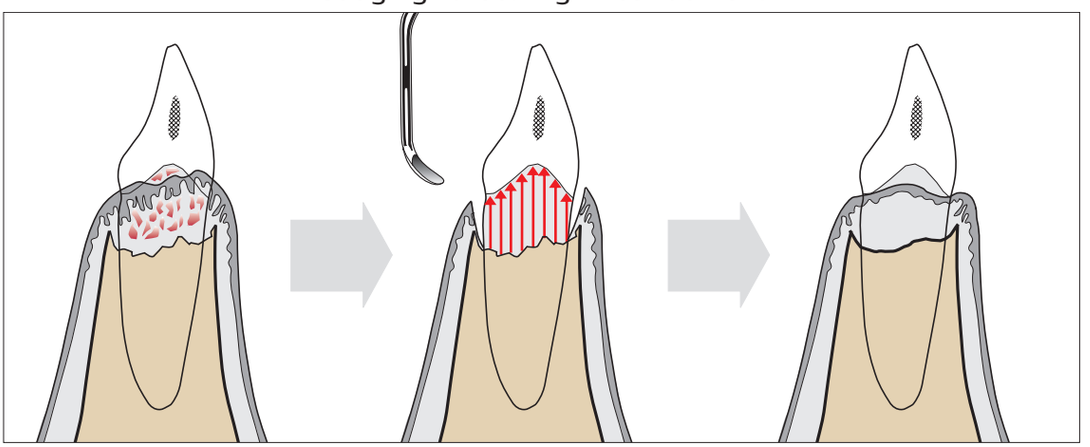
Subgingival Scaling/Root Planing
Advantages: Relatively hemorrhage-free, less gingival shrinkage (good esthetics), rapid healing—*Gold standard*!

Disadvantages: Treatment is performed without direct vision, and is not indicated for very deep and complicated pockets; healing occurs by means of a long junctional epithelium.

Curettage
Advantages: Removal of pocket epithelium and infiltrated (bacterially colonized) subepithelial connective tissue.
Disadvantages: Not a definitive therapy; when used, only in combination with subgingival scaling or surgical procedures.

677 Subgingival Scaling (Debridement), Closed Root Planing
Removal of the biofilm and any concrements from the root surfaces is often the only therapy necessary for shallow pockets (gold standard), and is the obligatory pretreatment in severe cases prior to surgical intervention (p. 253). The instruments indicated include primarily curettes and ultrasonic devices. The initial situation, the procedure itself, and the therapeutic result are depicted (left—middle—right).

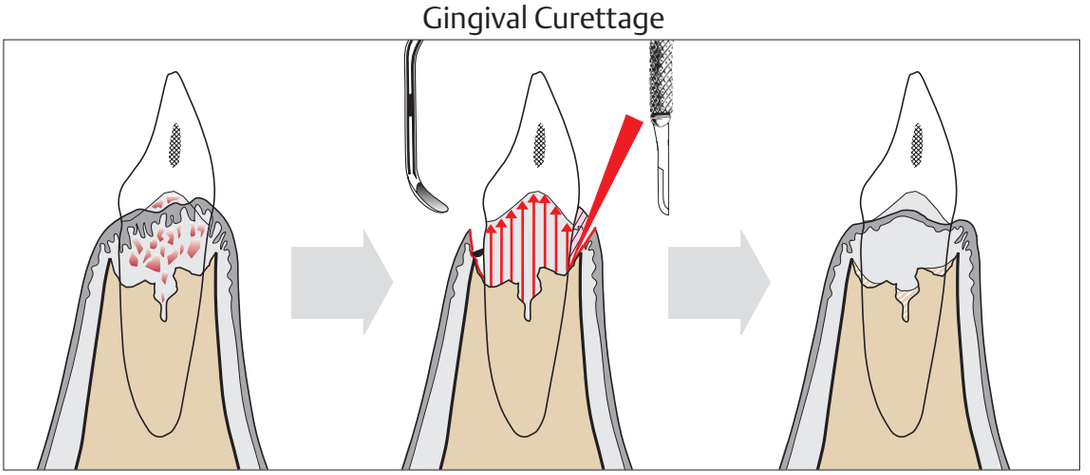
Subgingival Scaling—Debridement



678 Gingival Curettage
This includes removal of the pocket epithelium and the infiltrated/infected connective tissue. This procedure *never* represents definitive treatment for periodontitis. It is performed in combination with the essential therapeutic measures including closed and/or open root cleaning/planing.

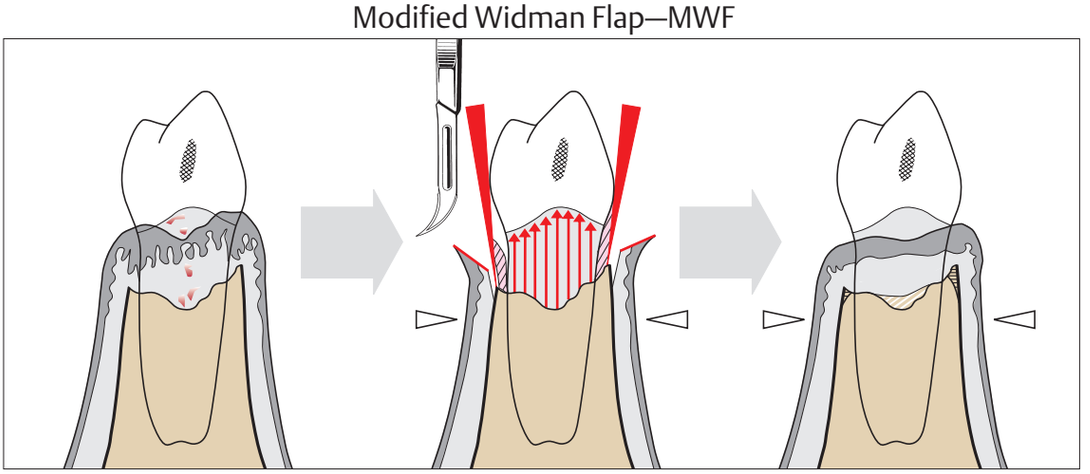
For gingival curettage, the double-edged universal curettes are indicated (*left* in the middle picture) or fine scalpels may be used for sharp dissection.

Gingival Curettage



679 MWF—Modified Widman Flaps
After performing a "scalloping" paramarginal incision (internal GV) and additional marginal and interdental incisions, a "*partially mobilized*" mucoperiostial flap is elevated to the level of the alveolar crest (p. 309).
Cleaning of the root surfaces using curettes or ultrasonic scalers is performed with direct vision ("access flap"). Re-adaptation of the flaps following treatment is accomplished with interdental sutures.

Modified Widman Flap—MWF



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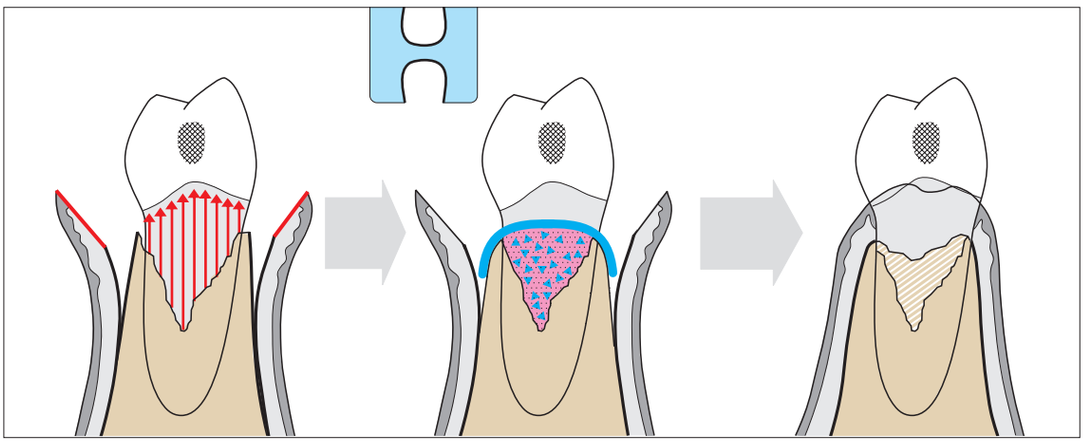
Access Flap, e. g., Modified Widman Flap Procedure (MWF)
Advantages: Scaling with direct vision, removal of pocket epithelium and infiltrated subepithelial tissues.
Disadvantages: Tissue loss (gingival margin, interdental) resulting from flap creation and the internal gingivectomy.

Regenerative Therapies, e. g., GTR
Advantages: An attempt to regenerate periodontal tissues; attachment gain.
Disadvantages: Usually only localized osseous defects can be so-treated; danger of infection of the membrane; predictability is uncertain; surgery is technically demanding; high financial burden.

Resective Therapy
Advantages: Elimination of pockets and their bacterial colonization (*Aa, Pg*; p. 23); predictable results; stable gingival margin; abutment teeth for planned reconstructions.
Disadvantages: Esthetics; exposed and possibly sensitive tooth necks.

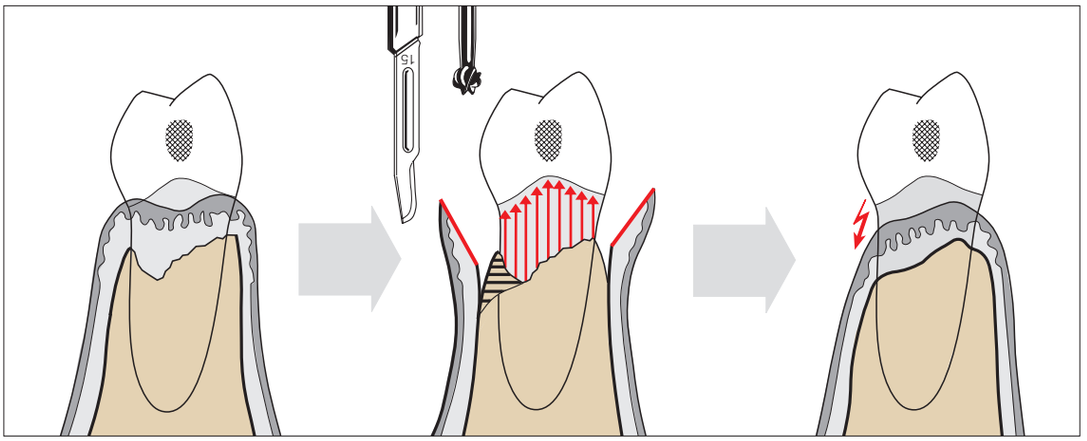
Gingivectomy/Gingivoplasty (GV/GP)
Advantages: Pocket elimination, gingival recontouring; above all indicated in cases of gingival hyperplasia.
Disadvantages: Possible only in the region of the attached gingiva; open wound; secondary healing; possible esthetic problems; no approach to osseous defects.

Regenerative Therapy



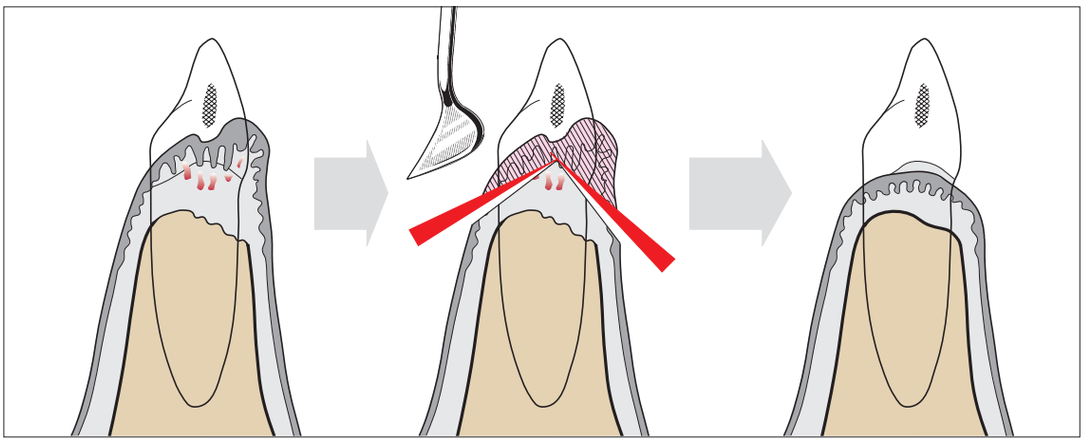
680 Regenerative Surgery
After making horizontal incisions, a mucoperiostial flap is raised. Debridement of the osseous defects and root planing are performed as usual, with direct vision, and the osseous defects are then covered with a membrane (GTR technique). In addition, the osseous defect may be filled with autogenous or heterologous material (*middle*). The goal of this treatment modality is not only new bone formation, but also at least a partial periodontal regeneration (p. 323).

Resective Therapy



681 Resective Procedure
Following paramarginal incisions and vertical releasing incisions, an expansive mucoperiosteal flap is reflected (*middle*). The interdental osseous craters are essentially eliminated by osteoplasty and ostectomy ("ramping"). The soft tissue flaps are apically repositioned. The results (*right*) are predictable and stable over the long term (important for dental reconstructions). Disadvantages include sensitive tooth necks and often compromised esthetics (anterior segments) (p. 355).

Gingivectomy/Gingivoplasty



682 Gingivectomy/ Gingivoplasty (GV/GP)
In cases of periodontitis, gingivectomy (GV) for pocket elimination is only rarely performed; however, gingival overgrowth (e. g., drug-induced) and the resulting pseudopockets (*left*) can be removed and sculpted by means of gingivoplasty (*middle*).
GV (*middle*) is performed with special instruments (p. 367).
Fine modeling of the tissue contour can be performed using the electrotome or, more recently, laser surgery.

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Pre-operative Treatment—Post-operative Care

The patient must be completely informed about the type of surgical procedure and its possible complications. The success of any periodontal surgical procedure—as with any other surgical procedure in the oral cavity—can be improved by appropriate pre- and post-surgical care.

Pre-operative

In principle, before every periodontal surgical procedure, the first phase of initial therapy—supragingival plaque and calculus removal—must be performed. In addition it has proven to be beneficial that a subgingival debridement also be performed. In addition to these treatment modalities by the dentist or the dental hygienist, patient education *must* lead to efficient *plaque control*, especially in the interdental areas.

Even in the initial medical history, alterable risk factors (lifestyle, smoking etc.; p. 167) must be minimized whenever possible. In the case of systemic diseases, conversation with the patient's physician must clarify whether antibiotic prophylaxis is required, or if there exists any alteration of the INR value (previously: Quick Test, p. 212), or if sedative medications are necessary.

683 Pre-operative
One day before the surgery, the patient is instructed to rinse every 12 hours with chlorhexidine mouthwash.

If there is a tendency to swelling or pain, the patient should take an antiphlogistic and/or a pain medication before the surgery and up to 3 days thereafter.



Pre-operative—Protocol
Important in every case:
- Patient selection (p. 297)
- Topical CHX rinse
- Plaque control: PI < 15–20 %
- Relative gingival health: BOP < 15–20 %

To be performed as indicated:
- Risk minimization:
  – e. g., smoking (p. 216)
  – endocarditis prophylaxis
- Pre-surgical systemic medication:
  – reduce anticoagulants?
  – antiphlogistics, sedatives

Post-operative

The patient must adhere to certain pre-operative agreements. The patient must be given written instructions, "procedures following surgery," describing the necessary action. *In general*, the patient should avoid corporeal exigencies (strenuous sports), and should avoid excessive exposure to the sun. The surgical site must be stabilized. Sutures and dressings must be protected. In the case of highly mobile teeth, it may be necessary pre-operatively to provide a temporary splint (p. 471). The pre-operative ingestion of antiphlogistics should be continued for an additional 3–4 days. Cooling measures (ice pack) are recommended upon the skin above the operation area. In addition, chlorhexidine rinses are indicated to eliminate the necessity for mechanical oral hygiene at the surgical site. Sutures and periodontal dressings are removed 7–10 days post-surgically. Thereafter, careful tooth brushing is initiated. Such mechanical tooth cleansing enhances chlorhexidine rinses.

Following these post-operative measures, it is important, during the subsequent *maturation* of the tissues, to provide repeated observation of the healing for several months (Westfelt et al. 1983): The area of surgery should be observed every four weeks, and should be professionally cleaned, and the patient's own plaque control efforts should be monitored.

684 Post-operative
Ice bags or cold packs are recommended immediately following the surgery.
Depending upon the type of surgical procedure, CHX rinses should be performed for 2–6 weeks. No mechanical oral hygiene should be performed in the surgical area, but toothpicks may be used to carefully remove any impacted food.
Antiphlogistics, p.r.n. Relatively high doses following mucogingival surgery in every case (despite the resultant slower healing!).



Post-operative—Protocol
To be performed in every case:
- Patient information about post-operative measures (written instructions)
- Cold packs
- Analgesic medication
- No mechanical oral hygiene in the surgical area for 10–40 days (depending upon the type of surgery)
- CHX rinsing
- Suture and/or periodontal pack removal in ca. 7–10 days

To be performed as indicated:
- Antiphlogistic medication

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Flap Procedures—Open Treatment

The almost fluid transitions between modern closed and open (surgical) periodontal therapy were discussed on page 253. Both types of therapy are targeted toward removal of plaque biofilm from the root surfaces, removal of pathogenic bacteria, and removal of calculus in order to create a bioacceptable root surface and therefore successful healing (p. 206). The procedures, however, should also simplify plaque control over the long term, and reduce the re-colonization of the treated pockets by periodontopathic microorganisms.

The reflection of *soft tissue flaps* makes it possible to directly view the tissues to be treated in the area of the periodontal defects such as bone (alveolar ridge, pocket morphology) and the morphology of the pocket; treatment can be rendered with direct vision. Soft tissue flaps can be repositioned at the original location or apically, coronally or laterally. One distinguishes between *full flaps* (mucoperiosteal flaps) and "split thickness flaps" (mucosal flaps). The borders of the flaps are determined by horizontal and vertical incisions (p. 304).

General Indications

Flap procedures are indicated in cases of periodontitis with active (inflamed) pockets over 5 mm deep, which do not respond satisfactorily to initial, closed therapy.

Special Indications

- Infrabony pockets, defects
- Implants and osseous transplants into infrabony pockets
- Guided tissue regeneration (GTR)
- Pronounced thickening of the marginal bone
- Hemisection of a tooth, or root resection
- Tooth extraction with immediate periodontal treatment of remaining adjacent periodontal structures
- Crown lengthening procedures to expose restoration margins for prosthetic reconstructions

Contraindications

- Pronounced gingival enlargement/overgrowth, which can be treated more efficiently by means of GV/GP
- Exposure of tooth preparations and restoration margins, usually better achieved by GV (exception: crown lengthening procedures).

Advantages

Flap procedures have the following advantages over closed root planing or GV/GP:

- Pocket epithelium is removed by the inverse bevel incision (internal gingivectomy); such pocket epithelium is usually colonized by pathogenic microorganisms.
- Optimum subgingival scaling and root planing to the base of the pocket can be performed with direct vision. Optimum subgingival scaling is also possible on difficult-to-access root surfaces.
- At the end of the surgery, the flaps can be replaced at the original location, or repositioned apically, coronally or laterally.
- The interdental bone or infrabony defects can be covered by the soft tissue flaps.
- No open wound persists post-operatively.

Disadvantage

With apical flap repositioning (resective techniques), and due to shrinkage of the gingiva, coronal segments of the root surface are often exposed, and this may be associated with poor esthetics, sensitive tooth necks, and the risk of root surface caries.

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Flap Design—Incisions

The reflection of every soft tissue flap begins with well-planned incisions. The type of periodontal defect, the goal of the surgical procedure and the desired result determine the course and direction of the incisions.

In every case, a *horizontal incision* will be performed. This may be purely an *intrasulcular* incision or it may course *paramarginally* at varying distances from the gingival margin. This incision will 1) remove the junctional epithelium and 2) shorten the gingival margin somewhat apically. This incision has been referred to an "internal gingivectomy." When viewed from either the facial or the oral aspect, the paramarginal incision courses in a scalloping form, approximately parallel to the (ideal) gingival margin. In order to protect the interdental papillae, in the case of relatively wide interdental spaces, the "*papilla preservation flap*" (Takei et al. 1985) has been recommended, with additional modification of the flap for narrow interdental spaces (Cortellini et al. 1995).

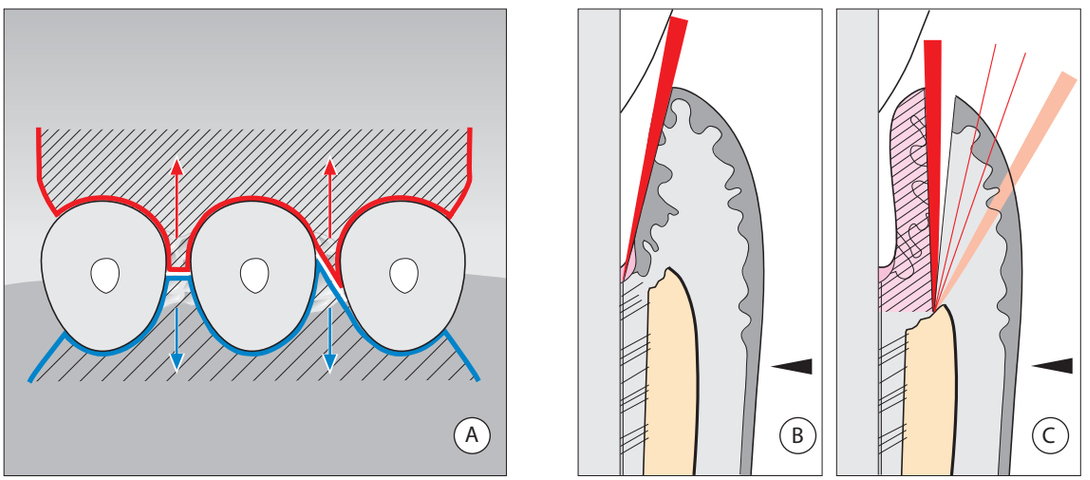
Vertical incisions are neither necessary nor desirable in every surgical procedure (mucosal scarring). If vertical incisions are necessary, they should be made in such a way as to prevent gingival recession or the loss of interdental papillae (Fig. 687).

685 Horizontal Incisions

A The *conventional* facial (red) and oral horizontal incisions (blue) usually meet each other interdentally at the height of the interdental papillae. The tissue surfaces in this area may approximate parallel or oblique.

B *Sulcular* incisions: Pocket epithelium is not removed. Maximum retention of soft tissues.

C *Paramarginal* facial (red) incisions at varying distances from the gingival margin. Tissue removal via "internal gingivectomy."

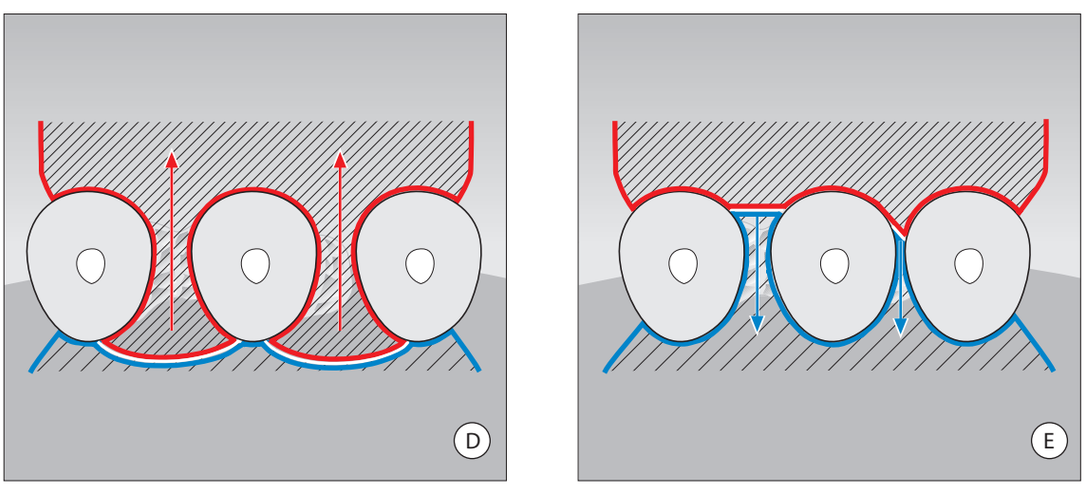


686 "Papilla Preservation Flap"

These incisions preserve the interdental papillae. Following wound closure post-surgically, soft tissue flaps cover the interdental region (the approximal defects), and "leakage" of the wound is inhibited. The papilla-protective flap can, however, only be instituted in cases with a relatively broad interdental space.

D The papillae (red incision) are mobilized facially.

E The papillae are mobilized orally with the flaps.



687 Vertical Incisions, Releasing Incisions

Unfavorable incision course?

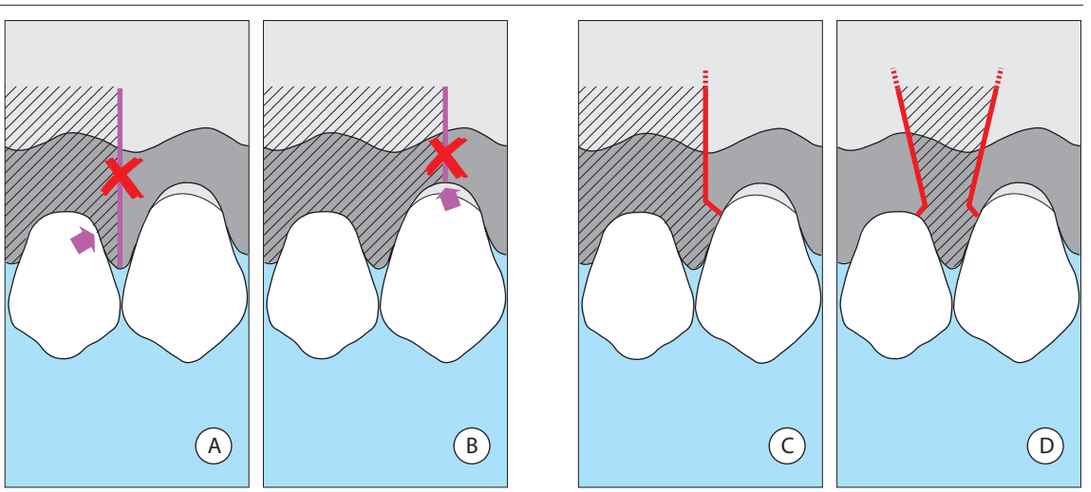
A Through the papilla tip: Danger of recession (loss of the papilla)

B Median: Danger of recession (shrinkage), undesirable in the case of facial pockets

Favorable incision lines:

C Paramedian: A better compromise, less shrinkage

D Triangular flap, paramedial incisions for the treatment of a localized periodontal defect



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Instruments for Flap Surgery

In general, the instruments used for periodontal flap surgical procedures are the same as those employed for other oral surgical procedures: *Scalpels*, *elevators* and *needle holders*. These instruments must be as fine as possible, to promote sensitive handling of the delicate tissues. The various instruments depicted and described below have proved useful in practice. Many other types and manufacturers are available that will yield satisfactory results if used correctly. If the flap procedure is to be combined with a gingivectomy/gingivoplasty, the gingivectomy knives described later in this text may also be required (gingivectomy knife, papilla knife; p. 368). The flap surgery "kit" also contains various forceps, explorers, periodontal probes, surgical scissors and clamps, and most importantly curettes for the debridement of root surfaces and osseous defects. In addition, bone burs or bone files or chisels (osteoplasty), and also fissure burs or diamonds for tooth hemisection, root resection, as well as specially indicated instruments will be depicted for individual surgical procedures.



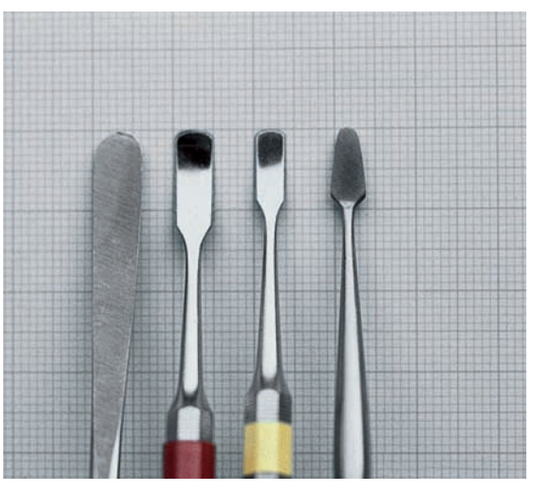


688 Scalpels
From right to left:

- **No. 1** (Martin)
- **No. 12 B** (Bard-Parker)
- **No. 15** (Martin)
- **No. 15 C** (Bard-Parker)

To insure sharpness, only disposable blades are utilized, providing a minimum of tissue trauma and enhanced healing.
Only fine and delicate scalpels are indicated for periodontal surgery, where precision is demanded and access is often limited.





689 Elevators

- **6 mm** (FK 300, Aesculap; white)
- **5 mm** (VT 24, 22, 23, Deppeler; red)
- **4 mm** (VT, Deppeler, yellow)
- **2.5 mm** (Special manufacture; Zabona, blue)

Small, narrow elevators (2–6 mm wide) are used to mobilize and reflect the soft tissue flaps. Larger/wider instruments may also be used during root planing or osseous recontouring to deflect the flaps from the field of operation, and improve visibility.





690 Needle Holders—Elasticity of the Closure (in gm)

- **Mathieu** (Aesculap; 1500 gm)
- **Boyton** (Hu-Friedy; 1200 gm)
- **Castrovieijo** (Aesculap; 500 gm)
- **Gillis** (Dufner; without closure, with scissors)

Needle holders must fulfill opposing demands: They must hold the needle securely, while allowing the surgeon to release the locking mechanism easily.

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Needles and Suture Materials

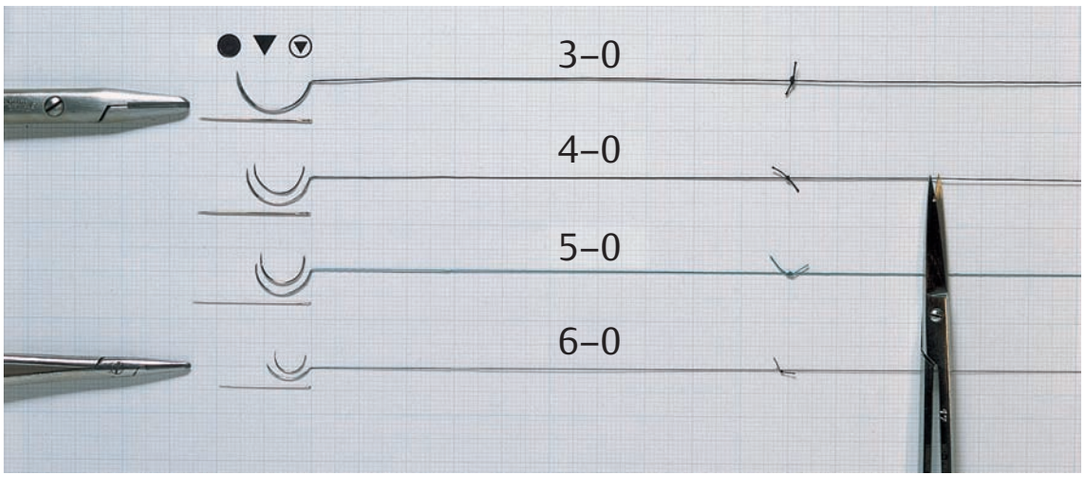
Following almost every flap surgical procedure in the periodontium, the wound is closed with sutures.

Today, atraumatic *needle-suture combinations* are used almost exclusively (optimally sharp, small diameter). Commercially available are straight or curved needles of varying thickness. The latter are available in various profiles (1/4, 1/2, 5/8, 3/4). If deeper tissue layers must be included in the suturing procedure, and if the access to the surgical site is difficult, sharply curved needles are recommended (5/8).

The finer the needle-suture combination is, so must also the needle holder be more delicate.

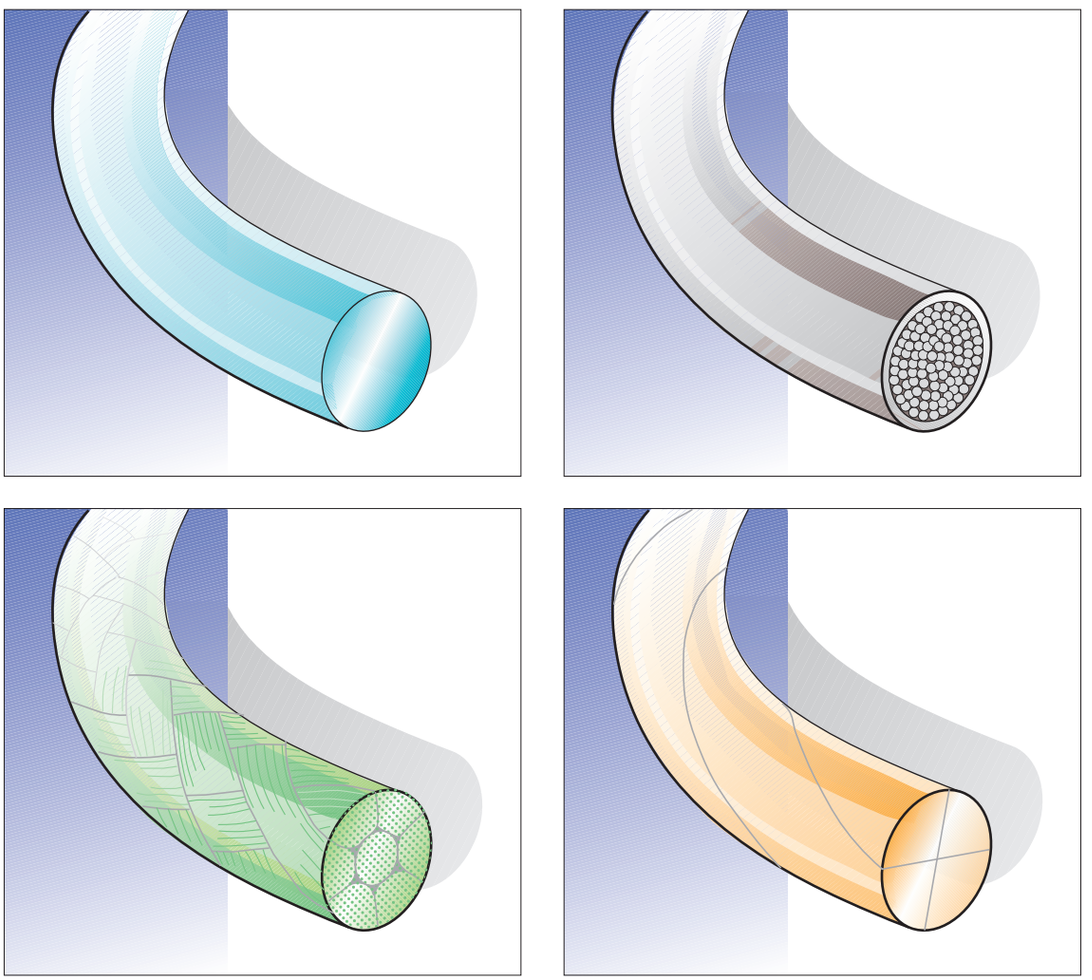
Suture material is available in many forms: Synthetic and natural, mono- and poly-filament, as well as resorbable and non-resorbable.

Depending upon the type and localization of the surgery, various suture materials are indicated for periodontal surgery. For example, a connective tissue graft will be affixed beneath the covering flap using monofilament, resorbable sutures, while the flap itself should be adapted using non-resorbable sutures.



691 Needles and Suture Material
Needles with a half-circular profile are depicted with varying thickness and the corresponding suture material.
According to the American Pharmacopia (USP), a suture with a 4–0 diameter approximates 0.150–0.199 mm.

The International Signets (logos, upper left) determine the diameter of needle points and needle bodies.



692 Suture Materials
Many types of sutures are available. The materials employed and their configuration determine characteristics such as strength, elasticity, bending strength, surface smoothness, capillary activity, susceptibility to contamination and resorbability. These four figures depict the characteristics of various sutures.

Above: **Monofilament or pseudo-monofilament Configuration**
Extremely fine (6–0 and thinner); used primarily in microsurgery.
Advantages: Ease of use, strength, and not enhancing bacterial colonization.
Disadvantages: Poor hold when simple knots are placed.

Below, left: **Complex fiber**
Advantages: Knots well, resistant, ease of insertion
Disadvantages: May enhance bacterial colonization

Below, right: **Twisted fiber, e. g., catgut**
Advantages: Soft, resorbable
Disadvantages: Animal source, promotes infection, not strong

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Sutures and Knots

The types of sutures and knots and loops that are used to affix and adapt soft tissue wound margins after periodontal surgery depend primarily on the goals of the surgical procedure. Additional criteria include the type of incision, the consistency of the tissue (attached gingiva or mucosa), as well as the type of suture material selected (type and thickness).

A knot always consists of at least two suture strands. The task of the first is to approximate the wound margins, and the second secures the knot and therewith also the definitive positioning of the tissues. The first loop can be doubled (see surgical knots, Figs. 694 and 695), which will secure a certain resistance to tissue displacement, and this can then be further secured by an additional loop. With certain surgical knots (e. g., the "one-one surgeon's knot"), using delicate, monofilament fibers, after placing the second loop, the sutures can be pulled tight to secure the soft tissue flap at the desired location. Securing the suture closure can then be accomplished with a third simple strand (for example, the Gore suture material).





693 "Sailor's Knot" (1–1)
This suture knot consists of a "right" and a "left around" loop. This knot—when it is formed by a thin, monofilament thread—can be tightened (careful with the direction!) and can therefore be displaceable upon the substrate (mucosa).
If it is necessary to secure this knot, a third simple loop can be incorporated (Fig. 696).





694 Surgical Knot (2–1)
A double and a single loop are used together in this simple surgeon's knot. This knot, most often used with braided sutures, is the most often used suturing technique in periodontal flap surgery when the repositioned tissues demand a certain tension in the opposite direction.





695 Double Surgical Knot (2–2)
This very secure knot consists of two double-loops. It forms and comprises relatively large, and sometimes troublesome, knots.





696 "Sailor's Knot" with Securing Loops (1–1–1)
The closable sailor's knot described in Fig. 693 can be secured with an additional loop.
Such three-times knots are often used with the "mildly maneuverable" Gore suture material (pseudo-monofilament).

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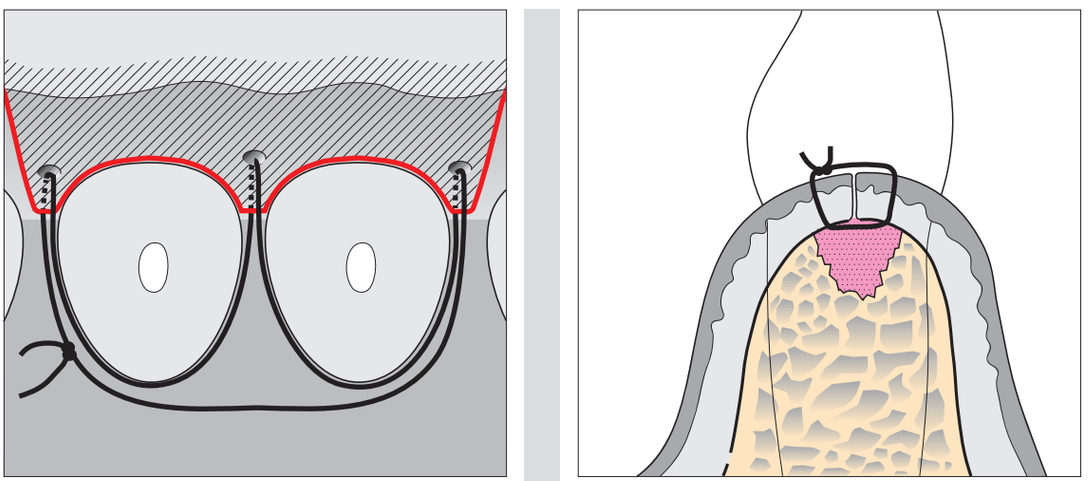
Frequently Used Suture Placements

Sutures have one purpose: To close the surgical wound by means of the repositioning of soft tissue flaps. If two wound margins are tightly approximated with sutures, healing by primary intention will occur within 24 hours. Healing originates from the basal cells of the epithelium, and later fibroblasts of the connective tissue, and is orchestrated by mediators and numerous other factors such as age, susceptibility to infection, and morphology (see Healing, p. 205).

Healing by primary intention unfortunately does not occur often in periodontology. It is not always possible to reposition two wound margins at the desired location and *secure that position*. In addition, subsequent to some special surgical techniques the *intention* is to reposition a soft tissue flap *not* at its original location, but rather, using appropriate sutures, to position that tissue flap coronally, apically or laterally. The consequence is that certain surgical areas will be reconciled to *healing by secondary intention*. The soft tissue flap does not always completely cover the wound. In a GTR procedure, the membrane may have a suture directly over the osseous defect.

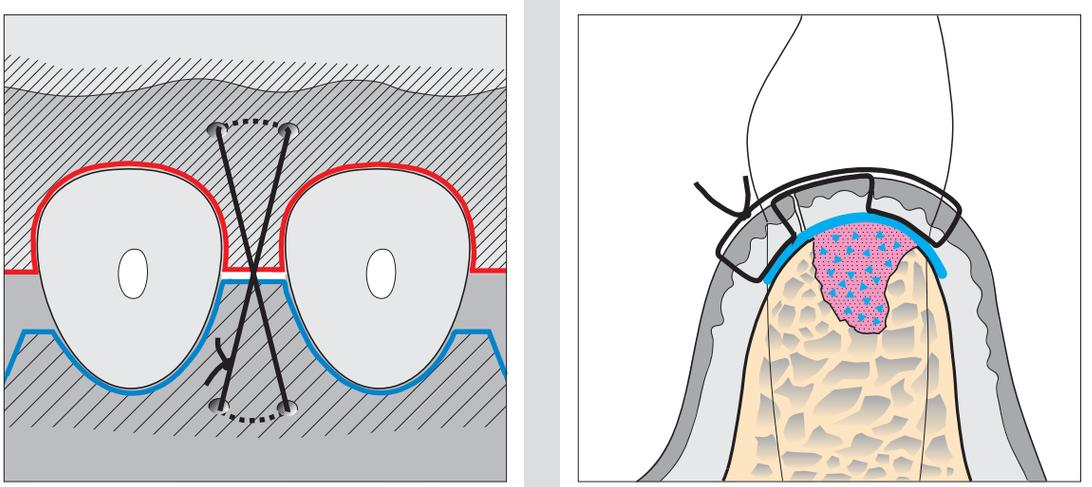
697 Continuous Suture (Surrounding Two Teeth; Left)
If the flap is reflected only on the facial surface (or only orally), it can be adapted using a continuous suture. One papilla of the flap is engaged, the suture is looped around the tooth, and the adjacent papilla is correspondingly included. No more than two teeth should be included, because the friction prevents equalization of the tension.

Interdental Individual (Interrupted) Suture (Right)



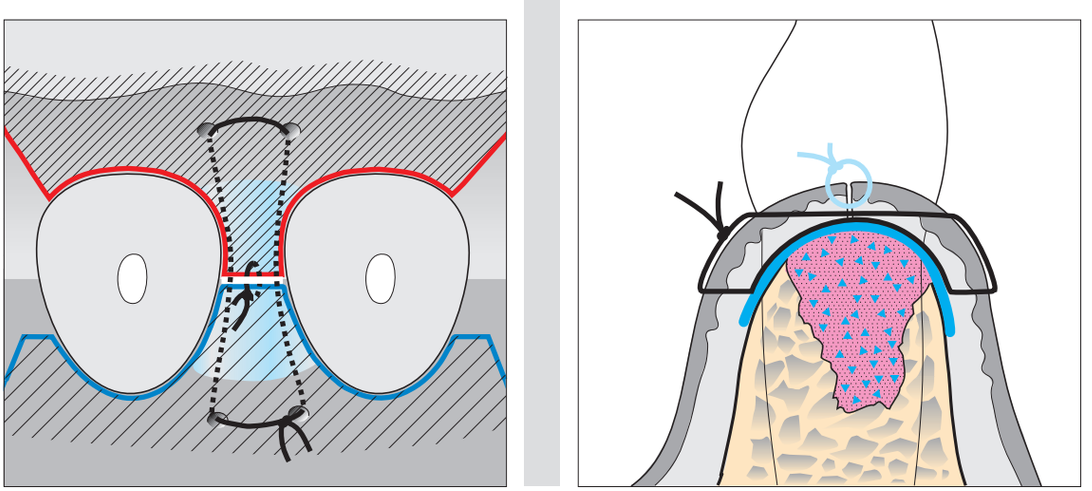
698 Horizontal Mattress Suture: External, Crossed (Left)
With this technique, there is no direct pull on the papilla tips, thus protecting against tearing the papillary tissues.

Vertical Mattress Suture: External, Closure of a "Papilla Preservation Flap" (Right)
The penetration sites are vertically below each other. The tissue flap is affixed directly onto the membrane. This provides closure for filled and/or membrane-covered osseous defects.



699 Horizontal Mattress Sutures: Internal, Parallel (Left)
This suture is scarcely visible (esthetics). It holds the flap at its base and is tension-free.

Vertical Mattress Suture: Internal (Right)
The submucosal location of this suture places it directly above the osseous defect or upon a membrane, which is held firmly in place. Coronal tissue segments (papillae) are not closed by the mattress suture, and require an additional interrupted suture.



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“Access Flap” Surgery, Open Flap Debridement (OFD)—Modified Widman Flap (MWF)

Of the numerous periodontal surgical techniques, the oft-modified Widman flap (“Modified Widman Flap,” MWF) remains the standard procedure for open periodontitis therapy (Widman 1918, Ramfjord & Nissle 1974, Ramfjord 1977). It is classified with the “access flap operations” because the goal of the flap reflection is primarily to provide improved visual access to the periodontally involved tissues.

The method is characterized by precise incisions, partial flap reflection and an atraumatic procedure, whose goal is not necessarily pocket elimination but “healing” (regeneration or a long junctional epithelium) of the periodontal pocket with minimum tissue loss. Because the alveolar process is only partially exposed, post-operative pain and swelling are rare.

The main goals of the procedure include optimum mechanical subgingival root planing and “decontamination” with direct vision, as well as healing by primary intention following close interdental flap adaptation. No ostectomy is performed. However, minor contouring osteoplasty can be performed to improve the facial or oral osseous morphology, primarily to achieve the desired interdental defect closure.

Indications

- The MWF is indicated for the treatment of all types of periodontitis, but is especially effective with pocket depths of 5–7 mm.
- Dependent upon the pathomorphologic situation on the individual teeth, the MWF may be combined with larger and fully reflected flaps (resective methods) and special procedures such as wedge excisions, root resection, hemisection, osseous implantations etc., and infrequently also with gingivectomy/gingivoplasty (combined surgical procedures, p. 366).

Advantages

- Root cleaning with direct vision
- “Tissue friendly”
- Reparative, with healing by primary intention
- Minimal crestal bone resorption
- Lack of post-operative discomfort

Contraindications

There are but few contraindications for the MWF:

- Lack of or very thin and narrow attached gingiva can render the technique difficult, because a narrow band of attached gingiva does not permit the initial scalloped incision (internal gingivectomy). In such situations, it may be necessary to employ the classic marginal or even an intrasulcular incision.
- MWF is contraindicated for osseous surgical procedures (expansive osteoplasty or ostectomy) with very deep osseous defects and *irregular* bone loss facially and orally, and if apical flap repositioning is planned (cf. resective surgical methods, p. 355).

Disadvantages

See contraindications.

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Principles of the Modified Widman Procedure—Ramfjord Technique

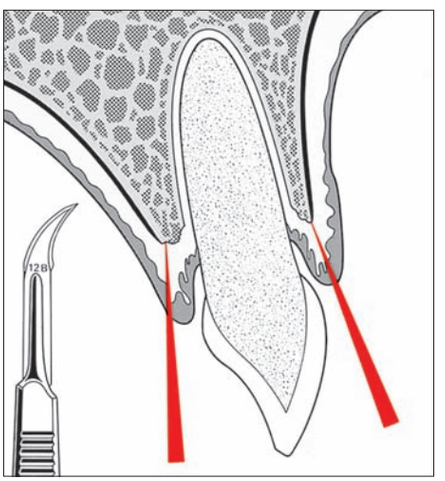
1 Initial incision—continuous, "scalloping," paramarginal (intragingival) incision; no vertical releasing incisions
2 Partial mobilization of the mucoperiosteal flap (full thickness flaps both facially and orally) within the attached gingiva to the alveolar crest
3 Second incision—sulcular incision
4 Third incision—horizontal incision, also interdentally; removal of the delineated tissue and all granulation tissue
5 Root cleaning and planing with direct vision
6 Flap adaptation, complete coverage interdentally.

The MWF technique differentiates the typical six treatment steps listed at the left:
The first incision sharply dissects the pocket epithelium and portions of the subepithelial infiltrate. This effectively thins the marginal gingiva, insuring ideal flap repositioning postsurgically. The gingiva is then reflected using an elevator, but only to the extent that the alveolar crest can be visualized.
The second incision is sulcular/intrasulcular around each tooth. This separates the pocket epithelium and junctional epithelium from the root surface, to the fundus of the pocket.

700 First Incision—Scalloping Inverse Bevel, Paramarginal
This incision determines the shape of the flap and is performed both facially *and* orally using a 12B scalpel. It is an inverse bevel incision, extending to the alveolar crest. The distance of the incision from the gingival margin will vary depending on the width of the interdental spaces that must be covered (0.5 to 1.5 mm). The incision becomes intrasulcular in the interdental areas.

Right: Incision, schematically.

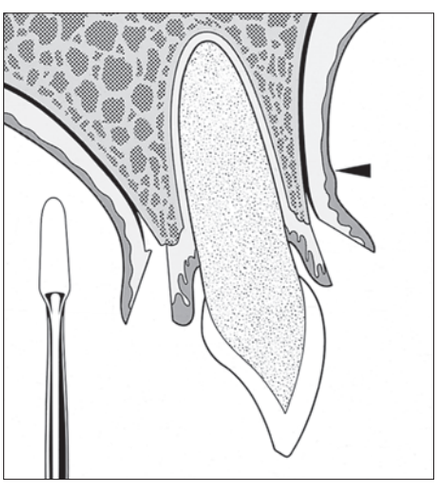




701 Flap Reflection
A small elevator is used to reflect a full thickness yet only partially mobilized mucoperiostial flap, as atraumatically as possible. The flap is reflected for one reason only: To permit direct visualization of the root surface and the alveolar crest.

Right: The schematic shows clearly that the facial flap is not reflected beyond the mucogingival line (black arrow): *Conservative flap reflection!*

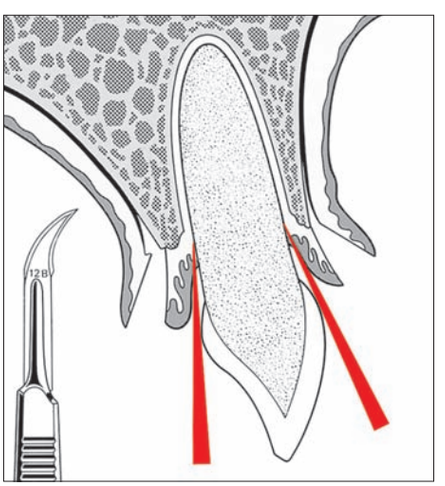




702 Second Incision—Intrasulcular
This incision is a purely intrasulcular incision that is carried around each tooth, between the hard structure and the gingiva, beyond the base of the pocket and extending to the apical extent of the pocket epithelium.
The 12B scalpel is also indicated for this second incision.

Right: Schematic depiction of the second, intrasulcular incision (red).



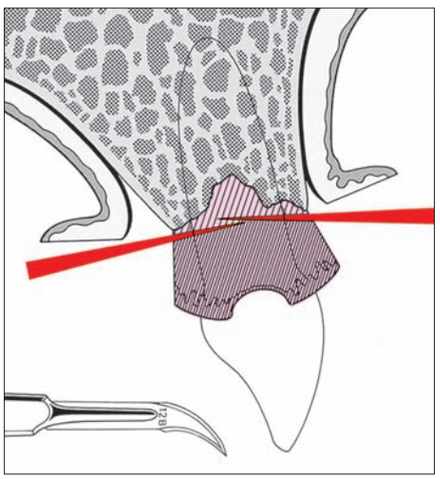


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The final incision is horizontal, and serves, especially in the interdental area, to release the pocket tissues sharply and atraumatically. The soft tissue and all of the granulation tissue within the pocket are thereby removed. The most important element of the procedure now follows: Systematic scaling and planing of the root surfaces using fine curettes or ultrasonic instruments with *direct vision*. This critical procedure requires significant time. Supportive alveolar bone is *not* removed, but minimal osteoplasty may be performed as necessary.

Finally, the flaps are repositioned. Because of the shape of the flaps created by the initial scalloping incision, secure and complete coverage of the interdental bone is possible. This enhances healing by *primary intention*.

Adherence to these principles is routinely associated with excellent long-term results in terms of maintenance or even true gain in periodontal attachment (Knowles et al. 1979; Fig. 727). The principles are therefore also applicable to other flap surgical procedures.

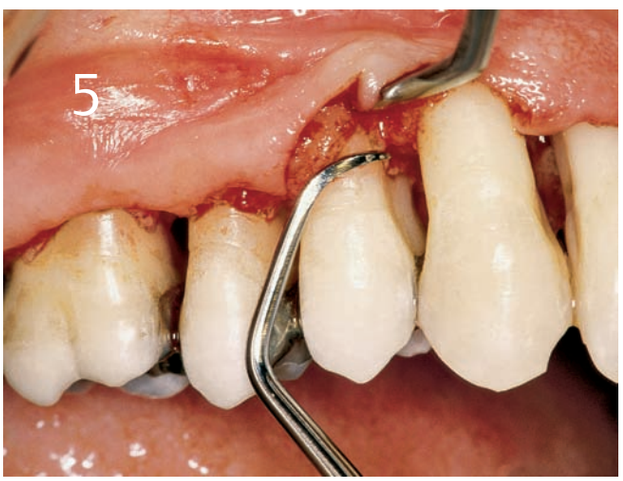




703 Third Incision—Horizontal
The horizontal incision is carried along the alveolar crest from the facial to the oral aspect, or the reverse, thus separating the supracrestal pocket tissue from its supporting subjacent tissues, especially in the interdental area.

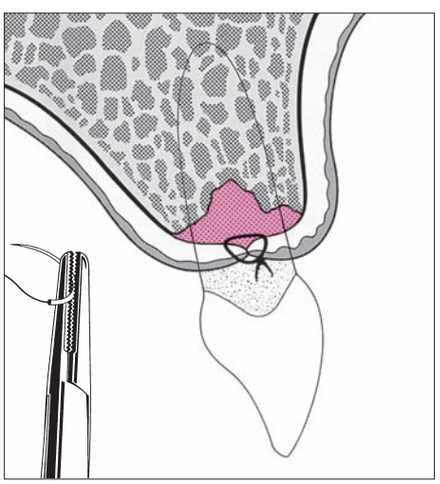
Left: The schematic clearly depicts the bony pocket, especially the interdental osseous crater. The horizontal incision does not approach the base of the pocket (note red spears and the profile of the bony architecture apical to it).





704 Root Planing with Direct Vision
Fine curettes are used in the depth of interdental craters to remove remnants of pocket epithelium and granulation tissue. Systematic root cleaning and planing is performed with repeated rinsing (NaCl 0.9 %) and evacuation by the dental assistant, with *direct vision*.

Left: Schematic depiction of the removal of granulation tissue and root cleaning/planing with a universal curette.





705 Tight Coverage of Interdental Defects
The facial and oral flaps are tightly adapted over the bone and between the teeth by means of interrupted interdental sutures. Because “new papillae” were created by the initial scalloping incision, it is usually possible to completely cover interdental bony defects.

Left: The schematic clearly shows the coverage of the interdental area. In the molar regions, this is not always possible (healing occurs by secondary intention).

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Principles of the MWF—Occlusal View

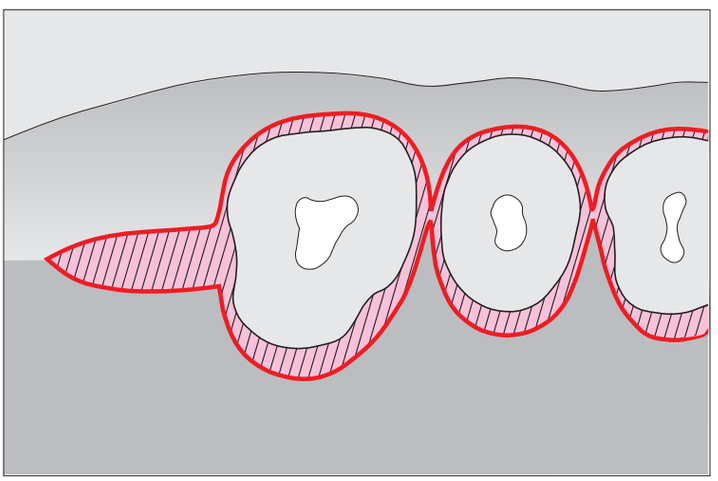
The surgical principles of the Ramfjord technique described above will be depicted here schematically from the occlusal view (horizontal section). The geometry of flap design and flap repositioning can be appreciated particularly well when viewed from the occlusal aspect. The initial incision is scalloped, somewhat removed from the free gingival margin buccally and palatally, creating "new papillae" for subsequent complete closure of interdental defects.

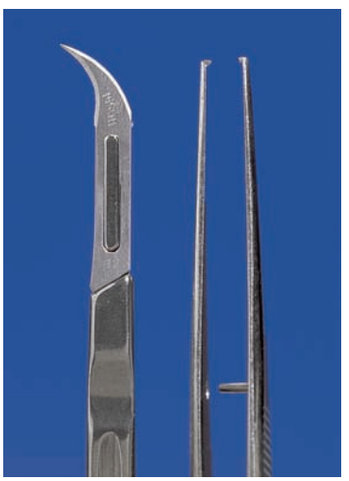
In the interdental regions, the incision may become a purely intrasulcular incision, to provide adequate tissue for subsequent complete closure of the interdental space. The scalloped margin of the flap provides tight closure of the curetted interdental osseous craters (e.g., two-wall bony pockets).

At the distal end of the arch, the primary incision may end as a wedge excision, providing access to and therapy for pockets or furcation involvement in this region (wedge excisions, p. 319). Osseous craters and furcation involvement in this region also may be treated using regenerative methods (p. 323).

706 Primary Incision – "Scalloping"
The first incision is a scalloped, inverse bevel incision (internal gingivectomy) in the maxillary posterior segment (Fig. 712). This incision continues into a wedge excision distal to tooth 16.
The second (intrasulcular) and third (horizontal) incisions are not depicted here.

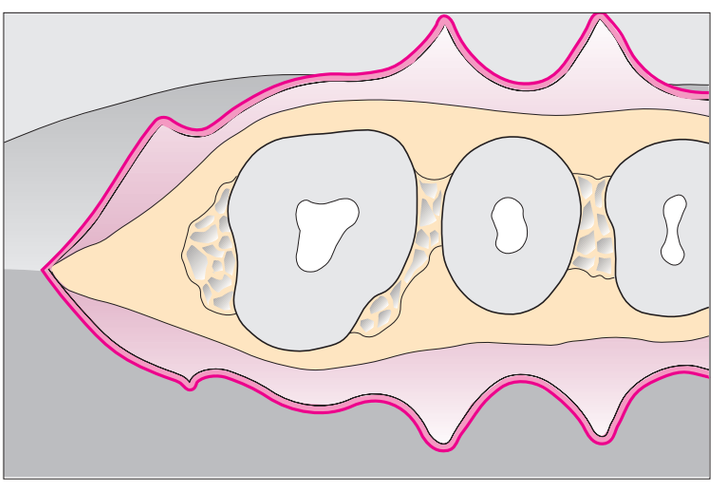
Right: Instrumentarium (I)—scalpel with blade 12 B, surgical forceps.





707 Partially Reflected Flaps
After reflecting mucoperiosteal flaps, the excised soft tissues are removed, the osseous defects are carefully curetted, and all root surfaces are cleaned and carefully planed with direct vision.

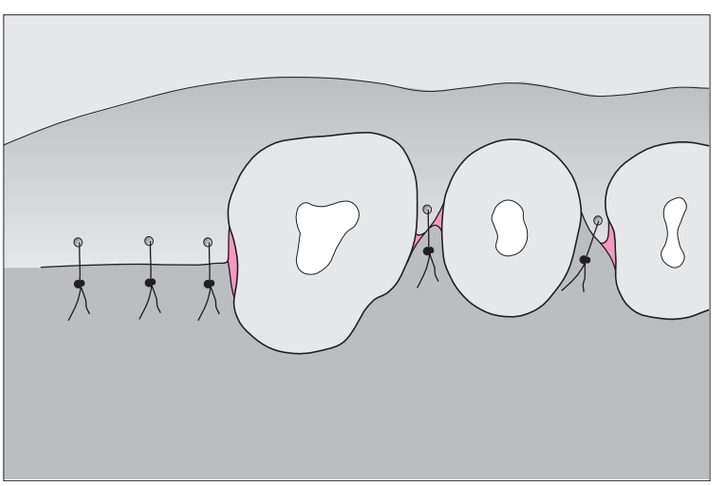
Right: Instrumentarium (II)—narrow and broad elevators (depicted: Prichard elevator).





708 Flap Re-Adaptation, Suturing
Complete coverage of the interdental bone is accomplished by tension-free fixation of the papilla tips using interrupted sutures. If sufficient tissue is available, the facial and oral papilla tips can actually be repositioned side-by-side before fixation. This ensures complete coverage of the interdental areas.

Right: Instrumentarium (III)—needle holder and needle-suture combination (4–0 silk).





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"Access Flap"/Modified Widman Procedure—Case Presentation

Surgical Procedure, Step-by-Step

The systematic procedure for conservative flap reflection according to the principles of the Ramfjord technique will be depicted in the maxillary right sextant.

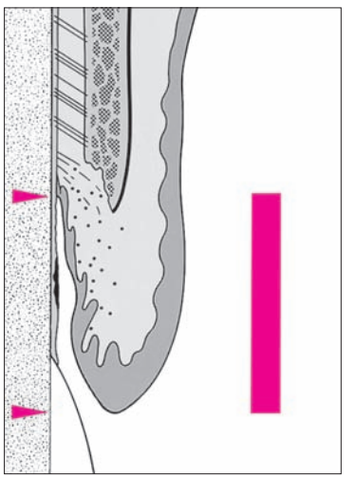
The 42-year-old female presents with mild to moderate chronic periodontitis. Phase 1 therapy (initial therapy) is complete. Patient cooperation/compliance was "only average." The goal of a PI and BOP index below 20% was not achieved.

Also in other sextants (not depicted here), minor surgical procedures were indicated.

Findings after initial therapy:

PI: 30% BOP: 32% TM: 0–2

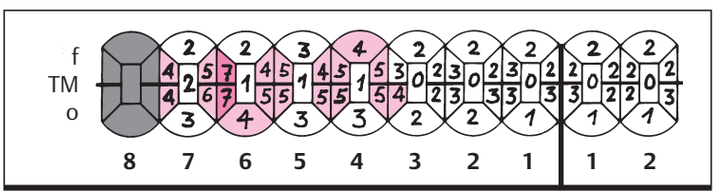
See accompanying figures for the clinical picture, probing depths, gingival contour and radiographic survey.



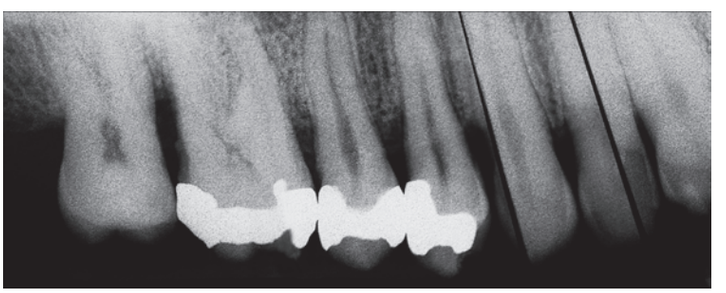


709 After Initial Therapy
The gingivae remain inflamed despite initial therapy. Mild hemorrhage occurs upon probing of the pockets. The patient's oral hygiene must be checked repeatedly and OHI provided even after the planned surgical procedures. Additional instructions and motivation must be provided to the patient during each maintenance phase appointment (recall).

Left: Sagittal diagram of the initial condition. Arrows: Pocket probing depth.



710 Probing Depths Following Initial Therapy; Tooth Mobility (TM)
Interdental probing depths up to 7 mm persist after initial therapy.



Radiographic Survey
Mild to moderate attachment loss is observed in the premolar region. Between teeth 17 and 16, the interdental septum has resorbed to the midpoint of the root.



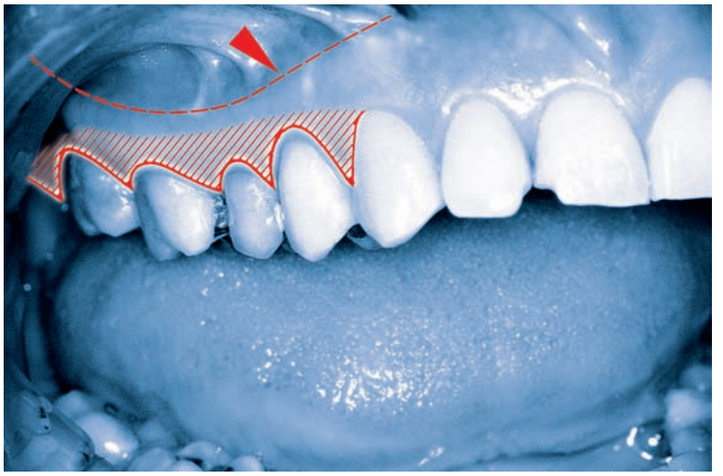
711 Occlusopalatal View
Mild inflammation is also obvious from the oral aspect in the sextant between 13 and 17, especially in the interdental area. Near the deep pocket around 16 and 17, signs of pocket activity (exudate) persist despite initial therapy.

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712 Planned First Incision and Flap Reflection
The planned initial scalloping incision and the extent of the flap to be reflected are indicated.

Solid red line: Initial scalloping horizontal incision.
Hatched area: The planned flap reflection ends *coronal* to the mucogingival junction (dashed red line and arrow).

Right: Surgical protocol.

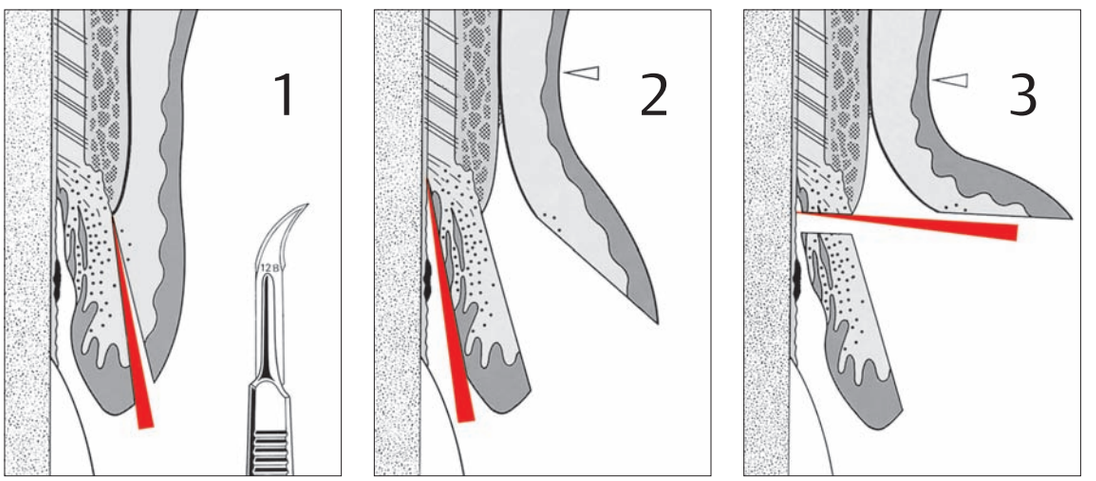


“Access Flap”—Surgical Protocol

- First incision—paramarginal, scalloping
- Flap reflection (full thickness flaps)
- Second incision—sulcular
- Third incision—horizontal
- Removal of the delineated soft tissues (pocket exudate), “osseous curettage”
- *Root planing with direct vision*
- Flap repositioning, suturing

713 Principles of the Three Incisions

1 Paramarginal first incision: Severing the soft tissue pocket wall with the 12 B scalpel
2 Flap reflection and sulcular incision
3 Horizontal incision: Extends especially into the interdental areas



Initial Incisions: Facial and Oral

714 Scalloping Paramarginal Initial Incision—Facial
The first “scalloping incision” is an inverse bevel incision (internal gingivectomy).

If teeth are crowded, this incision may become a purely intrasulcular one in interdental areas, to ensure sufficient tissue for the “new” papillae destined to completely cover the interdental region.



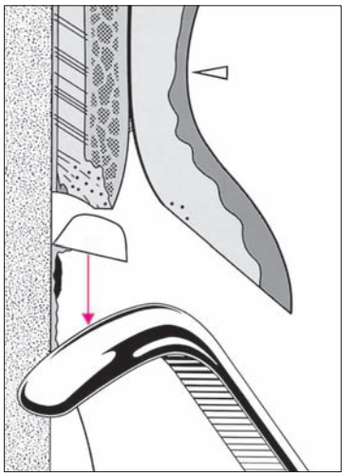
715 Scalloped Primary Paramarginal Incision—Palatal View
The incision is performed identically on the palatal surfaces. In order to ensure that the rather resilient palatal flaps can be re-adapted to the tooth and osseous surfaces, the primary incision should not be made too far removed from the height of the gingival margin (*maximum* 1–2 mm). The palatal flap is usually non-mobile, and this flap must be thin and even.

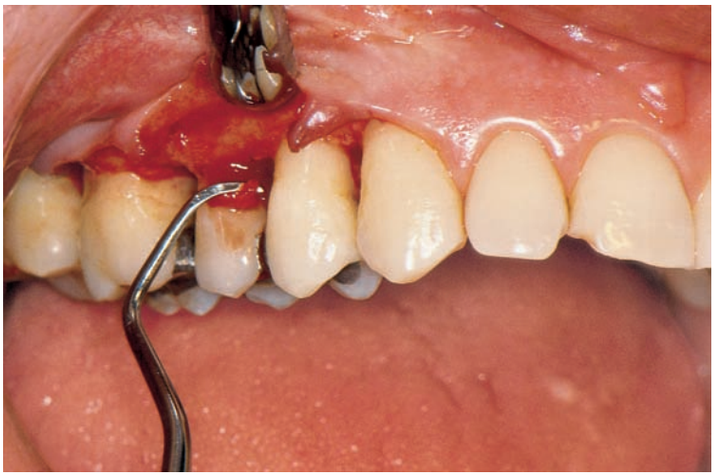


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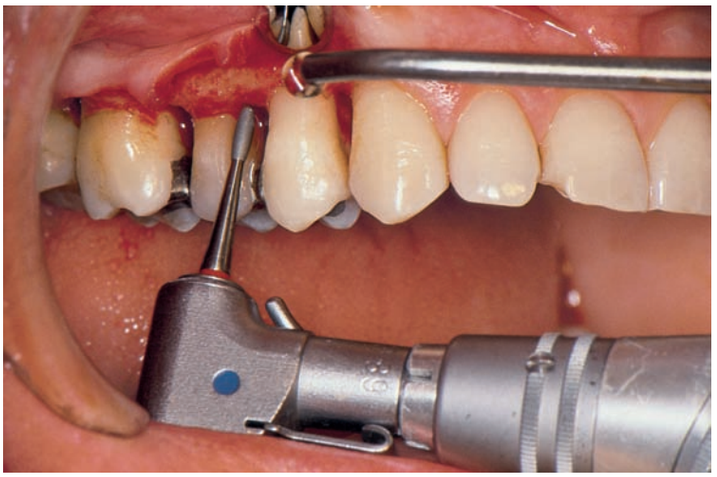


716 Removal of the Sharply Dissected Pocket Tissues
Once the flaps are reflected and the second and third incisions are performed (Fig. 713), the sharply demarcated tissue is easy to remove. Granulation tissue should also be removed to provide direct vision of the root surfaces to be cleaned and thoroughly planed. Complete elimination/removal of all granulation tissue is not *per se* necessary, but will reduce the risk that pathogenic microorganisms (e. g., *Aa* or *Pg*) will remain in the tissues post-surgically.





717 Debridement—Root Planing with Curettes
After removal of the pocket soft tissues, the root surfaces are cleaned and planed using fine universal or Gracey curettes, thus thoroughly eliminating the biofilm as far as possible. This procedure can also be accomplished using ultrasonic devices. *Root planing is the most time-consuming procedure during flap surgical procedures.*
Left: Schematic depiction of root surface cleaning with a universal curette (M23A; Deppeler).



718 Root Cleaning and Planing with a Fine Polishing Diamond
Hard deposits (calculus) can also be removed mechanically, e. g., with rotating diamond stones (Perio-Set; 40 µm abrasiveness), and root planing with the 15 µm diamonds. Low rpm and constant rinsing/cooling with Ringer's solution must always accompany the use of rotating diamonds. The diamond stones of the *Perio-Set* (Fig. 541) should not be considered as replacements for curettes, but enhancements!





719 Palatal View
The resilient palatal tissue must also be reflected to permit root planing with direct vision. Note the deep interdental crater between 16 and 17 (cf. Fig. 710).

Left: Rinsing solutions—physiologic NaCl (0.9 %) and H_2O_2 (3 %), or betadine for disinfection and to reduce hemorrhage (cf. FMT, Fig. 651).

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720 Flap Adaptation Using Interrupted Sutures

Close approximation of the flaps upon bone and tooth surfaces is a prerequisite for optimum healing. Fixation of the flaps is accomplished by means of interdental interrupted sutures. The scalloping form of the initial incision usually permits tight closure in the interdental areas. If the flap tissue is extremely thin, the use of mattress suturing is indicated for flap fixation (p. 308).

Right: Palatal view.



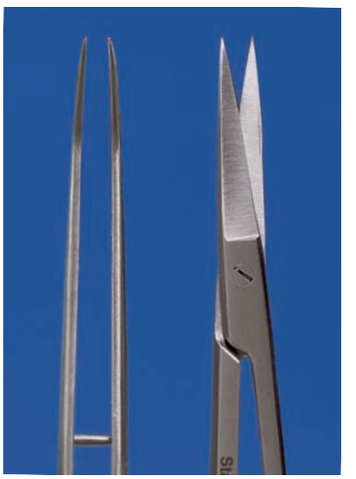


721 Suture Removal—Carefully!

Sutures should be removed at one week. By this time the wound margins have adhered to each other and the healing process is well underway; however, careless handling of the sutures could disturb the delicate attachments between and among the flaps, the osseous surface and the tooth/root.

Right: Pointed scissors, fine forceps and “two steady hands” minimize any risk of tissue damage.





722 Tooth Cleaning

Following suture removal, the surfaces of the teeth are cleaned using soft rubber cups and mildly abrasive prophy paste (or dentifrice) (cf. “RCP method,” p. 251). Since wound healing is not yet complete (regeneration at the depth of the pocket, new junctional epithelium) care must be exercised that no paste is forced into the sulcus, beneath the formerly reflected soft tissue flap.

Right: Protocol for the post-operative measures.



Post-operative Measures—Protocol

- Topical disinfection, e. g., CHX 0.1–0.2 % for at least two weeks
- Systemic medication only as indicated (p. 287)
  – antiphlogistic for 2–3 days
  – antibiotics
- Prevention of swelling: Topical application of cold packs or ice for 2–3 days
- Professional evaluation and cleaning 7–10 days post-op, and after suture removal every 2–3 weeks for two months

723 Following Suture Removal and Tooth Cleaning

Two weeks post-surgically, the patient must re-initiate home care, but carefully. It is recommended that the chlorhexidine rinses be continued. The field of operation should be *professionally* cleaned by the dental hygienist during subsequent appointments, ca. every two weeks.

Right: Despite the effort to close the interdental area, a small “crater” remains between teeth 16 and 17.





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"Access flap"/Modified Widman Procedure

Summary

The 42-year-old patient with mild to moderate chronic periodontitis exhibited deficient oral hygiene at the initial examination. Even after initial therapy, mild gingival inflammation was in evidence; between molars 16 and 17, signs of mild pocket activity (exudate) persisted.

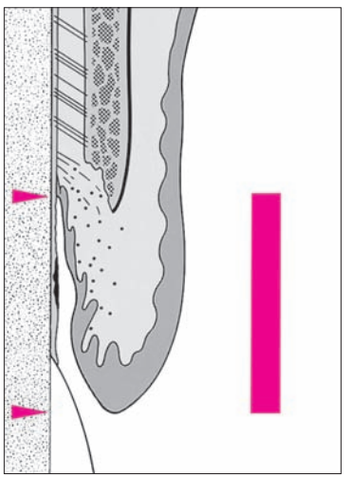
Probing depths returned to physiologic levels six months following the surgical procedure.

Even the deep defect between 16 and 17 exhibited a probing depth of only 4 mm.

These excellent results can also be attributed to the great improvements by the patient in home care. Because of the gingival shrinkage, mainly in the interdental region, special emphasis should be placed on interdental hygiene.

PI: 10 % BOP: 9 %

The initial recall was set at three months; if patient cooperation/compliance continues, subsequent recalls may be at 4–6 month intervals.

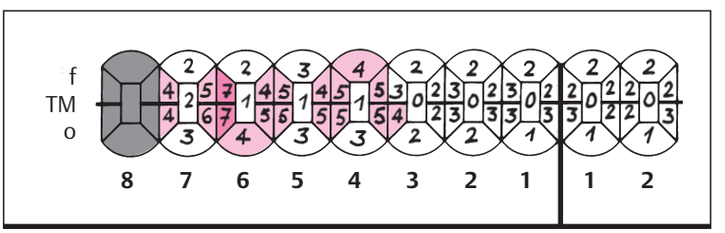




724 Before Surgery
Despite initial therapy, mild gingival inflammation persists (BOP: 32 %). Application of finger pressure between 16 and 17 elicits release of minimal exudate. The treatment of choice is *surgical* ("access flap") with direct vision.

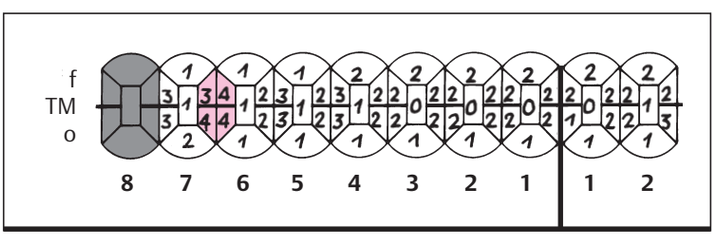
Left: An untreated pocket (facial surface of tooth 14). The probing depth is depicted in red (bar and arrows).

Before

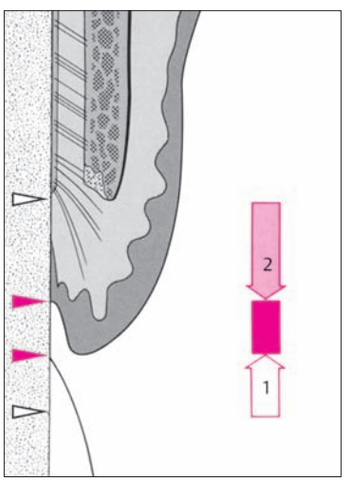


725 Pocket Probings and TM Following Initial Therapy—Before Surgery
Following initial therapy, which consisted of closed supra- and subgingival scaling, pockets of 4–7 mm persist, especially interdentally.

After



Pocket Probings and TM Six Months after Surgery
At the recall examination six months after the surgical procedure, no probing depths beyond the 4 mm level are detected.





726 Six Months Post-operative
Even after the careful and conservative MWF procedure, the teeth appear slightly "elongated" and the interdental spaces are more open. Recommendations for interdental hygiene are determined by the anatomic/morphologic situation.

Left: A slightly deepened sulcus persists ("residual pocket," red bar). The former probing depth has been reduced through shrinkage (**1**) as well as periodontal healing ("repair," **2**).

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Long-term Results Following Various Treatment Modalities

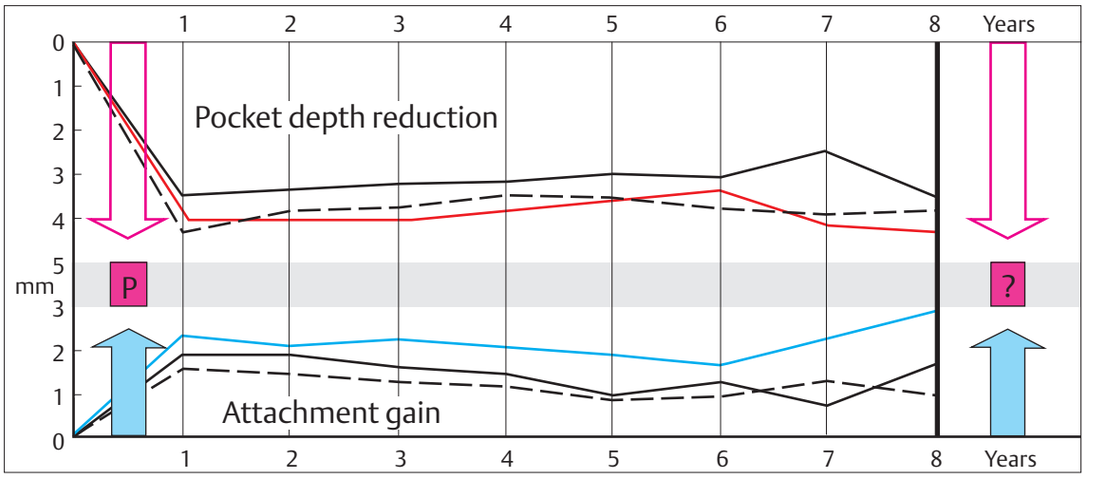
Periodontitis can be treated “closed” (*subgingival scaling and root planing*) or “open” (*surgically*). The therapeutic modality selected will depend upon the type of periodontitis (chronic, aggressive), the severity of the disease, and upon the many factors discussed previously (see etiology and pathogenesis, p. 21; diagnosis/risk factors, p. 165; treatment planning, p. 208). The goal of therapy, however, is the same for each type of treatment: Reduction of pocket depth and gain of attachment.

The clinical results that can be expected from various treatment methods and with variably progressive periodontal disease over the long term were described in the second half of the 1970s by Ramfjord et al. (1975), Ramfjord (1977) as well as Knowles et al. (1979). During those years, the authors compared closed root planing with the modified Widman procedure (described today as the “access flap” procedure), as well as with surgical pocket elimination. The latter is characterized by the *resective treatment methods* (p. 355). Eight years following treatment, probing depths of 7–12 mm pockets were reduced by 3–4 mm by *all treatment modalities*, with attachment gain less than 3 mm!

727 Pocket Depth Reduction and Gain of Attachment after Various Treatment Methods in 7–12 mm Pockets (Knowles et al. 1979)

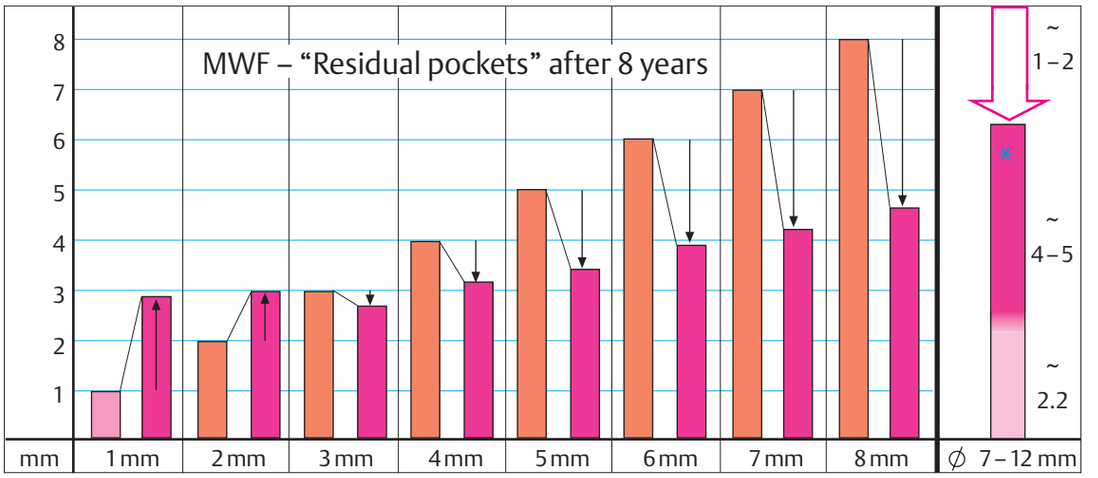
Modified Widman procedure
Root planing and curettage
Surgical pocket elimination

Compare also Fig. 644: Same study with shallower pockets. Modified from *J. Knowles et al. 1979*



728 Residual Pockets Following MWF

The deeper the pocket probing depths initially, the better will be any subsequent pocket depth reduction (*no pocket elimination*!). With 7–12 mm pockets (right), reduction was at least 4 mm (ca. 2 mm of *clinical attachment gain* and ca. 2 mm of gingival recession). There remains an average *probing depth* (“residual pocket”) of 4–5 mm; there is a risk of recolonization by pathogenic anaerobes if a strict professional recall is not maintained!



Conclusions

As one considers these long-term results, it is important to realize that during the entire 8-year follow-up examinations of the patients, 3-month recall intervals were observed: The plaque control by the therapist and patient was therefore quite beyond average. Under these strict conditions, it is not surprising that the long-term results with regard to pocket reduction (Fig. 727) did not differ dramatically among the three procedures.

If one considers the results following MWF therapy alone, it becomes clear that with physiologic probing depths of 1–3 mm, no surgery is necessary. To do so leads to clinical attachment loss! The deeper the probing depths, the more pronounced will be the pocket reduction and therewith the indication for surgical intervention, e. g., via MWF.

Note: *Regenerative methods* such as bone transplantation and GTR were only in the developmental phases when these large clinical trials were performed, and therefore were not taken into consideration; the same is true for the much-discussed combination of mechanical and medicinal therapies today (p. 287). The problem of deep *residual* pockets has not yet been successfully solved (incomplete regeneration, Fig. 728).

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Wedge Excision Distal to a Lone-standing Tooth—Principles

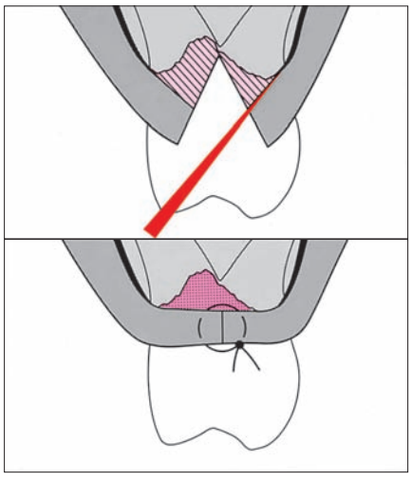
Surgical Principles

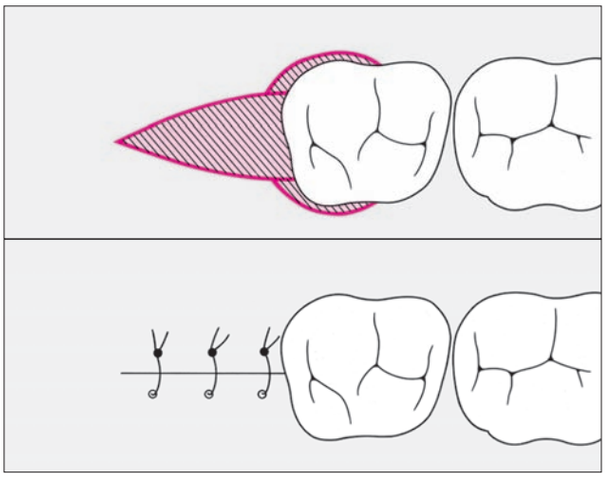
Periodontal pockets distal to the last tooth in an arch, as well as pockets on lone-standing teeth (p. 320) are often difficult to eliminate surgically. Such clinical situations can usually only be successfully treated if the surgery can be performed within the region of the attached gingiva.

The danger of recurrence is particularly high in the mandible, distal to the second molar, i. e., in the region of mobile mucosa at the periphery of the retromolar pad. If surgery in this area is unavoidable, care must be taken to avoid the lingual nerve, which often assumes a more superior position. For this reason, repeated root planing may be preferable to surgical intervention in some cases.

Wedge excisions are often combined with flap procedures in the same sextant or quadrant.

Three various possibilities for surgical pocket elimination distal to end-standing teeth are depicted diagrammatically in the figures below.

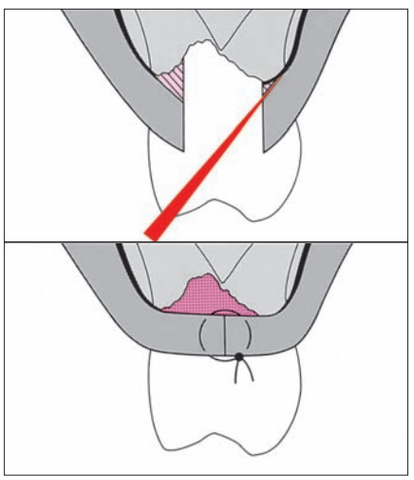


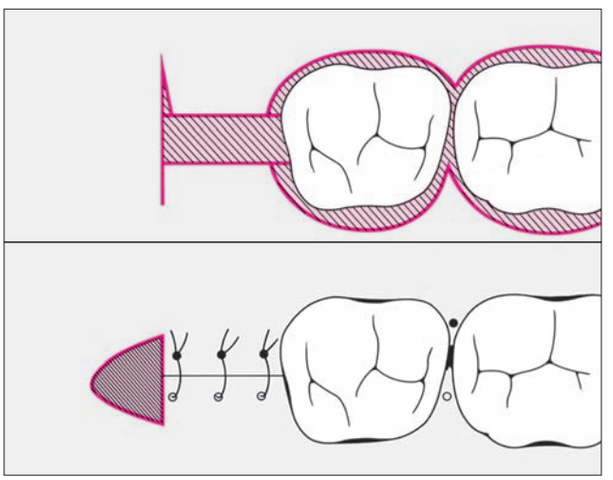


729 "Classic" Distal Wedge Excision

The triangular-shaped wedge excision is generally used for pocket elimination distal to the last tooth in an arch. The first incisions delineate the excision, and converge at the base of the pocket. The second incision (red arrow, left) serves to undermine and thin the buccal and oral flaps.

Repositioning the flaps with sutures essentially eliminates the distal pocket.

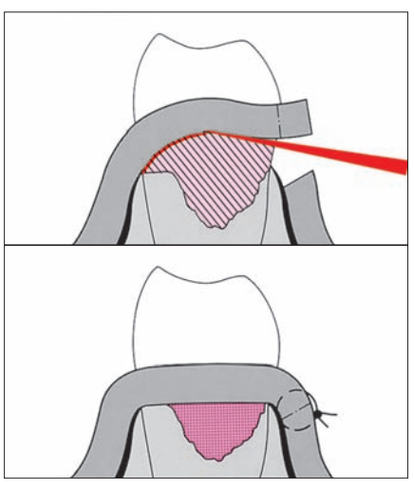


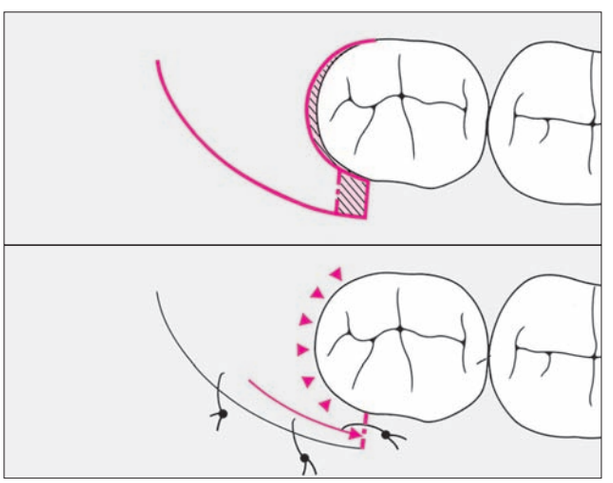


730 Modified Incision

If the elimination of a pocket distal to the last tooth in the arch is performed as one phase of a more extensive flap procedure in the region, the surgery described above (Fig. 729) may be employed, or a modified wedge excision can be used.

The modified technique considerably simplifies the reflection of buccal and oral flaps, and direct vision of the root surface and bone is improved. The procedure is depicted in the clinical situation that follows (p. 320).





731 Wedge Excision According to Chaikin—Facial Incision in the Mandible

Curved incision from the buccal around the distal of the tooth, marginally. Flap reflection. Subjacent tissue is excised, and root planing is performed.

The tip of the half-moon shaped flap is shortened, then sutured to the primary incision site.

Positive: Protection of the lingual nerve.

Caution: Danger of necrosis due to tension on the narrow flap.

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Wedge Excision—Most Distal Tooth

Surgical Procedure, Step-by-Step

In a 38-year-old female, mild inflammation and an active 6 mm pocket persisted distal to tooth 26 following initial therapy (deep scaling). The treatment plan called for elimination of the pocket by means of a modified wedge technique (Fig. 735). The operation is feasible because there remained sufficient attached gingiva distal to the tooth. The distal furcation was only slightly involved (F1).

No surgery is indicated for the remainder of the dentition, but repeated scaling will be performed. The temporary composite restorations in teeth 24 and 25 and the amalgam in 26 should be replaced by inlays after conclusion of the periodontal therapy.

Findings following initial therapy:

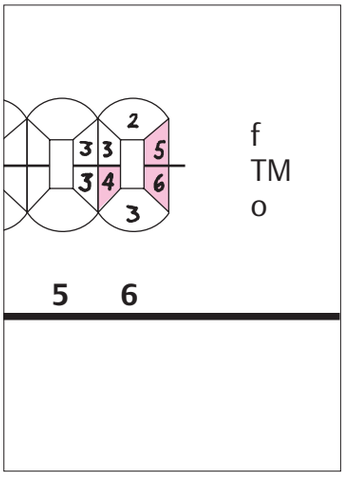
PI: 30 % BOP: 26 % TM: 0–1

Clinical findings and radiographic survey are depicted in the figures below.

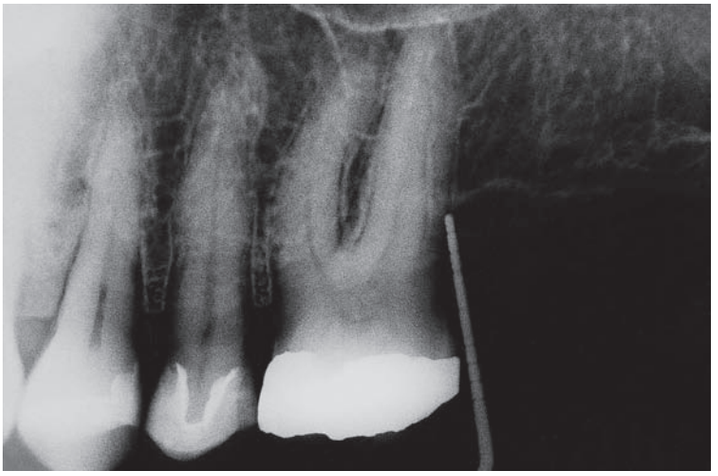
732 Active Distal Residual Pocket
The persistent 6 mm deep pocket extends into the region of the distal furcation. The furcation entrance can be probed in the horizontal direction using the Nabers probe (No. 2), ca. 2 mm deep (furcation involvement, grade F1).

Right: Pocket probing depths around teeth 25 and 26.





733 Radiograph with Periodontal Probe in Situ
The Williams probe encountered no resistance from the inflamed tissues, and penetrated to the alveolar bone crest with only slight probing force (ca. 0.25 N). The markings on this probe are at 1, 2, 3, 5, 7, 8, 9 and 10 mm. The thickness of alveolar tissues in relation to the clinical situation (Fig. 732) can be appreciated in this view.



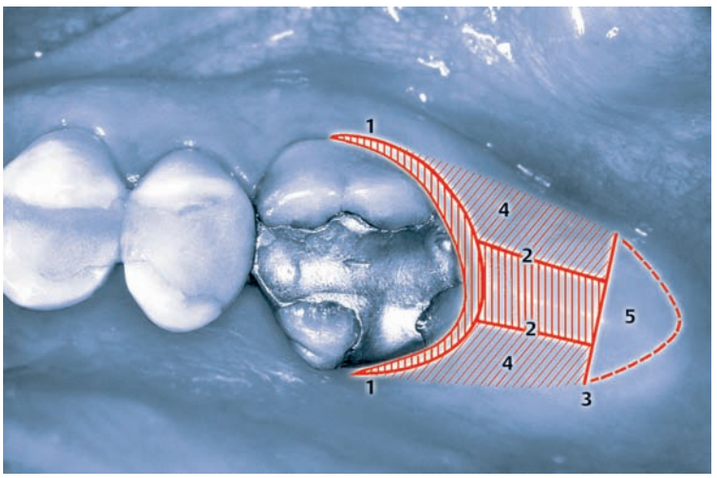
734 Occlusal View in the Region of Tooth 26
Sufficient attached gingiva is present distal to tooth 26. This makes the operation much simpler and virtually excludes any risk of recurrence.
One critical consideration could be a deep depression in the region of the distal molar furcation. The old amalgam restoration must be recontoured and polished before surgery. It could be replaced later by an inlay.



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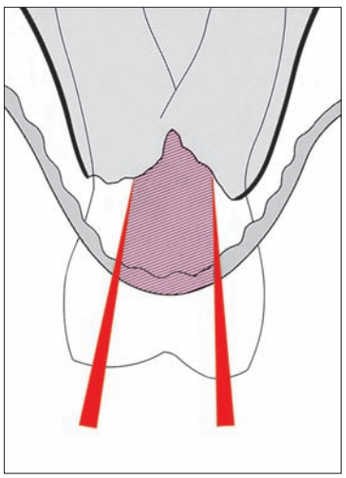
Wedge Excision—Surgical Protocol

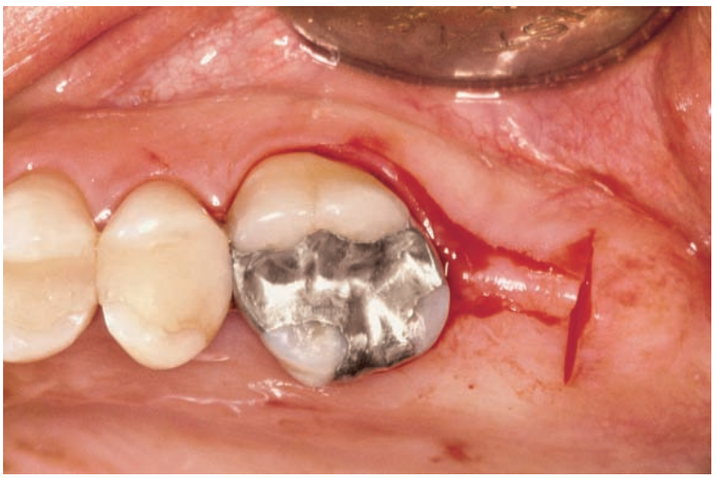
1. Half-moon shaped inverse bevel incision from the mid-buccal to the mid-palatal aspect, then a corresponding pure intrasulcular incision
2. Parallel incisions carried ca. 10 mm distal to the tooth
3. Distal perpendicular incisions
4. Undermining incisions for flap creation (hatched area, Fig. 735, right)
5. Contouring gingivectomy to eliminate redundant tissue



735 Occlusal View of the Surgical Site
The temporal sequence of the incisions, flap creation and distal gingivectomy that comprise the wedge excision are indicated.

Left: The surgical procedure is described step-by-step.

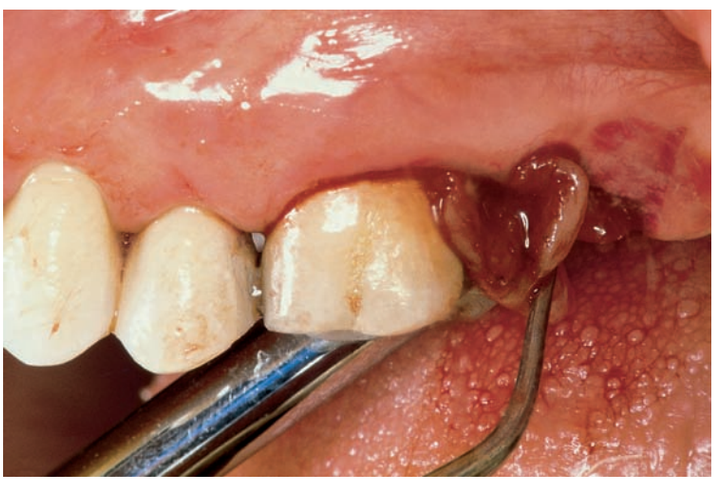




736 Incisions
Clinical view after performing the incisions depicted above.
The old amalgam restoration was recontoured and polished before surgery.

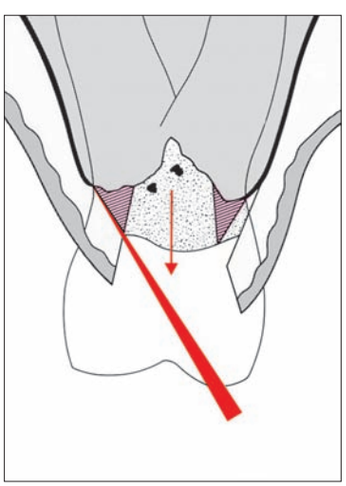
Left: Parallel incisions depicted in an orofacial schematic (second incision, Fig. 735).

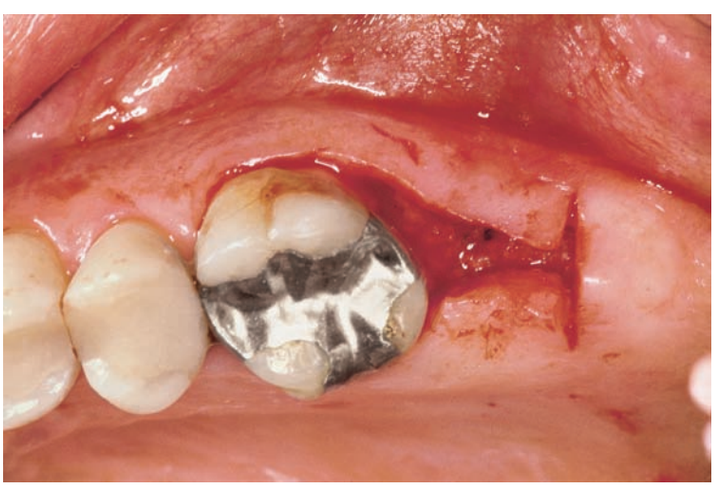




737 Removal of the Wedge
A lingual scaler (ZI 12; Deppeler) may be used to advantage for freeing the tissue wedge from subjacent bone.

Left: Wedge removed; lingual scaler (Fig. 526).





738 Flap Reflection—Root Planing
The undermining incisions (incision 4, Fig. 735) parallel to the gingival surface create flaps of uniform thickness (2–3 mm) both buccally and orally.

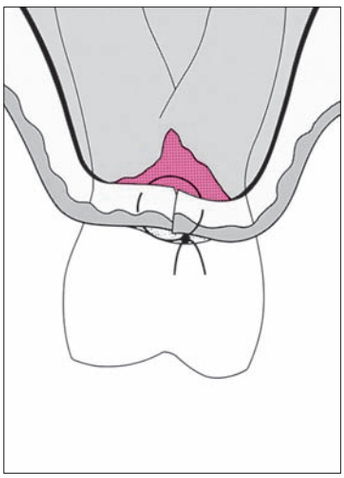
Left: Undermining incision (red spear). The exposed root surface is thoroughly planed, especially in the region of the furcation entrance (thin red arrow).

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739 Suturing the Thinned Flaps
The technique of wedge excision with modified incisions permits particularly effective flap adaptation and secure closure of the defect; however, redundant tissue remains distal to the tuberosity.

Right: Sutured flaps, coagulum in the osseous crater. A suture that encircles the tooth, as described by Yuodelis, is indicated with deep concavities. The suture draws the tissue better into the furcation area and around the tooth (Fig. 740 R).

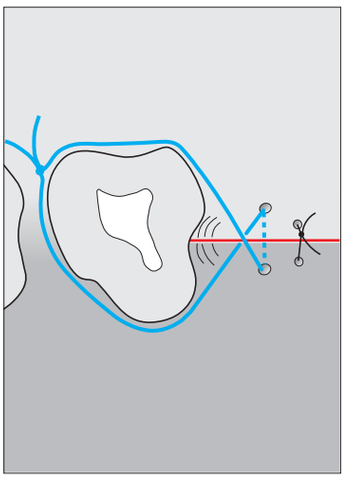




740 Gingivectomy of the Redundant Distal Tissue
The excess tissue distal to the molar may be removed by electrosurgery, scalpel or laser. This small open wound is left to epithelialize without any periodontal dressing; the patient may experience some discomfort. Alternatively, the wound may be covered with a tissue adhesive (Histoacryl).

Right: Alternative suturing technique, as described by *Yuodelis*.

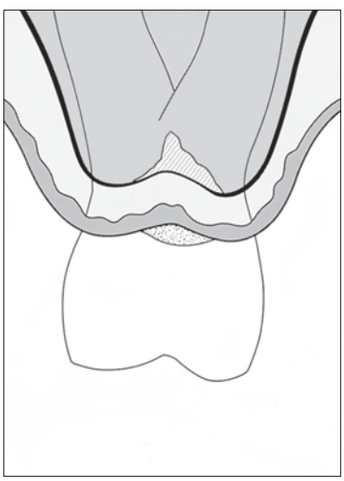




741 Two Months Post-Operative
In comparison to the initial situation, pocket depth has been reduced by about 4 mm (note restoration margin and gingival contour). The sulcus can be probed to a depth of 3 mm, and there are no signs of disease activity.

Right: Some degree of osseous regeneration may be expected. The slight depression in the gingiva distal to the molar (furcation region) must be given special attention during home care.





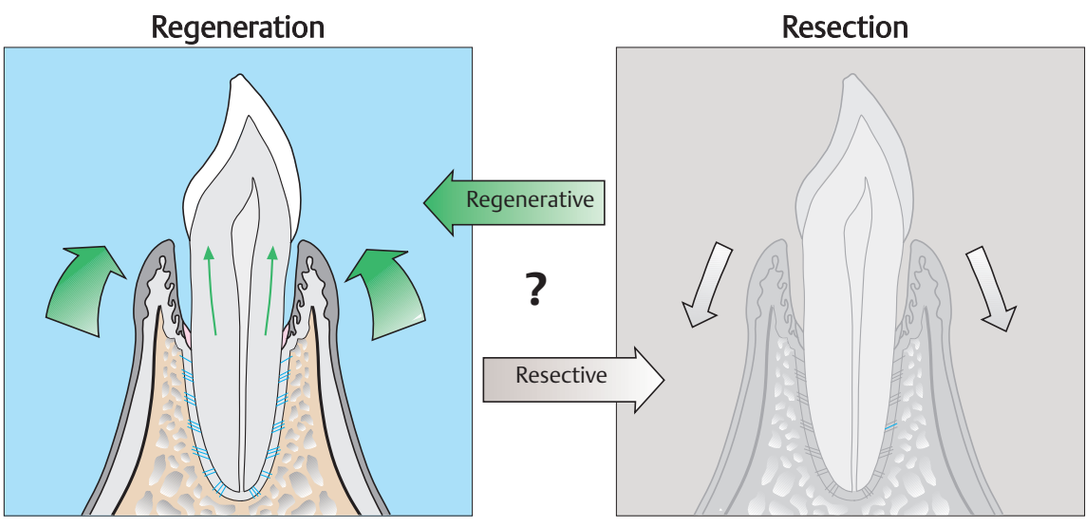
742 Occlusal View—4 Months Post-Operative
At the first recall appointment, an inflamed papilla was noted facially between teeth 25 and 26. This was due to inadequate plaque control.
Motivation and re-instruction of the patient led to elimination of this gingival inflammation within a few days.



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Regenerative Methods

For many years, attempts have been made to treat periodontitits by means of regenerative rather than resective methods. But *new bone formation*, "defect filling," especially in multi-walled osseous pockets often can be achieved by means of closed or open root planing (Rosling 1976, Lang 2000). Early on, Ramfjord championed the "modified Widman flap" (p. 309) to induce healing in the sense of regeneration of all *periodontal tissues;* however, the results consisted of "repair," with the formation of a long junctional epithelium, and not true regenerative healing with the creation of new cementum, periodontal ligament and alveolar bone. True regeneration of all periodontal structures is strived for today by means of various procedures (C, D, E; p. 324). While the results and successes following such methods are striking, they remain inconsistent and, as always, they cannot yet be "predictable" with any certainty.



743 Regeneration Versus Resection
One of the most important goals of periodontitis therapy is the elimination of deep pockets, because in such pockets the anaerobic milieu contains increasing numbers of periodontopathic microorganisms.
Pocket depth reduction (pocket elimination) can be achieved by *resective* methods or *regenerative* methods; the latter are preferred today. In many cases, the two methods are combined.

Regenerative Healing – Prerequisites

The previously described treatment methods (closed and open therapy) are based almost exclusively upon the thorough antimicrobial treatment of the periodontal lesion: mechanical/instrumental therapy, and selective use of antimicrobial agents.

However, in order to make possible a truly biologic and functionally complete regeneration, additional important prerequisites must be fulfilled in the region of the periodontal defect:

- A biocompatible root surface
- Presence of precursor cells
- Exclusion of the epithelium and in some cases also the gingival connective tissue from the root surface
- Stabilization of the wound area (protection of the coagulum and its adhesion to the biomodified root surface)

These additional prerequisites were discussed in more detail previously (Periodontal Healing, pp. 205, 351).

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Intra-Alveolar Pockets—Anatomy of the Defect

The success of any regenerative therapy depends not only upon the very important patient factors (p. 297), but primarily also upon the anatomy of the periodontal defect and the choice of the treatment technique. Of special importance is the morphology of the bony pocket (classification, see p. 78). In addition, the consistency, width and thickness of the gingiva, as well as tooth position (crowding) and the amount of remaining periodontal bone (above all, interdentally) are important for the planning and the predictable outcome of any regenerative technique.

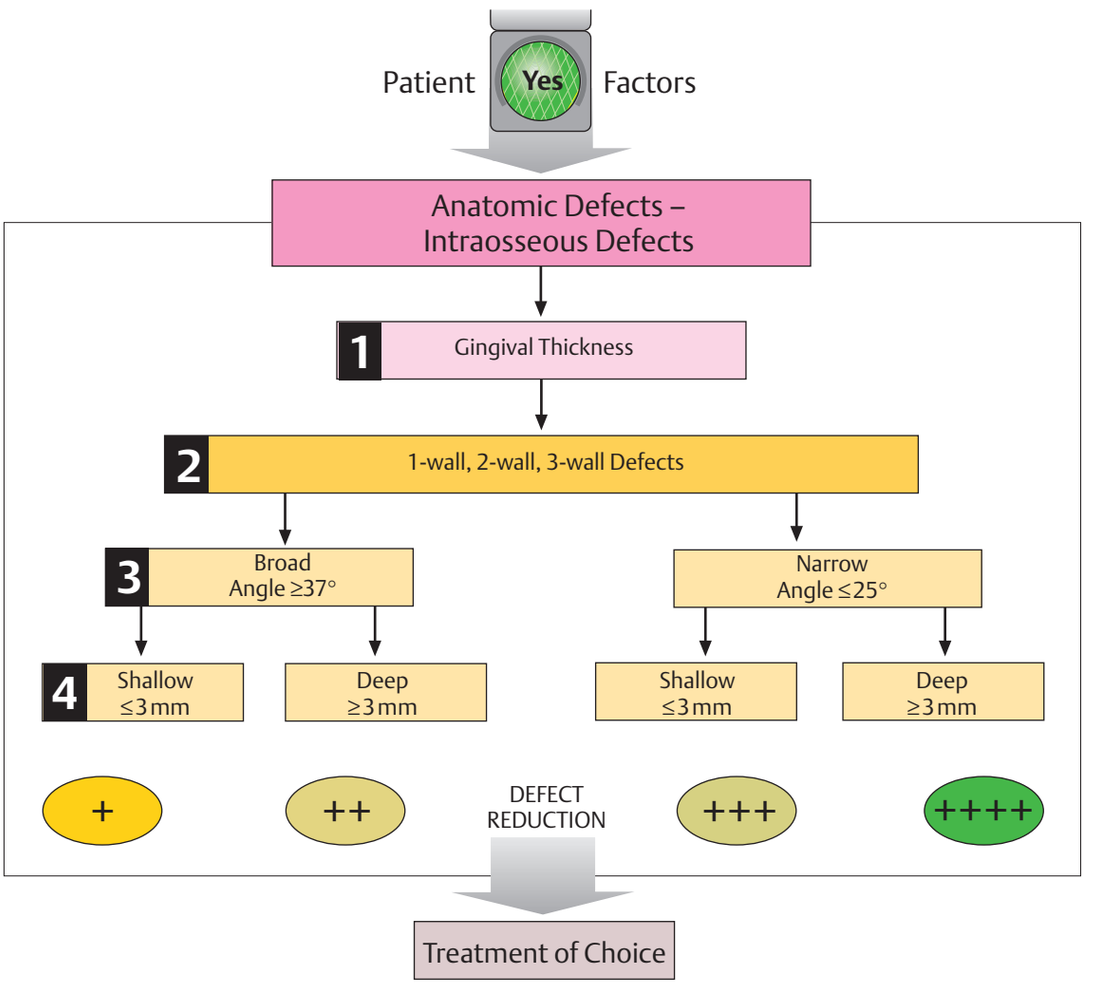
Regeneration (new formation) of *horizontal* bone loss in a *periodontally involved* dentition, e. g., between adjacent teeth, is rarely possible today. The same is true for the most part for gingival papilla regeneration, which can only be successful if the papillae are *supported by* interdental bone (p. 496). The situation may be quite different, however, in *edentulous alveolar segments*, in which the height and/or width of the alveolar process have been lost. In such instances, soft tissue enhancement (p. 505) or "*guided bone regeneration*" (GBR) can effectively correct the defect and create a sufficient fundament for dental implants or a favorable initial situation for esthetic, optimum bridgework.

744 Anatomy of the Periodontal Defect

For the planning of a surgical intervention in cases with *bony pockets*, pre-operative and also *intra-operative* consideration must be given to the anatomy of the osseous defect as well as that of the overlying gingiva (**1–4**).

It is clear that the success of treatment will always depend upon precise root cleaning and planing. Other factors that influence significantly the treatment result include the shape, width (angular degree) and depth of the bony pockets. Filling of the defect appears to be most successful with deep, narrow bony pockets. This does not mean, however, that the final result will be better the deeper the pocket! If a 5 mm pocket (gingival plus osseous defect) is reduced by only 3 mm, in practical terms a healthy sulcus of 2 mm in depth results. On the other hand, if a 10 mm pocket is reduced by 5 mm, the result remains a 5 mm residual pocket.

Modified from *P. Cortellini & G. Bowers 1995*



Selection of Treatment Materials for Regenerative Therapy

In order to "regenerate" periodontal structures, various methods and materials are available:

- **A** Osseous regeneration alone by means of closed or open root planing, without additional methods
- **B** Use of bone and bone replacement materials
- **C** Incorporation of membranes; guided tissue regeneration (GTR)
- **D** Use of matrix proteins and growth factors/differentiation factors
- **E** Combinations of the above-listed methods

While the methods listed as **A** and **B** primarily are directed toward the reformation of periodontal bone, the methods listed as **C**, **D** and **E** are targeted toward new formation of all periodontal structures, including cementum and periodontal ligament in addition to alveolar bone. While these efforts have been associated with limited successes, the clinical developments in this direction are not yet concluded.

Final word, 2004: Long-term healing of periodontitis and the complete regeneration of the destruction caused by this disease remain today a wonderful possibility for the future! Stay tuned …

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A Osseous Regeneration by Means of Closed or Open Root Cleaning/Planing, without "Additives"

As demonstrated by Rosling and co-workers (1976), following thorough root planing, and especially following open treatment of two- and three-wall defects, a significant gain of alveolar bone can result, but only if the patient maintains the strictest oral hygiene regimen. New cementum and periodontal ligament were, however, not demonstrated. As a consequence, the "Gothenburg School" continued the search for methods to achieve regeneration of *all* periodontal tissues (GTR, p. 338).

B Bone and Bone Replacement Materials

Previous studies have demonstrated that, with the exception of autogenous bone, the implantation of osseous and/or osseous replacement materials into intra-alveolar pockets does not lead to regeneration of *all periodontal tissues*. Such materials are really only "space maintainers" within the osseous coagulum, but often exert an *osteoinductive* effect. With or without implantation, however, the most important prerequisite for new bone formation (osseous defect filling) remains perfect and precise root cleaning/planing, secure wound closure post-surgically, monitored healing (Westfeld et al. 1983) and optimum oral hygiene.

Autogenous (autologous) bone from a healthy patient is variously osteoinductive, but nevertheless is the most promising substance for enhancement of new bone formation (p. 327; Rosen et al. 2000).
Favorable *intraoral* donor sites include edentulous arch segments as well as the tuberosity and the chin. Autogenous bone can be implanted as an "osseous coagulum" into a bony pocket after the simultaneous performance of osteoplasty/ostectomy into a filter (p. 328).
In contrast to autogenous bone, which is usually harvested from a distant (secondary) surgical site in the alveolar bone, *allogenic bone* and *bone replacement materials* are commercially available in unlimited quantities (p. 332).
Research has demonstrated that allogenic, human, demineralized, freeze-dried bone is *osteoconductive*, and possibly also *osteoinductive*, and is therefore clinically promising. In contrast, the *xenogenic* and *alloplastic* materials, such as Bioglas, are only osteoconductive; these materials may improve pocket probing depth, attachment level and osseous niveau, which are positive results, but which are not significantly better than the results achieved following optimum open root cleaning/planing. Because it is seldom possible to harvest sufficient quantities of autogenous bone (second surgery), it is common to mix autogenous tissue with alloplastic substances or xenogenic materials (Camelo et al. 2001).

C Barrier Membranes—Guided Tissue Regeneration (GTR)

The first clinical investigations using the GTR technique were promising of success (Nyman et al. 1982a, b; Gottlow 1986; Gottlow et al. 1986; Ponteriero et al. 1987): The purpose of the membrane was to *prevent* healing via "repair," via a long junctional epithelium. The purpose of the mechanical barrier was to inhibit the rapid epithelial downgrowth as well as contact of the root surface with gingival connective tissue, thus giving the deeper periodontal structures sufficient time for regeneration. The GTR technique has become common practice in periodontology, and in certain cases provides better results in terms of attachment gain vis-à-vis previous regenerative methods (Cortellini & Tonetti 2000, Pontoriero; p. 354).

D Growth Factors and Matrix Proteins

Complete regeneration of periodontal tissues, including cementum, periodontal ligament, bone and gingiva, demands the coordination of pluripotent cells, extracellular matrix, systemic hormones, growth factors, certain matrix proteins and signal molecules (Cochran & Wozney 1999).

The *future of periodontitis therapy* probably resides in the use of these biologic substances. Nevertheless, the primary mechanical and adjunctive medicinal elimination of the primary etiologic bacteria and the biofilm will nonetheless remain essential components of comprehensive treatment.
Some substances are already available for clinical use, or will soon be introduced, including growth and differentiation factors, proteins such as "bone morphogenetic protein" (BMP) and amelogenins: Matrix proteins (amelogenins/emdogain) and the blood platelet growth factor (PDGF, "platelet-derived growth factor") are already used in periodontal practice today.

E Combinations of Various Regenerative Methods

It is only natural/understandable that the individual treatment methods **B** through **D**, might be combined in order to optimize the regeneration of periodontal structures. For example, an osseous or a bone replacement material used to fill a defect may also be covered by a membrane in order to prevent premature epithelial downgrowth.

The anatomy of the osseous defect, the surgical procedure and the treatment of choice will determine the ultimate procedure based upon the surgeon's preferences and his/her experience with the individual materials and combinations thereof.

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Bone Regeneration without "Transplantation"

Alveolar bone regeneration following closed and, above all, open periodontal treatment is "under certain conditions" possible even *without filling materials* (Rosling et al. 1976, Lang 2000). Bone regeneration can be enhanced by filling materials and GTR—individually or combined—and modified by growth factors etc.

Regardless of the pathway toward osseous regeneration, the most important aspect for success is *perfect root planing* during the surgical procedure and continuous, optimum plaque control by the dental team (recall) and the patient.

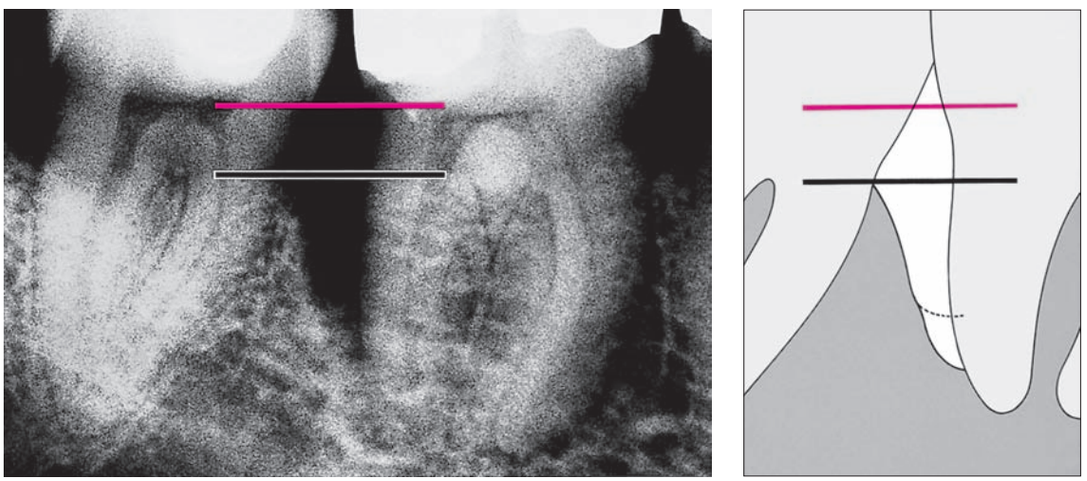
Osseous regeneration does not, however, indicate that true periodontal regeneration with new cementum and the formation of a new periodontal ligament has occurred. In many cases, only a long junctional epithelium results, extending apically beyond the newly formed bone, even to the depth of the original pocket. Nevertheless: New bone formation may be viewed as a therapeutic success (reduction of the osseous defect, freedom from infection).

The radiographs of a 35-year-old patient, depicted below, reveal "new bone formation" without any transplanted substances.

745 9 mm Pocket with a Deep Vertical Osseous Defect

The pocket distal to tooth 46 was debrided and thoroughly root planed with direct vision (flap surgical procedure).

Right: The schematic representation of the radiograph depicts the level of the CEJ (red line), the level of the original bony margin (black line) and the expanse of the osseous defect.

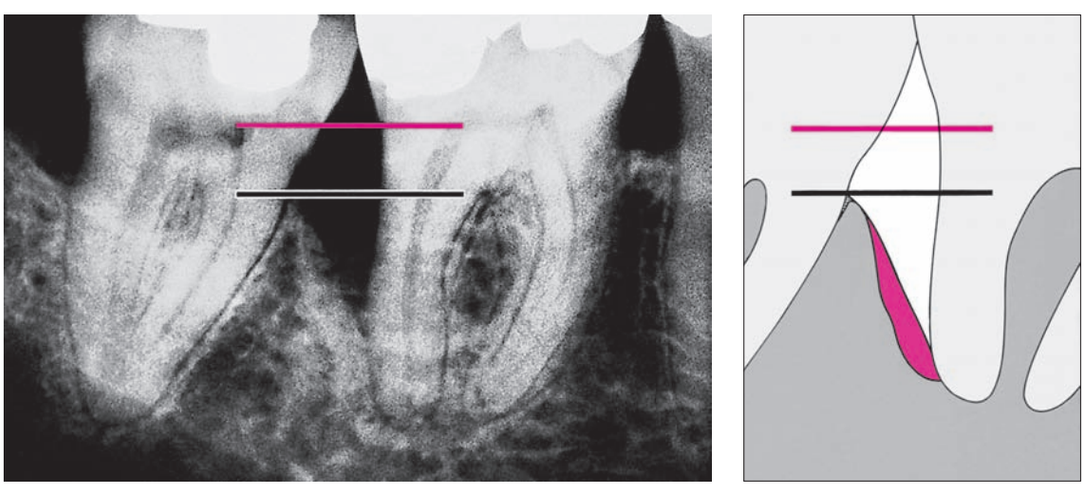


746 Beginning Osseous Regeneration

Several weeks after the surgery, the defect appears to be $^1/_3$ filled.

Right: In the schematic, the initial osseous formation is shown in red.

Note: True osseous regeneration can only be demonstrated *histologically*.

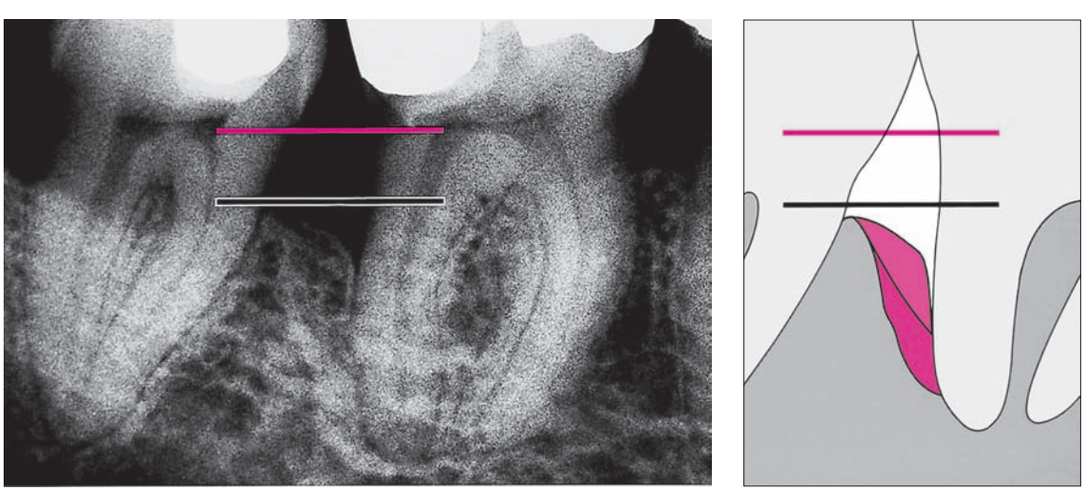


747 Bone Regeneration 6 Months Post-Operatively

The original vertical defect has almost completely filled in. The clinical probing depth is 3 mm. The osseous regeneration observed in the radiograph, however, cannot be taken as a qualitative indication of the degree of true new attachment.

Right: The schematic depicts the extent of new bone formation (red).

Courtesy *G. Cimasoni*



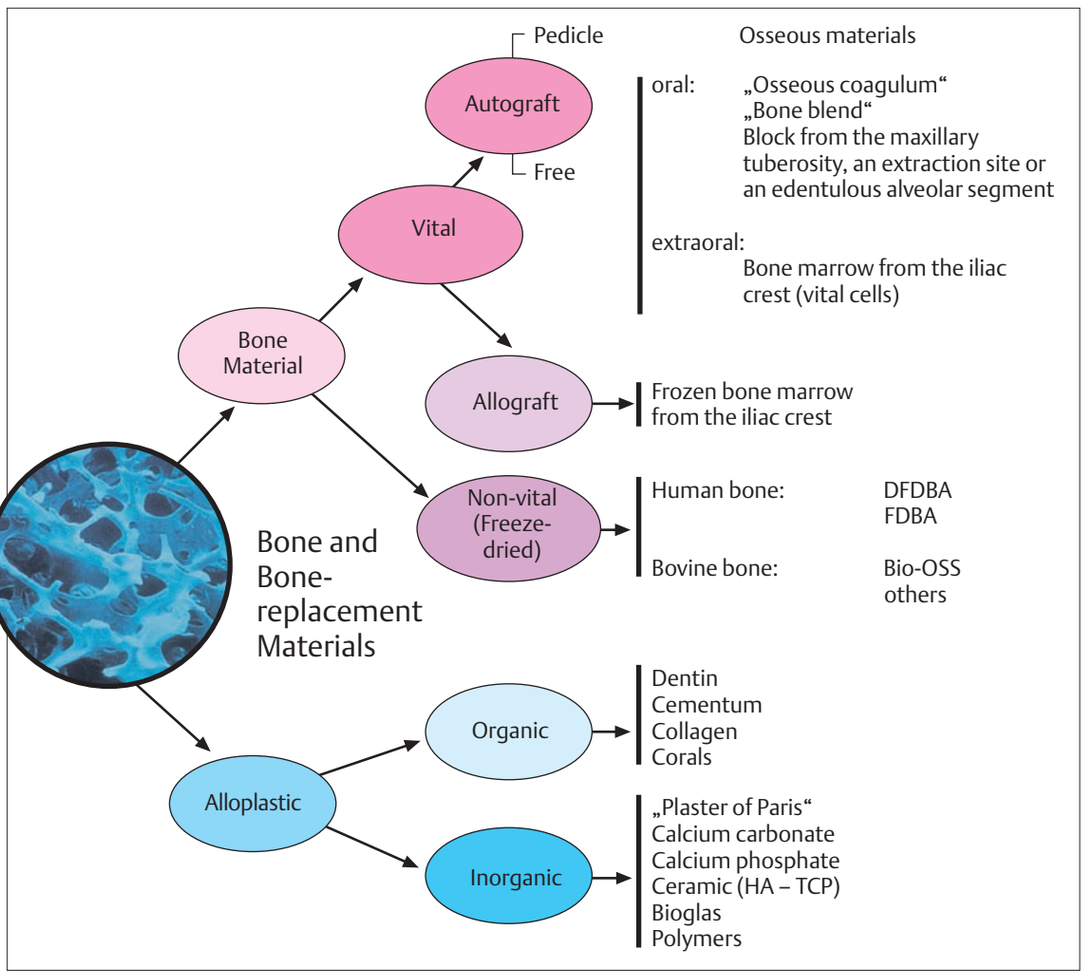
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Filling Materials for Intra-Alveolar Pockets—Transplants/Implants

- *Transplantation* is the surgical transfer of living tissue or whole organs. The tissue (organ) must remain vital in the transplantation bed.
- *Implantation* is the surgical transfer of non-vital or dead tissue, or other tissue-foreign material.

In regenerative periodontal surgery, an autogenous piece of bone could also be termed a transplant; however, even "filling materials" (bone chips) from the same individual will be resorbed within the osseous defect and eventually replaced with newly formed bone.

For decades, attempts have been made to fill periodontal osseous defects with various materials. The therapeutic efficacy of such endeavors appears to be better than closed or open therapy, but remains unimpressive. In addition, it is important to remain cognizant of the fact that osseous regeneration within intra-alveolar pockets does not signify the regeneration of all periodontal tissues.



748 Bone and Bone Replacement Materials
It is important to differentiate among the implant materials for the filling of intra-alveolar defects:

- True bone
- Bone-derived materials
- Bone replacement materials

The most effective material, with true *osteoinductive* potential for new bone formation is autogenous bone, with can be harvested *intraorally* from the alveolar bone, or in special cases *extraorally*, e. g., from the iliac crest.

At the present time, secondary materials include allogenic and xenogenic bone materials, and a third choice of alloplastic materials, including organic and inorganic, natural and synthetic materials not derived from bone (Nomenclature; see p. 332).

Modified from *M. Congé et al. 1978, J. Ouhayoun 1996*

In addition to the effectiveness of the various materials, it is important to remember that such treatment methods require/demand additional effort, and demand also special knowledge and experience of the clinician. Thus, not every dentist, and not even every specialist will be prepared to harvest trabecular bone from the iliac crest. While such methods may bring good results, there must be a comparison of "cost and benefit," and consideration of side effects, that may result in a less than realistic solution. For example, a necessary second surgery in the patient's mouth for harvesting a bone transplant, e. g., from the tuberosity or from an edentulous jaw segment, is more complicated than the use of other available materials such as freeze-dried bone or inorganic filling materials such as tricalcium phosphate (TCP), hydroxyapatite (HA), bioglasses etc.

Only autogenous bone (possibly also allogenic material) appears to have any inductive potential for the creation of new periodontal bone. All other materials appear to have only a conductive effect as a "space maintainer" or stabilizer of the blood coagulum (Urist 1965, 1973).

For organic and inorganic material not derived from bone, it has not yet been possible to verify any clinical efficacy.

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Instruments for Harvesting Autogenous Bone ...

Because of its osteoinductive effect, autogenous, vital bone remains the best material for the filling of periodontal osseous defects. Such bone can be harvested during periodontal surgery in several ways:

During osteoplasty or ostectomy (flap surgery) with copious saline solution rinsing, a filter inserted distal to the evacuation tip can trap particles of bone for subsequent use in infrabony pockets (Robinson 1969, Dayoub 1981). Various filter systems are commercially available.

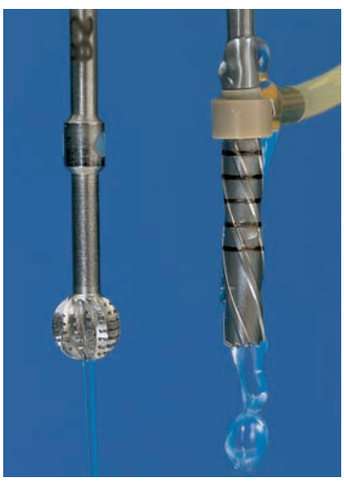
Additional instruments for the removal of autogenous bone include the trephine drills (hollow drills). These are available in various sizes for use manually or in the contra-angle handpiece.

Finally, normal fissure burs or special Lindemann drills, separating disks etc. can also be used to harvest bone from the edentulous ridge, from exostoses etc., which may necessitate a second surgical site.

749 Internally Cooled Burs and Carbide Burs
When performing osteoplasty or ostectomy, cooling with NaCl or Ringer's solution is imperative. The fluid can be directed through the bur itself (left) or must be externally applied when normal burs are used (small carbide burs, right).

Right: Enlargement of an internally cooled bur and a hollow drill.





750 Bone Filters—Bone Traps
Three bone trap systems depicted in mirror image (visible undersurface, from left to right):

- **Titan filter sieve**
  With casing; Frialit Co.
- **Osseous coagulum trap/OCT**
  Quality Aspirators Co., Duncanville, TX
- **Bone trap**
  Heico Dent, Switzerland

Right: Common suction cannulae, with three different filters systems.





751 Additional Instruments for Harvesting Bone—Trephines
Instruments indicated for harvesting bone include hollow burs, such as those used in implantology, carbide burs, internally cooled burs and trephines (Hu-Friedy), commercially available in various sizes.

Right: Trephine bur of 10 mm diameter. Large pieces of bone must be reduced in size before insertion (to ca. 1 mm^3), e. g., using commercially available bone mills.





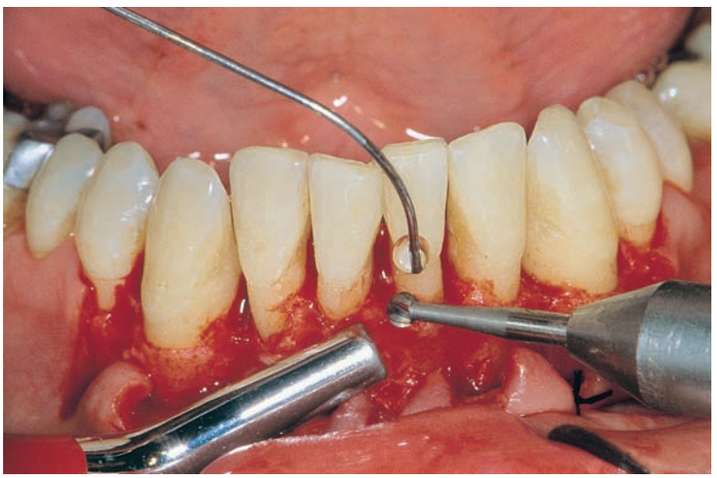
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... and Their Use

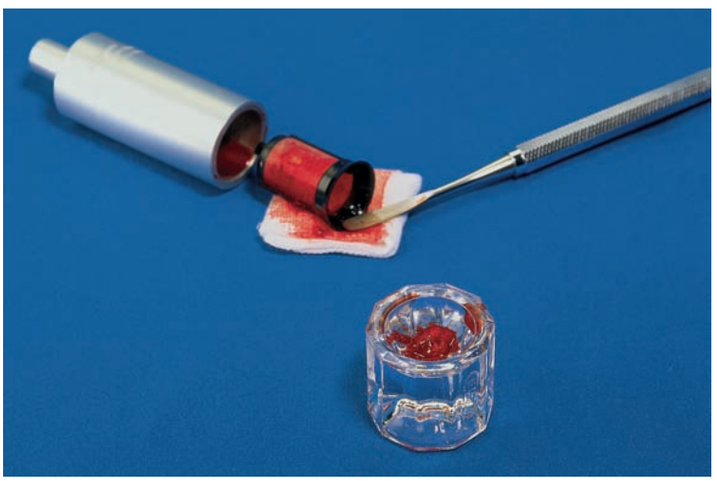
In cases exhibiting extremely irregular bone loss and/or variable osseous architecture, resective and regenerative techniques are frequently combined. On the one hand, bulbous exostoses are modeled, sharp osseous edges are rounded and any intra-alveolar defects are somewhat reduced; on the other hand, deeper osseous pockets are filled using autogenous material.

In this way, the autogenous bone captured in a filter during osteoplasty/ostectomy can be used to fill deeper bony defects; the filled material must sometimes be "stretched" using commercially available materials.

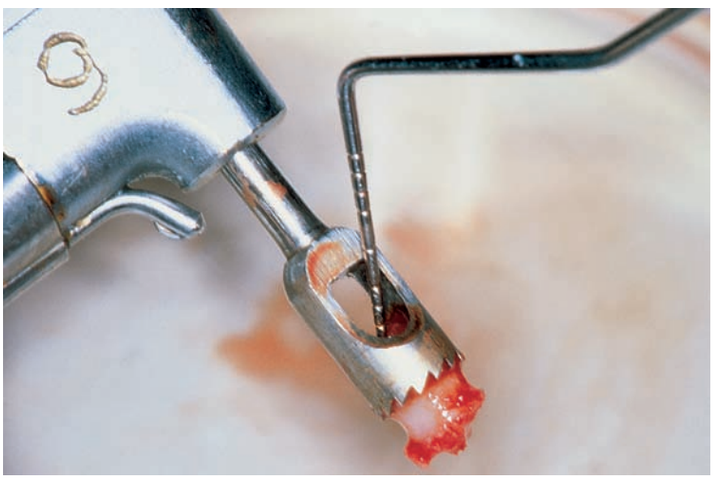
If osteoplasty/ostectomy is not indicated, autogenous bone may be harvested from a "distant" site on the alveolar ridge by means of a (small) second surgery. The material that is removed must contain not only compact bone but also trabecular bone. Before transplanting the harvested bone into the bony defect, it must be reduced to small chips of not greater than 1 mm^3. This can be accomplished with a hefty scissors or a "bone nipper"; the procedure is often difficult and associated with loss of harvested material; a small bone mill (e.g., mortar and pestle) may be used with less loss of transplant material!



752 Osteoplasty—Cooling and Evacuation
The bone chips created by osteoplasty become mixed with the coolant fluid and blood and are then trapped in the filter within the evacuation device. This bone-coagulum can then be implanted into a 2- or 3-wall bony pocket.



753 "Osseous Coagulum Trap" (OCT)
The osseous material harvested during osteoplasty/ostectomy is removed from the filter before being placed into a vertical osseous defect.



754 Harvesting Bone with a Trephine Bur
The stiff Williams periodontal probe is used to eject the harvested bone from the trephine bur. This bone was harvested from the mandibular retromolar area. This piece of bone must be reduced to chips, which can be accomplished using a scalpel or scissors.

A "bone mill" (e.g., "Karr-Dental," or "Quetin") simplifies the procedure but is expensive because it is seldom employed.

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Implantation (Transplantation?) of Autogenous Bone

Surgical Procedure, Step-by-Step

A 30-year-old female wished to have the space in her upper left quadrant closed by means of a fixed bridge. Because the mandible was completely dentulous it was prudent to maintain the lone-standing maxillary second molar (27) as an abutment, but a 7 mm infrabony pocket could be probed on the mesial aspect of this tooth. The clinical examination led the practitioner to suspect a multi-walled defect.

The treatment plan called for filling this defect with autologous bone. The edentulous area between 24 and 27 was available as a site for harvesting the transplant material; however, because of the proximity of the maxillary sinus, this is a "risky" donor site.

Findings after initial therapy in the entire dentition:

PI: 9% BOP: 11%

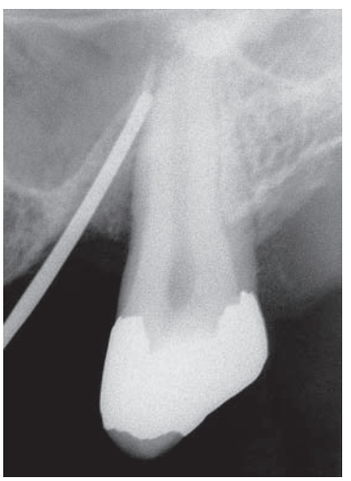
TM: Grade 2 for tooth 27, grade 1 for teeth 23 and 24.

The clinical picture and radiograph are presented in the figures below.

755 After Initial Therapy
The persistent 7 mm infrabony pocket mesial to the second molar is depicted clinically with a Goldman periodontal probe *in situ*.
A 5 mm gingival pocket was present on the distal surface of tooth 24.

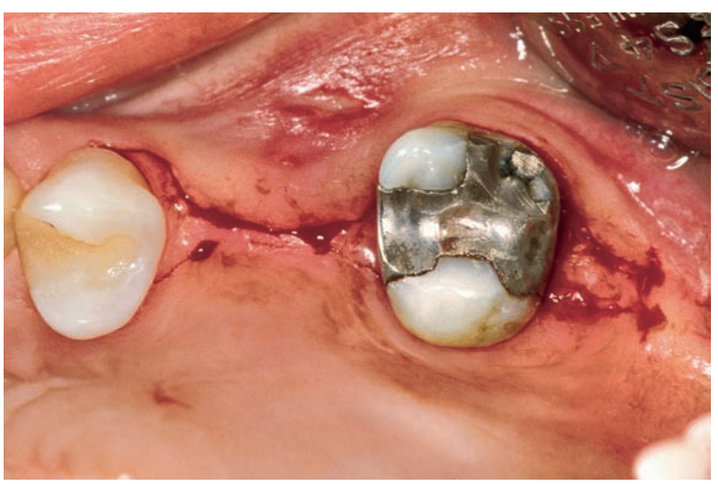
Right: The deep pocket mesial to tooth 27 is depicted in the radiograph even more clearly than in the clinical view.

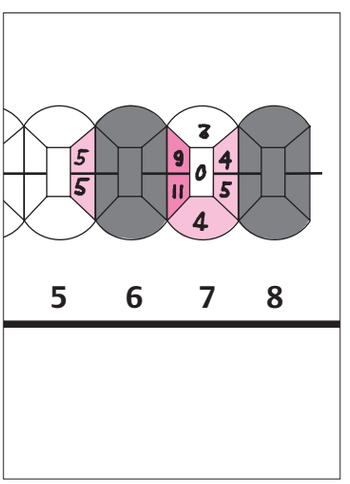




756 Incisions
Buccal and oral flap reflection is necessary in order to perform periodontal treatment on teeth 24 and 27, with simultaneous harvesting of autologous bone. The incision courses parallel to the alveolar crest. As the incision approaches the teeth it is "wedge form" and becomes a sulcular incision on both buccal and oral aspects. Distal to tooth 27, the incision ends as a modified wedge procedure (cf. Fig. 730).

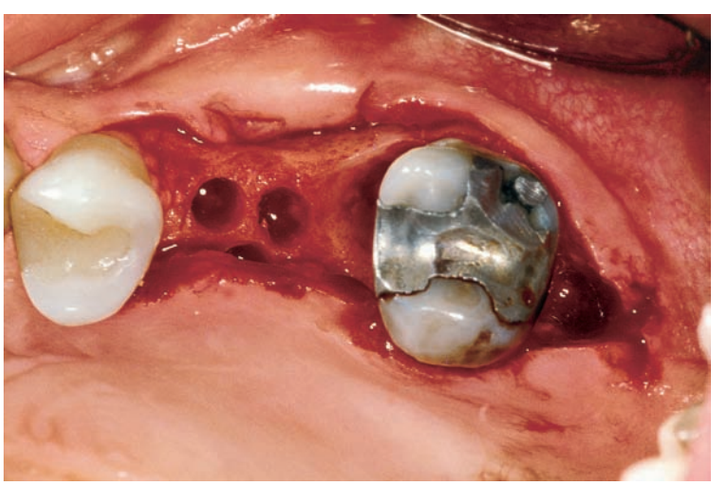
Right: Pocket probing depths.

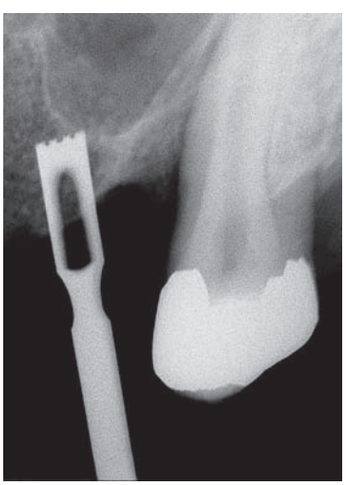




757 Surgical Field Exposed
Following root planing and debridement of the infrabony pocket, the 3-wall defect and two of the donor sites are visible. Three small (2.8 mm) pieces were harvested, without altering the contour of the alveolar ridge. The three pieces of bone were further reduced in size before transplantation.

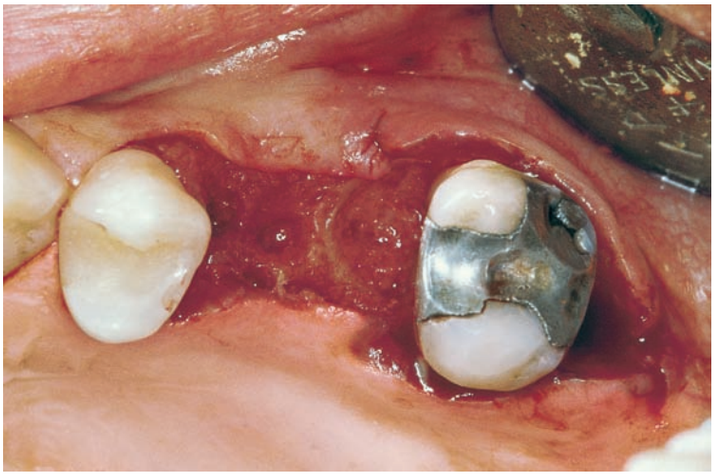
Right: Radiographic view of the trephine drill during bone removal.
Caution: Maxillary sinus!





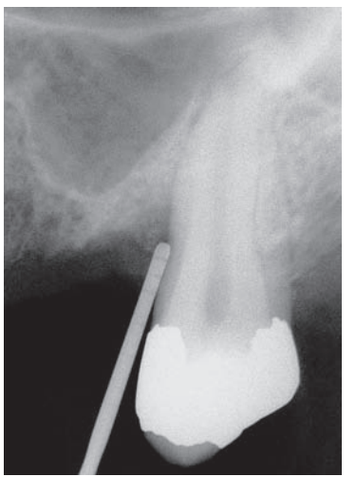
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758 After Transplantation of the Bone
Bony chips with a maximum size of 1 mm^3 are tightly wedged into the 3-wall defect.
The crater is completely filled with blood and bone chips, and blood clots fill the donor sites. Such sites re-ossify quickly, similar to a small extraction site.

Left: Trephine bur with the harvested bone cylinder.



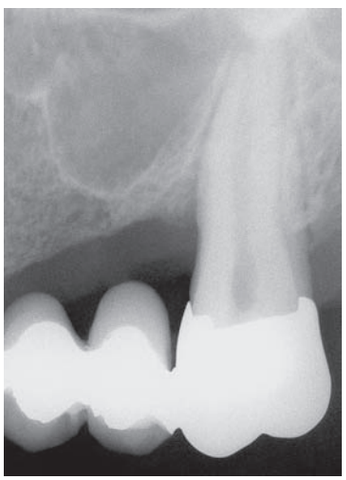


759 Tight Closure of the Surgical Site
Healing of the site can only occur optimally if the flap closure is complete and secure.
The surgical site should be protected from mechanical trauma by a periodontal dressing, or as in this case, with a tissue adhesive (Histoacryl).

Left: Radiographic check of the osseous fill. Compare the original radiograph (Fig. 755, right).



760 3 Months Post-Operative
Healing proceeded without complications.
Fabrication of the new fixed bridge can begin in a few more months even though the maturation phase of the tissues (e. g., the gingival margin) usually takes a bit more time. The supragingival position of the crown margins permits such early seating of the definitive bridge.





761 7 Months Post-Operative—Seating the Bridge
The 4-unit bridge is first seated temporarily. The bridge abutments at that time (1982) were *three-quarter crowns*. Interdental hygiene can be performed using spiral brushes. The periodontal pocket on the mesial of tooth 27 was eliminated via apical repositioning of the gingiva and possible (?) osseous regeneration.

Left: Radiograph—Defect mesial 27 filled in with new bone.

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Bone Replacement Materials—"Fillers"

Filling materials for intra-alveolar pockets were listed on page 327. The practical use of autogenous bone was described (p. 328), and the high success rate with this method of filling bony defects was noted. However, nothing could be said about the formation of *new attachment.*

There have been no reports that equally good results would be expected if *syngenic* bone from identical twins were transplanted. This possibly is mentioned here only *theoretically*, although it is hardly of any great significance, because in dentistry—neither in practice nor in the literature—is the practice of vital bone *trans*plantation for periodontitis therapy mentioned, described or acknowledged. On the other hand, in general medicine, there is of course no better organ or tissue donor than an identical twin.

In this section, we will discuss only *bone replacement materials*. Their names and synonyms (former and contemporary nomenclature), the source of the material and examples of several of these materials are presented in Figure 762 and on page 333.

762 Name, Origin and Examples of Bone and Bone Replacement Materials

Autogenous Bone
An autogenous transplant consisting of cortical bone, trabecular bone and bone marrow cells is the most promising material. It is both *osteoinductive* and *osteoconductive*.

Bone Replacement Materials
Allogenic material, bone derived from human cadavers, is available through authorized and certified tissue banks:
- DFDBA: demineralized freeze-dried bone allograft
- FDBA: freeze-dried bone allograft

Xenogenic filling material (from other species), e. g., bovine bone, as well as *alloplastic*, synthetic material have been used in recent years more and more, not least because they are available in virtually unlimited quantities.

The various materials may often also be combined.

Graft type	Synonyms	Origin	Examples
Autograft	Autogenous (previously: "autologous")	Same-person transplant	Cortical bone Trabecular bone Combinations Bone marrow cells
Isograft	Syngenous	Monozygous twins, related persons	Cortical bone Trabecular bone Combinations
Allograft	Allogenic, homologous	Same species	DFDBA FDBA Freeze-dried bone marrow, iliac crest
Xenograft	Xenogenic, heterogenous	Other species	Collagen Bone Hydroxyapatite
Alloplastic graft	Alloplastic, synthetic	Chemically foreign body	„Plaster of Paris" Calcium carbonate Calcium phosphate (-ceramics) HA β-TCP Bioglasses Polymers
Combined techniques		e. g., autogenous + alloplastic	Cortical bone – Bio-Oss collagen

Natural or Synthetic?

Allogenic, xenogenic and alloplastic material is widely available commercially and has a long shelf-life, but is often also relatively expensive. The use of such materials is often not accepted by dental insurance companies.
Regardless of their usefulness, one must always consider the fact that systemic infections by viruses cannot be completely eliminated when using natural implantation materials. With allografts, there have been discussions about the minimal possibility of transfer of HIV or hepatitis, and with xenografts fears of BSE ("mad cow disease") and swine fever. The manufacturers of bone replacement materials ensure that their materials are subjected to the most strenuous control so that any danger of infection is ruled out.
In conclusion it is worth repeating that *only* autografts can be expected to routinely induce the formation of new bone; allogenic bone is only a poor inducer. The age of the anonymous donor, and the preparation of the material for commercial distribution appear to elicit variability. All of the other materials also have a conductive potential or the material is osteogenic, "bioactive" (e. g., bioglasses). The formation of new bone does *not* provide the periodontal "holy grail": The regeneration of *all* periodontal tissues. The intensive search for the "ideal" *inductive* replacement material continues (p. 351).

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Allogenic—Human DFDBA



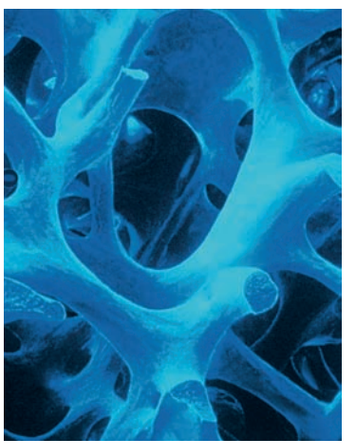


Bone Replacement Materials

763 DFDBA—Freeze-Dried Bone (Human)
This allogenic (homologous) material can be obtained from a tissue bank (e. g., University of Miami). Because of sterility considerations, a single portion should only be used for *one* patient. For implantation into a bony pocket, this material must be well mixed with blood.

Left: SEM-enlarged trabecular structure.

Xenogenic—Bovine HA



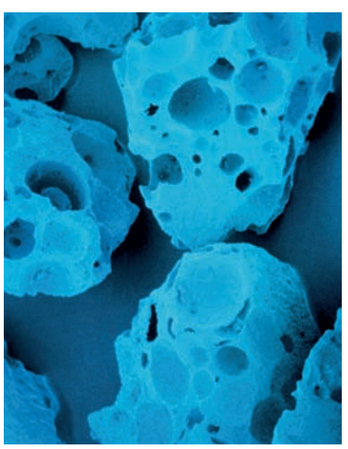


764 Xenogenic (Heterologous) Material
Bovine bone material. The granulated tissue derives from the cortical or trabecular bone and consists primarily of hydroxyapatite (HA). This figure depicts various particle sizes of the Bio-Oss product.

Left: Bovine trabecular bone is similar to human bone in terms of pore size and trabecular configuration.

TEM courtesy *Geistlich Co.*

Alloplastic—β-TCP





765 Alloplastic (synthetic) Tricalcium Phosphate
β-TCP (“Ceros,” particle size 0.5–0.7 mm) has been used for a very long time as an osteoconductive “space maintainer” in bony pockets. In contrast to hydroxyapatite, it is resorbed relatively quickly and replaced by autogenous bone. The “powder” must be mixed with blood and/or saline solution before application.

Left: SEM picture of the porous TCP granules (Ceros Co.).

Alloplastic—Bioglass





766 Alloplastic Bioglass
The synthetically produced “PerioGlas” (US Biomaterials; particle size ca. 0.3 mm) has established itself as a filler for osseous pockets. It has a strongly basic character, which gives it anti-infectious and anti-inflammatory properties. It is slowly resorbed, then replaced by the host’s own bone.

Left: The “sticky” PerioGlas material can be easily placed into osseous defects.

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Alloplastic Implant—Combined Surgical Procedure

Flap Surgery (Facial)—GV and Flaps (Palatal)

Surgical Procedure, Step-by-Step

In the case presented here, various periodontal surgical procedures were combined. The primary goal of the surgery was to attempt to enhance bone regeneration in the 8 mm pocket mesial to the left central incisor, by placement of an alloplastic implant material (resorbable beta-tricalcium phosphate, β-TCP). Secondary goals included minimization of post-operative gingival recession and the shrinkage of interdental papillae, for esthetic reasons.

The patient was a 27-year-old female, manifesting generalized mild and localized severe periodontitis. The 8 mm pocket on the mesial of tooth 21 persisted even after initial therapy.

Findings before surgery in the entire dentition:

PI: 15 % BOP: 25 %

TM: Average of 1, but grade 3 on tooth 21

Clinical picture, probing depths and radiographic survey are depicted below.

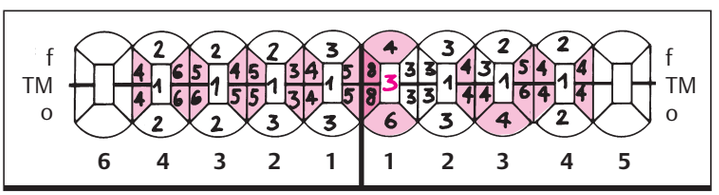
767 After Initial Therapy
The gingivae are essentially free of clinical signs of inflammation following initial therapy (supra- and subgingival debridement). However, several active residual pockets persist, the deepest (8 mm) 2- and 3-wall bony defects mesial to tooth 21. Mild occlusal trauma (clenching with premature contact) was eliminated by selective grinding.

Right: The periodontal probe indicates the depth of the periodontal pocket.

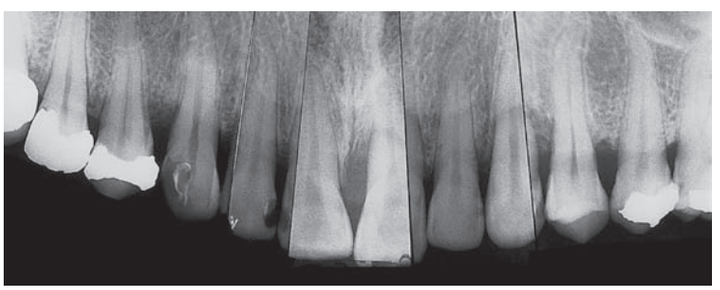




768 Probing Depths and Tooth Mobility (TM) Following Initial Therapy
The highly mobile tooth 21 must be approached surgically, with its mobility of grade 3.

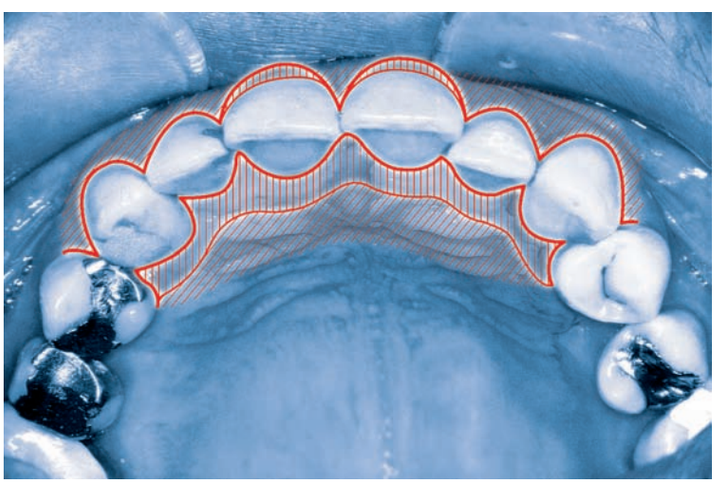


Radiographic Survey
The relatively "soft" radiograph does not provide an entirely clear picture of the deep defect mesial to tooth 21.



769 Treatment Plan—Clinical, Occlusal View Following Initial Therapy
Despite initial therapy, mild gingivitis persists. The relatively steep anterior palatal vault permits gingivectomy without the creation of an expansive wound surface (hatched area). After the gingivectomy, a sulcular incision is performed to delineate a soft tissue flap.

Right: Protocol/plan of the surgical procedure.



Combined Surgical Procedure using β-TCP—Surgical Protocol

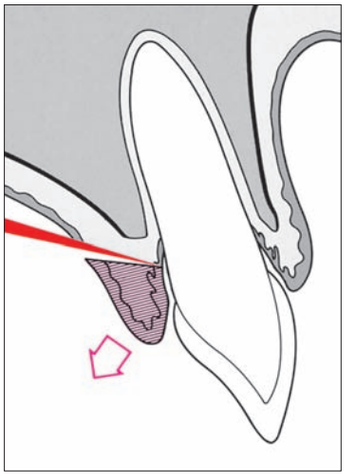
Palatal

- GV, oblique incision, followed by a sulcular incision
- Flap reflection

Facial

- Paramarginal incision 11; 21
- Sulcular incision 22 and 23
- Full-thickness flaps
- Minimal osteoplasty
- Curettage of the osseous defect, root planing
- Suture placement between 11 and 21
- Defect filling with β-TCP
- Suture placement and periodontal dressing

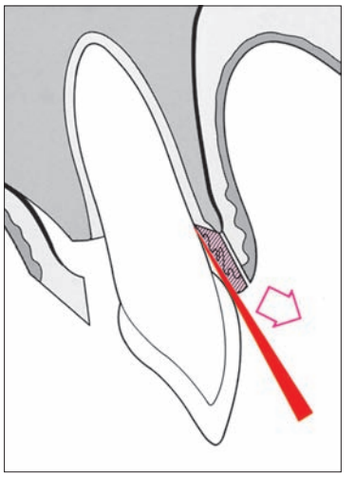
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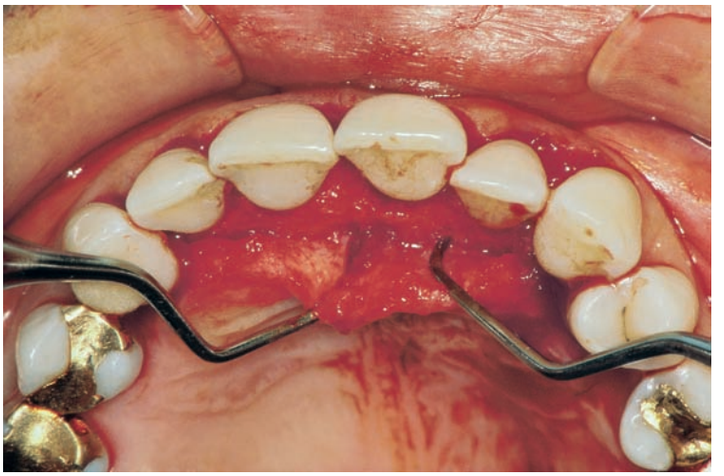




770 Palatal Gingivectomy
The first step is a gingivectomy (oblique incision; p. 367) without contacting the palatal bone.
By using the double-angled periodontal knife (GR 1L/1R; Deppeler) access is easy and any potential technical difficulties are overcome.

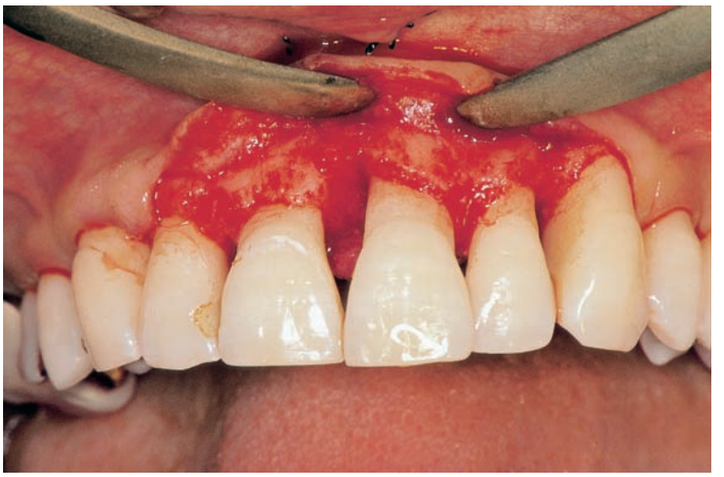
Left: Schematic view of the oral gingivectomy



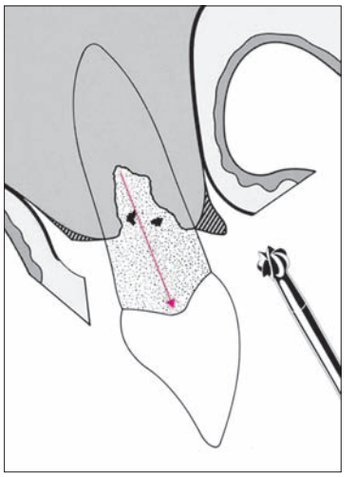


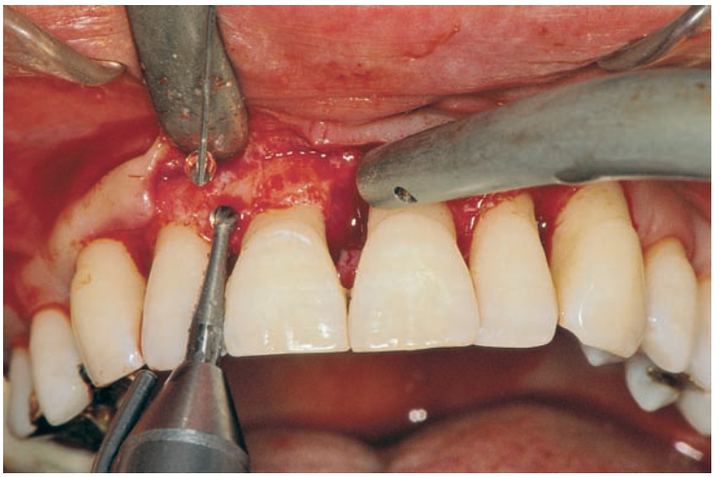
771 Mobilization of the Palatal Flap
Following the gingivectomy, a crevicular incision (not shown) is made using the scalpel. The palatal flap is reflected and direct access is gained to the root surfaces, alveolar bone crest and the interdental osseous crater.

Left: On the *facial* aspect, a paramarginal incision (internal gingivectomy) has been performed to delineate a soft tissue flap. The sulcular incision (depicted) then follows as a second incision.



772 Root Planing
The deep osseous crater mesial to tooth 21 has filled with blood following thorough but careful debridement and root planing (coagulum).
Regeneration of new bone in the depth of this crater may be anticipated, without the use of an implant of any kind.





773 Osteoplasty
The bulbous bony margin between 11 and 12 is carefully recontoured without reducing the *height* of the supporting alveolar bone.

Left: The anticipated osteoplasty is indicated (hatched area). After recontouring the bone, definitive planing of the root surfaces (red arrow) is performed on the mesial aspect of tooth 21.

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774 Bone Replacement Material; Beta-Tricalcium Phosphate (β-TCP)
TCP implantation materials (Ceros) in varying particle sizes (cf. p. 333).

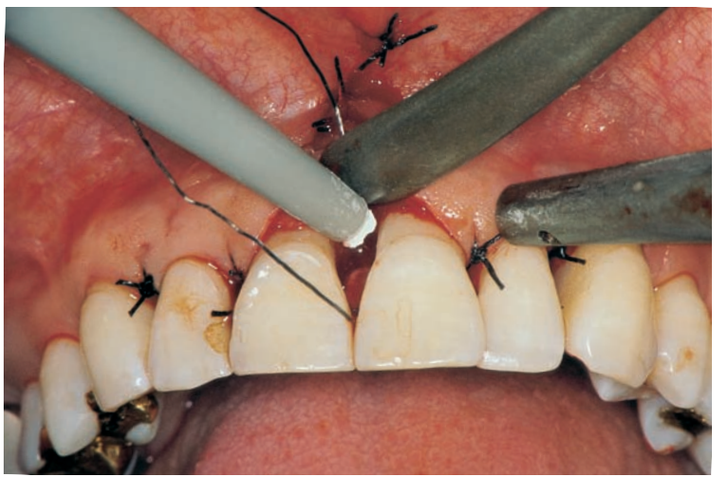
Right: The porous, synthetic material is mixed with Ringer solution to form a thick paste.
It can be conveniently placed into the osseous defect. The defect must be mechanically stimulated to cause hemorrhage (using a scaler, or even a rotating bur into the marrow spaces).





775 Filling the Bony Defect with β-TCP
The facial and oral flaps have been secured using interdental sutures. In the area of the osseous defect mesial to tooth 21, the suture has been placed but not yet tied.
The TCP is forced into the bony pocket. It must become well mixed with blood. The osseous defect must not be overfilled.

Immediately thereafter, the preplaced sutures are used to adapt and secure the soft tissues flaps.



776 Tight Flap Adaptation
To preclude loss of the implanted material, flaps must be closely adapted and completely closed over the interdental defect.
Caution: The suturing must be completely *tension-free*. Tension can lead to ischemia and necrosis of the flaps, especially the papillary segments (loss of interdental papillae and resultant esthetic dark appearance of the interdental space). An additional consequence might be loss of the implant material or loss of the sutures, with similar consequences.

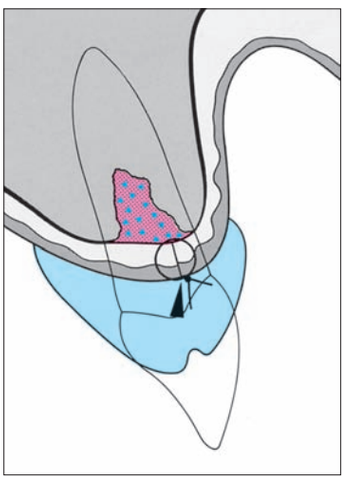


Wound Healing

777 Periodontal Dressing
In order to guarantee undisturbed wound healing, the surgical site is protected from mechanical trauma by placement of a well-adapted periodontal dressing. Coe-pak rolled in chlorhexidine-HCl was employed in this case.

Right: The coagulum (red) with the TCP particles (blue) fill the infrabony defect completely. The suture (arrow) is protected by the dressing (light blue).





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Combined Procedure—Intra-Osseous Implant

Summary

The young female patient responded positively to initial therapy and demonstrated a high level of motivation even before the surgical intervention. Despite this, some active pockets persisted, especially in the interdental areas (active "residual pockets").

By means of a combined surgical procedure, the deepest residual pocket, a vertical osseous defect between teeth 11 and 21 was successfully treated. The 8 mm pocket on the mesial of tooth 21 was reduced to a depth of 3 mm.

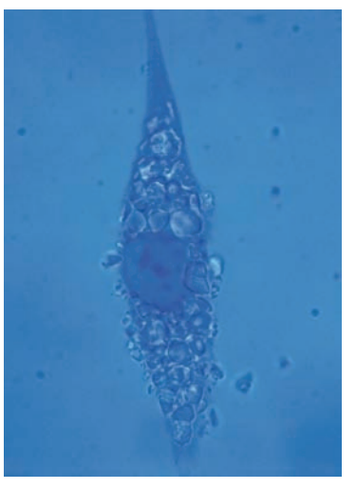
Some of this pocket depth reduction, of course, was due to a significant amount of gingival shrinkage. Nevertheless, true osseous regeneration occurred to a large degree in the intra-osseous pocket. Whether or not the tricalcium phosphate implant accelerated or increased bone regeneration cannot be determined. Similarly, neither clinical examination nor radiographic appearance can reveal whether new formation of cementum and periodontal ligament occurred at the depth of the pocket.

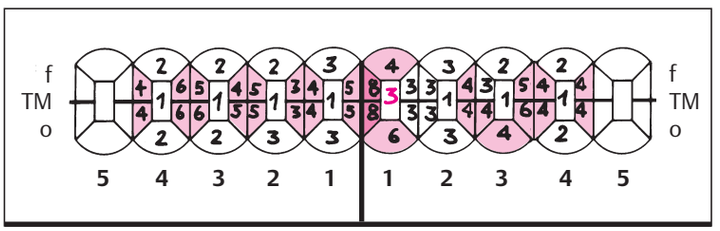


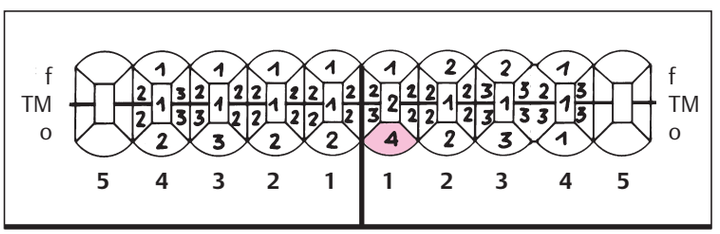


778 After Initial Therapy
The gingival contour is satisfactory following initial therapy. A deep infrabony pocket (8 mm) is present on the mesial aspect of the left central incisor.

Left: The radiograph reveals a 2- to 3-wall defect on the mesial aspect of tooth 21.







779 Pocket Probing Depths and Tooth Mobility (TM) Following Initial Therapy (above) and One Year Post-operatively (Below)

Before

After

Left: This culture shows a vital fibroblast with phagozytosed TCP particles (glass-like particles within the cell). This demonstrates the biocompatibility of β-TCP. Courtesy *A. Kallenberger*





780 One Year Post-Operative
The combined surgical procedure led to a reduction of pocket depth from 8 to 3 mm. This reduction could be accounted for by gingival shrinkage and possibly some osseous regeneration.

Left: The radiograph reveals what appears to be some filling of the osseous defect mesial to tooth 21 (cf. pre-operative radiograph, Fig. 768).

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Guided Tissue Regeneration—GTR

To understand regenerative processes such as the new formation of all periodontal tissues (cementum, periodontal ligament, alveolar bone), one must recall the original formation of these structures during embryologic development of the tooth, its root and the surrounding tissues (Hammarström 1997, Selvig & Wikesjö 1999, MacNeil & Somerman 1999).

The histologic development of the root via the Hertwig epithelial sheath is well understood. As is also the case with other embryologic tissue formation and healing processes, guidance of the developmental processes is via genetically determined mediators such as growth factors and differentiation factors including bone morphogenetic proteins (BMP) as well as especially matrix proteins of the Hertwig epithelial sheath.

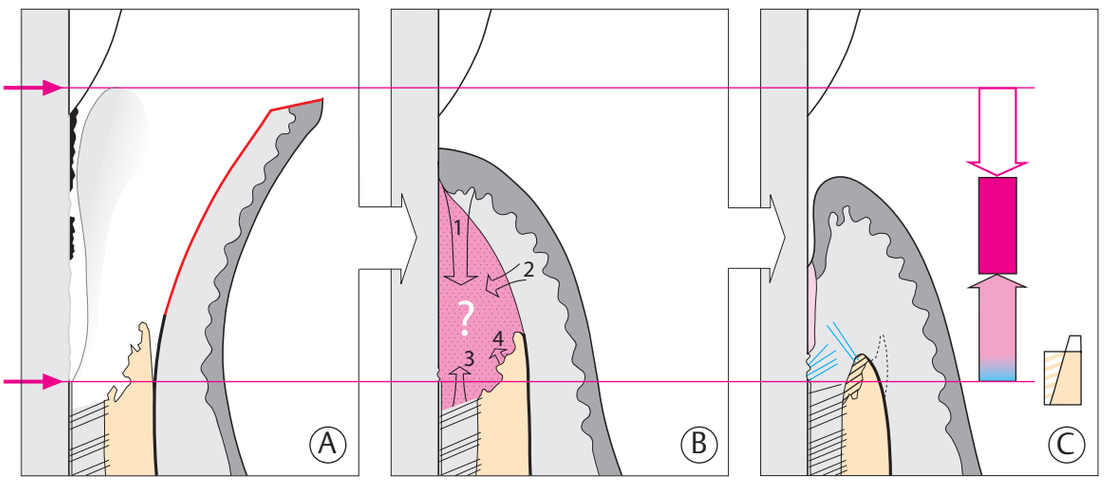
The classic GTR procedure involves only *physical* membrane barriers, without *biological* factors. Growth factors, proteins, adhesins etc. will surely soon be combined with the physical membrane techniques, and perhaps even replace them! Physical barriers that are *in situ* during the healing phase prevent epithelium proliferation apically, and thus prevent the formation of a long junctional epithelium.

781 Healing after "Classical" Treatment—"the Race Among the Tissues"

A Following flap reflection, the root surfaces are planed.

B Healing of the tissues occurs with varying degrees of rapidity (arrows **1–4**).

C *Reparative* healing with a long junctional epithelium (pink). At the fundus of the pocket, there may be some slight new formation of attachment (blue), and with marginal shrinkage of the gingiva (open arrow) a small residual pocket (red) remains.

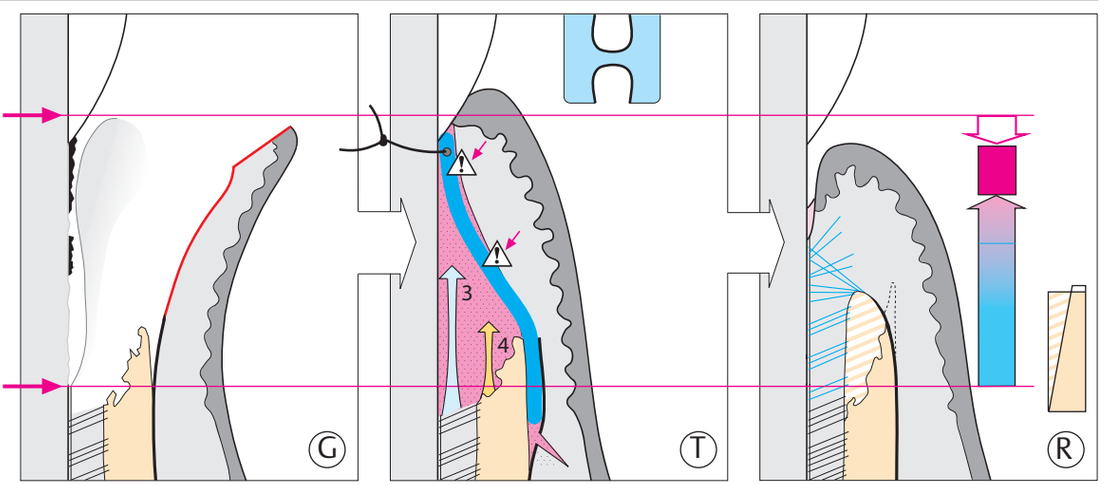


782 Healing after Guided Tissue Regeneration (GTR)

G Following flap reflection and root planing.

T The membrane (blue) separates oral epithelium and gingival connective tissue from the tooth surface, the periodontal ligament and bone. The ligament (**3**) and bone (**4**) can regenerate undisturbed.

R *Regenerative* healing: Subjacent to a short JE (pink), new cementum, periodontal ligament (blue) and bone (yellow) have formed.



More than 40 years ago, Björn (1961, 1965) demonstrated in his experimental work the prevention of rapid epithelium downgrowth; after removal of the clinical tooth crown and treatment of the periodontal defect, the remaining root was completely covered with mucosa, effectively eliminating epithelial proliferation and permitting undisturbed regeneration of the subjacent periodontal tissues. Ellegaard (1976) used the free gingival graft to cover osseous periodontal defects after they had been filled with autogenous bone. Both of these authors described effective inhibition of epithelium downgrowth, and demonstrated true periodontal regeneration.

It was only in 1982, however, that Nyman and co-workers first described the use of a Millipore filter as a barrier membrane to prevent epithelial downgrowth. It was then that the term "guided tissue regeneration" (GTR) was born, and found fertile soil in the treatment of periodontitis.

The commercial dental market reacted quickly, and offers today a wide variety of barriers (pp. 340–350), some of which are pre-formed, some that can be individually cut at chairside, and others that represent viscous initiation materials.

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Non-resorbable—Gore-Tex Periodontal Material

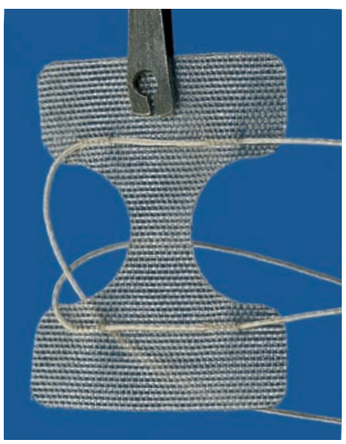




Barrier Membranes

783 Gore-Tex Periodontal Material (Gore Co.)
This membrane consists of polytetrafluoroethylene (ePTFE, Teflon). Gore-Tex is relatively stiff (*left*) and can therefore be positioned over most bony defects whether or not such defects are previously filled with bone or bone replacement materials. Gore-Tex is not resorbable and must be carefully removed in a second surgical procedure 4–6 weeks later.

Resorbable/Synthetic—Vicryl





784 Vicryl (Ethicon Co.)
This synthetic, resorbable membrane consists of fibers of polyglycolide and polylactide in the relationship of 9:1 (polyglactin 910). It is relatively soft, and may collapse into an osseous defect; it must therefore be used in combination with a defect-filling material. The membrane offers pre-selected, resorbable sutures (*left*), which, however, do not always adapt ideally to the individual intraoral situation.

Resorbable/Synthetic—Atrisorb





785 Atrisorb (Atrix Co.)
This material is commercially available as a gel consisting of poly-DL-lactic acid. It can be cut and fabricated, chairside, into an individually crafted membrane with the application of a saline infusion.

Atrisorb can also be used as a fluid material, directly from the "tube" via a canula (*left*), and applied *in situ*, using a moist cotton pellet for shaping, in anticipation of polymerization (p. 345).

Resorbable/Xenogenic—Bio-Gide





786 Bio-Gide—Perio-System (Geistlich Co.)
In contradistinction to the other synthetic barriers, this membrane is xenogenic, derived from porcine collagen. Upon contact with moisture, it becomes very soft and therefore can readily fill an osseous defect. Suturing is usually not necessary because the material is "sticky" and remains securely at its site. In selective cases, this membrane can be secured with resorbable "pins" (*left*).

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Selection of Membranes and Barriers

The following groups of membranes can be differentiated:

- Synthetic, non-resorbable
- Synthetic, resorbable
- Natural, biodegradable

If resorbable membranes are used, it is not necessary to perform a second surgical procedure to remove them; resorbable membranes are therefore preferred in *periodontitis therapy*. An array of membranes is available, but the ideal membrane "for all cases" has not yet been invented.

Long-term and stable space maintainers are necessary for "guided bone regeneration" (GBR, p. 513).

Requirements for the optimum membrane:

- Safety—no disease transmission
- Biocompatibility—non-toxic, non-immunogenic
- Adaptation to the root and bone surfaces—simple, secure
- Rigidity—no collapse into the osseous defect
- Permeability—important molecules, but no cells
- Tissue integration—immobility
- Space maintenance—reliable duration
- Biodegradation—controlled
- Additional mediators—antimicrobial, biostimulatory

787 Membranes for GTR and GBR Techniques

Synthetically produced, non-resorbable membranes

- The *GoreTex membranes* are seldom used today in regenerative periodontal surgery, but appear to be reconciled to "guided bone regeneration" (GBR), as the titanium-reinforced method of choice (gold standard).

Synthetically manufactured resorbable membranes

- Vicryl is a readily available material whose indication is dependent upon the osseous defect, with or without a filler material.
- Guidor is a material that was welcomed by practitioners, but which is no longer available today.
- Resolut is also produced by Gore Co. (Arizona). See Gore-Tex PM and Osseoquest.
- Atrisorb has a different approach as a finished membrane because after filling an osseous defect ("free flow") it provides an individually contoured membrane at chairside.
- The Osseoquest membrane is particularly indicated because of its 6-month resorption time; perfect for the GBR technique.

Natural, biodegradable membranes

- Bio-Gide and Biomend are easily manipulated xenogenic collagen materials deriving from porcine and bovine collagen.
- Alloderm is an allogenic material, consisting primarily of human skin collagen.

Membrane *	Substance/Origin	Characteristics	Comments
● Synthetic – non-resorbable			
GORETEX [1] Periodontal Material	GTPM – ePTFE Extended polytetrafluoroethylene	Good space maintainer Relatively stiff "Handling"?	Longest clinical experience; therefore, "STANDARD"
GORETEX-TI [2] Titanium-reinforced GTPM	Titanium-reinforced ePTFE, Ti-PTPE	Most stable space maintainer. Requires no filler material	Titanium must not be exposed! For recession, ridge augmentation
● Synthetic – resorbable/bioabsorbable			
VICRYL [3] Periodontal Mesh	"Polyglactin 910" = Polyglycolid/ Polylactid 9:1	Relatively soft Well adaptable Resorption: 4–12 weeks	Woven membrane. Four prefabricated shapes Resorbable sutures incl.
GUIDOR [4] Matrix Barrier	Poly-DL-lactid/ Poly-L-lactid/ + acetyltributylcitrate	Double-layered membrane Outer: large pores Inner: fine pores	Production/sales halted! Literature reports excellent results
RESOLUT [5] Regenerative Material	Poly-DL-lactid/ Co-glycolid	Resorption: 10 weeks Functional integrity Good space maintainer	Good tissue integration Separate suture material "Gore and Resolut" sutures
ATRISORB [6] Bioabsorbable Barrier	Poly-DL-lactid and solvent (N-methyl-2-pyrrolidone)	Soft, well-adaptable Interesting resorptive characteristics	Customized membrane fabrication with → "Barrier Kit"
ATRISORB FF [7] free flow/direct	see above	Adherent, completed ATRISORB membrane becomes the final membrane	For use only in combination with filler substances. "Innovation 2000"
OSSEOQUEST [8] Regenerative Membrane	Polylactid/ Polyglycolid/ Trimethylene carbonate	Reinforced pattern Function: 6 months cf. RESOLUT	Indicated for "guided bone regeneration" → implants, ridge augmentation
● Natural – biodegradable			
BIO-GIDE [9]	Xenogenic collagen Type I (100 %); porcine skin	Barrier function At least 6 weeks bioactive	Usually employed in combination with filler substances (e. g., Bio-Oss)
BIOMEND [10]	Xenogenic collagen Type I (100 %); bovine tendon	Resorption: 4–8 weeks. Collagen complexed with formaldehyde	Collagen network extends the resorption time
ALLODERM [11]	Allogenic skin matrix Type I collagen, human skin	Cell-free membrane. Skin matrix must be rehydrated	Still new. Possibly favorable results covering multiple areas of recession

*As of 2000

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GTR Using Non-resorbable Membranes

Step-by-Step Procedure with GoreTex Periodontal Material

This 22-year-old female presented with a chief complaint of esthetic problems in the anterior segment (protruded maxillary anterior segment with elongation of tooth 11); she sought a comprehensive appraisal of her dental situation. Since the age of 16, she had suffered under an insulin-dependent diabetes mellitus (Type 1). She also was allergic to nickel, Peruvian balsam, rosin and various aromatic substances. The patient was a non-smoker. At age 10, all of her second premolars had been extracted.

The clinical findings led to a diagnosis of aggressive periodontitis (Type III). The periodontal involvement was especially pronounced on teeth 16, 26 and 36, as well as on the anterior teeth; this is a clinical situation that would have been diagnosed as LJP—"juvenile periodontitis"—previously.

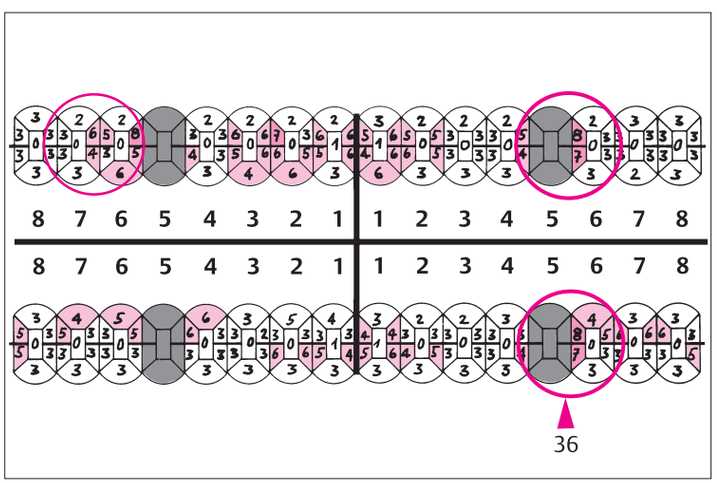
Initial findings:

PI: 30% BOP: 52%

Clinical picture, probing depths, tooth mobility and radiographic findings are depicted in the figures below.



788 Initial Findings
Even though dental plaque is not conspicuous, the gingivae appear somewhat swollen and erythematous; there is little evidence of stippling and the gingivae appear "glassy." Interdental hemorrhage occurred after probing.
This clinical photograph demonstrates the mild open bite which, during the course of periodontitis, may correlate with the obvious skeletal/dental anomalies, e. g., the pronounced protrusion of the maxillary anterior segment (cf. Fig. 799).



789 Pocket Probing Depths and Tooth Mobility (TM)
Indicated are localized deep pockets up to 8 mm (circled in red) on 3 of the 4 first permanent molars. Tooth mobility was elevated only on the maxillary and mandibular central incisors.

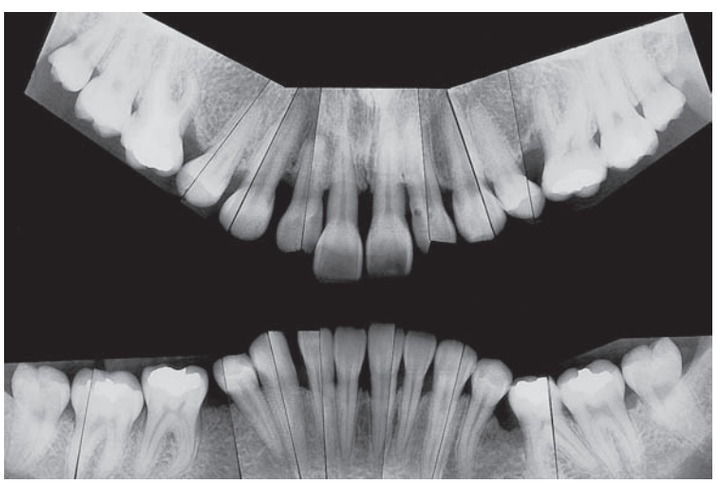
The surgical procedure will be depicted only on the indicated tooth 36 (red arrow).



Left: GoreTex interdental membrane, which was employed in the following surgical procedure.

GTR Membrane Technique

- Sulcular horizontal incision
- Vertical incision distal to the canine
- Mucoperiosteal flaps buccal and oral
- Root cleaning/planing
- (no filling of the defect)
- Positioning of the Gore membrane
- Coronal repositioning of flaps, after periosteal incision
- Secure coverage of the membrane
- Post-operative instructions to the patient



790 Radiographic Picture
The radiographic appearance confirms the clinical measurements. The attachment loss is pronounced on the maxillary anterior teeth and the mesial surfaces of teeth 16, 26 and 36. These teeth also exhibit a pronounced mesial tipping (second premolar was extracted before age 12).

Left: Following conventional initial therapy, the regenerative procedure was planned and a *surgical protocol* was prepared.

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GTR Procedure—Intra-Osseous Defect on the Mesial of Tooth 36

791 Initial Radiographic Appearance
Mesial to the severely tipped tooth 36 is a vertical bony defect extending to about $^1/_3$ of the root length. Coronal to the deepest site, one notes structures that indicate a multi-walled bony defect.

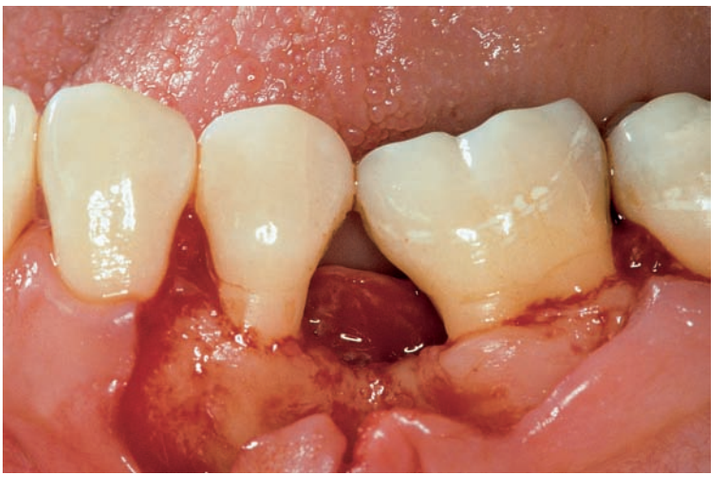
Right: Clinical situation following initial treatment, immediately before the surgical procedure.

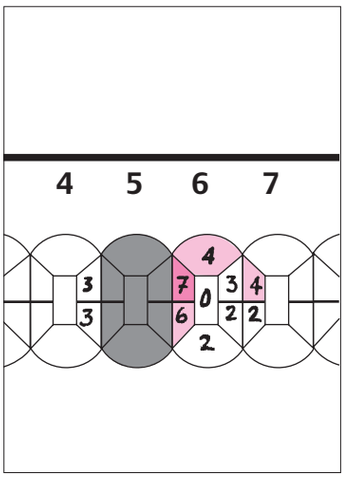




792 Flap Reflection and Root Planing
Following a sulcular horizontal incision extending from tooth 33 to tooth 37, and a paramedial vertical incision distobuccal from tooth 33, the mucoperiosteal flap is reflected. A lingual flap was also reflected.
The granulation tissue was removed to reveal the osseous defect, and root planing was possible optimally with direct vision.

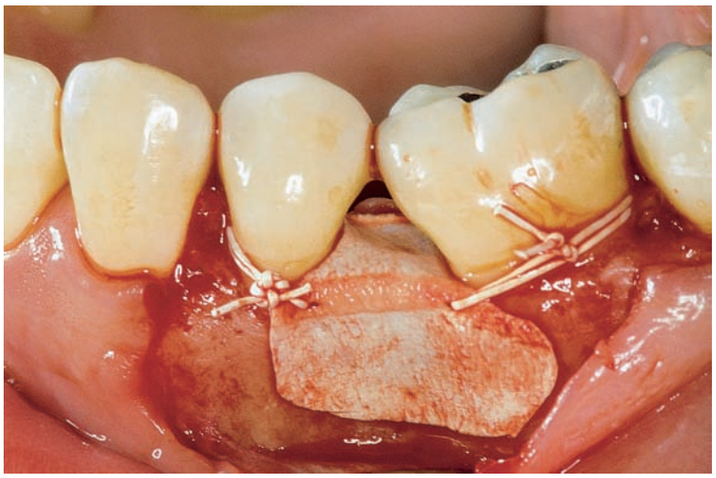
Right: Pocket probing depths after initial therapy, before surgery.

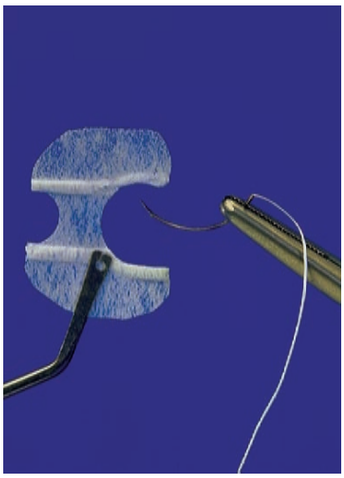




793 Membrane in Situ
The relatively stiff membrane, cut to fit the precise localization, was affixed using Gore sutures to teeth 34 and 36. The goal was to completely cover the osseous defect. Actual filling of the bony pocket (filling material; see p. 332) was a matter of discussion. The soft tissue flaps were "extended" in order to insure complete coverage of the membrane with soft tissue.

Right: GoreTex interdental membrane showing the forceps and needle-suture combination.





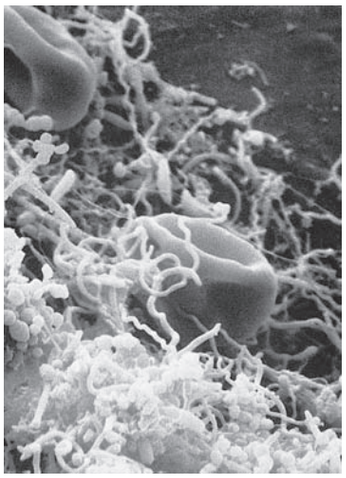
794 Sutured Surgical Site
By means of additional individual sutures, the wound was tightly closed, and the membrane was completely covered.

Until "re-entry" (removal of the membrane in a second procedure, Fig. 796) the patient did not perform any mechanical oral hygiene, but rinsed twice daily with chlorhexidine solution.



Courtesy *R. Oberholzer*

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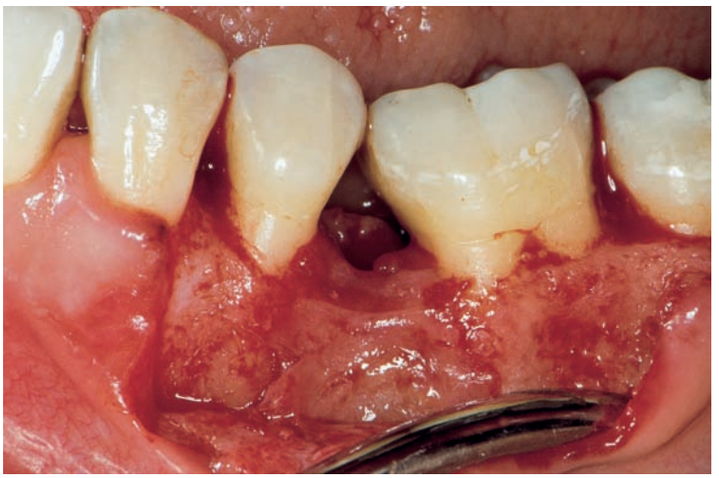




795 9 Tage nach Erstoperation – Nahtentfernung
Eine leichte Freilegung der Membran und Rötung des Marginalsaums sind sichtbar; eine Reaktion, die bei der Anwendung nicht resorbierbarer Membranen häufig zu beobachten ist. Zu diesem Zeitpunkt kommt es häufig zu einer bakteriellen Besiedelung der Membranaußenfläche.

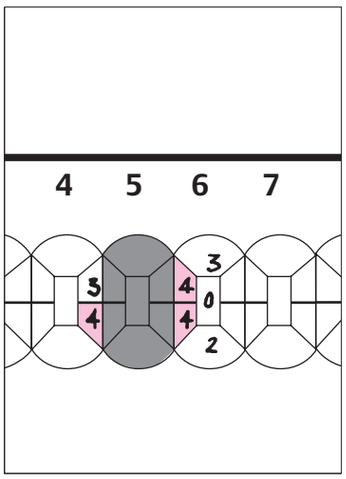
Links: Infizierte Außenfläche einer Gore-Membran im REM. Neben verschiedenen Mikroorganismen sind Erythrozyten sichtbar.

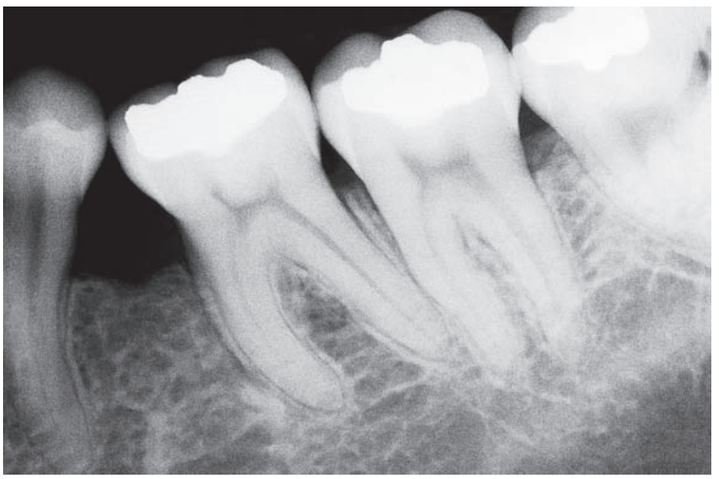




796 Zweitoperation nach 5 Wochen
Der ursprüngliche Lappen wird sehr vorsichtig „remobilisiert“ und die Haltenähte der Membran entfernt. Die kaum sichtbare Membran ist interdental leicht in den Defekt kollabiert, weil sie nicht durch „Füller“ gestützt war. Die Membran wird entfernt (Schonung der nichtmineralisierten Knochenmatrix), der Lappen reponiert und vernäht.

Links: Grazile Instrumente zur Lappenbildung, Lösen der Nähte und Entfernung der Membran.





797 Röntgenbefund 11 Monate nach GTR-Eingriff
Die Knochentasche mesial von Zahn 36 ist weitgehend ausgeheilt. Vgl. Röntgen-Anfangsbefunde (790/791).

Links: Die ursprüngliche Sondierungstiefe von 8 mm im Anfangsbefund wurde auf 4 mm reduziert, eine Sondierungstiefe, die einer Besiedelung mit parodontopathogenen Anaerobien (*Pg, Aa*) kaum mehr Vorschub leistet.



798 3 Jahre nach Operation
Die parodontale Situation ist konsolidiert. Leider ist der Sechser noch stärker nach mesial gekippt als im Anfangsbefund.
Im Rahmen der früheren kieferorthopädischen UK-Frontzahn-Behandlung wurde der Siebener zur Fixation der von frontal her laufenden Ligatur bebändert. Es kam zu einem Mesialdruck auf den Sechser. Eine indizierte, noch umfangreichere (bessere) kieferorthopädische Therapie wurde unterlassen, weil die Patientin nur die Frontsituation (Ästhetik) verbessert haben wollte.

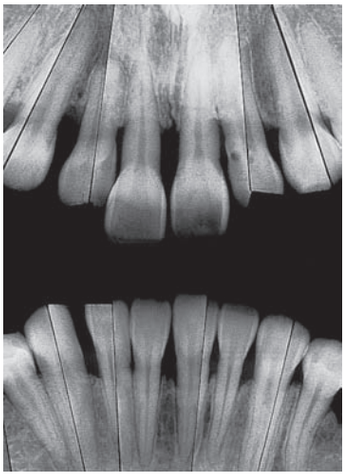
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Summary of the Case—Initial Findings

799 Maxillary Occlusal View
Gingival swelling and erythema. Severe protrusion of the maxillary anterior teeth, and formation of diastemata.

Right: The radiographic view depicts pronounced vertical defects in the maxillary anterior segment, extending to 50 % of the root length. Bone loss in the mandible is less pronounced.

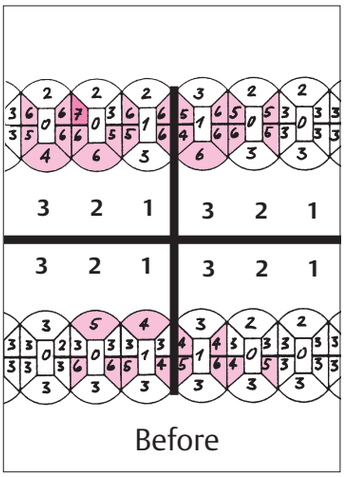




800 Anterior View of the Maxilla and Mandible
Gingival inflammation is clearly visible, but the severity of periodontitis cannot be ascertained by this clinical view. Of note is the fact that the anterior maxillary teeth appear to have "migrated."

Right: Pocket probing depths to 7 mm were measured.



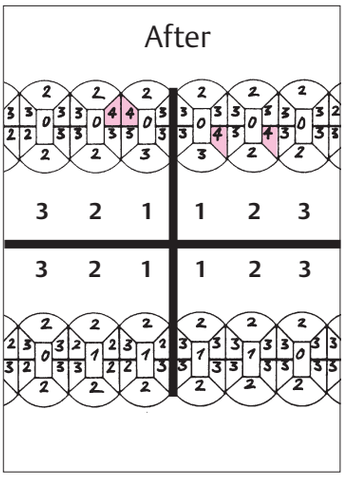


Clinical Findings 3 Years later

801 Anterior View
Healthy gingival conditions: salmon pink, slight gingival shrinkage. This positive result was achieved despite the risk factor of diabetes mellitus. The patient's compliance was very good. Because the patient's primary concern was improvement of esthetics, the patient was quite satisfied by the ultimate result.

Right: Probing depths remain at the physiologic level, max. 4 mm.

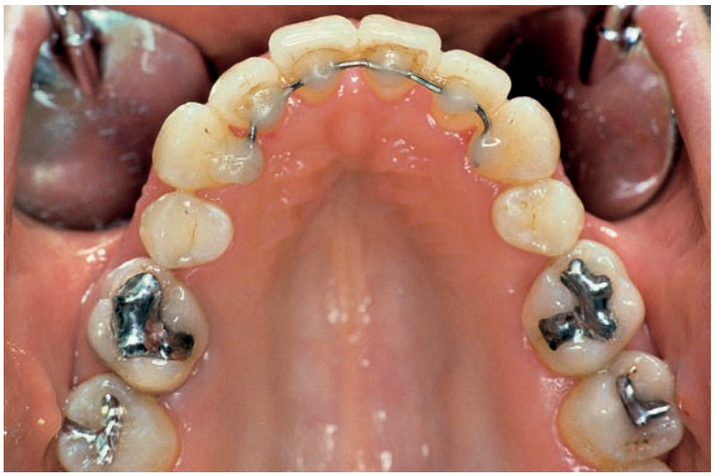


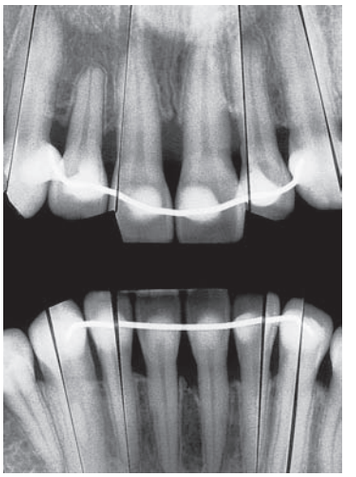


802 Occlusal View, Maxilla
Following successful orthodontic treatment, a retainer wire was affixed using composite resin. This rendered oral hygiene of the palatal surfaces more difficult, and led to mild gingivitis in this segment. The patient remained in a 4-month recall.

Right: The osseous situation completely consolidated; the interdental regions exhibited compact osseous structure.

Courtesy *R. Oberholzer*





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Individual Immediate Membrane—Atrisorb/Atrigel Technology

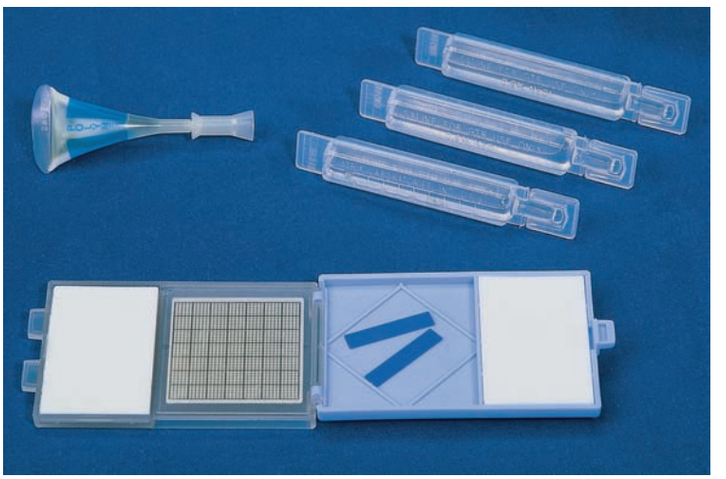
The Atrisorb membrane represents a practice-friendly further development in the arena of resorbable barriers.

The *Atrigel technology* (Atrix Co., Fort Collins, Colorado) provides the clinician the possibility to create polymers with variable resorption times. The ready-to-use *Atrisorb* is a powder-form polylactid in a liquid carrier substance (N-methyl-2-pyrrolidone/NMP).

The viscous product can be directly applied by the dentist (“*free flow*”). In the presence of moisture (NaCl 0.9 % or tissue fluids) a membrane is formed, which guarantees a barrier function for at least 20 weeks.

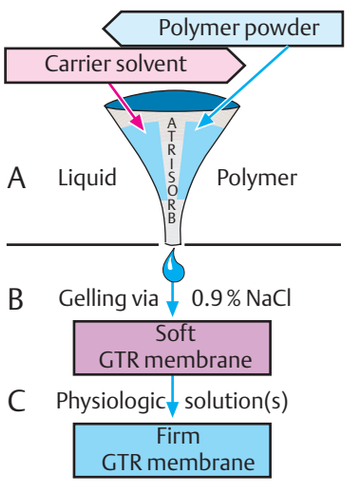
As an alternative, the kit provides a “waffle iron” that permits the chairside fabrication of a custom membrane. In the initial phase (moisture, temperature), within 5 minutes a flexible membrane can be custom-cut to any desired shape. After the custom-fabricated membrane is applied to the osseous defect, it continues the setting reaction. The custom membrane covers any filling material that was previously placed in the osseous defect.

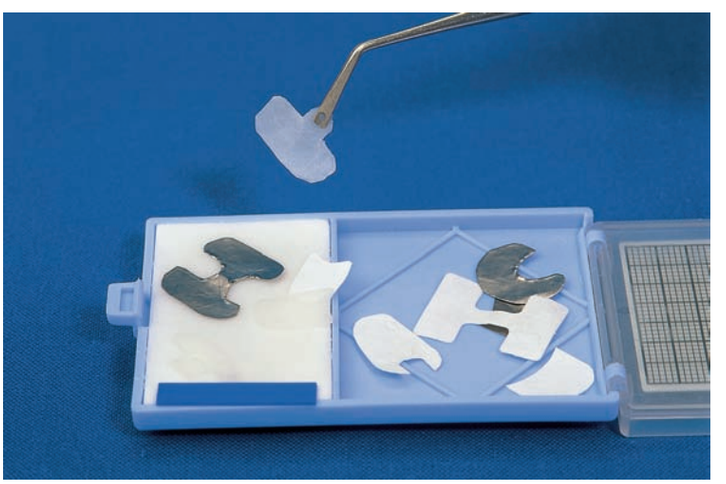




803 Atrisorb Kit
The commercial package contains the plastic-sealed kit, a closable device for preparing the membrane, NaCl solution, as well as the Atrisorb gel in a small, funnel-shaped receptacle.

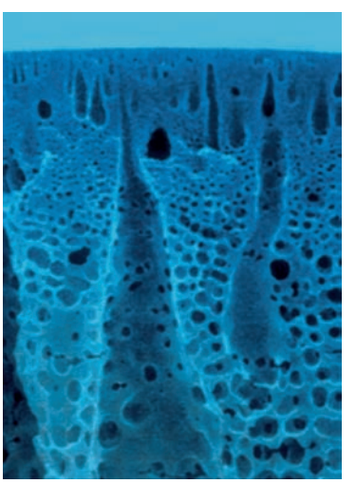
Left: A cannula can be affixed to the funnel to permit direct application of the material in the patient’s mouth (“*Atrisorb free flow*”).

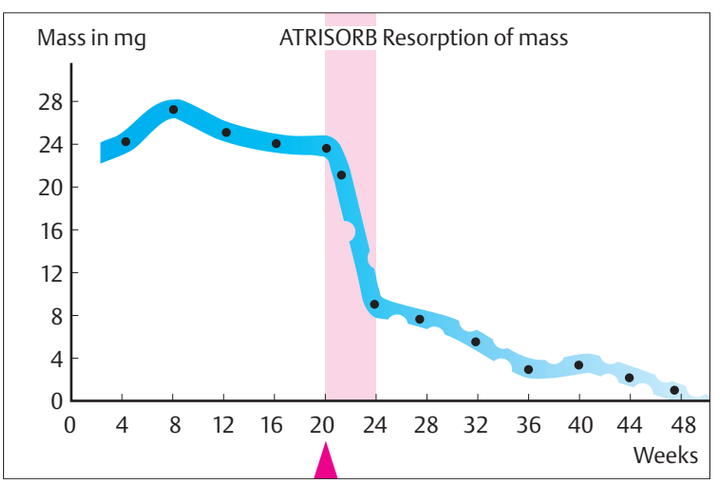




804 Preparing the Membrane
Both sides of the porous segments of the “waffle iron” are coated with saline solution. Excess fluid is removed, and the viscous Atrisorb is applied to the moist device, and the device is immediately closed. Space maintainers (blue strips) guarantee an even thickness of the membrane. After 4 minutes 30 seconds, the device is opened and the soft membrane is cut to fit the defect.

Left: Schematic depiction of membrane fabrication.





805 Bioresorption—Functional Longevity of the Barrier
An Atrisorb membrane is functional for ca. 20 weeks. Thereafter it is relatively quickly metabolized. The primary metabolite is lactic acid, which has an antimicrobial effect.

Left: Cross-section through an Atrisorb membrane. The material exhibits a certain porosity, but cannot be penetrated/perforated by connective tissues cells or epithelial cells from the soft tissue flap (SEM; *Atrix Co.*).

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GTR with Membrane and Filler—“Atrisorb Free Flow,” “Bio-Oss Collagen”

The Atrisorb product can be used clinically in two different ways: It can be formed into a membrane and then cut to fit the periodontal defect, or it can be applied directly in fluid form (“free flow”). Bone or a bone replacement material must prevent the Atrisorb from flowing *into* the osseous defect. The viscous Atrisorb is applied onto the filling material using a special cannula. Moist pellets are used to adapt the still viscous Atrisorb onto the adjacent root surfaces and onto the filling material. The liquids elicit hardening of the membrane in a few minutes.

This 67-year-old male is systemically healthy and a nonsmoker. The patient presented with generalized chronic periodontitis (Type II) with localized, very deep osseous defects. The pronounced areas of dental abrasion indicate a long-standing occlusal parafunction.

Findings around teeth 32 and 33 following initial therapy:

PI: 12 % BOP: 12 %

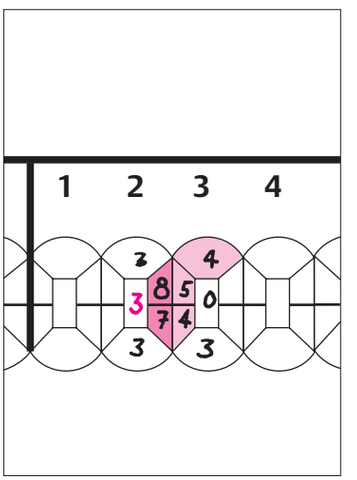
Probing depths, tooth mobility and radiographic findings are depicted in the figures.

806 Initial Clinical View—Osseous Defect between Teeth 32 and 33

Following initial therapy, neither plaque nor BOP are in evidence. The gingiva is pale pink in color and exhibits healthy shrinkage. A deep pocket persists on the distal surface of tooth 32. Chlorhexidine rinsing has stained the exposed root surface.

Right: Pocket probing depths to 8 mm were recorded. The tooth mobility of tooth 32 is highly elevated (TM: 3; p. 174).





807 Clinical View of the Bony Pocket Distal to Tooth 32

Facial and oral mucoperiosteal flaps were reflected from tooth 41–34, without vertical releasing incisions.

The granulation tissue was removed and the root surfaces were cleaned and planed. A deep and narrow bony pocket was clinically obvious.

Right: The radiograph demonstrates that the distal surface of tooth 32 has lost ca. $^{2}/_{3}$ of its alveolar bony support.

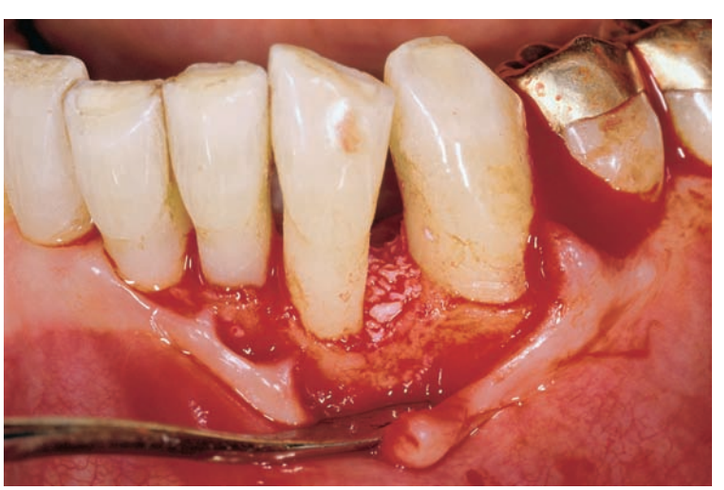




808 Filled Osseous Defect

The bony pocket, filled with blood, is filled to the highest level of the approximal bone on tooth 33 with Bio-Oss collagen (xenogenic implant material).

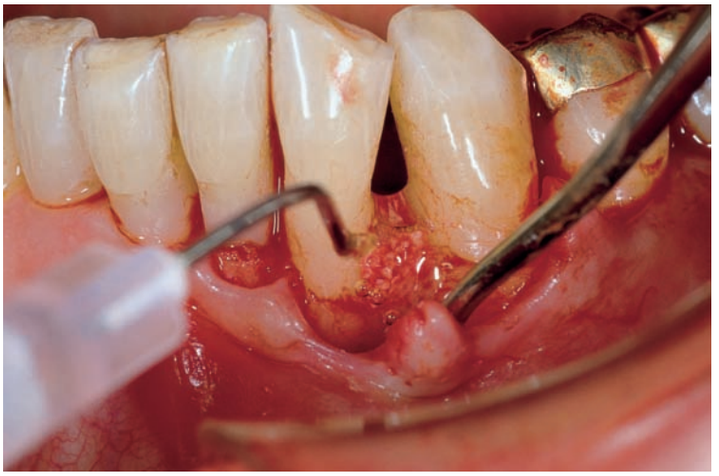
Right: The Bio-Oss collagen block, bovine trabebular granulate (hydroxyapatite/HA) plus 10 % porcine collagen is softened in Ringer saline solution and reduced to small chips.





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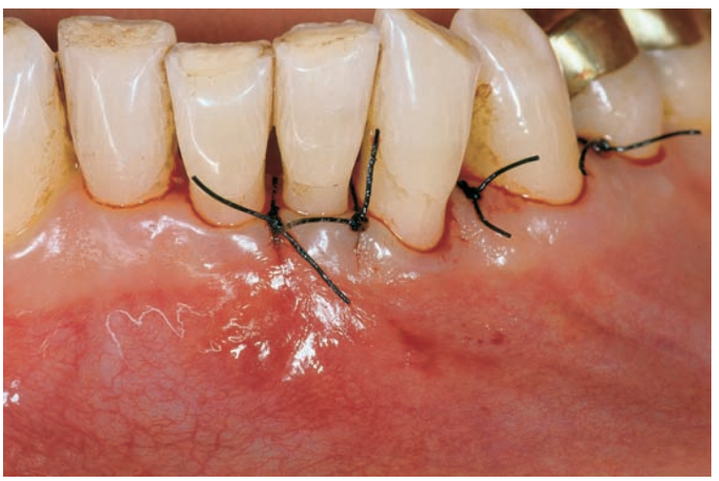


809 Application of the Fluid Membrane Material
Atrisorb-Direct fluid is applied drop-wise onto the filling material and the adjacent bone. The setting reaction is initiated by blood, tissue fluid or saline solution. Adequate rigidity is achieved in about 5 minutes. During this period of time, there should be no manipulation of the wound.

Left: Plastic tube with Atrisorb and a mounted cannula.



810 Definitively Adapted Atrisorb-Direct
The material was shaped using a moist, sterile cotton pellet. It now is perfectly adapted onto the Bio-Oss and the root surfaces. It is not necessary to stabilize the membrane with sutures.



811 Wound Closure
An incision is made through the periosteum to create a facial split-thickness flap, and the wound is then closed using interrupted sutures.

It is important that the suture needle not contact or displace the membrane!





812 Fifteen Months Post-Operatively
In comparison to the initial situation (Fig. 806) no significant gingival shrinkage occurred. Probing depths of only 3–4 mm remain, indicating substantial healing of the defect (attachment gain?). The brown staining resulted from CHX rinsing.

Left: Compared to the original radiograph (Fig. 807, right) pronounced new bone formation distal of tooth 32 can be observed.

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GTR with Filling Material and a Membrane—"Bio-Gide" and "Bio-Oss Collagen"

In contrast to the previously described pure trabecular bone filling material, the Bio-Oss material consists of granular trabecular bone (bovine; 90%) plus collagen (porcine; 10%) in *block form* as a filling material for periodontal osseous defects. The commercially available product provides a Bio-Gide membrane that can be cut pre-surgically using the templates provided (Fig. 816).

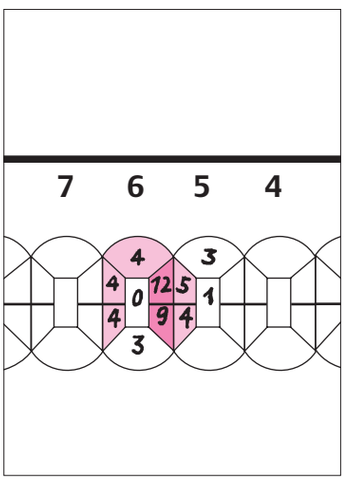
The 39-year-old male is for the most part periodontally healthy. The localized deep osseous defect on the mesial of tooth 46 could not be clearly defined etiologically. The defect may have arisen following extraction of the second premolar; it is also possible that when the composite MOD restoration was placed in the first molar, the liquid bonding material entered the sulcus and led eventually to the localized bony defect. This danger is often underestimated (virtually invisible bonding material).

Following initial therapy, very little plaque was present in the area of the defect, but bleeding on probing persisted. Probing depths, tooth mobility and bone loss are depicted in the figures.

813 Quadrant 4 after Initial Therapy
There is virtually no stippling, the gingivae present a "glassy" appearance, and there was bleeding on probing on the mesial aspect of the tipped first molar. The old, relatively large composite restoration (with radiographic contrast) had probably been in place for many years. Following completion of periodontal therapy, it will be replaced with an inlay.

Right: The probing depth on the mesial of tooth 46 was 12 mm.





814 Radiographic View
Only on the mesial surface of tooth 46 does one note a pronounced vertical defect. It was suspected that a 3-wall bony pocket was present at the depth of the lesion. The defect extends far beyond the midpoint of the root.

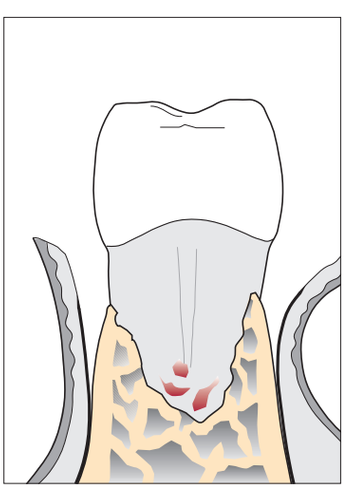


815 Exposing the Defect
After performing a marginal incision (without vertical releasing incisions) mucoperiosteal flaps were reflected buccally and lingually, the granulation tissue was removed and the root surfaces cleaned and planed. Before filling the defect, bleeding must be provoked by perforating the osseous crater walls (coagulum, nutrition).

Right: Diagram of the localized bony pocket.

Courtesy *M. Marxer*





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816 Bio-Oss Perio-System—Combination Packaging
The package contains a 16×22 mm Bio-Gide membrane, a Bio-Oss block (granulated trabecular bone and collagen 90:10 %), as well as templates for the membrane.

Left: Section through a porcine collagen membrane. The rough side (left in the picture) is applied to the tooth or osseous aspect.

Modified from *Geistlich Biomaterials Co.*

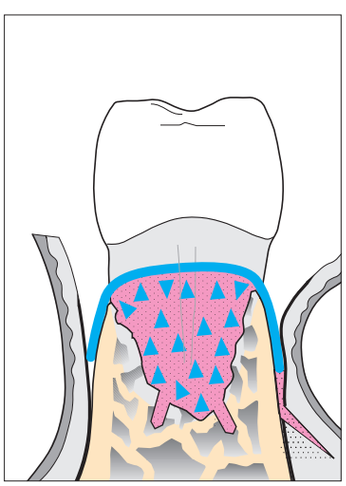


817 Filled Defect
The defect is completely filled with moistened granular trabecular bone/collagen, up to the cementoenamel junction. It is also possible to mix tetracycline powder into the filling material to provide some antimicrobial activity. Before applying the membrane, the filling material should be very well saturated with blood (more than shown in this clinical view).

Note: Gross overfilling of the defect is counterproductive!



818 Applied Membrane
When moistened, the membrane becomes slightly sticky, and is used to completely cover the filled defect; no sutures are employed. Fixation of the membrane with sutures or resorbable pins is only indicated if there is a danger that mechanical influences might lead to dislodging or abrading the coagulum on the root surface.





819 Wound Closure
The wound and the membrane are securely covered with the mucoperiosteal flaps, and interdental sutures are placed.
The patient is instructed to rinse for the next 6–8 weeks with chlorhexidine solution, or to apply CHX gel (1 %). Mechanical oral hygiene in the surgical site must not be reinstated too early.

Left: Diagram of the filled osseous defect, covered by a membrane (blue). Note the incision in the periosteum: tension-free flaps!

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Summary of the Clinical Case "Xenogenic Implant/Collagen Membrane"

The localized defect on the mesial of tooth 46 was successfully treated in the 39-year-old male. The 12 mm pocket was reduced to 4 mm. The intra-alveolar portion of the osseous defect regenerated completely (Fig. 820, right). Nevertheless, these clinical and radiographic results do not permit the conclusion that new cementum and new periodontal ligament were also formed in addition to the new bone.

Clinical Study: "Xenogenic Implant/Collagen Membrane"

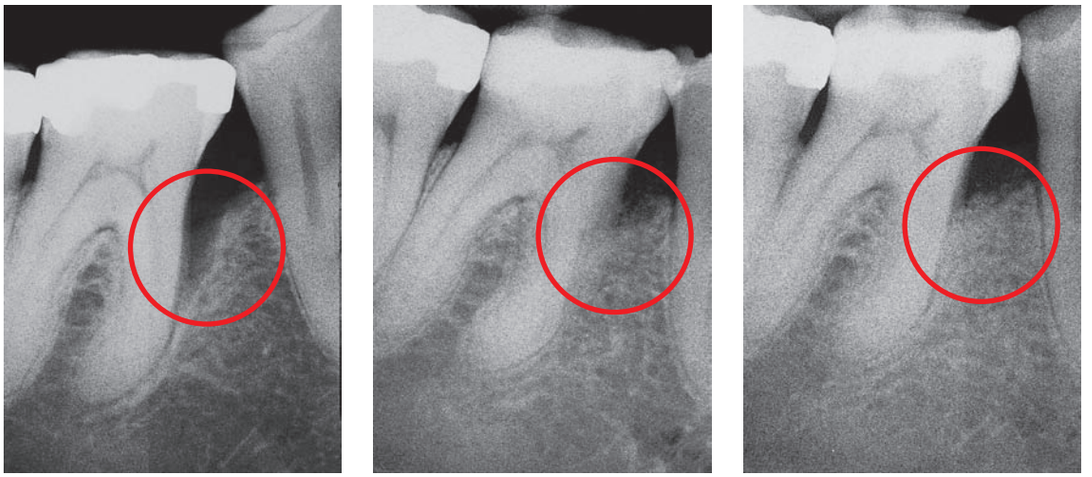
The treatment methods described in the previous two cases were studied extensively in 14 patients with a total of 27 periodontal osseous defects (Zitzmann et al. 2002). At the initiation of the study, the osseous defects were at least 5 mm deep, and attachment loss was 6 mm. Follow-up examinations were performed over the course of two years. The results are depicted in Fig. 822.

820 Clinical Case—Bony Defect Filling: Radiographs before Surgery, Immediately Post-surgical and Two Years Later
Left: The localized 3-wall defect extends to about the midpoint of the root length.

Middle: Defect immediately after "filling" with Bio-Oss collagen.

Right: Defect two years later.

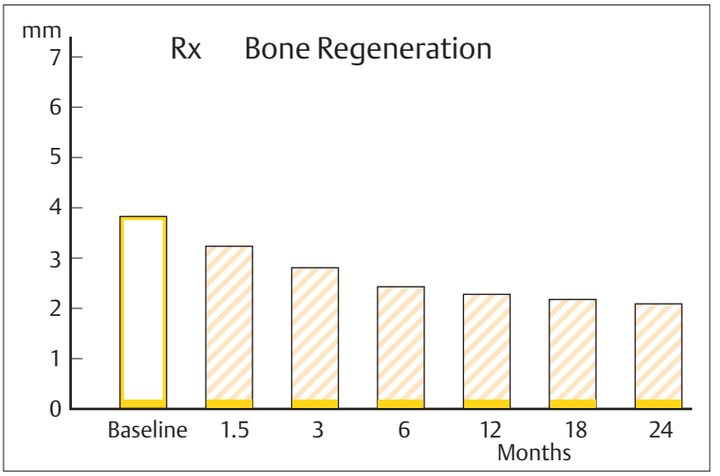
Courtesy *M. Marxer*

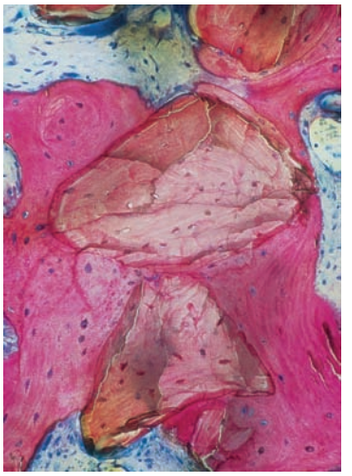


Clinical Study by Zitzmann et al. 2002

821 Radiographically Visible Bone Fill
If a bony pocket is filled using a xenogenic filling material covered with a collagen membrane, radiographs taken two years later reveal over 40 % loss of the filling material.

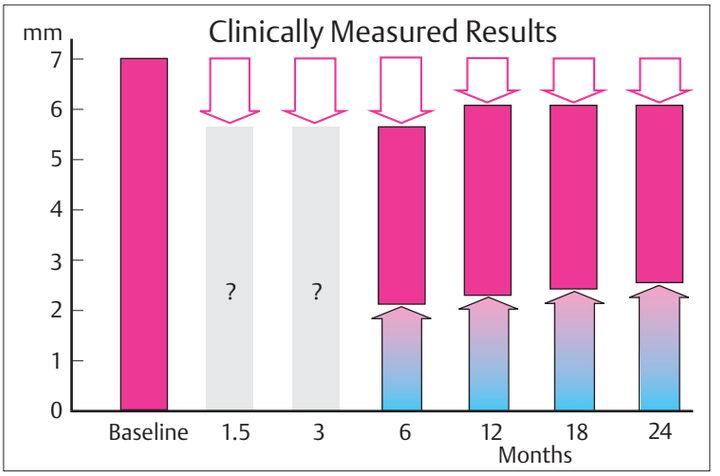
Right: New bone can be seen around the implanted, non-resorbed Bio-Oss particle.

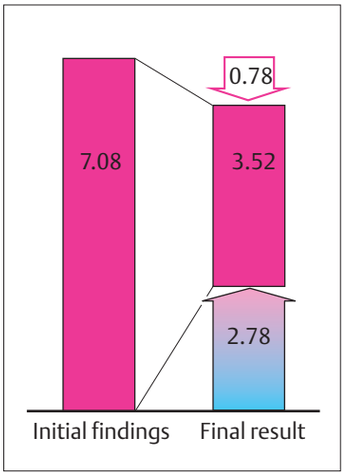




822 Probing Depths, Clinical Attachment Gain and Recession of the Gingival Margin
Surgical sites should not be probed for at least 6 months! (See the question marks).
Probings 6–24 months after surgery demonstrate significantly reduced probing depths (ca. 3.5 mm or 50 %). The clinical attachment gain was about 2.8 mm, and recession ca. 1 mm.

Right: Overview of the results.
- Initial findings
- Final result





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Regeneration Using Proteins, Growth Factors and Differentiation Factors

The complete regeneration of periodontal tissues – including cementum, periodontal ligament, bone and gingiva – requires the interplay among pluripotent cells, extracellular matrix and matrix proteins, systemic hormones as well as growth and differentiation factors (local signal molecules).

Growth factors are mitogenic signal molecules (polypeptides), which can influence in a variety of ways the local growth (proliferation) and function of many different cell types.

Differentiation factors determine the phenotype of immature cells: Pluripotent "stem cells" develop under the influence of differentiation factors into functional, mature cells. Osteoblasts develop from undifferentiated mesenchymal cells (p. 205) under the influence bone morphogenetic proteins (BMP).

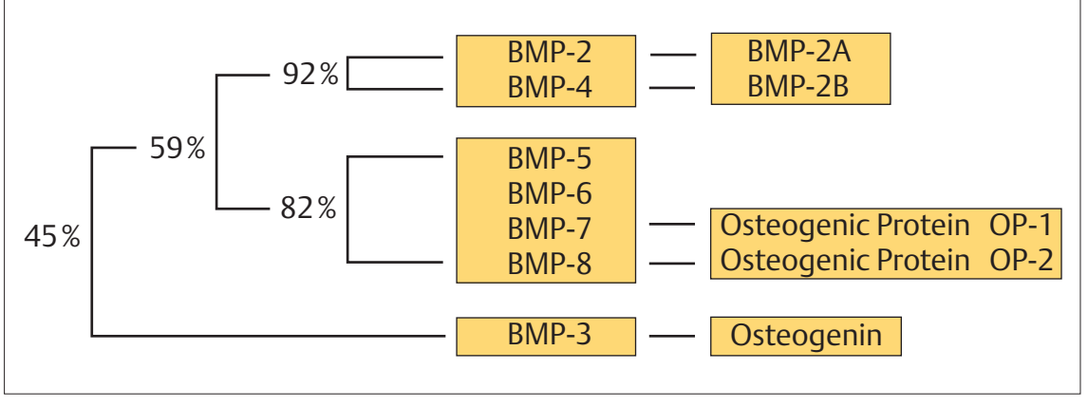
Such factors are being used more and more often to accelerate and improve periodontal healing and post-implant healing, using various carrier systems (bone or collagen fillers).

Effects of Important Growth Factors / Signal Molecules

Factor		Fibroblast proliferation	Osteoblast proliferation	Synthesis of matrix proteins	Mesenchymal cell differentiation	Vascularization
PDGF	Platelet-Derived Growth Factor (GF)	++	++	–	–	+*
IGF	Insulin-like GF	+	++	++	–	–
TGFβ	Transforming GF beta	±	±	++	–	++*
BMP	Bone Morphogenetic Proteins -2, -4, -7	–	±	±	++	++*
FGF	Fibroblast GF	++	++	–	–	++

823 Activity of Growth Factors (Modified from Cochran & Wozney 1999, Müller 2001)
Signal molecules exert their effects strictly locally (autocrine/self-stimulating and paracrine/to neighboring cells). This means that the target cells must be nearby.
Table: Activity/effect
++ Highly elevated
+ Elevated
± Possibly elevated
– Without effect, or negative effect
* Indirect effect

Homology of hBMP – "Human Bone Morphogenetic Proteins"



824 Bone Morphogenetic Proteins (BMP)
The group of BMP molecules are categorized within the large family of transforming growth factors (TGF). The table shows the various relationships within the BMP family.
Some BMPs have already been used in orthopedic surgery. Of those molecules studied, BMP-2 and –4, as well as BMP-7 and –8 have shown the most efficacy for bone *induction*.

Factors and Proteins in Dental Practice

The following certificated mediator systems have been used to date in practice:

- "Platelet-rich plasma" (PRP) in various types of applications (Marx 2001). The concentrated plasma fraction from a patient's own blood is rich in thrombocytes with large quantities of platelet-derived growth factor (PDPF) and transforming growth factor (TGFβ).
- P-15 (synthetic polypeptide), administered in bovine filler granulate.
- Emdogain (Straumann): Porcine amelogenins are a fraction of enamel matrix proteins ("enamel matrix derivatives," EMD), which enhance cementum formation in the proper environment: Emdogain initiates a process upon human root dentin that mimics the processes of natural tooth development (mimicry; Zetterström et al. 1997). The use of Emdogain in regenerative therapy is based upon the application of this knowledge. The histologic demonstration of newly formed root cementum and periodontal ligament (cf. p. 200!) along a previously diseased root surface supports the appropriateness of these considerations.

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Flap Surgery Combined with Enamel Matrix Proteins—Emdogain

Surgical Procedure—Step-by-Step

The refrigerated, ready-to-use Emdogain is brought to room temperature 15 minutes before use. The goal when Emdogain was being developed was to achieve the very good regeneration results that had been achieved with the very technique sensitive membrane method, in combination with simple access flaps (MWF; Hammarström 1997, Heijl et al. 1997). In the original clinical research (Fig. 826, right) the Emdogain provided no support for the soft tissue flaps; therefore, today's product also contains supportive filler particles for stabilization, especially in expansive defects. This was anticipated in the case depicted below.

In this 56-year-old female, following initial therapy (Phases 1 and 2) a 9 mm pocket persisted on the mesial surface of abutment tooth 43, and the radiograph revealed a bony defect of at least 6 mm. The patient was systemically healthy and wanted to maintain her "old bridge."

Conditions following initial therapy of the entire dentition:
PI: 16 % BOP: 18 % TM: 0

Flap Procedures Using EMP Derivatives/Emdogain

825 Before Initial Therapy
Two-wall, 9 mm bony pocket on the mesial of tooth 43.
When flaps are reflected, no direct injection of anesthetic solution should be used into the papillae or the surgical site, because blood flow is critical to success and must be initiated quickly following the surgical procedure.

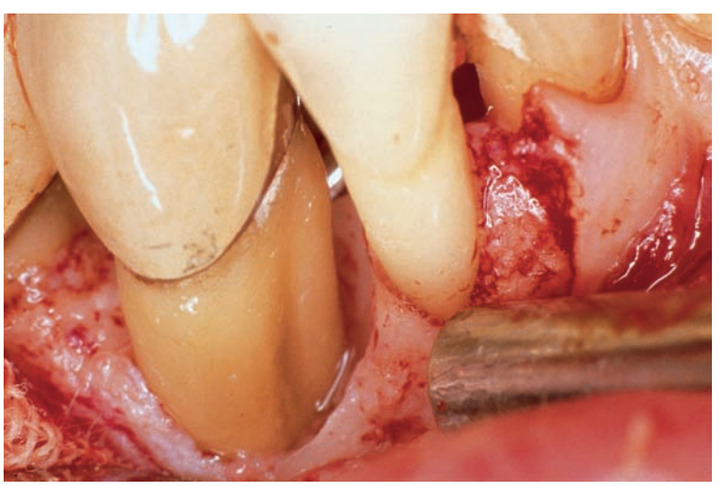
Right: Radiograph of tooth 43.





826 Clinical View Following Flap Reflection
Following *sulcular* and if necessary *vertical* incisions, full mucoperiosteal flaps are reflected on the buccal aspect.
The bone and the pocket are completely curetted and the root surface is planed and debrided with direct vision.

Right: Protocol for a flap procedure using Emdogain.

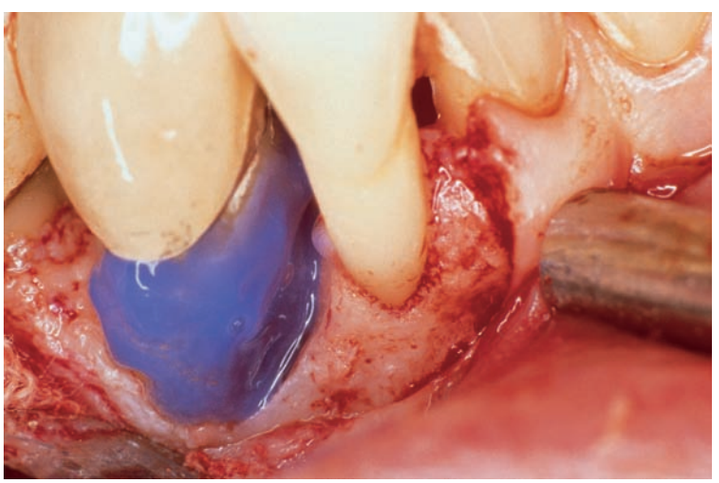


Flaps with Emdogain—Original Surgical Protocol
- Anesthesia—*relatively* blood-free
- Incisions, flap reflection (full flaps); incision of the periosteum
- Curettage of the bony defect
- Debridement of root surfaces
- Two minute application of EDTA 24 %
- Copious rinsing (NaCl 0.9 %)
- Application of Emdogain onto the blood-free root surface; filling of the defect
- Tightening the previously placed suture
- Placement of additional sutures

827 Biomodification of the Root Surface
An alternative is to "biomodify" the root surface by application of phosphoric acid (blue) and etching for 10 seconds. This significantly shortens the long working time necessary with EDTA (2 min).

Right: Biomodification factors:
- **Citric acid** **ca. 30 %** pH1
- **Phosphoric acid** **37 %** pH3
- **EDTA** **24 %** pH 7

Courtesy *M. Imoberdorf*





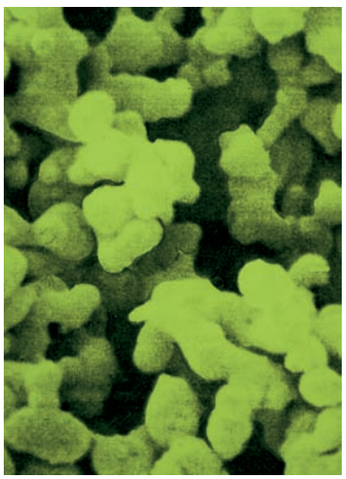
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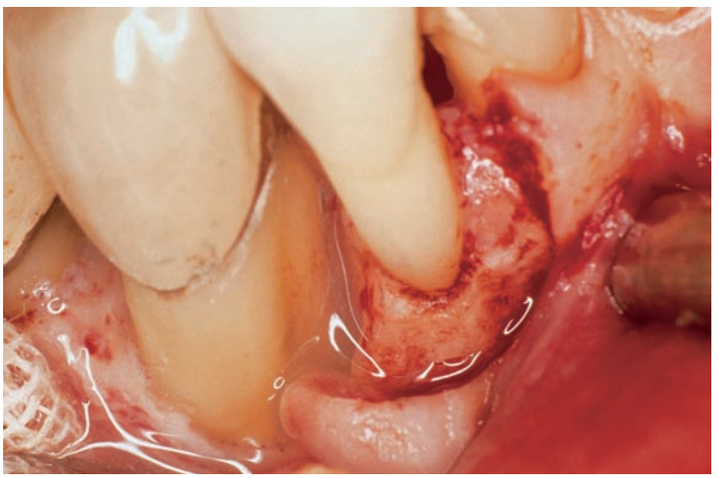




828 Emdogain-Gel (Straumann)
Ready-to-use fraction of enamel matrix proteins. The acid solution and the solvent propylene glycolic acid-alginate (pH 2.6) maintains the gel soluble and it can be applied with a syringe and cannula. Warming and a neutral pH value permit the gel to precipitate. Quantities available:
- **0.3 ml**
- **0.7 ml**
- **0.7 ml/TS: with filling material**

Left: 0.3 ml Emdogain is sufficient to fill 2–3 average bony defects.

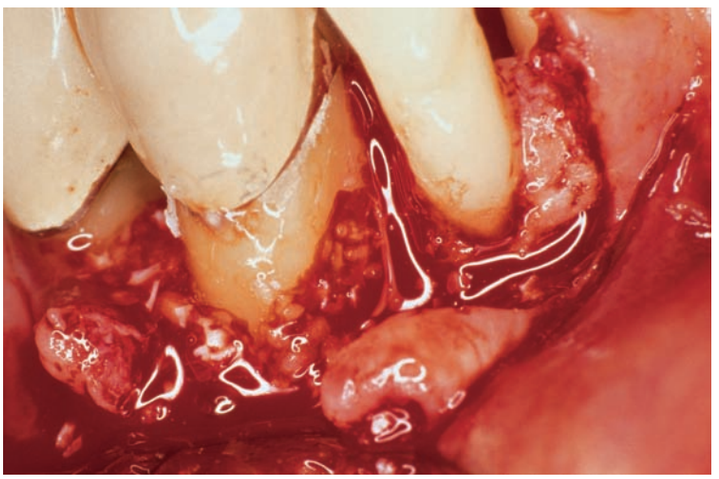




829 Application of the Gel—Filled Bony Pocket
Beginning in the apical depth, the blood-free defect is slightly overfilled. Immediately before application, the defect is copiously rinsed with NaCl solution. Its neutral pH value enhances rapid precipitation of the gel (before blood re-enters the defect).

Left: Precipitated globular proteins of Emdogain.

Courtesy *Biora Co.*, section from advertising brochure



830 Additional Stabilization of the Defect Using Filler Materials
In this case the defect was filled *afterwords* with bovine trabecular bone particles. This prevents the repositioned tissue flaps from collapsing into the defect, and also serves to stabilize the blood coagulum.

Finally the flaps are repositioned over the surgical site and affixed with monofilament, soft suture material (e. g., GoreTex).





831 Suturing
Thanks to the periosteal incisions, the flaps completely cover the defect, without tension. The sutures are left in place for 14 days in order to guarantee the necessary time for the initial phase of healing and to prevent dislodging the coagulum from the root surface. A dressing can be used as desired.

Left: Radiographic view six months post-surgical

Courtesy *M. Imoberdorf*

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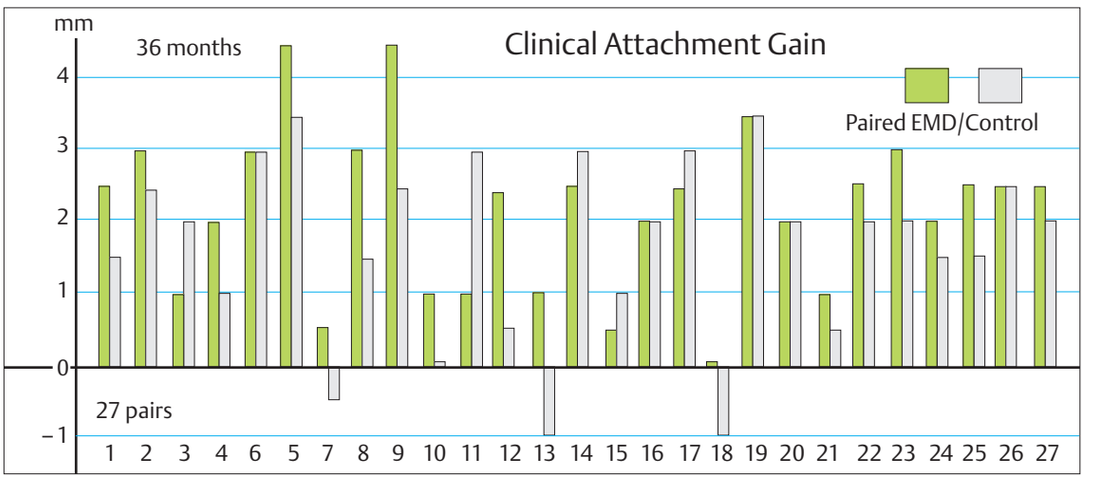
Regeneration Using Emdogain

Regeneration in the periodontal area occurs under extremely difficult circumstances (Healing, p. 205). The central problem remains the creation of a syndesmotic *de novo* connection between hard, cell-free tooth root surfaces and the surrounding tissues (cementum, periodontal ligament, bone and connective tissue).

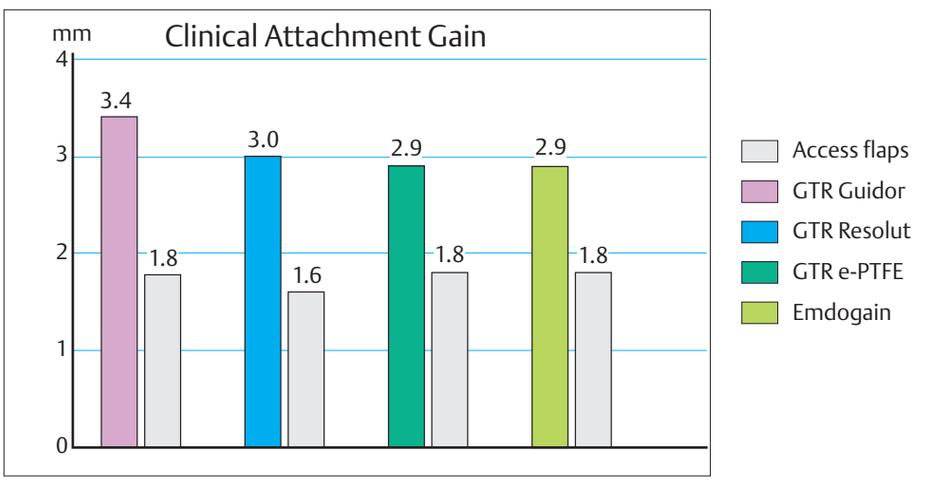
The "mechanistic" regeneration methods (filling materials, membranes) only partially fulfill the expectations, and usually achieve 50–60 %, but not *complete* regeneration.

"Biological" or "biologically supported" regeneration methods such as the use of growth and differentiation factors and morphogenetic proteins (e. g., BMP), and also "tissue engineering," will surely find a bright future, but these methods are not yet ready for routine practice.

The Emdogain technique, developed in 1997, results in excellent but not always similar results (Fig. 832). One questions whether all factors such as the patient, the osseous defects and other variables were taken into consideration during these clinical studies.



832 Emdogain Studies—Clinical Attachment Gain Following Flap Surgery (MWF) with or without Use of Emdogain (Heijl et al. 1997)
"Split mouth" study of 27 patients; paired results. Immediately apparent are the inconsistencies in results of both methods: The results do not appear to be predictable for the individual case. It varies from significant attachment gain (cases 5, 9) to attachment loss (case 18). The "average" result, however, indicates dramatic improvement of the surgical result.



833 Emdogain—Comparison with Additional Regenerative Methods (GTR) (Pontoriero et al. 1999)
Emdogain in combination with the modified Widman flap (MWF) was undertaken to *simplify the surgical technique*, and to achieve the same degree of tissue regeneration that results from the much more difficult and technically sensitive membrane techniques (GTR; p. 338).
The clinical attachment gain depicted in this graft indicates that the effort was successful.

Regenerative Therapy—*Quo Vadis?*

Regenerative treatment methods based on purely mechanical means (exclusion of epithelial downgrowth etc.) have become well established in dentistry and have proved effective especially in the disciplines of oral surgery, periodontology and implantology.
In a closed surgical situation, the desired results are quite predicable (ridge augmentation; "guided bone regeneration," GBR; sinus lift procedures etc.). In open, contaminated wounds (e. g., periodontal pocket surgery), the results are often more modest, sometimes not even surpassing the purely open debridement methods.

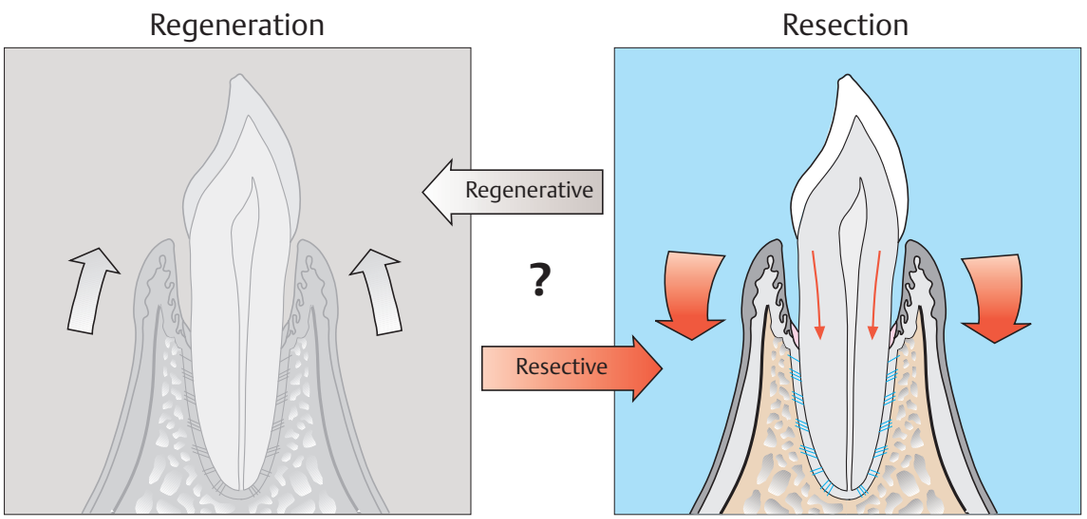
But today there is new knowledge concerning the stabilization of the coagulum and the role of adhesin molecules and their ligands in the regeneration of all periodontal tissues. Regenerative surgery—with the exception of Emdogain use—is indicated for single-rooted teeth and not entire sextants! The question remains open concerning the "handling" of persistent residual pockets postoperatively.

Today's question remains: Should the skeptical practitioner once again (or still) depend upon the older resective methods despite their acknowledged disadvantages?
(See the next chapter.)

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Resective Methods
Pocket Elimination—Osseous Surgery

The ultimate goal of periodontitis therapy is the regeneration of *all* disease-elicited defects. Latin: *Restitutio ad integrum*. Unfortunately, our profession still remains far away from this utopic but noble goal, even though various *regenerative* methods (p. 323) provide at least *local* healing, and elicit some new formation of destroyed periodontal structures. However, as long as the results of regenerative treatment methods remain unpredictable, the *resective* surgical methods will continue to be unavoidable, particularly in certain *generalized* cases that have not yet progressed extensively. The goal of pocket elimination surgery is healing with a sulcus depth of 0–2 mm, i. e., the achievement of overall healthy periodontal conditions, even though the soft tissue is positioned at a somewhat more apical niveau; in such cases, the tendency toward recurrence is low if plaque control is adequate; this is especially important if fixed dental replacements (crowns, bridgework) are planned.



834 Resection Versus Regeneration
Deep periodontal pockets, with their microflora defended by the biofilm represent the *primary risk* for the progression of periodontitis (gram-negative anaerobes). Such periodontal pockets can be eliminated or reduced by the regenerative or resective methods available today, and both methods are associated with advantages and disadvantages.
The indication for one or another surgical procedure will vary from case to case.

Indications for Resective Periodontal Surgery
- "Generalized periodontitis" with pronounced and irregular bone loss on several teeth to be treated simultaneously
- Necessary osteoplasty/ostectomy with deep intra-alveolar defects exhibiting sharp bony margins or exostoses
- Tissue architecture that prevents adequate plaque control
- Hemisections and resections of individual molars or roots
- Abutment teeth for planned prosthetic replacements

Contraindications
- In *every* case where closed or regenerative therapy can provide sufficient results, and where esthetics is important

Advantages
- Direct vision, improved access to all root surfaces and to furcations, grooves etc.
- Frequent post-operative "re-growth," without residual pockets

Disadvantages
- Post-operative edema, pain, attachment loss
- Clinically elongated teeth
- Exposure of tooth necks (esthetics, sensitivity, caries)

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Instruments for Osseous Surgery ...

Even in cases where resective periodontal surgical techniques are necessary, alveolar bone must always be gently treated! Above all it is important to *not* remove *tooth-supporting* bone.

For the contouring and resection of alveolar bone, a myriad of various instruments is available. The choice depends upon the morphologic configuration of the bone, and the desired result.

There is a very basic difference between power-driven burs and hand instruments. When using burs, copious cooling solution is of primary importance, because the osteocytes within vital bone will be irreversibly damaged at temperatures above ca. 45 °C.

In addition, it is important during each and every osseous surgery to avoid any damage to the root surfaces of adjacent teeth. This is a danger that exists primarily during the use of rotating instruments.

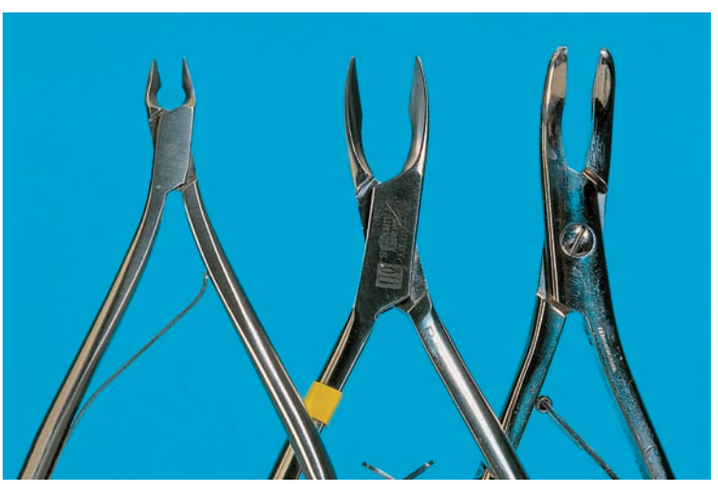
835 Burs
Depicted at the left are two internally cooled burs; on the right, two high-speed carbide round burs. When using high-speed burs, cooling solutions (saline) must be copious from an external source.

Note: During osteoplasty/ostectomy, resulting bone chips can be captured in filters ("bone traps") and used to fill other osseous defects.



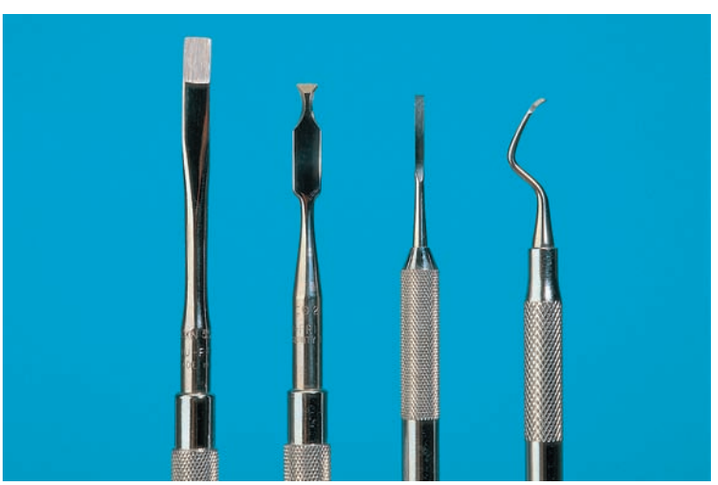
836 Rongeurs/"Nippers"
The depicted instruments, as well as additional types, are available in various sizes, with pointed or rounded working ends.

Left: **Fine nipper**
Goldman Fox
Hu-Friedy NIPS
Middle: **Concave chisel nipper**
Mini Friedman
Hu-Friedy RMF/RKN
Right: **Concave chisel nipper**
Luer Friedman
Aeskulap



837 Chisels
Sharp chisels are commercially available in various sizes and shapes. For each planned osseous correction, the appropriately sized and angled instrument should be selected.
Bone chisels, from left to right (from Hu-Friedy):

- **Kramer-Nevins** (CKN 55) 5.5 mm
- **Ochsenbein No. 2** (CO 2)
- **Kramer-Nevins** (CKN 1/2) No. 1/2
- **Ochsenbein No. 3** (CO 3)



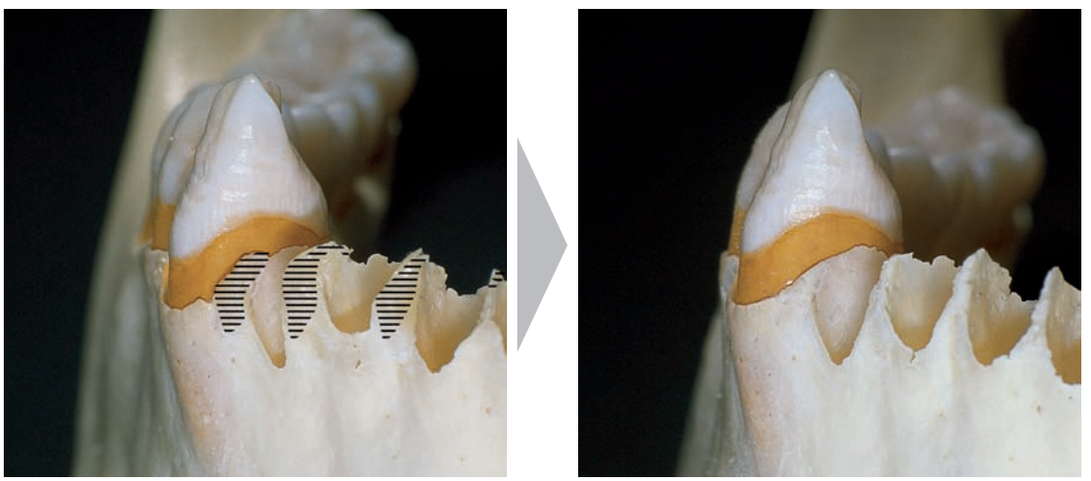
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... and Their Use

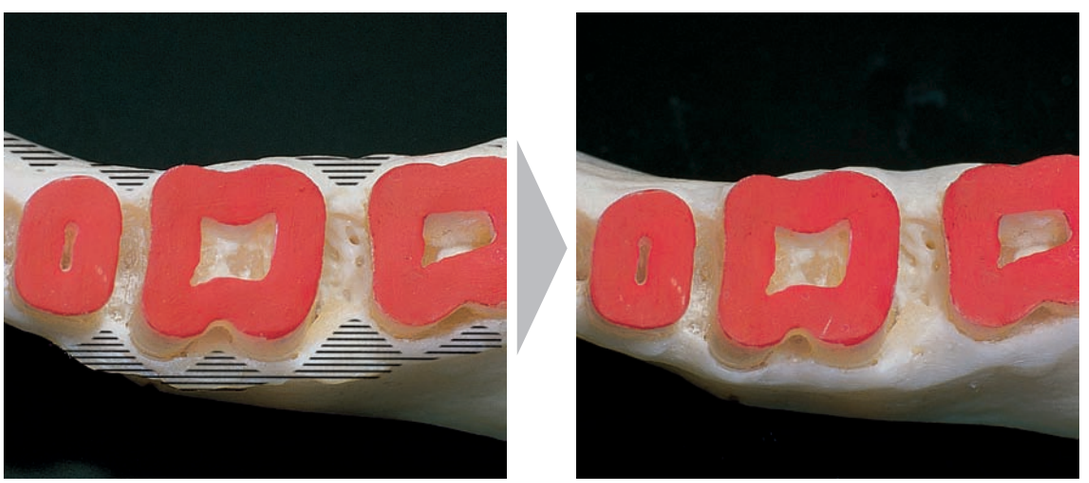
Today, aggressive ostectomy for the complete elimination of vertical osseous defects is seldom employed even during resective therapy. Nevertheless, indications remain for selective osteoplasty:

- Correction of sharp, irregular bony margins; the goal is a harmonic *crestal architecture*.
- *Contouring of bulbous bone*: Particularly in the interdental region, it is important to create a physiologic morphology. The goal is natural-appearing interdental papillae between the individual teeth. Following osteoplasty, it is easier to optimally adapt the mucoperiosteal flaps and to completely cover any slight interdental craters that remain.
- *Elimination of intra-alveolar bony craters*: "Ramping" is a radical procedure that permits maintenance of the alveolar bone height while "opening" the bony pocket facially or orally.

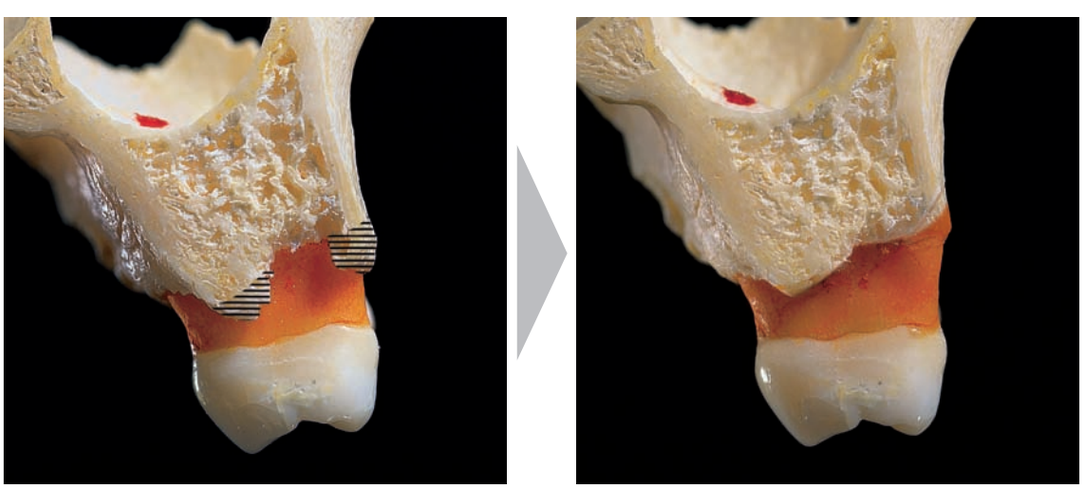
The treatment results of such surgical procedures are predictable and the initial healing occurs rapidly; however, the maturation phase for the bone takes much longer.



838 "Harmonization" of the Alveolar Crest
On buccal surfaces and in interdental areas, sharp bony margins and protuberances are recontoured and smoothed (hatched areas). Especially on the facial and mesial surfaces of the canine (tooth 43), the contour of the bone parallel to the cementoenamel junction is clear (right).



839 Protuberant Interdental Bone
The protuberant bone in the interdental regions (*left*, hatched) is given a more physiologic contour form both facial and oral aspects. The bone is also carefully recontoured in the furcation areas. Here, it is important that the furcation entrances be neither damaged nor further opened.



840 "Ramping"
In the maxillary posterior segments, the bony pockets facially and palatally are opened interdentally. No bony craters persist (*right*), only an oblique bony surface.
After healing, the proximal tooth and root surfaces can be adequately cleaned, e. g., using interdental brushes, thus preventing any re-infection. This surgical procedure results in longer teeth, and therefore the ramping method is not recommended in the visible anterior regions.

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Evaluation of Various Resective Methods

Comparison of Flap Surgical Procedures with or without Osseous Surgery

Numerous professional publications have reported the results, both clinical and histological, of various surgical treatment methodologies, particularly *regenerative* and *resective*. These clinical studies have shown that the results following radical surgical procedures are better than following conservative techniques in terms of *pocket reduction*. Pocket reduction is especially important when extensive reconstructive dentistry (bridgework, crowns) is planned post-surgically (minimal pockets, stability of the gingival margin).

The disadvantages of resective surgical procedures include esthetic compromises in the visible regions (facial recession, loss of papillae) and the possibility of tooth neck sensitivity and root surface caries if oral hygiene is not optimum (cf. gerodontology, p. 515).

While changes in attachment level and pocket probing depths have often been compared in residual pockets, changes in the amount and composition of *microbial biofilm* (microbial colonies/colonization) have not been.

Results

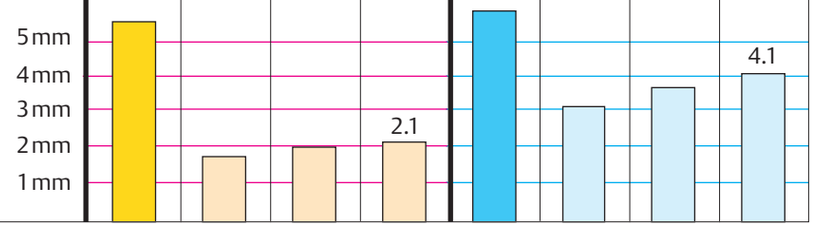
841 Pocket Probing Depths Following Flap Surgery with and without Osseous Surgery

Following osteoplasty/ostectomy and apical repositioning of flaps, pocket depth reduction is significant and stable over the long term. In similar flap surgery, but without osseous surgery, the soft tissue flaps must be re-adapted over the remaining, cleaned osseous defects. Deep *residual pockets* remain, and these have a poorer long-term prognosis (recolonization, difficult plaque control).

Pocket Depths in Periodontal Lesions *with ..... without* Osseous Surgery

	Osseous surgery				without osseous surgery			
	Initial	1 month	3 months	6 months	Initial	1 month	3 months	6 months
Average probing depths (mm)	5.5	1.9	2.0	2.1	5.9	3.1	3.8	4.1

Individual probing depths



842 Comparison of the Methods with Regard to Colony Formation of Periodontopathic Microorganisms

The reduction and partial elimination of periodontopathic microorganisms (*Aa*, *Pg*) are remarkable following osseous surgery. This can be explained by the fact that in shallow pockets (aerobic, O_2) the well-known, disease-inducing gram-negative *anaerobes* can no longer thrive. Such dramatic results must be considered for *every* type of periodontal treatment planning.

Microorganisms in Periodontitis Lesions *with ..... without* Osseous Surgery

Species	Osseous surgery				Without osseous surgery			
	Initial	1 month	3 months	6 months	Initial	1 month	3 months	6 months
A. actinomycetemcomitans	1	0	0	0	1	3	3	2
P. gingivalis	5	0	0	0	4	2	3	3
T. forsythensis	5	0	1	1	2	1	2	1
P. intermedia	6	2	1	1	5	3	3	3
Fusobacterium sp.	6	2	2	1	6	4	4	4
P. micros	5	1	1	1	6	2	6	5
C. rectus	6	0	0	0	5	1	3	4
Capnocytophaga sp.	2	0	0	0	0	1	2	1
„Enteric rods/Pseudomonas“	1	0	0	0	0	1	0	1
Yeasts/Fungi	0	1	1	1	1	0	1	0

Studies Performed by the Slots Research Team

Mao-Chin Tuan and co-workers (2000) studied a group of 14 adult patients with interdental osseous craters, including the clinical results as well as microbial parameters (microbiologic cultures from the 3 deepest pockets) *after* conventional professional (supragingival) tooth cleaning, but before any subgingival treatment. Following subgingival debridement and surgical therapy (*with or without* osseous surgery) the post-operative changes in the residual pockets were measured (bacterial type, number of colonies in the *pooled samples*). These evaluations were repeated after 1, 3 and 6 months.

In addition to the expected significant pocket depth reduction following osseous surgery, there was also a significantly reduced BOP index, even in the face of identical plaque accumulation! Most significant, however, were the microbiologic differences: Following osseous surgery, they observed significantly fewer pathogenic bacterial colonies, especially *Aa* and *Pg*. The risk of recurrence could therefore be reduced (cf. Socransky et al. 1997, 2003).

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Resective Therapy
Apical Flap Repositioning—Pocket Elimination

Surgical Procedure, Step-by-Step

We have demonstrated that, even today, in special cases, resective procedures for the surgical elimination of periodontal pockets may be indicated. In the clinical case described below, the patient was a 33-year-old male with advanced, aggressive periodontitis. A radical surgical procedure was indicated in this case because patient compliance was only moderate, esthetics played no role, and the fees were of consequence. Following initial therapy in the mandible, all of the sextants were treated surgically. Here, the surgical procedure in the *anterior segment* is portrayed.

Situation before initial therapy:

PI: 50% BOP: 75%

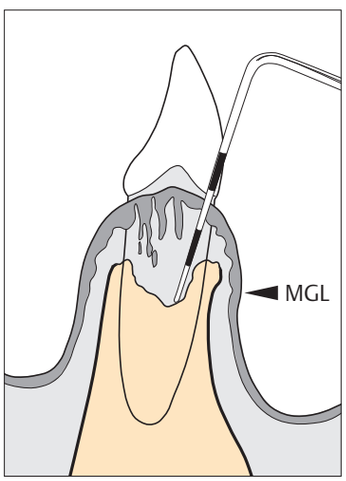
Clinical picture, probing depths, tooth mobility and radiographic view are provided in the figures below.

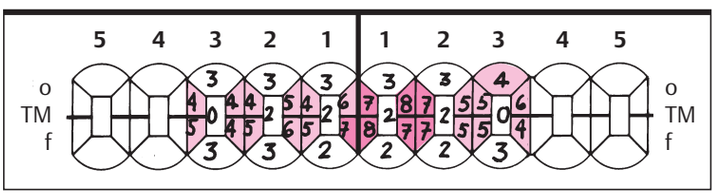


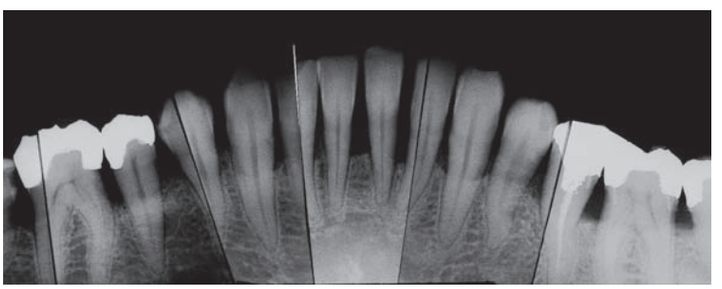


843 Clinical View of the Mandibular Anterior Region before Initial Therapy
Inflamed, hyperplastic gingiva. The pocket probing depth on the mesial surface of tooth 31 was 8 mm. Diastema formation between teeth 41 and 31, as well as between 31 and 32.

Left: The periodontal probe positioned on the mesial surface of tooth 31 indicates the depth of the periodontal pocket, whose fundus is apical to the mucogingival junction!







844 Initial Findings

Pocket Probing Depths and Tooth Mobility:
In the proximal regions, there is pronounced probing depth to 8 mm. Tooth mobility to grade 2.

Radiographic Findings:
The radiographs confirm the pronounced attachment loss in the interdental regions, but do not indicate an irregular alveolar course on the facial aspect (Fig. 849).
Left: Diagram of the initial situation.

Resective Therapy—Protocol

- Paramarginal, horizontal incision
- Vertical releasing incisions extending into the mobile mucosa
- Reflected full thickness flaps
- Incision of the periosteum apical to the mucogingival junction
- Exposing the crestal bone
- Removal of granulation tissue
- Root cleaning/planing
- Osteoplasty/ostectomy
- Apical repositioning of the soft tissue flaps
- Interrupted sutures



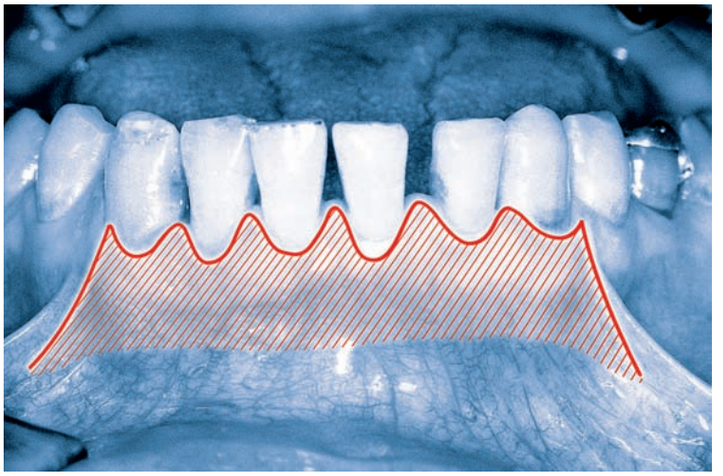
845 Lingual Clinical View
Severe gingival inflammation, expansive plaque and calculus.

Left: Surgical procedure, step-by-step (protocol).

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846 Surgical Planning after Supra- and Subgingival Debridement
The gingival inflammation was greatly reduced, but pockets up to 7 mm remained interdentally. For esthetic reasons, tooth 31 was shortened and tooth 33 was re-contoured (selective odontoplasty).

The planned incisions are indicated (red).



Facial View

Surgical Planning

847 Incisions
The *horizontal incision* is scalloped and intragingival (intrasulcular). Because the treatment plan includes apical repositioning of the flaps, paramedial *vertical releasing incisions* are made distal to each canine.



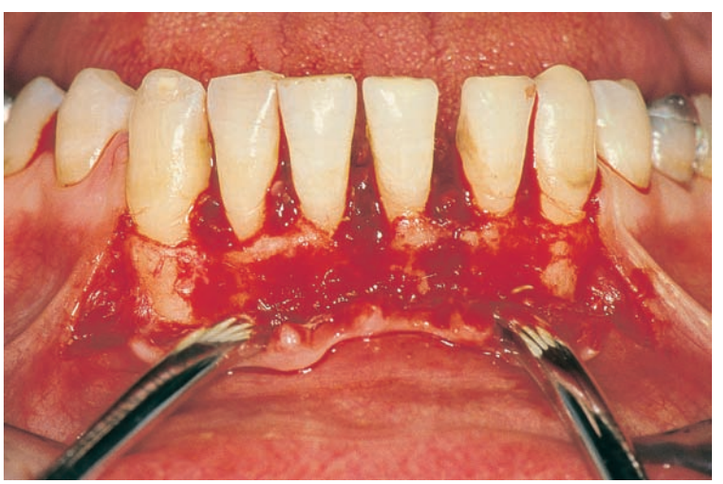
Incisions

848 Flap Reflection
A sharp elevator of the appropriate size is used to mobilize and reflect the expansive mucoperiosteal flap beyond the mucogingival junction.



Flap Reflection

849 Exposing the Alveolar Bone
Even before removing the superficial granulation tissue, the irregular course of the alveolar bone is clearly visible.

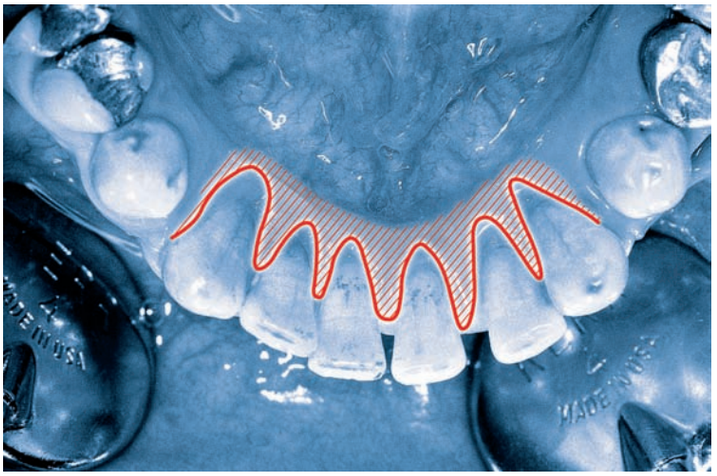


Exposing the Bone

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Lingual view

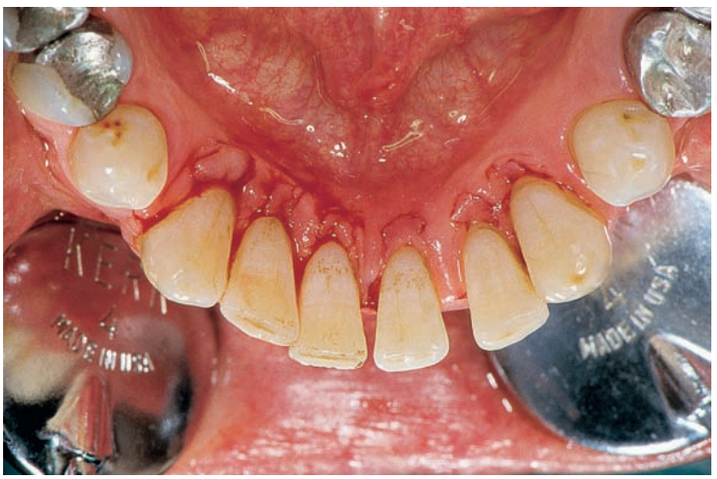
... Following Phase 1 Therapy



850 After Supra- and Subgingival Debridement
The gingival inflammation was also reduced on the lingual aspect following Phase 1 therapy.

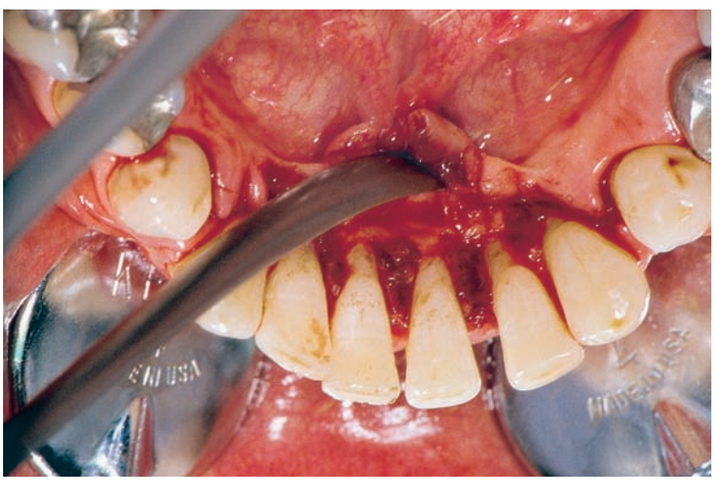
(The red line indicates the planned incision; cf. Fig. 846.)

Incision



851 Incision
The scalloped intragingival incision courses on the lingual surface further removed from the gingival margin than on the labial surface, because in the lingual area the flap is more difficult to reflect carefully, i. e., with certain risks (muscle attachments, vasculature etc.). This broader excision of tissue assists in pocket depth reduction and provides for apical repositioning of the gingival margin.

Flap Reflection



852 Flap Reflection
This is performed without any vertical incisions and the flap is only partially mobilized; nevertheless, the alveolar bone must be rendered clearly visible in order to provide adequate direct vision of the bone and root surfaces.

Caution: Vasculature in this lingual area!

After Root Scaling/Planing

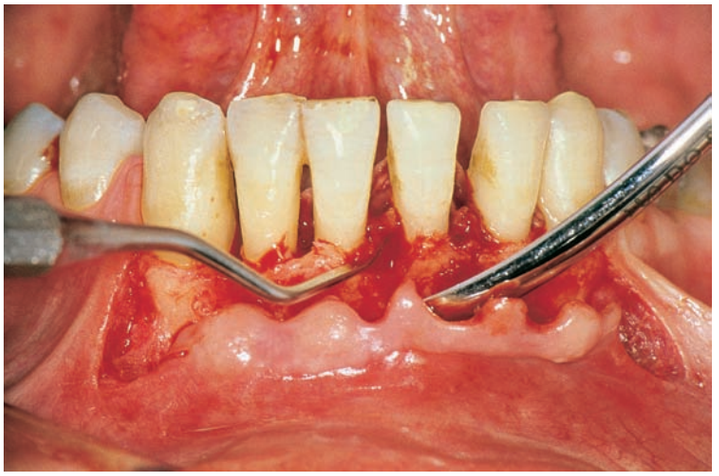


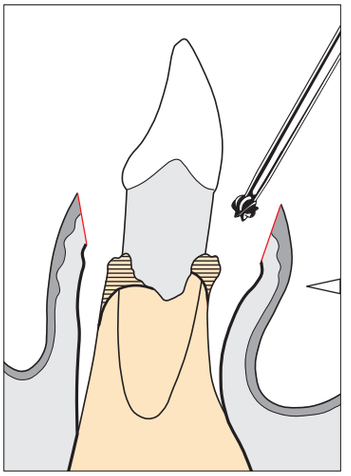
853 Following Root Cleaning/Planing
The infiltrated pocket tissue and granulation tissue that was delineated by the initial incision have already been removed; this permits planing of the root surfaces with direct vision (see also Figs. 854–858).

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854 Removal of Soft Tissues on the Facial Surfaces
Granulation tissue is removed from the tooth surfaces using fine curettes, and from the osseous surface and vertical bony defects using fine excavators. This provides clear and complete vision for cleaning and planing the root surfaces.

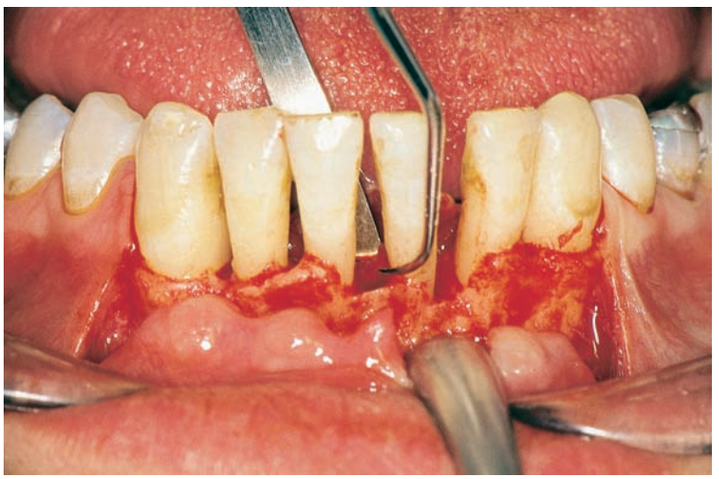
Right; Bone before ostectomy/ osteoplasty (hatched areas).





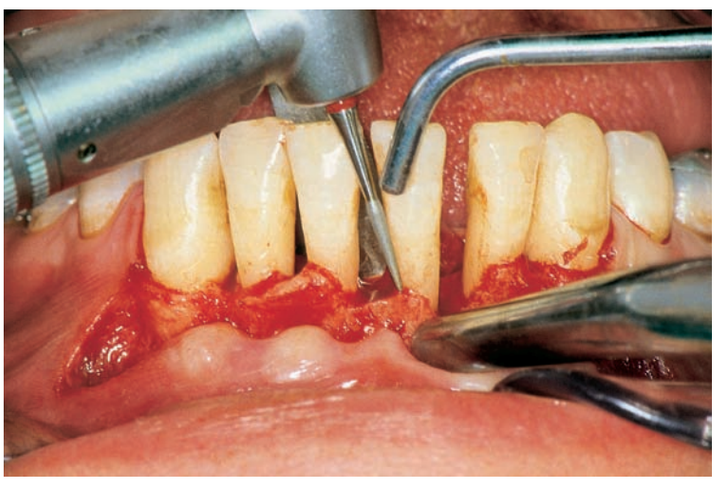
855 Root Planing with Curettes
The root surfaces are scaled and planed systematically, stroke-by-stroke, with extreme attention to detail. The working area is repeatedly rinsed with saline solution. Root planing is an *obligatory phase* of therapy and it is one of the most important measures of the entire surgical procedure!

Right: Excavator for the removal of granulation tissue; curette for root planing.



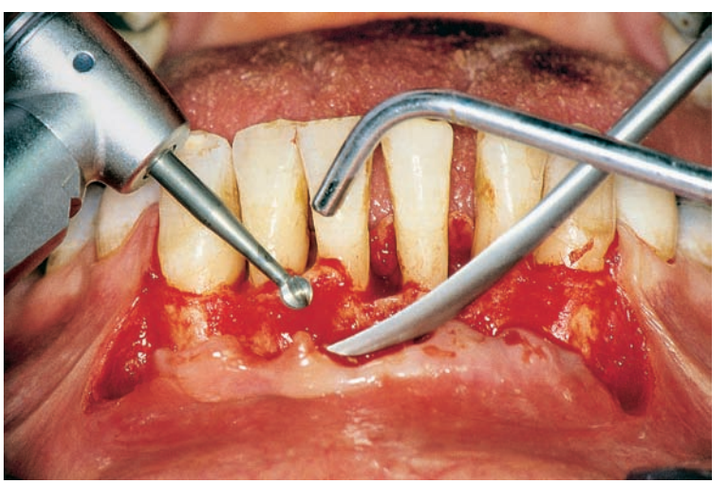


856 Use of a Rotating Diamond to Enhance Root Planing
Especially depressions and irregularities of the root surface can be cleaned/planed thoroughly using the finest polishing diamonds (Perio-Set 15 µm).
If areas of the root have to be recontoured via odontoplasty (open grooves etc.), diamonds up to 40 µm can be employed. Such areas are then finished using fine polishing diamonds (15 µm) or with curettes.



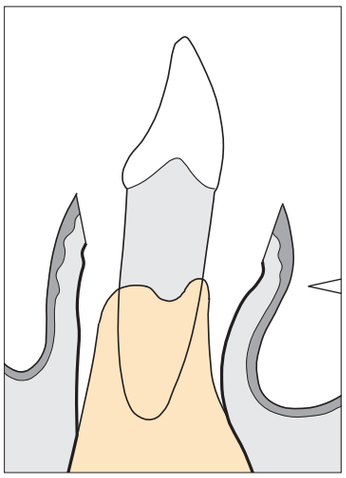
857 Osteoplasty
Using round burs (steel or carbide), sharp and protuberant bone is removed or recontoured under constant cooling with saline solution. Recontoured bone is then smoothed using fine hand chisels.
Any interdental craters are opened slightly. Tooth-supportive bone should never be removed.

Right: Instruments for osteoplasty: Steel or carbide round burs; chisel (CO1; Deppeler).





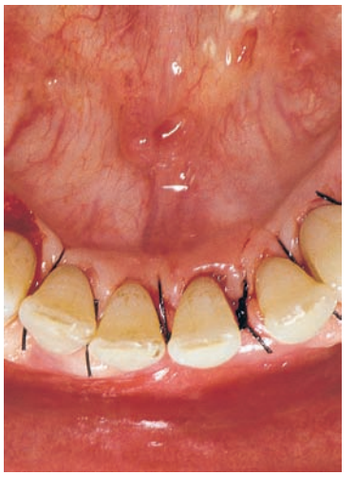
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858 Following Root Planing and Osteoplasty
Note that the tooth-supportive bone on the facial surfaces of all incisors was preserved. This preservation led to a less than overall positive architecture of the alveolar crest (attachment).

Left: Bone contour following ostectomy/osteoplasty.





859 Apically Repositioned Flaps
The vertical incisions and each interdental space are closed using interrupted sutures. The extent of the apical repositioning can be seen by comparing with the initial view (Fig. 849).

Left: Incisal view. Because of the deeper scalloping initial incision on the lingual aspect, with preservation of the interdental papillae, it was possible to almost completely cover the interdental craters.





860 Ten Days after Surgery
The situation has already consolidated for the most part. However, the course of the marginal gingiva remains irregular.

Left: During the first 10–14 days, when mechanical oral hygiene is not possible, the patient must rinse with chlorhexidine solution. Thereafter, healing is monitored as frequently as possible, with professional plaque removal.



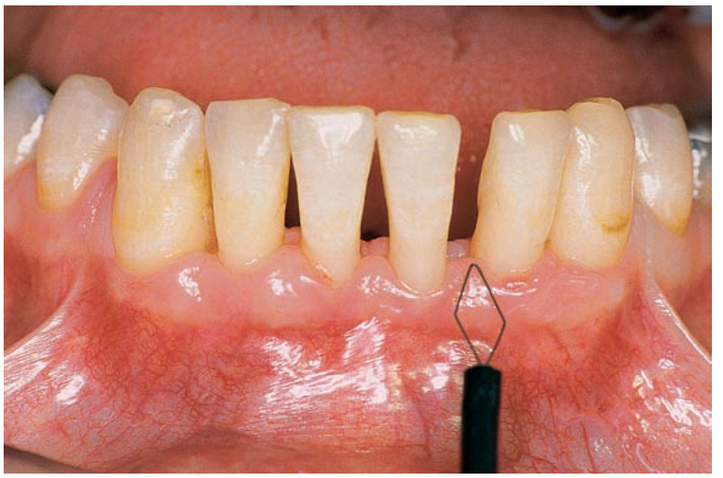


861 Three Months Following Surgery
There are no gingival depressions in the interdental areas. These occur frequently if the labial and lingual papilla tips become necrotic after suturing. In addition, note that the gingiva in the region of 41–43 has become fibrous and somewhat thickened.

Left: This incisal view clearly shows the small crater between teeth 31 and 32.

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862 Follow-up Gingivoplasty by Means of Electrosurgery
The small interdental gingival depression shown in Figure 861 resolved satisfactorily following rigorous interdental hygiene (Stimudent, interdental brushes). If such minor gingival defects persist, they can be corrected via "mini-gingivoplasty" using the rhomboid electrosurgical loop.



863 Following "Mini-Gingivoplasty"
The small gingival craters have been eliminated and at the same time the somewhat bulbous gingival tissue on the facial surfaces of 41–43 was recontoured.

Right: Incisal view following gingivoplasty.

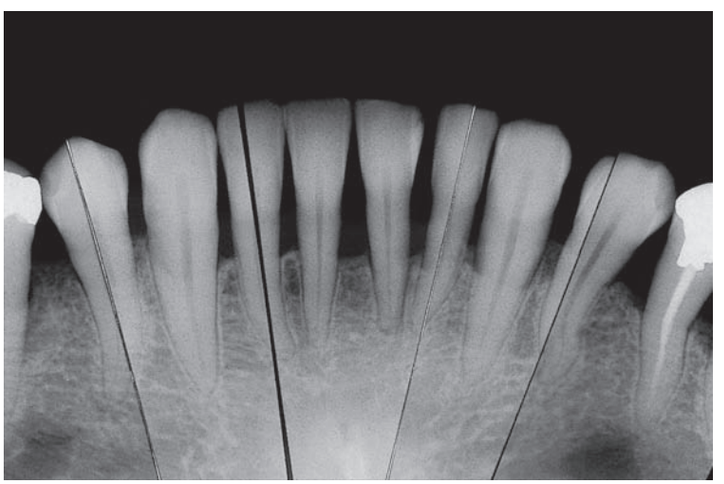




864 Ten Months after Gingivoplasty, 13 Months Post-Surgical
The gingival morphology is much improved. Teeth 31 and 32 were widened somewhat using composite resin in order to reduce the size of the diastemata between these teeth (esthetics).



865 Final Radiographic View
As expected, following apical flap repositioning, the osseous level and attachment was not significantly altered. At several sites, however, there appears to be some new compact bone formation interdentally.



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Resective Therapy
Apical Flap Repositioning—Pocket Elimination

Summary

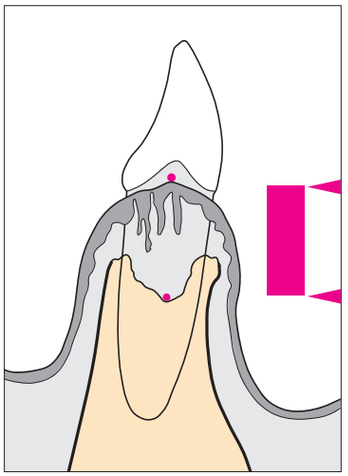
In the introduction to this chapter on "Resective Therapy," the advantages and disadvantages of this treatment modality were described. "Resective therapy" is slowly being reconciled to the periodontological past, rather than future.

In the case presented here, a 33-year-old male with only moderate oral hygiene compliance, the treatment of choice was a rapid, predictable result and therefore a radical treatment method in the form of resective therapy. There were no esthetic considerations. Moderate tooth neck hypersensitivity post-therapy could be successfully treated in only a few weeks. Following surgical therapy, the compliance of the patient improved significantly, thanks to a short-interval recall.

Situation 13 months following treatment:

PI: 20% BOP: 12%

"Healthy," normal periodontal probing depths were in evidence and these were anticipated following treatment procedures involving resective therapy and apically repositioned flaps.

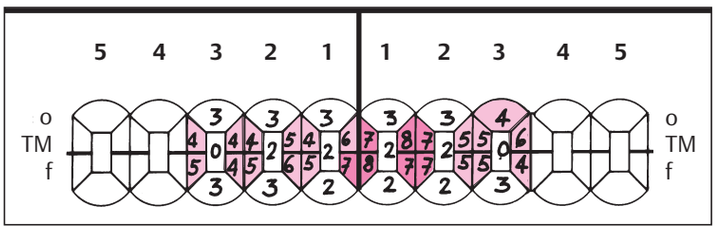




866 Clinical View before initial Therapy
Severe gingival inflammation. Irregular course of the incisal edges. Diastema formation between teeth 41, 31 and 32.

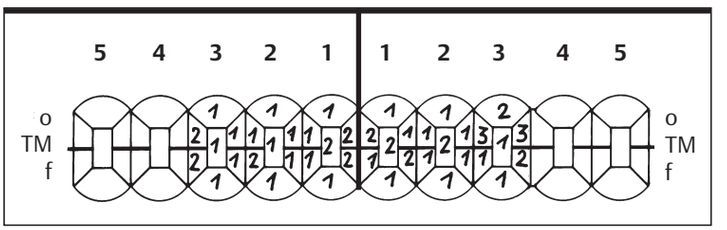
Left: The gingival margin remains at its normal level. The interproximal areas exhibit deep periodontal pockets and osseous defects (long red bar).

Before

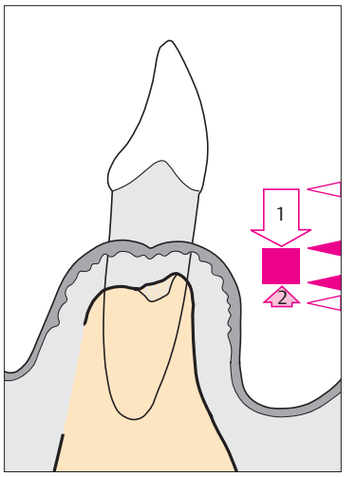


867 Pocket Probing Depths and Initial Tooth Mobility
Probing depths up to 8 mm in the proximal areas. The incisor teeth exhibit tooth mobility of grade 2.

After



Pocket Probing Depths and Tooth Mobility 13 Months after Surgery
Physiologic probing depths. As expected, there was little change in tooth mobility.





868 Clinical View 13 Months after Surgery
Healthy periodontal conditions exist, with the gingival margin at a more apical niveau. Pockets have been eliminated; the teeth appear somewhat longer.

Left: Dramatically reduced pocket probing depths; minimal residual pockets (short red bar).

1 Apical repositioning and gingival shrinkage
2 Minimal new attachment (bone formation?)

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Combined Flap Surgical Techniques

It is very rare that advanced periodontitis effects the entire dentition. Inflammation, attachment loss and pocket probing depths must be determined for each tooth and for each surface of each tooth ("single tooth diagnosis"). It is not unusual to see, in any individual case, that in one region only gingivitis is present, while in other areas severe periodontal destruction is in evidence, while in a third location one observes only localized gingival recession.

Theoretically, based upon the tissue loss configuration, the treatment methodologies within an individual patient might include: For individual teeth, initial therapy and subsequent home care by the patient may be all that is needed; in other patients, minor surgical procedures such as gingivoplasty or a modified Widman flap procedure may be indicated; while in other segments of the dentition, it may be necessary to perform expansive flap surgical procedures in order to perform osteoplasty, root resection, tooth hemisection, and/or implantations using autogenous or alloplastic materials in bony defects.

It is important to realize that during a single surgical procedure one technique may be required on the facial surface and another may be indicated on the oral surface.

869 Possible Combinations of Flap Surgery and Other Techniques

"Flap surgery": This term is used for all types of surgery involving mucoperiosteal flaps, whether full thickness or split thickness, completely mobilized or only partially so.

AF "Access flap"
FMF Fully mobilized and reflected flaps
GTR Guided tissue regeneration

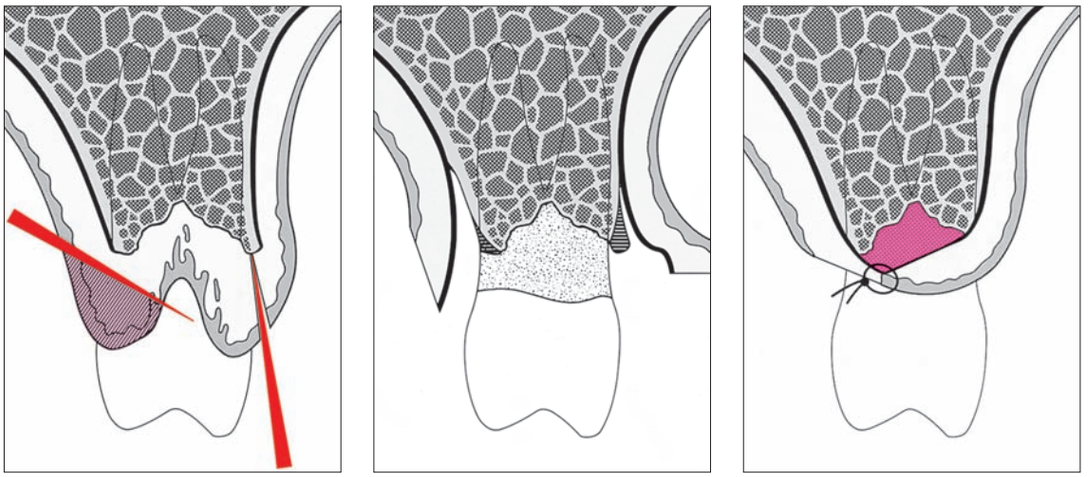
Combined with flaps	Surgical techniques	Page
Flaps (AF*/FMF*)	GTR**	338
	Pocket implant	327
	Root resection	387
	Hemisection	392
	Extraction	481
	Wedge excision	319
	Gingivectomy	334

870 Example of a Combined Operation

Left: Facial, paramarginal incision (internal gingivectomy); oral gingivectomy

Middle: Facial flap reflection, minimum osteoplasty (hatched area), subsequent oral flap reflection following gingivectomy.

Right: Wound closure with interrupted, interdental sutures. The oral gingivectomy wound re-epithelializes secondarily.



Because of its firm consistency, a palatal flap is difficult to reposition apically (pocket elimination); thus, in many cases gingivectomy (oral) and flap reflection (facial) are often combined.

In addition, for example, one might partially eliminate the pocket via gingivectomy and subsequently at the same location create a soft tissue flap (Fig. 770). In addition to these purely anatomical indications, esthetic considerations can also determine the treatment procedures. For example, in the visible maxillary anterior segment it may be possible/reasonable to perform only closed root cleaning/planing (minimal gingival shrinkage), and to intervene surgically from the palatal aspect exclusively.

The often-necessary combination of "access flap" and fully mobilized mucoperiosteal flaps (in resective procedures) is depicted in Fig. 869 in combination with other techniques.

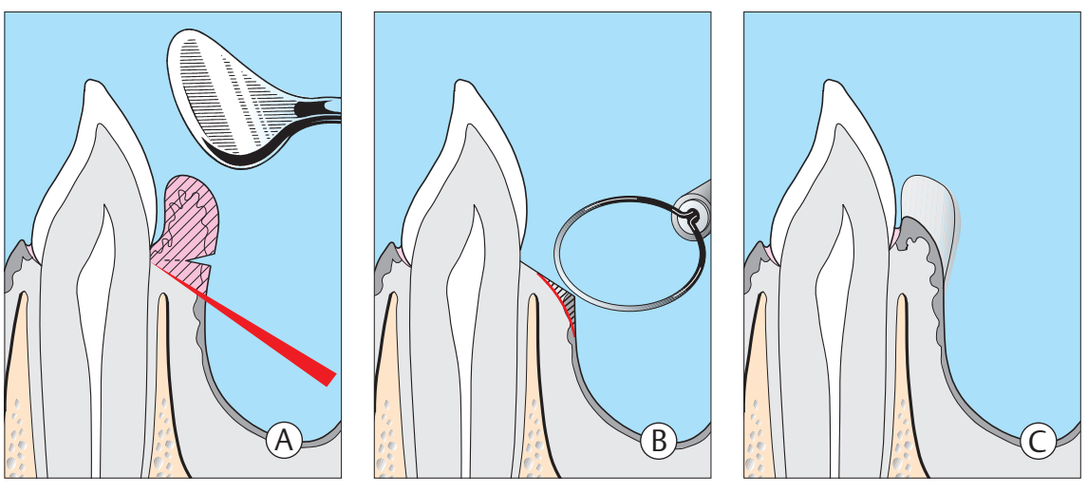
Surgical procedures combining periodontal flap procedures with the simultaneous removal of an impacted or retained third molar are seldom indicated because it is not possible to exclude contamination of the wound after the surgical procedure.

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Gingivectomy and Gingivoplasty

The *gingivectomy* (*GV*) was formerly a frequently performed surgical procedure; however, it has lost most of its significance as a procedure in the treatment of *periodontitis* today. The goal of the GV operation is elimination of gingival pockets by resection of gingival tissue. But the primary goal of modern periodontitis therapy is *pocket elimination* mainly by means of *regenerative* methods, i.e., the true healing of diseased periodontal structures; only in very few cases will this be attempted by means of simple resective methods.

Nevertheless, the GV procedure maintains its place as a *minor periodontal surgical procedure*, e.g., for the exposure of crown and cavity margins, crown lengthening procedures etc. Clinical indication still exists also for the *gingivoplasty* (*GP*). For example, GP will be indicated for the reduction of gingival hyperplasia/overgrowth, and for recontouring the gingival surface to a more physiologic form. In some clinical situations, GV and GP are also combined with flap surgery (p. 334).



871 Principles of Gingivectomy and Gingivoplasty

A After marking the pocket fundus, the hyperplastic gingiva adjacent to the markings is excised (GV) with a 45° angled incision.

B Subsequently, the tissue contour can be improved (GP) using the electrotome or laser.

C The covered (periodontal dressing) wound epithelializes secondarily, and new junctional epithelium is created with a gingival sulcus exhibiting minimal probing depth.

Indications

- Gingival enlargement or overgrowth (caused by medicaments or hormonally)
- Idiopathic fibrosis
- Suprabony pockets in areas with limited access
- Minor corrective procedures during the course of flap surgery
- Crown lengthening in patients with adequate attached gingiva (e.g., a “gummy smile”, p. 493)

Suprabony pockets are, however, best treated conservatively (closed) by scaling and root planing, or by means of the modified Widman procedure!

Contraindications, Disadvantages

- Narrow or absent attached gingiva
- Infrabony pockets
- Thickening of marginal alveolar bone (exostoses)
- Large open wound; post-operative pain
- Healing by secondary intention (epithelium: ca. 0.5 mm per day)
- Danger of exposing alveolar bone
- Loss of attached gingiva
- Exposed cervical areas of teeth (sensitivity, esthetics, caries)
- Phonetic and esthetic problems in the anterior area

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Instruments for Gingivectomy/Gingivoplasty ...

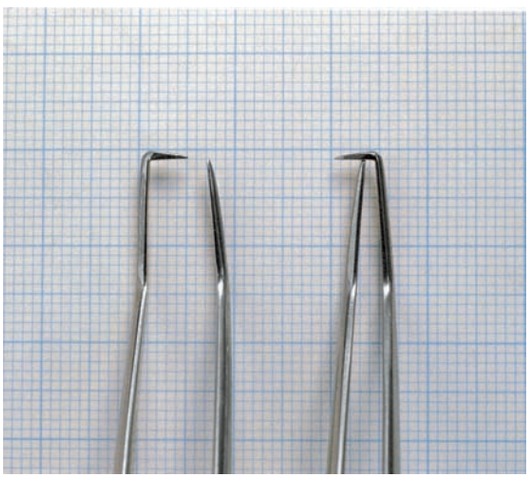
A wide variety of instruments is indicated for GV/GP. Virtually every manufacturer of dental instruments offers the tools for periodontal surgical procedures, especially the gingivectomy. The original GV/GP instruments are decades old (e. g., Kirkland and Orban knives), and continue to be modified and improved. The most important aspect is that the size, shape and angulation of the working end provides the most uncomplicated, rapid and clean performance.
Generally speaking, a GV/GP procedure requires a gingivectomy knife and a papilla knife, each of which may be single- or double-angled.

A sharp incision with a GV knife is "cleaner" than the layer-by-layer tissue removal with a laser device. Only for the superficial recontouring of the gingiva as well as for minor procedures such as exposing crown or cavity margins, the time-proven GV knife may be replaced or enhanced by electrosurgery or a laser device. Forceps for measurement and marking of the pocket fundus are not obligatory. Such measurements and markings can be performed with the conventional periodontal probe.

872 Pocket Marking Forceps
Left: The paired pocket marking forceps (Deppeler, L & R) are used to indicate the base of the gingival pocket. This can also be done using a fine periodontal probe.

Right: Working end of marking forceps. The straight arm is inserted to the bottom of the pocket, then the forceps is closed. The point thus penetrates the surface of the gingiva and creates a bleeding point corresponding to the base of the pocket.





873 Gingivectomy Knives

- **GV knife** (GX 7 L & R), single bend
- **Papilla knife** (ZI 14 L & R), single bend
- **Universal knife** (ZL 19), single bend

(all instruments from Deppeler).

These knives are also available with two bends, and as double-ended instruments.

Right: Working ends of the gingivectomy knives.

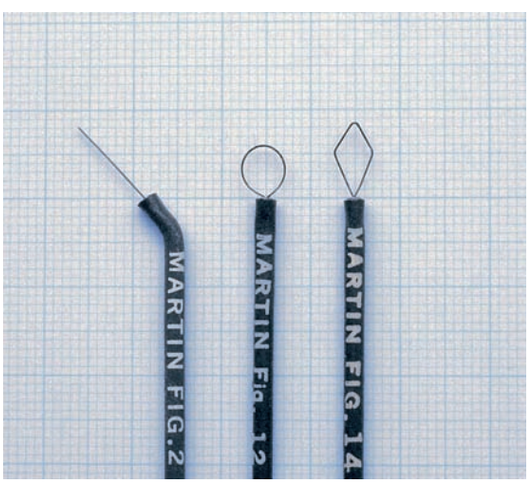




874 Electrosurgery—Device and Tips (Martin, Ellman etc.)
Electrosurgery (and possibly also laser) finds its primary function in the gingivoplasty procedure (GP) for contouring soft tissues, and exposing the margins of restorations. For the primary incision, however, in extensive gingivectomy procedures, it is not recommended because of the possibility of injury to the tooth root, periosteum and bone.

Right: The three most important tips for the Electrotome.





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... and Their Use

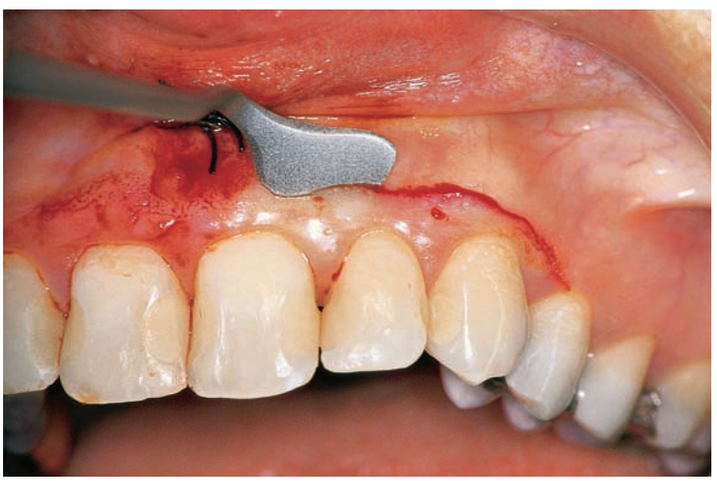
More important than design and manufacturer is the *sharpness* of the instruments: Gingivectomy knives must be sharpened prior to each surgical procedure (Arkansas stone with oil, p. 268). This requirement can only be avoided through use of instruments with disposable blades.

For contouring the gingival surface (GP), fine electrosurgical tips or even a laser device may be indicated. These devices may also be indicated for minor procedures such as exposing the margin of a tooth preparation before taking an impression, or before seating a restoration. Because electrosurgery and lasers exert a certain hemostatic effect, they may be used to advantage for the excision of highly vascular, edematous soft tissues.

The use of the specific gingivectomy instruments is described here "in principle." They are described in more detail in the following "step-by-step" surgical procedure, which is performed using the few instrument types depicted below.



875 Marking the Pocket Depth
The straight arm of the forceps is guided into the mesiobuccal pocket on tooth 33, much as a periodontal probe would be. Upon closing the forceps, a bleeding point is created, revealing the depth of the pocket (4 mm) on the external surface of the gingiva. This type of pocket depth marking is particularly valuable with extremely varying pocket depths.



876 Gingivectomy Using a Kirkland Knife
At an angle of 45° to the tooth long axis, the single-bend gingivectomy knife (GX 7; R) is used to make a continuous incision along the marked pocket depths. To perform a gingivectomy on the less accessible palatal or lingual areas, a double-angled instrument is often useful.



877 Gingivoplasty Using an Electrosurgical Loop
Using the rhomboid No. 14 loop, the hyperplastic and thickened gingiva between teeth 43 and 42 is removed from the facial, interdental approach. The excision must be definitive, because a portion of the wound will fill with granulation tissue during healing, and re-epithelization must occur unimpeded. The alveolar bone must not be touched or exposed!

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Tissue Dressings and Tissue Adhesives

The open gingivectomy wound must always be covered with a periodontal dressing. In rare instances, a dressing may be placed following flap surgical procedures, less to enhance wound healing than to ensure stability and protection of the flap for patient comfort.

Pain during the first days following GV/GP will be lessened by the wound dressing. The dressing is usually left in place for 7–10 days, without change. If re-epithelialization proceeds slowly, a second dressing may be placed for several additional days.

Only eugenol-free dressings are recommended, e. g., Perio-Care, Peripak, Coe-Pack, Barricaid. A periodontal dressing alone does not prevent microbial colonization of the wound. However, if the dressing is enhanced with a disinfecting agent, e. g., CHX powder, plaque formation under the dressing can be almost completely inhibited (Plüss et al. 1975).

Minor injuries such as those created by GV to expose the margin of a crown or restoration can be covered by a cyanoacrylate-based tissue adhesive (Histoacryl, Octyldent etc.).

878 Three Periodontal Dressing Materials that Remain Soft
These have the advantage that they are easy to apply and above all they can be easily removed without eliciting pain. If the dressing is placed over sutures (e. g., following flap procedures), the sutures are not excessively stressed upon removal of the soft dressing.

Right: Old *Peripac*, with a plaster basis. Upon contact with moisture, it becomes relatively hard. The new material, with two components, remains soft.





879 Barricaid
This material remains elastic; it is a light-hardened composite material. The angled tip of the syringe simplifies application of the dressing.

Right: For esthetic reasons, Barricaid is especially indicated in the anterior sextant. It is partially transparent, pink in color, and light-hardened. However, its removal from deep interdental spaces, especially in the presence of sutures, can be problematic.



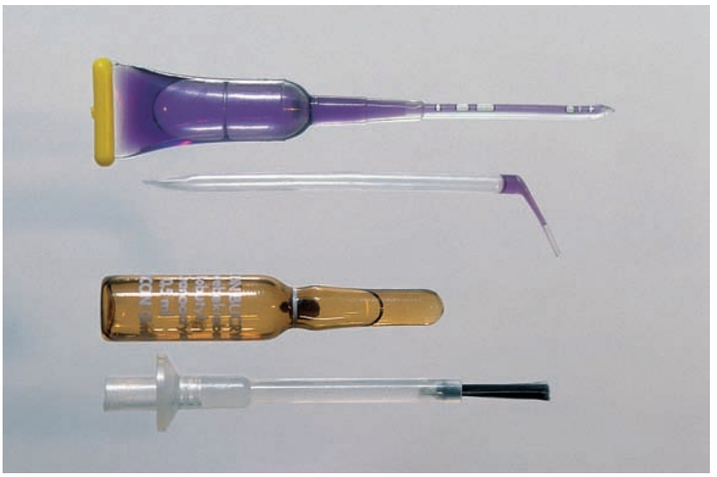


880 Tissue Adhesive—Cyanoacrylate

- **Histoacryl** (Braun, above)
- **Bucrylate** (Ethicon, below)
- **Octyldent** (Tri-Point Medicals)

Cyanoacrylate materials are truly versatile. Following minor GV/GP procedures, they can be used in place of a conventional periodontal dressing (application using a cannula, paint brush etc.

Right: Octyldent and its pipette ("Squeez-Ett," Ellman).





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Gingivectomy/Gingivoplasty

Surgical Procedure, Step-by-Step

The systematic procedure for a GV/GP operation is depicted below in the case of an 18-year-old male exhibiting severe, non-medicine induced hyperplastic gingivitis in the anterior segments of the mandible and maxilla. Etiologic factors included enormous plaque accumulation, mouth breathing, crowding in the maxilla and anterior malocclusion.

Orthodontic therapy was recommended to simplify oral hygiene, but was not accepted at that time by the parents or the patient, but was performed years later.

Initial findings:

PI: 89% BOP: 80% TM: 0

Findings after initial therapy:

PI: 27% BOP: 22% TM: 0

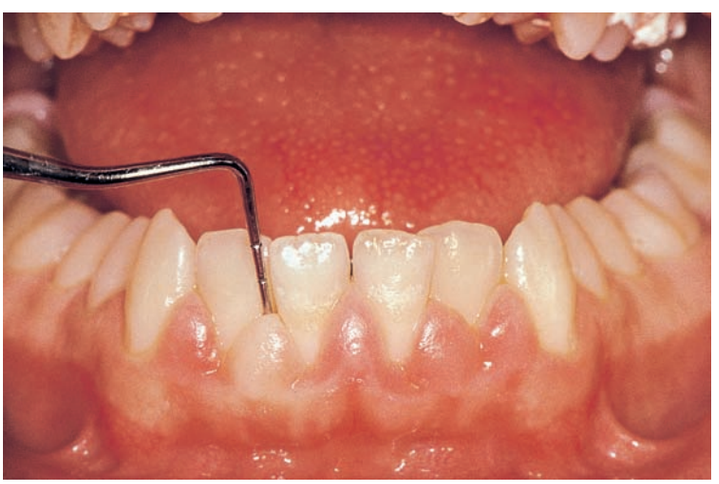
The clinical picture, gingival contour, pocket probing depths and radiographic findings are presented in the figures below.



881 Clinical Appearance before the Hygiene Phase (Initial Therapy I)

Extreme gingival hyperplasia with pseudopockets to 6 mm (pure gingival pockets).

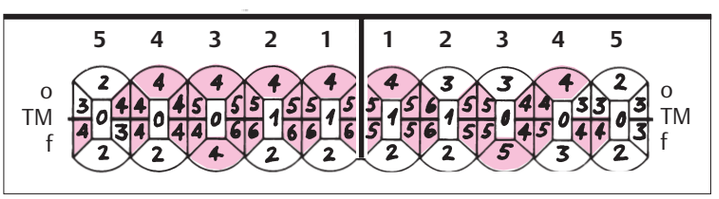
Plaque and calculus mainly in the anterior region, tooth position anomalies, mouth breathing. The situation prevented effective home care. The patient stated that the gingival problems had already begun during puberty.

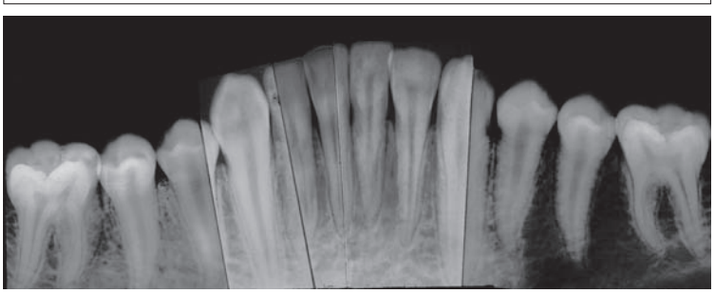


882 Mandible Following Initial Therapy

Following the successful elimination of inflammation by professional plaque and calculus removal as well as improved individual oral hygiene by the patient, the probing depths of the pseudopockets were reduced to 3–5 mm.

The gingivae remain fibrotically enlarged, and home care is still difficult.





883 Pocket Probing Depths, Tooth Mobility and Radiographic Appearance following Initial Therapy

The remaining pseudopockets are the result solely of redundant tissue (there is no true attachment loss or osseous defect).

Tooth mobility is only slightly elevated (TM).

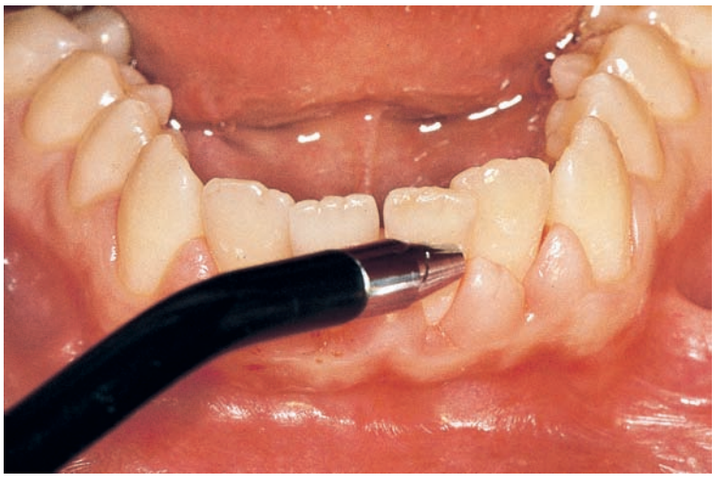
Radiographic Findings

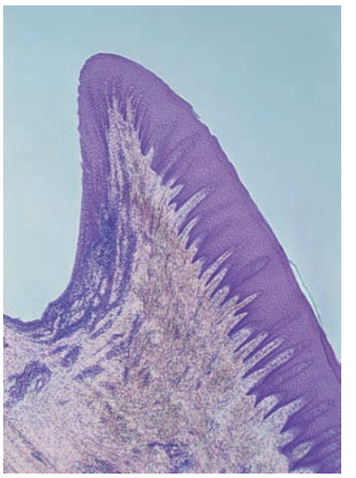
No bone loss is detectable in the radiograph.

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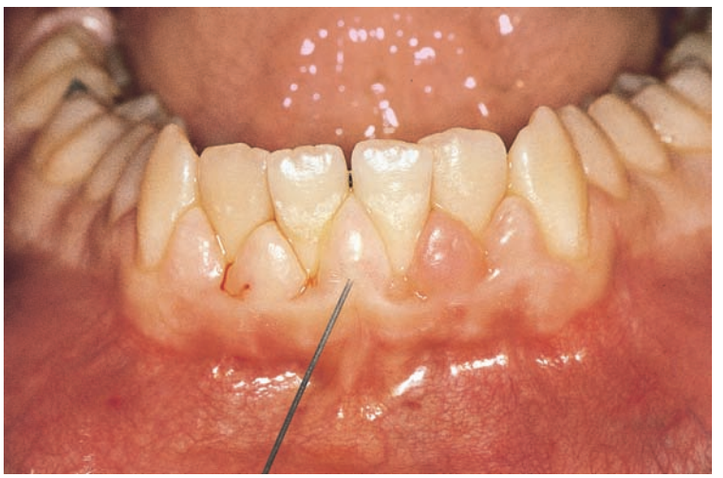
884 Gingival Hyperplasia and Pseudopockets
A blast of air from the syringe reflects an enlarged papilla away from the tooth surface.

Right: Histologically, the apical end of the junctional epithelium is at the cementoenamel junction (cf. black line on the facial surface of tooth 33 in the clinical picture). A mild inflammatory infiltrate within the connective tissue is still apparent even after initial therapy; a sign of the host response.



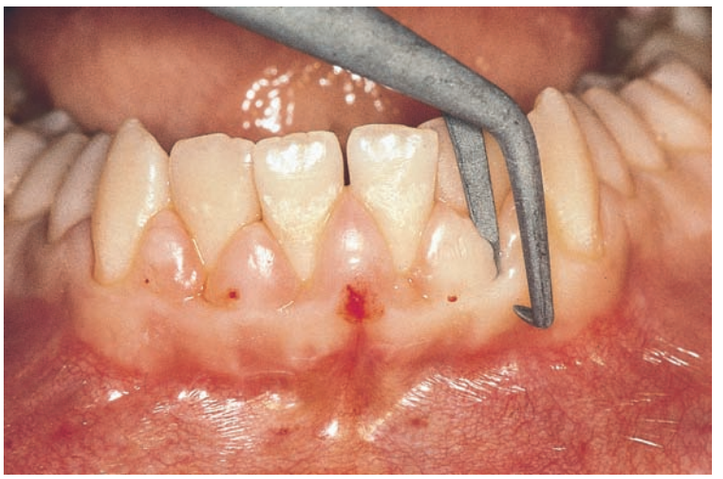


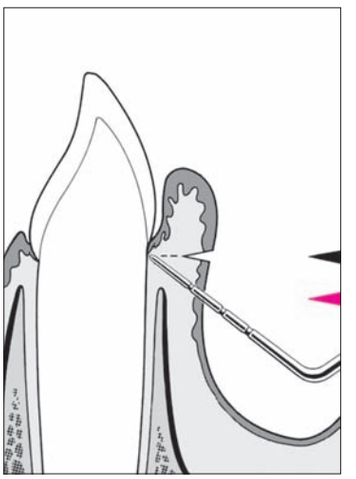
885 Anesthesia
Profound anesthesia is accomplished by injections into the mucobuccal fold ("terminal" anesthesia). To reduce hemorrhage during the procedure, each interdental papilla destined for resection is injected directly. This provides a virtually hemorrhage-free surgical site.



886 Marking the Base of the Pocket
The pocket marking forceps is used on the marginal gingiva and also the interdental papillae to create bleeding points indicating the *vestibular* course of the pseudopocket fundus between teeth 43 and 33.

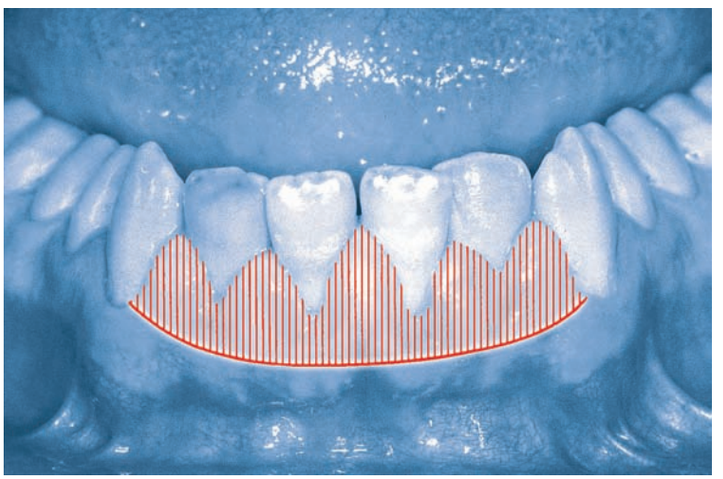
Right: The diagram shows the bleeding points at the level of the bottom of the pocket (black arrow). The periodontal probe depicts the incision (red arrow) and the incision line.





887 Planned Gingivectomy/Gingivoplasty
The hatched lines indicate the hyperplastic tissue that will be removed by an oblique incision, with subsequent recontouring. The *lingual* aspect of the mandible in this case exhibited no pseudopockets and the gingival contour was normal so the gingivectomy was limited to the facial aspect; in the maxilla it was also necessary to treat the palatal aspect (p. 377).

Right: Surgical protocol for GV/GP.

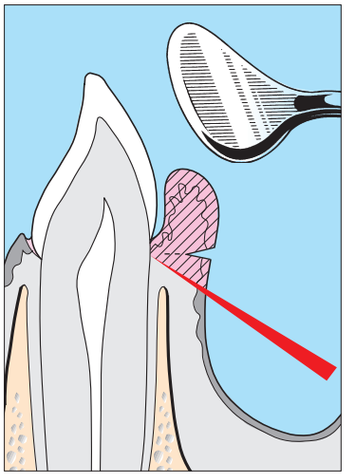


Gingivectomy/Gingivoplasty—Surgical Protocol

- Profound anesthesia (hemorrhage)
- Marking the pocket fundus
- 45° incision with the GV knife
- Sharp dissection of interdental papilla using the papilla knife
- Scaling, root planing
- Contouring the incision edge
- (Staunching any hemorrhage)
- Placing a dressing

- Post-operative instructions to the patient

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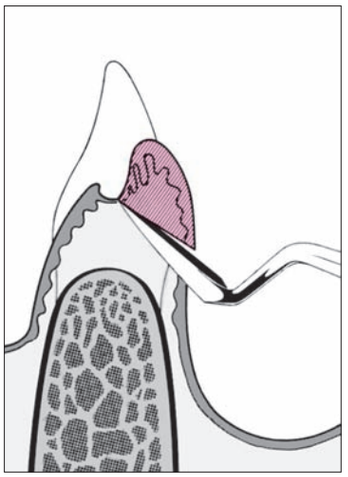


888 Uninterrupted, Beveled Incision with the GV Knife (Kirkland Knife)
The incision line is totally within the attached gingiva. The mucogingival line is nowhere approached.

Left: The diagram shows the marked pocket fundus and the incision line (red; GX 7, Deppeler).



889 Use of the Orban Papilla Knife
The strong and rigid, pointed papilla knife (GX 11, Deppeler) is carried along the contours of each tooth at the same 45° angle as the GV knife, cutting through the papilla to the interdental col region, thus releasing the excised tissue at its base. The tissue must not be *torn* away (delayed wound healing).





890 Removal of the Tissue
Using gentle pressure on the surgical forceps the incised tissue is teased free. The papilla knife can be used to sever any remaining attachment.

Left: Definitive release of the excised tissue (red, hatched) using the papilla knife in the interdental area.



891 Excised Tissue
In this case, it was possible to remove the redundant tissue as a single piece.
If the etiology of the lesion is known, histopathologic evaluation is seldom indicated.

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892 Scaling and Root Planing with Direct Vision

Even when scaling during initial therapy has been performed with great care, residual plaque and calculus may be revealed within the pseudopockets after soft tissue removal.

Therefore, the most important step in treatment during gingivectomy is cleaning and planing of any exposed root surfaces. In this way, a biologically compatible surface is created for the new junctional epithelium that must form, and the resulting gingival contour.



893 Gingivectomy Wound after Root Planing

The teeth are clean.

The incision line/wound margin remains relatively sharp, despite the 45° angulation of the initial incision. This edge must be rounded in order to provide ideal gingival contour following definitive healing (simplied oral hygiene, plaque control).

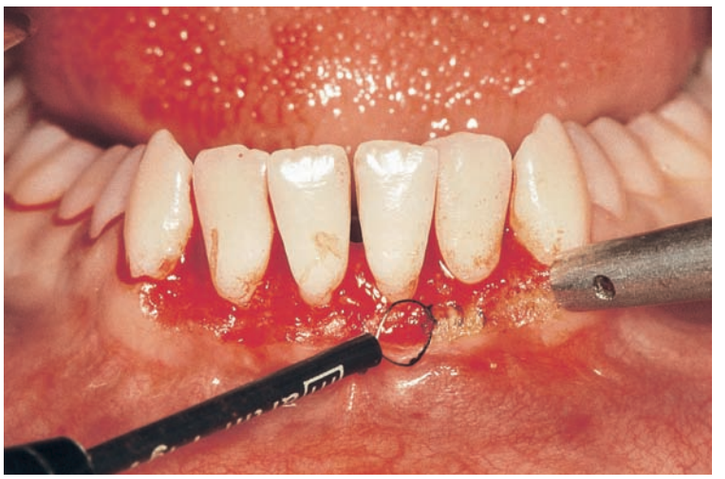


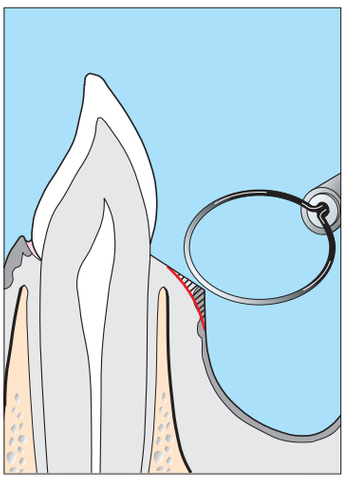
894 Smoothing the "Sharp Edge"

For this fine contouring procedure, an electrosurgical loop is indicated (or possibly also a laser procedure).

The *entire* GV/GP (p. 373) procedure will be more successful following a clean incision with the GV knife than following layer-by-layer removal of hyperplastic tissue using electrosurgery or laser.

Right: Rounding of the incision margin using an electrosurgical loop.





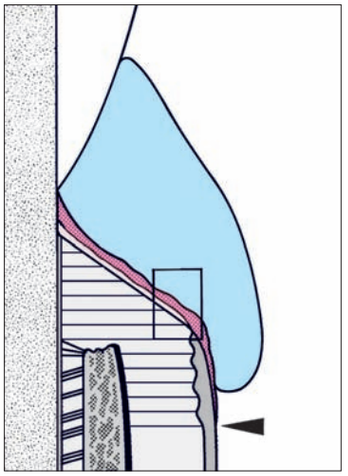
895 Cleansing the Wound

The gingivectomy knife is used to scrape away residual tissue tags or debris; this also serves to round off any sharp tissue margins.

This gingivoplastic contouring is performed from the midline, distally to the first premolars.



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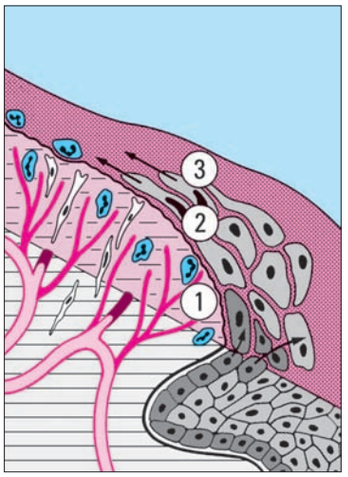




Wound Healing

896 Clinical View Immediately Following GV/GP
The surgical procedure creates a relatively expansive wound surface that must be protected by a periodontal dressing.

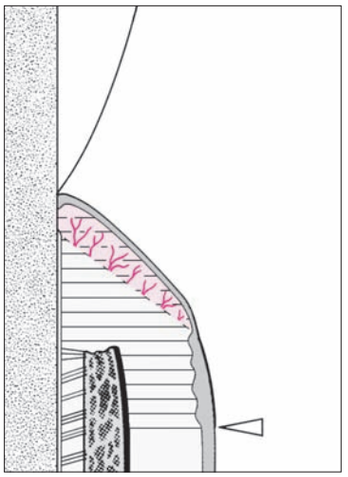
Left: The GV wound is in the region of the attached gingiva. The black arrow indicates the mucogingival junction.
A very thin blood coagulum separates the dressing from the wound.





897 Dressing *In Situ*
The depicted periodontal dressing ("Peripac") must not encroach upon the mobile mucosa, to prevent pressure and ulceration. The dressing should be left *in situ* for 7–10 days.

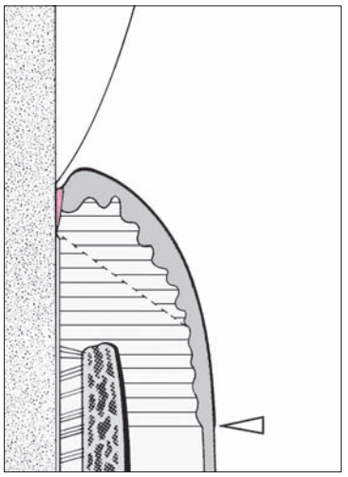
Left: Healing two days after GV.
1 Granulation tissue (PMNs, fibroblasts and new vessels) emigrates from the wound surface into the coagulum
2 Epithelial cells proliferate from the basal cell layer
3 Coagulum





898 Dressing Removal and Dental Prophylaxis after 7 Days
Careful tooth cleansing by means of rubber cups and very fine abrasive paste. The wound surface is cleansed using H_2O_2 (3 %) and a mild spray. The patient can now re-initiate careful home care.

Left: The granulation tissue matures into normal connective tissue; it is covered by a thick epithelial layer. A new epithelial attachment begins to form upon the tooth (root) surface.





899 Six Months after GV/GP
The gingivae are inflammation-free and exhibit a generally physiologic morphology. A very slight recurrence of gingival hyperplasia can be detected between teeth 43, 42 and 41 (papillae).

Left: The tissue is completely regenerated. Normal oral epithelium with rete ridges and a shallow sulcus. A new, short junctional epithelium has differentiated upon the tooth surface.

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Gingivectomy/Gingivoplasty

Summary

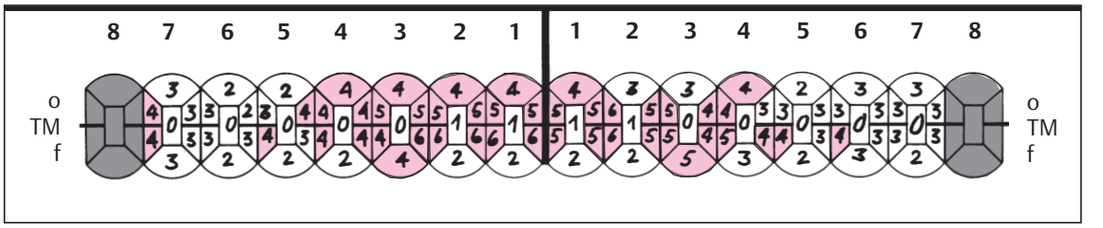
This 18-year-old patient presented with very pronounced hyperplastic gingivitis. During the phases of initial therapy, it became clear that this young man was difficult to motivate. Oral hygiene was miserable, especially in the maxillary interdental regions, also due to the crowding. Orthodontic treatment was recommended, but was initially rejected by the patient. Therefore, the occlusion and articulation were improved somewhat by selective odontoplasty. Despite these unfavorable circumstances, initial therapy and a subsequent surgical procedure (GV/GP) led to an acceptable result in terms of gingival contour and freedom from gingival pockets.

In such cases, it is crucial to maintain a short-interval recall for the repeated motivation and for maintenance of the treatment results in those first years following therapy.

Two years later, and motivated by the success of the gingival treatment, the patient sought orthodontic therapy to correct the malocclusion.



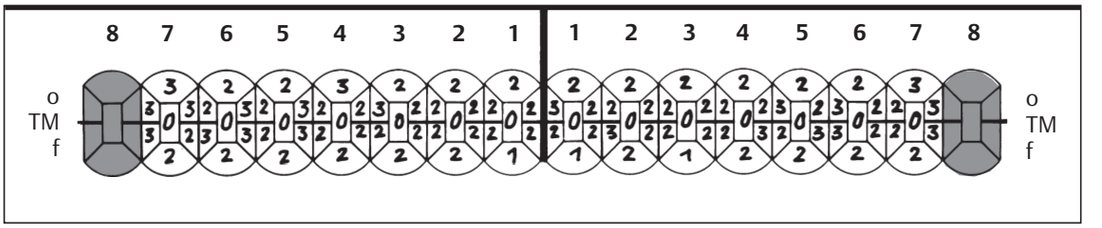
900 Clinical Appearance before Initial Therapy—Gingival Hyperplasia
Severe, non-medication induced hyperplastic gingivitis; plaque, calculus, poor oral hygiene.
The clinical symptoms are limited primarily to the labial surface in the anterior segment of the maxilla and mandible (cf. Fig. 903). Mouth breathing was also an etiologic factor. Poor occlusal relationships, plaque-retentive niches related to tooth position anomalies.



901 Pocket Probing Depths and Tooth Mobility in the Mandible before Initial Therapy
Deep pseudopockets! No attachment loss, i. e., absence of periodontitis.

Before

After



Pocking Probing Depths and Tooth Mobility 7 Years later
Normal probing depths following periodontal therapy and subsequent orthodontic treatment.



902 Clinical Findings 7 Years Post-Operatively
Despite irregular recall there was no recurrence of the hyperplastic gingivitis even though home care by the patient was only moderate. The patient has been able to maintain the area with only minor inflammation. The orthodontic result, while less than optimum, was satisfactory.
The maxilla was also treated by means of GV/GP (see next page).

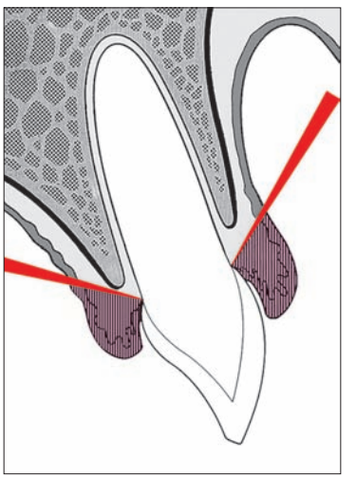
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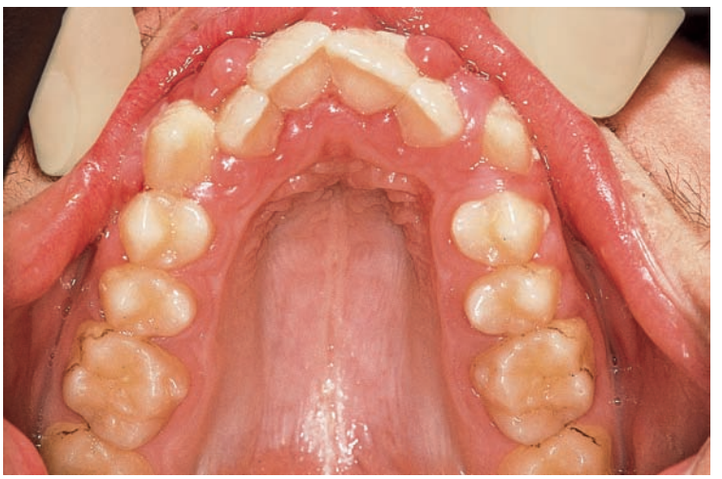
GV/GP, Maxilla: Facial and Palatal

In this 18-year-old patient who presented with extremely severe hyperplastic gingivitis (p. 376), gingivoplasty was performed in the area of the *maxillary* incisors, canines, and premolars both *labially* and *palatally*. The figures below depict primarily the *palatal* aspects of this case. The occlusal photographs demonstrate the associated problems (crowding). A GV/GP surgical procedure can be performed routinely if the palatal vault is high; however, if the palate is flat, a GV results in an *expansive* wound surface that heals slowly (epithelialization occurs at ca. 0.5 mm per day).

Hemorrhage from the incisal foramen can be readily controlled by means of electrocoagulation or injection of a vasoconstrictor-containing anesthetic solution under pressure directly into the canal.

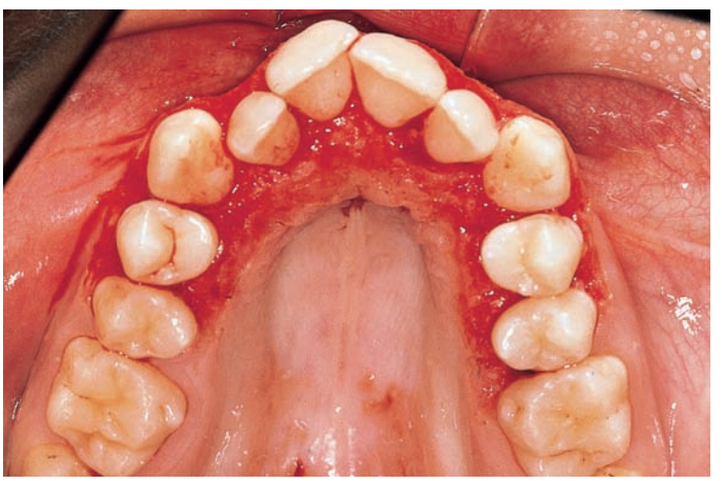
For the maintenance of treatment results in the palatal as well as lingual areas, proper and consistent oral hygiene is especially important (individualized OHI and frequent recall). In this regard, observe the difficulties in the final evaluation (Fig. 905).



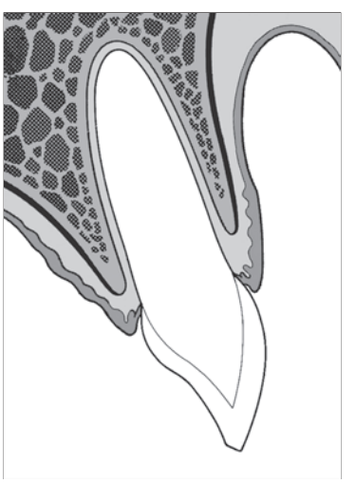


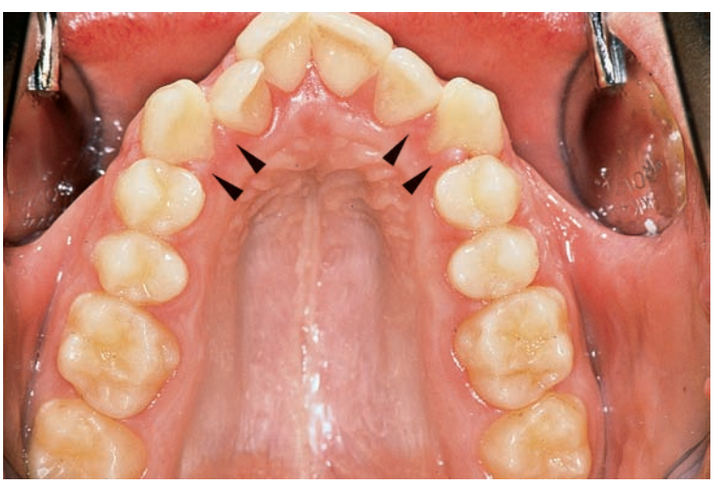
903 Before Initial Therapy
The hyperplastic gingivitis was enhanced on the palatal aspect by the anterior tooth crowding, which also rendered oral hygiene very difficult and led to plaque accumulation and retention.

Left: The diagram shows the anticipated GV/GP incisions. The surgical procedure is performed only after Phase 1 therapy (not depicted here).



904 Following Gingivectomy
The ideal 45° angle incision is also applied in the palatal area if the anatomic situation permits. This can result in expansive open wound surfaces, which are painful and take longer to completely heal. In such cases, it is often necessary to place a second periodontal dressing one week after the surgical procedure.





905 Three Months following Gingivectomy
The patient's home care was inadequate, particularly in difficult-to-reach areas occasioned by tooth position anomalies. In these areas, hyperplastic gingivitis has begun to recur (arrows). Motivated by the success of periodontal therapy, two years later the patient elected to undergo orthodontic treatment (Fig. 902).

Left: The desired healthy situation was achieved following GV/GP.

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GV/GP—Minor Surgical Procedures, Corrective Operations Exposing the Margins of Restorations and Cavity Preparations

Over the years, GV/GP has lost its significance as a radical surgical therapy for periodontitis (*pocket elimination*). However, these procedures maintain their value as corrective, local measures.

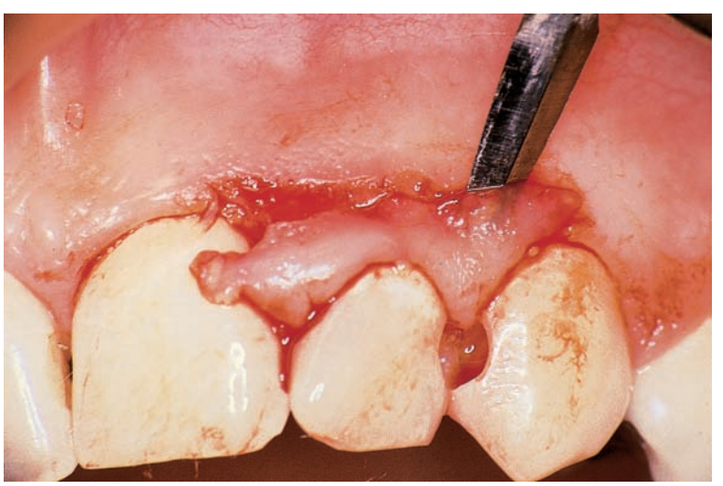
For purely preventive reasons, the margins of restorations and crowns should be located supragingivally (also for esthetic reasons). Precise tooth preparation and impression taking in the subgingival area is also difficult, and for many restorations requiring the rubber dam it is easier to place the dam if the margins of the preparations are supragingival.

In the case presented here, carious lesions that extend into the subgingival area are exposed by a labial and palatal gingivectomy procedure. If properly performed, there should be no subsequent esthetic compromises.

The female patient is otherwise periodontally healthy. Mild gingivitis is obvious near the carious lesions on teeth 21 and 22.



906 Subgingival Caries on Teeth 21, 22 and 23
Without a gingivectomy, it would be impossible to create a proper cavity preparation or apply the rubber dam, which is necessary for elimination of moisture when placing a composite resin restoration.
When treatment is complete, the margin of the restoration should be supragingival.





907 GV/GP Using a Papilla Knife
The gingiva is excised using a GV knife or—as in this case—using a papilla knife. Electrosurgery may also be used to advantage for such minor procedures. Minimal hemorrhage results, and restorations can be placed at the same appointment with a rubber dam in place.

Right: Paired, angled papilla knives (Orban knife GX 11 L & R; Deppeler).



908 After Restorative Treatment and Healing
The apical margins of the four scarcely visible composite restorations lie supragingivally and are therefore accessible for oral hygiene by means of toothbrush and dental floss. This prompts prevention of recurrent caries as well as gingivitis.

Courtesy *A. Bachmann*

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GV/GP—Phenytoin-Induced Gingival Overgrowth

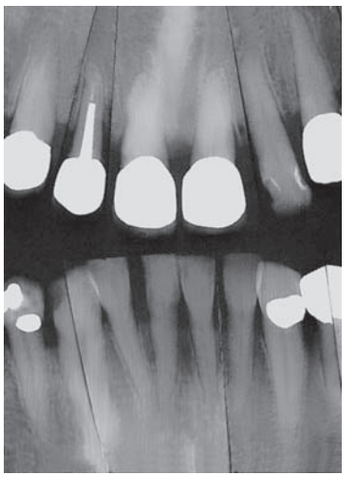
For the treatment of *epilepsy* and other central nervous system disturbances, *diphenylhydantoin-containing drugs* (Hydantoin, Phenytoin) are used. About half of all patients who take these medications on a chronic regimen exhibit esthetically objectionable gingival overgrowth (pharmacogenetic factor; Hassell & Jacoway 1981 a, b). The development of such gingival lesions is enhanced by plaque-induced inflammation. Following initial therapy, GV/GP is the treatment of choice in such cases (GV instruments, electrosurgery, especially also lasers etc.). Plaque control is often difficult and should be supported by chemical plaque-reducing agents.

The case presented below is a 20-year-old slightly retarded female, who had been under chronic hydantoin medication for many years, and who was unable to perform adequate oral hygiene.

Initial findings:

PI: 93%
BOP: 100%
PD: up to 8 mm, mostly pseudopockets

Clinical pictures and radiographs of the anterior teeth: See Fig. 909.



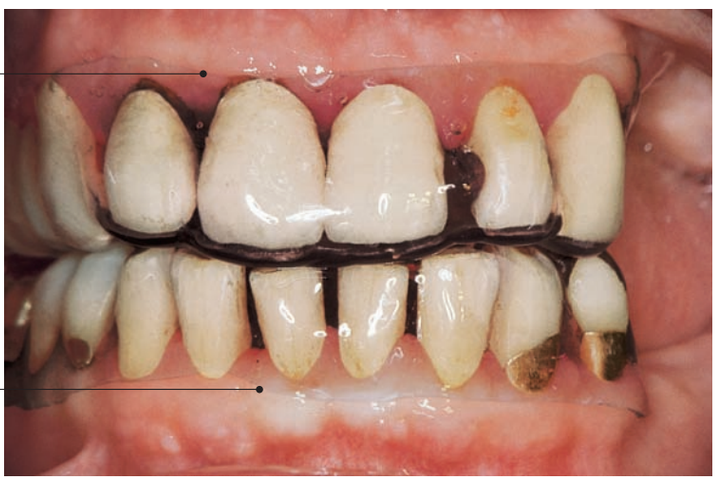


909 Phenytoin-Induced Gingival Overgrowth with Severe Secondary Inflammation
The fibrous gingival overgrowth may have been an etiologic factor in tooth migration.
Following initial therapy (plaque control, reduction of inflammation) the treatment plan included resective gingivectomy (GV) as well as recontouring of the gingival margin via gingivoplasty (GP).

Left: The radiograph reveals incipient bone resorption.



910 Customized Acrylic Tray Used as a Carrier for Chlorhexidine Gel
To prevent plaque accumulation and post-surgical recurrence of the gingival enlargement, customized transparent acrylic trays were fabricated for use as medicament carriers.
The patient, who lacked the dexterity to adequately perform oral hygiene, filled each tray with chlorhexidine gel (1 % chlorhexidine digluconate, e. g., Corsodyl-Gelee). The patient wears these trays for 30 minutes each evening.



911 One Year after GV/GP—Trays *In Situ*
The maxillary incisors have spontaneously regained their proper alignment in the dental arch. Recurrence of the gingival overgrowth was completely inhibited by chemical plaque control (CHX gel, once daily).

The acrylic trays extend beyond the attached gingiva and are well adapted, thus preventing any seepage of the gel into the oral cavity.

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Limitations of GV/GP—Cyclosporine-Induced Gingival Overgrowth

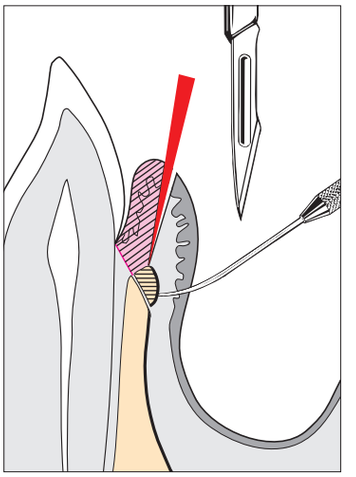
If exostoses exist beneath overgrown gingiva, the situation cannot be treated simply via gingivoplasty, because such treatment would expose bone and periosteum. The possible consequences include delayed healing, and in severe cases even osseous necrosis. Furthermore, in such cases gingivoplasty alone cannot lead to optimum clinical morphology.

A 26-year-old female suffered from pronounced gingival overgrowth, especially in the maxillary and mandibular anterior segments.

Several years previously she encountered an echinococcal infection that led to liver damage that was untreatable. At the young age of 23, she underwent liver transplantation. Since the surgery, she had been on a chronic regimen of cyclosporine-A (Sandimmune, p. 124).

5–6 mm pseudopockets were present at the *initial visit*:
PI: 75 % in incisor and canine regions
BOP: 83 % in incisor and canine regions
TM: 0





912 Severe Gingival Hyperplasia with Cyclosporine Medication
Note severely inflamed and hyperplastic gingival tissues in the mandibular and maxillary anterior segments.

Right: Following supragingival debridement and patient motivation, the condition improved. Under local anesthesia, the enlarged gingival tissues were probed, revealing thickening of the alveolar bone (exostosis). Gingivectomy was contraindicated.







913 Internal Gingivectomy and Osteoplasty
Left: The horizontal incision to create the mucoperiosteal flap was intragingival (internal gingivectomy) in order to thin the gingival tissues.

Middle: Note the bulbous alveolar ridge was visible.

Right: Using small round burs (with copious cooling!) the exostosis was thinned. The tissue flap was also thinned, then repositioned and sutured to place.



914 Final Result
Three months later, one observes healthy gingiva. In the maxilla, where there were no exostoses; a pure gingivoplasty was performed. This excellent therapeutic result can only be maintained by intensive oral hygiene and short-interval professional recall. All pseudopockets have been eliminated. Clinical indices in the anterior sextants:

- PI: 8 %
- BOP: 10 %

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Furcation Involvement—Furcation Treatment

Because of its often bizarre anatomy and morphology, the furcation has been called a *locus minoris resistentiae* (Schroeder and Scherle 1987), a "special case," and a "freak of nature" (Lindhe).
Once the furcation becomes involved with periodontitis, it is a significant risk factor for the tooth because of its macro- and micromorphology (plaque-retentive niche); also, periodontitis in the furcation area often progresses rapidly. For this reason, furcation involvement must be diagnosed at the *earliest possible time.* For clinical assessment, color-coded furcation probes are helpful (NP 2C). The two-dimensional radiographic picture provides but meager information about furcation involvement, especially in the maxilla. This situation may be improved in the future through high resolution spiral CT.
The basic principle of furcation treatment is the same as that for single-rooted teeth—cleaning of the root surface. Depending upon the severity of involvement and the complexity of the furcation morphology, supportive topical application of medicaments may be indicated during purely mechanical treatment.
Advanced/severe furcation involvement may demand radical treatment methods (root amputation, resection). The first step is to clarify and decide whether the involved tooth must be maintained at all. Also worthy of consideration is the possibility of a shortened dental arch (premolar occlusion) or the replacement of a molar by a dental implant.
Last but not least, it is important to realize that costly furcation treatment with endodontic and prosthetic care (F3) can be *avoided* if the furcation involvement is diagnosed early (F1).
This chapter is arranged as follows:

- Embryologic development of multi-rooted teeth—furcations
- Furcation involvement—classifications
- Treatment planning, problems, long-term results
- Furcation therapy—aids in decision making
- Therapeutic possibilities for various case types

The following cases will be depicted:

- F1 in the mandible—scaling /debridement
- F2 in the maxilla—furcationplasty (tooth model)
- F2 in the mandible—GTR technique
- F3 in the mandible—hemisection with extraction
- F3 in the maxilla—root resection without reconstruction
- F3 in the maxilla—root resections with reconstruction
- F3 in the maxilla—"trisection" and maintenance of all roots

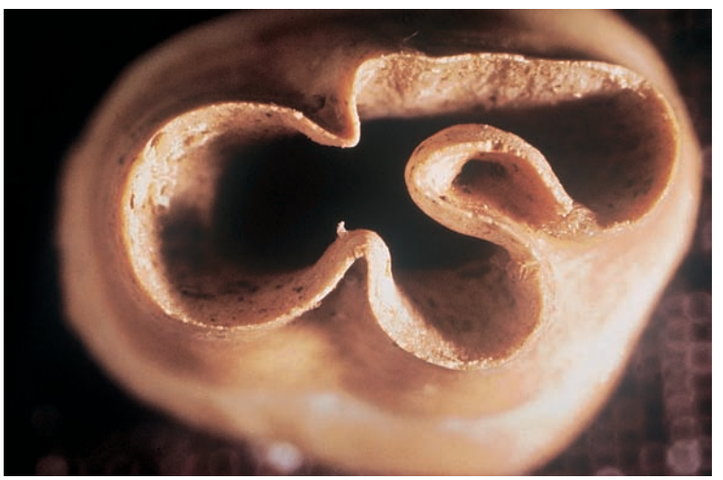
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Embryologic Development of Multi-Rooted Teeth—Furcation

As is the case with all other organs and organ systems in the body, the development of multi-rooted teeth is genetically controlled. Following formation of the tooth crown (enamel), inner and outer enamel epithelia fuse. These become the *Hertwig epithelial root sheath*, which is the basis for development of the tooth root.

During the development of a multi-rooted tooth, this sheath (diaphragm) inverts centrally as a tongue-like process. Depending upon the number of roots, two or three of these tongues unite in the region of the future furcation ceiling. After this fusion, the individual roots are formed as "tubes" identical to single-rooted teeth (Schroeder 1986, Müller & Eger 1998). The fact that the furcation-forming Hertwig epithelial sheath derives from the fused enamel epithelia explains why it is deflected from its new function—root formation including cementum matrix; this embryologic quirk explains why enamel may be formed *apical* to the cementoenamel junction in the form of *enamel projections* and *enamel pearls*, sometimes directly in the furcation.

915 Initial Embryologic Development of the Roots and Furcation Area of a Maxillary Molar
After the formation of a more or less long root trunk, the Hertwig epithelial sheath becomes inverted and leads to dentin formation in the direction of the furcation/furcation roof. The illustration shows the initial development of a three-rooted tooth.



916 Furcation Closed
Following contact or fusion of the three embryologic "tongues," if insufficient closure occurs, minute *pulpoperiodontal canals* may remain.

In addition, enamel pearls may develop in this region (Fig. 917). In the course of time, the furcation region often becomes covered by a thick, rough layer of mixed-fiber cementum.



917 Enamel Pearls
Left: An enamel pearl is visible in the radiograph in the distal furcation area of tooth 16. The hopeless tooth 17 was extracted.

Middle: After extraction, the enamel pearl in the distal furcation of tooth 16 became visible. During periodontal pocket therapy, enamel pearls and projections should be removed.

Right: Histologic view of an enamel pearl. It is covered by a layer of mixed-fiber cementum.







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Furcation Involvement—Classifications

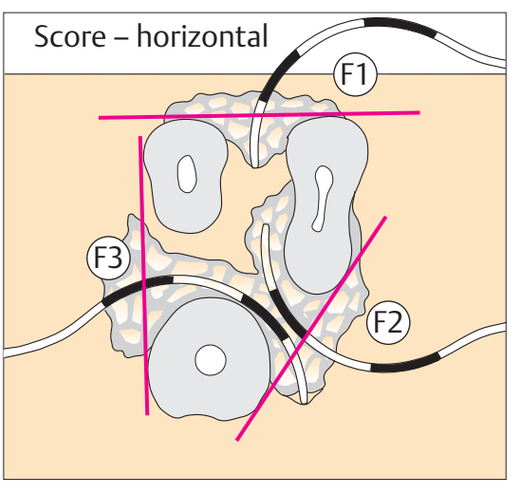
The varying degrees of severity of furcation involvement in multi-rooted teeth have already been discussed in the chapters "Forms" (p. 102) and "Data Collection—Diagnosis—Prognosis" (p. 172).

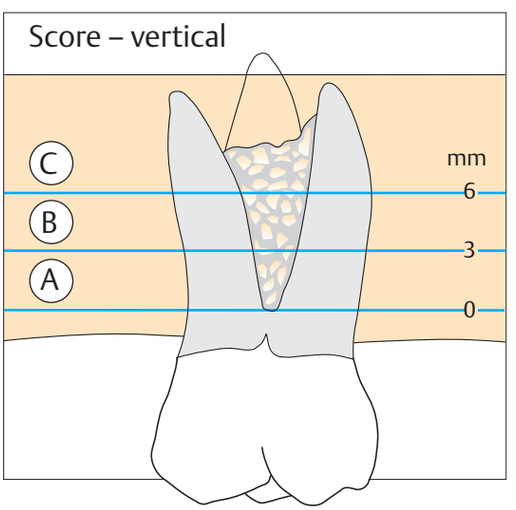
On the basis of horizontal probing, in this *Atlas* we will differentiate degrees of severity F1 to F3, as described by Hamp et al. (1975). It is also important for both diagnosis and treatment planning whether the root trunk between the crown and the divergence of the roots is long or short. Furthermore, the vertical interradicular dimension of the defect between the roof of the furcation and the alveolar bone niveau is important. Tarnow & Fletcher (1994) have categorized this vertical dimension in three subclasses:
A = 1–3 mm, B = 4–6 mm and C > 6 mm. This classification can only be precisely verified during surgery.

Also important for treatment planning is the angle between/among the roots and their proximity to each other. With a long root trunk or with close root proximity, hemisection is seldom possible, and totally impossible if the roots are fused.

Score – Horizontal measurement	
Measured from (an imaginary) tangent (Hamp et al. 1975)	
F0	No horizontal probing depth
F1	1–3 mm
F2	> 3 mm, but not through-and-through
F3	Through-and-through

Score – Vertical measurement	
Measured interradicularly from the furcation roof (Tarnow and Fletcher 1984)	
A	1–3 mm
B	4–6 mm
C	> 6 mm



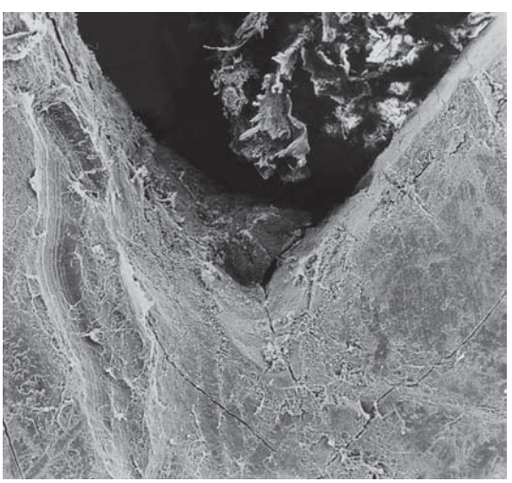


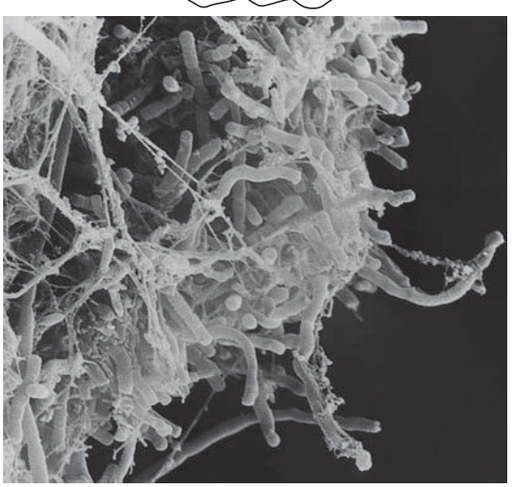
918 Classifications – Horizontal Severity
Left: Horizontal probing depth/severity (F0-F3), measured from the tangent of the three roots, along the roof of the furcation (red tangents in Fig. 919, left).

– Vertical Severity
Right: Probing severities (A, B, C) in the vertical direction measured from the roof of the furcation to the niveau of the alveolar bone of the adjacent two roots (cf. Fig. 919, right).

919 Maxillary Molar—"F-Degree"—Horizontal and Vertical
Left: The furcation probe (Nabers; Hu-Friedy) reveals a complex situation: Between the two buccal roots, **F1**, between the mesiobuccal and palatal root, **F2,** and a class **F3** furcation around the palatal root.

Right: Determining the *vertical* bone loss emanating from the roof of the furcation.
A precise measurement, with direct vision, is only possible during surgery.





920 Roof of the Furcation, SEM—Details
Left: Tooth 16 exhibits class F3 furcation involvement. After curetting the furcation, the tooth was extracted and prepared for SEM examination. Between the palatal (left) and the distobuccal root (right) the furcation is less that 1 mm wide (the width of a curette is 0.95–1.2 mm!).

Right: Plaque bacteria in the furcation region that were *not* eliminated by mechanical therapy.

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Treatment Planning—Problems—Long-Term Results

If a potentially severe furcation involvement is diagnosed during data collection, subsequent treatment planning must include a myriad of factors. These relate not only to the affected tooth, but also to the patient's own feelings about general systemic health and the oral cavity in particular.

As with every treatment for periodontitis, the therapy for furcation problems demands understanding and cooperation/compliance by the patient (patient evaluation, p. 297). For the dentist/periodontist, the treatment of furcation involvement is often truly a "hard nut to crack." *Systemic* and *local* factors, as well as problems that are specific to the furcation therapy, will be of significance (Al-Shammari et al. 2001):

General and Systemic Considerations

- General systemic health (diseases, risk factors)
- Patient's own feelings about oral health (oral hygiene, compliance)
- Patient's own desires
- Financial circumstances

Local Factors

- Overall condition of the dentition: Periodontitis, overall caries incidence, missing teeth, quality of existing restorations
- Prosthetic significance of the tooth or its individual roots
- Condition of the crown of the affected tooth: Carious destruction, expansive restorations
- Involvement of maxillary or mandibular molars
- Severity of the horizontal involvement (F1, F2, F3)
- Severity of the vertical involvement (A, B, C)
- Length of the root trunk?
- Length of the individual roots and level of periodontal attachment
- Angle of opening between the roots, i. e., broad root divergence or tight root proximity, or fusion
- Tooth mobility (p. 174)
- Crown-root length relationships
- Root surface irregularities (hour-glass shape), depressions, overhangs
- Enamel projections, pearls
- Fissured mixed-fiber cementum in the furcation region
- Mini-canals between the furcation roof and the pulp
- Endodontic treatment possibilities
- Possibilities for stable build-ups (following resective treatment methods)
- Bone quality and localization (maxilla/mandible) for a possible dental implant as an alternative
- Long-term prognosis of the various treatment possibilities?

921 Resective Furcation Treatment—Long-Term Results

The success rate following resective treatment in terms of maintaining individual roots is actually quite high. Failure is seldom due only to periodontal circumstances, but relatively frequently to endodontic and prosthetic problems.

Perio Periodontitis
Endo Endodontics
R-Frac Root fracture
St abut Strategic abutment

Modified from *J. Lindhe et al. 1997*

Success rates Authors		Observation time – years	Number of teeth	Success ▼	Failure ▼	Cause of failure: Perio	Endo	R. frac.	St. abut.
Bergenholtz	1972	2 – 10	45	94%	6%	4%	2%	–	–
Hamp & Nyman	1975	5	87	100%	0%	–	–	–	–
Langer et al.	1981	10	100	62%	38%	10%	7%	18%	3%
Erpenstein	1983	4 – 7	34	91%	9%	3%	6%	–	–
Bühler	1988	10	28	68%	32%	7%	18%	3.5%	3.5%
Carnevale et al.	1991	3 – 11	488	96%	4%	0.5%	0.9%	1.8%	0.9%
Basten et al.	1996	2 – 23	49	92%	8%	2%	4%	–	2%

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Furcation Treatment—Aids for Decision Making

Furcation involvement F1

Subgingival scaling and root planing is usually adequate therapy with shallow pockets and F1 furcation involvement. Any enamel projections or pearls in the furcation should be removed by selective grinding. In some cases the furcation entrance should be widened somewhat and then polished (odontoplasty, p. 388).

In the maxilla, however, these minor treatments are not always unproblematic, when the F1 involvement is between a buccal and the palatal root, accessible only palatally ("distal" or "mesial" furcation).

Depending upon the clinical findings and treatment plan for the adjacent teeth, treatment for an F1 involvement (pocket) may be combined with a gingivectomy or an access flap (modified Widman procedure). Adequate oral hygiene using marginal brushes must be demonstrated and re-evaluated.

Furcation involvement F2

The therapeutic possibilities for this class of furcation involvement are broad, indeed. In most cases, the treatment of choice is flap surgery to provide direct vision of the furcation morphology and the depth of involvement, and to permit scaling and root planing with *direct vision*. Important decisions such as whether to treat the furcation involvement using bone or bone replacement materials, and/or a membrane technique can only be made following flap reflection. Corrective odontoplasty in the marginal area can also serve to widen the furcation entrance and make it more accessible for plaque control. In cases with *deep* F2 involvement (especially in the maxilla), tooth hemisection or trisection may be indicated.

Furcation involvement F3

With a "through-and-through" furcation involvement, *conservative* procedures typically used for F1 or F2 cases are considerably less promising. The same is true for the use of regenerative methods, which have so far not provided promising results. There are several *resective* therapeutic possibilities: Resection of one root; hemi- or trisection with maintenance of one or more roots (p. 392, p. 396). One additional method, particularly indicated for advanced (F3) furcation involvement in the mandible, is "tunneling," which brings with it the advantage that no endodontic pre-treatment is required; the disadvantage is the difficulty for plaque control by the patient and the subsequent danger of caries as well as pulpal infection via pulpoperiodontal canals. The failure rate after tunneling procedures is relatively high over the long term.

	Furcation diagnosis	F1		F2		F3	
		Mand.	Maxilla	Mand.	Maxilla	Mand.	Maxilla
A	**Completely dentulous**	●	●	● R	● R*	● T / ▲	● ▲
B	**Prosthetic abutments** Strategically important teeth	● (R)	●	● R	● R / ▲	▲	▲
C	**Incomplete dentition** Missing teeth, diastemata	●	●	● △	● △	▲ ex	▲ ex
D	**"Palliative therapy"** Compliance, inadequate oral hygiene	○	○	○	○	○ ex	○ ex

922 Aids for Decision-Making—Possibilities for Treating the Furcation

●	Tooth maintenance
○	Tooth maintenance?
R	Regenerative
(R)	Regenerative?
T	Tunneling
▲	Radical
△	Radical?
ex	Extraction (dental implant?)

Therapeutic Possibilities—Consideration of the Furcation Defect

Careful consideration of the local and systemic conditions permit the recommendation of the procedures denoted above, for various initial clinical situations and varying furcation involvements (F1 to F3) and also with regard to maxillary or mandibular involvement.

A In fully dentulous patients, conservative treatment methods and maintenance of teeth should be attempted.

B If the case involves furcation invasion on a tooth that is planned as a prosthetic abutment, the therapeutic success must be predictable, i. e., the treatment method most likely of success should be selected.

C If some type of dental replacement is planned, more severe furcation involvement should be considered for tooth extraction.

D If the patient lacks interest and desire (compliance, oral hygiene), only palliative/conservative treatment (scaling) is indicated, followed by more radical procedures (extraction).

Possible alternatives to the therapies listed above include extraction of the affected tooth if it is not necessary for masticatory function (shortened dental arch), or its replacement with a dental implant, and if necessary eventually even ridge augmentation or a sinus lift procedure.

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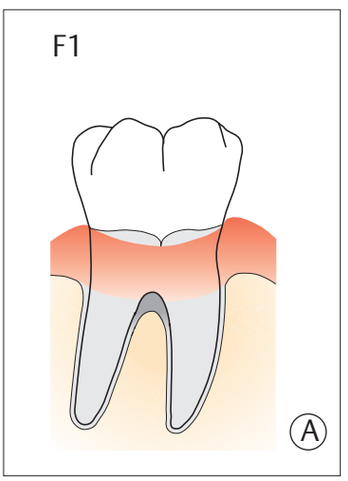
Therapeutic Possibilities for Various Cases

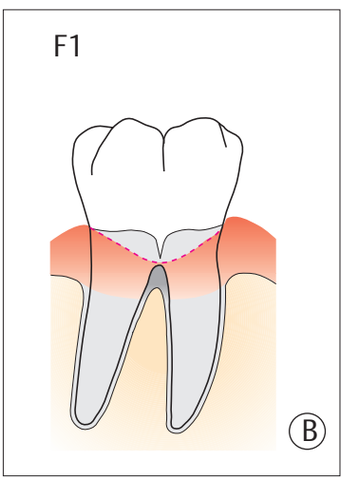
Depending upon the severity of furcation involvement (F1-F3; A-C), as well as the tooth position in either the maxilla or the mandible, various therapeutic methods may be attempted. Even with identical involvement in the same arch or even on the same tooth, diverse treatment methods are possible, depending upon the situation in a dentulous arch (abutment tooth), systemic health of the patient, and his/her oral hygiene compliance.

Depicted below in tables and diagrams are the various possible treatments:

- *Closed or open scaling*, with odontoplasty and possibly osteoplasty
- *Tunneling* procedures
- *Regenerative methods*:
  – GTR, "fillers," growth factors, matrix proteins
- *Resective methods*:
  – Hemisection with maintenance of one root
  – "Premolarization," with maintenance of both roots
  – Root amputation
  – Trisection in the maxilla, with maintenance of all roots
- Tooth extraction, replacement with a dental implant?

923 Closed Therapy—Scaling
Initial furcation involvement (F1) with a *deeply lying furcation* (long root trunk): The region can be *conservatively* treated via scaling and root planing with hand instruments or ultrasonic devices (**A**). If the furcation is relatively *superficial* in its location, it can readily be treated *radically* by pocket elimination (GV/GP). It can then be easily cleaned (**B**). At the same time, enamel projections or pearls can be removed, also enhancing access to the furcation area for oral hygiene.





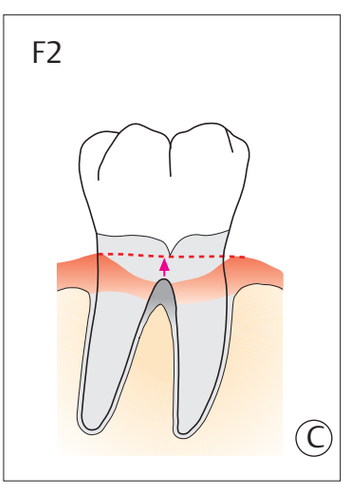
Maintenance of the Entire Natural Tooth

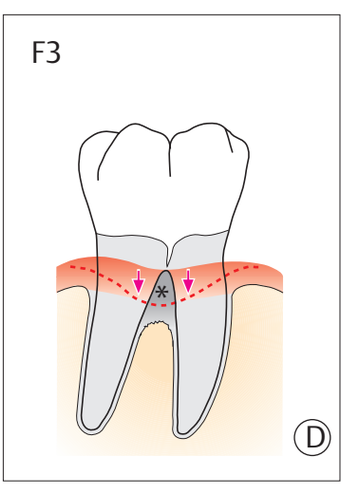
F1

Scaling
– conservative
– radical

924 Open Treatment in the Mandible—Access Flaps
With deeper furcation invasion (F2), calculus is often present upon the rough cementum surface. In such cases, open treatment is recommended. After reflecting soft tissue flaps, the furcation surfaces are cleaned and planed and the flaps are repositioned *coronally* to the furcation entrance (**C**).

In the case of through-and-through furcations (F3), one may consider a tunneling procedure with *apically repositioned* flaps (**D**).





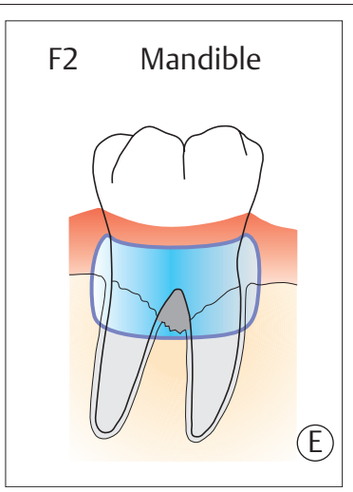
F2

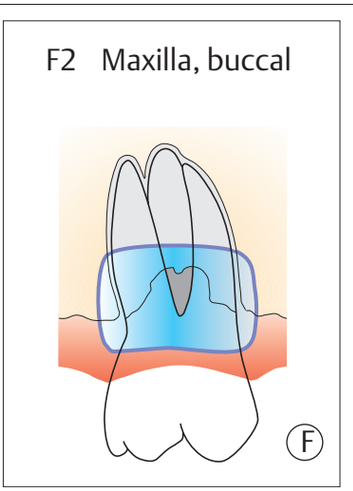
Flap Surgery

F3

"Tunnelling"

925 Furcation Treatment by Means of Regenerative Methods
With F2 furcation invasions, it is reasonable to attempt regenerative methods using GTR techniques, and also filling the defect with bone or bone replacement materials, and even the incorporation of growth factors or matrix proteins to completely heal the furcation defect. The tooth, itself, remains stable and vital.
Such "heroic" measures are generally more successful in the mandible (left) than in the maxilla (only in the buccal furcation!).





F1/F2

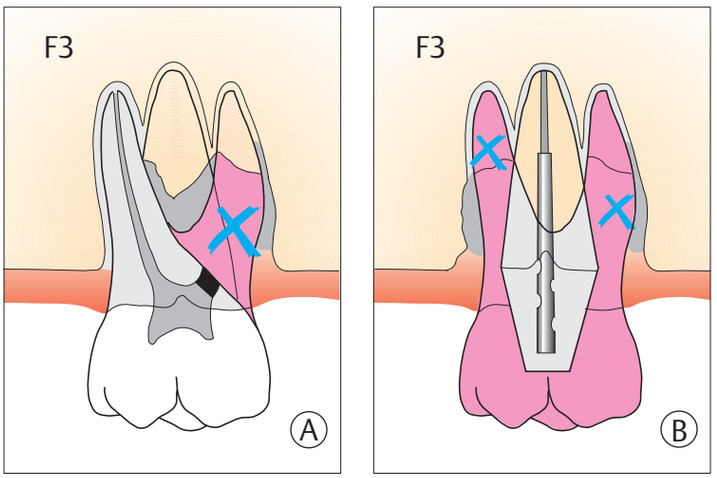
Regenerative—GTR

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Maintenance of Non-Vital Roots

F3

Root Amputation



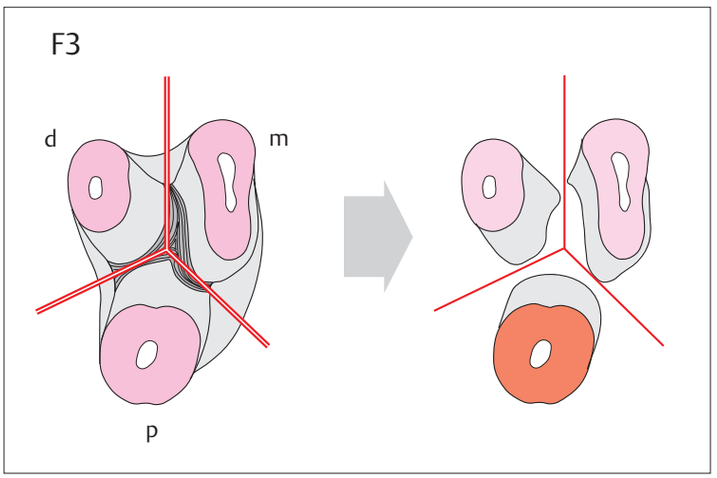
926 Amputation of Individual Roots in the Maxilla
If a class F3 furcation involvement exists on a maxillary molar involving two or three roots, it may be possible to remove a single root (**A**), with maintenance of the entire tooth crown, or two roots may be removed (**B**).

A Amputation of a buccal root (left)
B Amputation of both buccal roots; pin build-up upon the palatal root (right)

Maxilla

F3

Trisection
– diagnostic
– therapeutic

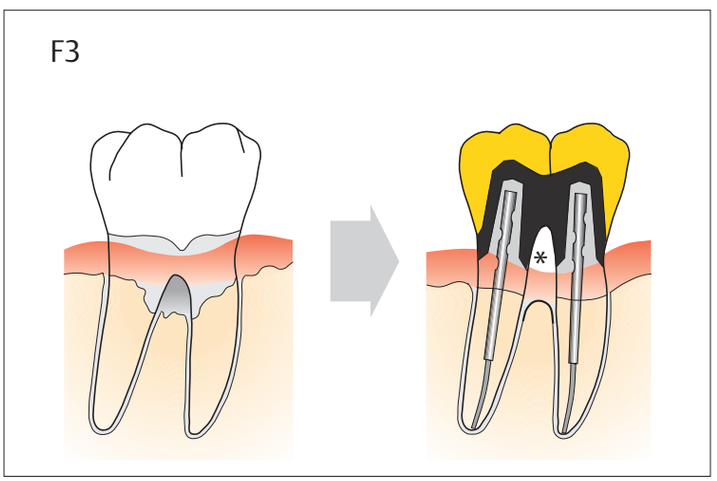


927 Trisection in the Maxilla—Maintenance of all Three Roots
If there exists through-and-through furcation involvement between/among all three roots, one should *always* consider trisection of the tooth. After the separation, it is possible to evaluate the remaining fundament of each individual root and to evaluate its value in terms of any anticipated prosthetic reconstruction. Because the individual roots are usually highly mobile and tend to migrate, stabilization is required.

Mandible

F3

Hemisection
– "Premolarization"

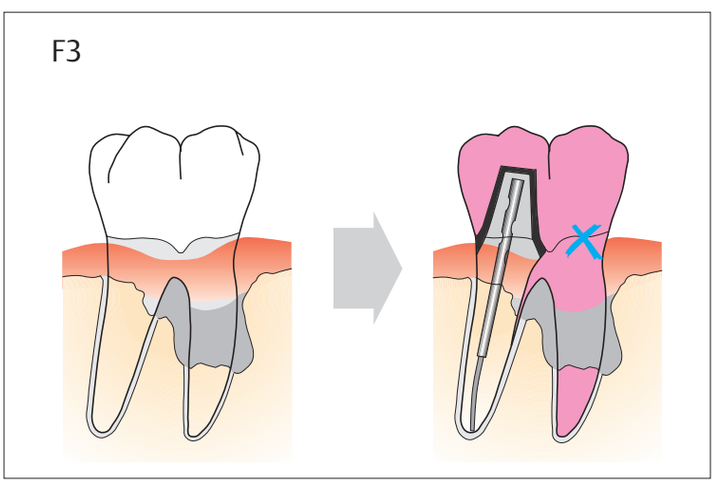


928 "Premolarization" in the Mandible—Maintenance of both Roots
If a class F3 furcation involvement is present in the mandible, and if there are no periapical considerations, the two roots are not in close proximity, are relatively long (remaining support), and the molar has a short root trunk, it should be possible after endodontic therapy to "premolarize" the tooth by means of hemisection. Subsequent definitive prosthetic reconstruction essentially splints the two roots.

Mandible

F3

Hemisection
– with extraction



929 Hemisection in the Mandible—Maintenance of one Root
Extraction of the poorly supported root is often indicated in class F3 involvement in the mandible and sometimes even in advanced F2 cases, as well as with pronounced, difficult to treat periapical lesions that are often combined with furcation defects. Extraction of one root can be performed after hemisection of the tooth. Definitive prosthetic treatment is subsequently required (cf. Fig. 948).

Right: Distal root = abutment.

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Furcation Involvement (F1) in the Mandible—Odontoplasty and Scaling

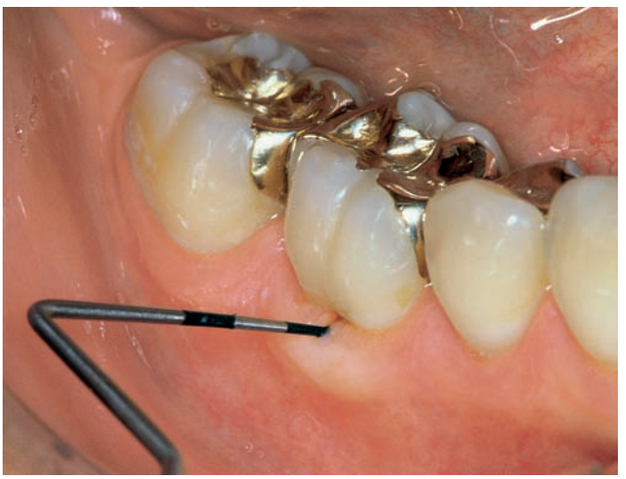
Even mild periodontal attachment loss can bring with it more severe problems if the furcation is relatively high coronally (short root trunk). Plaque-retentive niches exist that are often scarcely attainable with oral hygiene aids. In addition, the common enamel projections and enamel pearls apical to the cementoenamel junction at the entrance to a furcation represent weak points for the epithelial attachment. This complicates plaque control yet further.

As each tooth is examined, these sorts of "furcation anomalies" must be diagnosed. In most cases they can be corrected by means of simple odontoplasty/furcationplasty, without reflection of soft tissue flaps. This permits access to the furcation entrance for adequate hygiene. Any tooth surfaces that are adjusted must also be immediately well polished (15 µm diamond, as well as rubber cup polishers and prophy paste). Finally, the treated surfaces should be fluoridated.

Following such odontoplasty, hypersensitive dentin often persists for a short time.

930 The Furcation as a Plaque-Retention Niche—F1 Case
The furcation can be probed to ca. 3 mm (horizontal) from the buccal approach. Behind an overhanging enamel projection, there is a crater-like retentive area, which cannot be mechanically instrumented without a furcationplasty.

Right: A similar mandibular molar has been hemisectioned to reveal the furcation region. The plaque-retentive area (*) and the anticipated furcationplasty are shown.

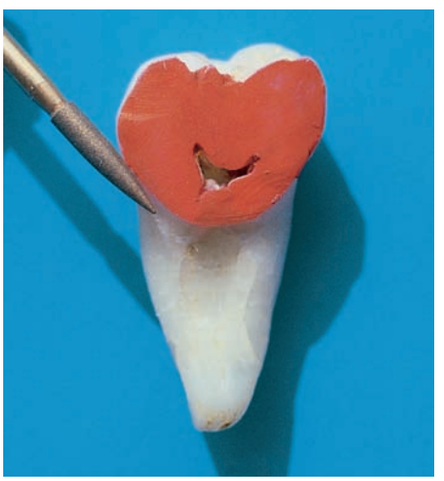




931 Odontoplasty of the Furcation Entrance
Using coarse diamond burs initially, the buccal overhang is removed and the entrance to the furcation is widened.
The furcation is now accessible for professional plaque control (scaling).

Right: Polishing of the furcation entrance using a 15µm diamond.





932 Oral Hygiene Using Marginal Brushes
The entrance to the furcation is now easy to clean by the patient using marginal brushes, solobrushes or single tuft brushes (depicted is the Lactona No. 27). These critical regions must be regularly checked at each recall appointment.

Right: This section through the tooth clearly shows that the single-tuft brush can effectively access the furcation entrance.





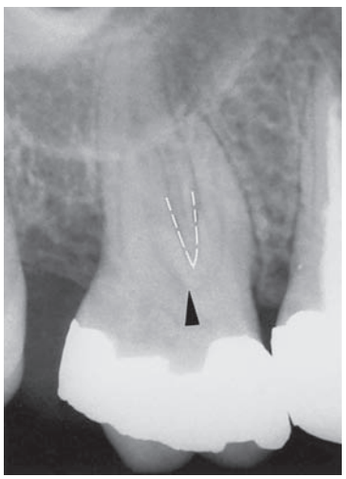
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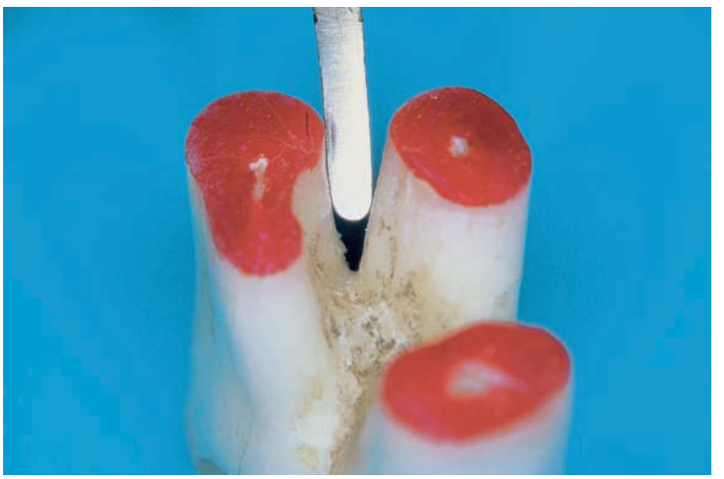
Furcation Involvement, F2, in the Maxilla—Furcationplasty

Several therapeutic options are available for the treatment of F2 furcation involvement in both maxilla and mandible, depending upon the morphologic situation. One may employ purely *conservative* methods as with F1 cases and simply open the plaque-retentive areas (Fig. 934), or one may intervene *surgically*, e. g., using GTR techniques (see next case) in an effort to induce tissue regeneration in the furcation. If the F2 involvement is on a maxillary molar between/among all three roots, some authors suggest hemisection or trisection (Carnevale et al. 1995).

It is common in F2 involvement that the roots join the root trunk forming narrow interradicular areas. In such circumstances, root planing is impossible even with the narrowest curettes. If the overall treatment plan calls for the maintenance of the tooth, the problem can often be solved by odontoplasty (furcationplasty). Initially using course diamonds (75–40 µm), the furcation entrance is widened such that it can be effectively treated with a slender curette. The area is then polished with the 15 µm diamond.

It is best to perform grade 2 furcationplasty carefully and with direct vision, i. e., after careful reflection of the gingiva.

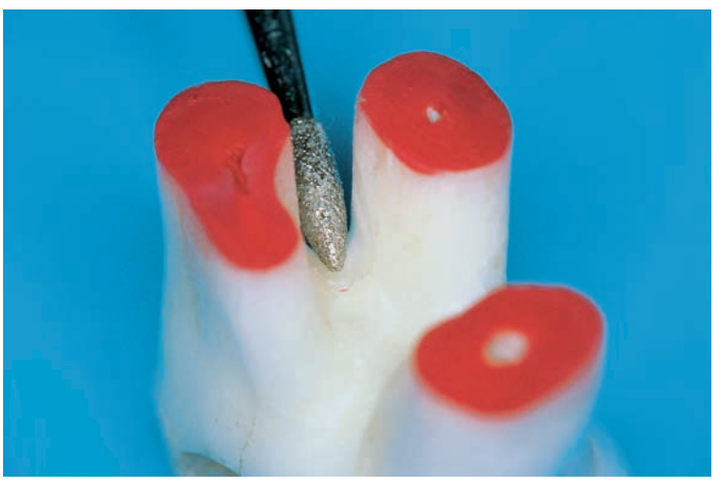




933 Narrow Buccal Furcation
Even the extremely slender (only 0.9 mm wide) curette cannot reach the roof of the affected furcation.

Left: The radiograph reveals the closely spaced buccal roots (arrow). However, the severity of the furcation involvement (periodontal pocket, plaque, calculus) cannot be ascertained radiographically especially in three-rooted maxillary molars.

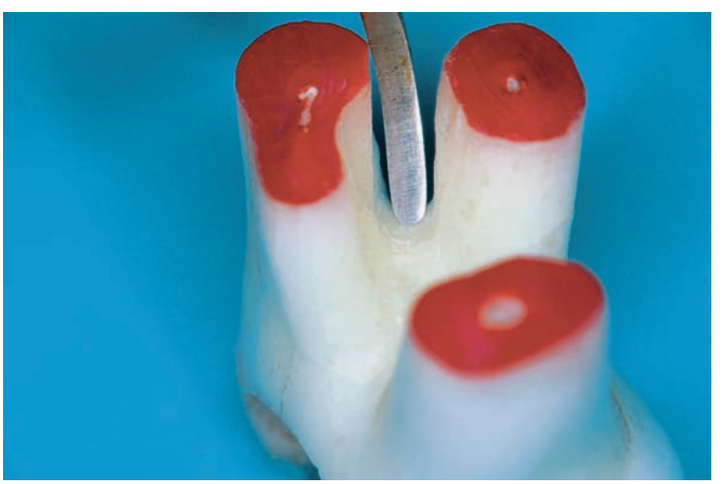




934 Furcationplasty
If clinical access is good, the procedure can be performed "closed," but it is easier to do after reflecting a small flap. Various instruments can be used to open such an involved furcation so curettes can be used for cleaning.

Left:
- **PerioSet diamonds**
- **KaVo sonic scaler**
- **Polishing diamonds**

Furcations that have undergone odontoplasty are particularly susceptible to caries!



935 Hygiene made Possible
Debridement and root planing with fine curettes can now be performed during the course of periodontal therapy as well as at each recall appointment.

In such cases, cervical sensitivity may be particularly troublesome and persistent. The treated furcation must be polished and fluoridated. This will enhance surface remineralization, reduce plaque formation and lessen cervical sensitivity.

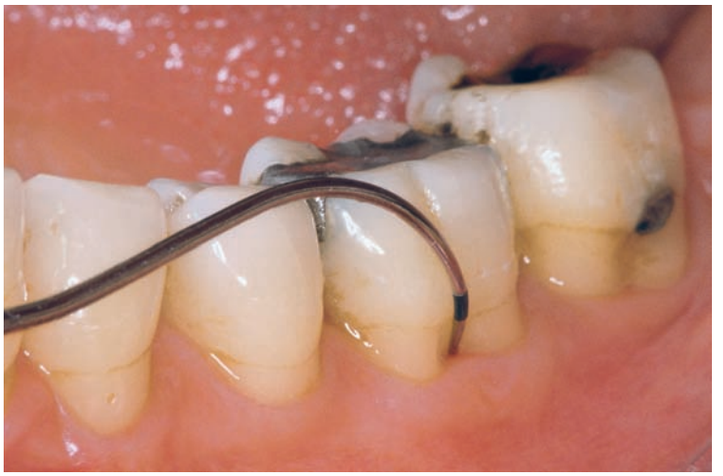
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Furcation Involvement, F2, in the Mandible—GTR Technique

Various treatment methods are available for treating an F2 furcation involvement in the mandible. In cases where the involvement is not too deep, simple flap reflection and cleaning and planing of the furcation region is usually successful. A further possibility includes efforts to use regenerative techniques (in this case GTR) to close the furcation or at least to change the F2 defect into an F1 involvement.

With *very deep* F2 cases (e.g., F2/C), even more radical methods such as hemisection have been recommended (Carnevale and Kaldahl 2000). A disadvantage is the necessity for endodontic therapy before the periodontal procedure. A prerequisite for retaining both roots is that they are not in close proximity (root angle) or fused.

In this case, a 45-year-old female, furcation treatment by means of GTR will be depicted. After initial therapy, tooth 36 exhibited a class F2 furcation involvement. An enamel projection was present in the furcation, emanating from the cementoenamel junction (Fig. 937). Attachment loss in the interdental region was only slight. Tooth 37 is somewhat super-erupted, because there was no maxillary antagonist.





936 Initial Clinical View Prior to Initial Therapy
The gingivae are for the most part inflammation-free. On tooth 36, however, a buccal furcation involvement (F2) was detected. The Nabers probe penetrated ca. 6 mm into the furcation, which was not, however, through-and-through.

Right: The radiograph reveals a radiolucency in the furcation area, but this appearance does not reveal the horizontal extent of the furcation invasion.





937 Soft Tissue Flap Reflection
After removal of the granulation tissue, the expansive enamel projection near the cementoenamel junction within the furcation becomes visible. This was removed using diamond burs. The roof of the furcation and all root surfaces and osseous surfaces were cleaned and planed.

Right: Fully prepared furcation prior to application of the membrane. The enamel projection was eliminated and the furcation itself slightly widened.



938 Gore Membrane *In Situ*
The non-resorbable membrane (ePTFE; p. 339) is placed with its coronal border at the level of the CEJ, and secured with Gore sutures. In the apical area, the membrane should extend at least 2–3 mm beyond the defect.

Important: Successful regeneration requires *stem cells* deriving from the periodontal ligament and interradicular bone. These tissues must be “freshened” before placing the membrane (blood supply).

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939 Flap Repositioning—Sutures
In particular the Gore membranes must be completely covered by the repositioned soft tissue flaps; the wound must be closed *without tension* on the flaps, but must nevertheless be as *tightly* closed as possible. The black 4–0 silk sutures are removed after 10 days. Until the time of membrane removal, the patient rinses with CHX solution, and continues this hygiene regimen for ca. 2 months; during this time, no tooth brushing at the *surgical site!*





940 Second Surgical Procedure—Membrane Removal
After 4–6 weeks, during a second surgical procedure at the same site, soft tissue flaps are again reflected. The non-resorbable Gore-Tex membrane is then carefully removed.

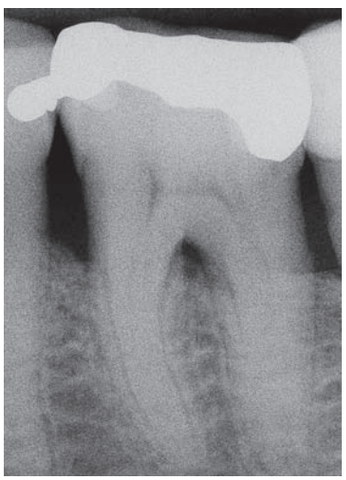
Left: 2–3 weeks following the initial surgery, there is minimal exposure of the membrane at the gingival margin. The mild erythema of the gingiva indicates a foreign body reaction or an incipient infection.





941 Immediately Following Membrane Removal
Within the furcation and adjacent portions of the buccal aspect of the root surface, one notes a young and immature tissue, and the hope is that it will differentiate into mature periodontal structures. Following membrane removal, the surgical site is again securely covered for at least 10 days.

Left: The CHX treatment (spray or gel) must be continued for 2–3 months.





942 Clinical and Radiographic Views, 10 Years later
The furcation showed clinical measurements horizontally and vertically of 2 mm (furcation F1, A). No additional attachment loss had occurred.

Left: Ten years after the initial surgical procedure, the radiograph depicts a minimally more dense osseous structure compared to the initial condition (Fig. 936, right), but not complete osseous regeneration within the furcation.

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Furcation Involvement, F3, in the Mandible—Hemisection with Extraction

If the furcation of a mandibular molar exhibits through-and-through involvement (F3), there are three therapeutic possibilities: 1. tunneling, 2. premolarization, and 3. hemisection with extraction. The indication for tunneling is very limited because there is a high risk of caries formation in the furcation roof if optimum oral hygiene is not possible. Hemisection with maintenance of the prognostically better root is, today, the most frequent method of choice. This procedure will be demonstrated in a 45-year-old female. The mesial root will be removed because in addition to periodontal involvement it exhibited endodontic problems. The hemisection and extraction were performed following gingival flap reflection. During the phase of consolidation, it was necessary to place a temporary bridge to prevent migration of the remaining root. Seven months after the surgical procedure, definitive restorative work could be performed. The patient exhibited good oral hygiene.

Clinical findings before hemisection, in the entire dentition:
PI: 10% BOP: 15% TM: 1
Clinical photographs and radiographs are depicted below.

943 Molar Tooth 46 Exhibiting Furcation Involvement, F3
The tooth crown is extensively destroyed, and the furcation is through-and-through (F3).

Right: The radiograph depicts a pronounced radiolucency on the mesial root of tooth 46. From a purely endodontic standpoint, it is reasonable to maintain only the distal root.
The distal root must be endodontically treated before the clinical hemisection of the tooth.

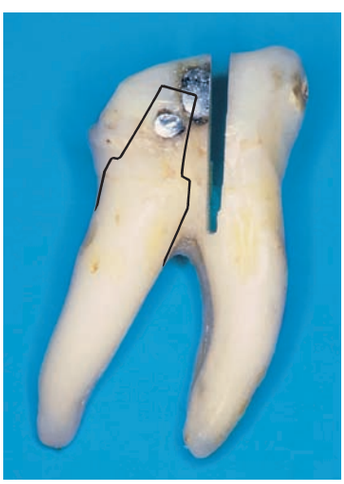




944 Flap Reflection, Hemisection
Sectioning of the tooth crown is performed using a coarse diamond bur.

Right: The point of sectioning is purposefully located nearer to the mesial root, which will be extracted, in order to avoid any damage to the distal root. A result of this procedure is that following hemisection a detectable overhang persists. This is removed during preliminary tooth preparation.

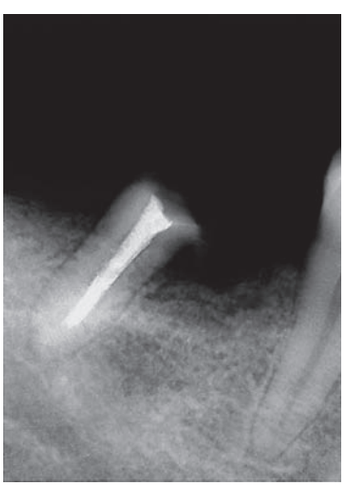




945 Following Extraction of the Mesial Root
The sharp edges of the remaining part of the tooth must be smoothed in the sense of a preliminary tooth preparation.

Right: The radiograph depicts the distal segment of the molar tooth, which appears as a single-rooted premolar with a distinct mesial inclination.





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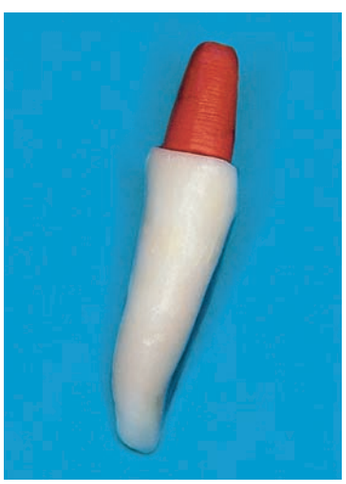


946 Six Weeks after Hemisection
After the initial healing of the extraction wound, the distal portion of tooth 46 is prepared to receive a full coverage crown including an intrapulpal screw and a composite resin build-up. The mesial bridge abutment, tooth 45, for the planned fixed bridge replacement is prepared to receive a $^{3}/_{4}$ crown.



947 Temporary Acrylic Bridge
The temporary bridge should be seated as soon as possible because of the tendency for the remaining abutments to migrate.

Contour and occlusion of the temporary bridge should anticipate the definitive restoration.





948 Seven Months after Hemisection—Definitive Bridge Placement
More than 6 months following tooth hemisection, the definitive fixed bridge can be seated. It extends from premolar tooth 45 to the distal root segment of tooth 46.

Left: The model clearly indicates the premolar-like appearance of the prepared distal root of molar tooth 46.





949 Final Appraisal—Prognosis
Fixed bridge: The pontic ("modified ridge lap"; p. 502) barely touches the alveolar ridge. The interdental spaces are configured such that interdental plaque control is possible using spiral brushes.

Left: Radiograph depicting the fixed bridge upon the single-rooted molar. This result has been in the patient's mouth for 12 years!

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Furcation Involvement, F3, in the Maxilla—Root Resection with Reconstruction

When dealing with furcation involvement in three-rooted maxillary molars, it may be possible to retain one or two roots, or to retain all three roots, depending on the situation.

The case presented here is a 39-year-old male, who presented with severe mobility and class F3 furcation involvement in the maxillary left sextant. Tooth 27 had to be extracted. Both buccal roots and the buccal portion of the crown of tooth 26 were resected. The remaining palatal root of 26 and the two premolars (24 and 25) were first treated with temporary crowns and later with definitive full-coverage restorations. Because of the severe mobility of the palatal portion of tooth 26, it was splinted with three crowns.

Clinical findings after supragingival scaling in quadrant 2:

PI: 50% BOP: 37% TM: 3 on 26, 27

Clinical views and radiographic status are depicted below.

950 Initial Clinical View
Before initial therapy, the pocket probing depth on the proximal surfaces of molars 26 and 27 was 7 mm. On both of these teeth, all furcations could be probed through-and-through (F3). Both teeth were highly mobile (TM = 3).

The premolar teeth 24 and 25 exhibited old and inadequate restorations.



951 Radiographic Findings
Note the especially pronounced bone loss on the buccal roots of the molars. Note also open furcations between all of the roots; F3-involvement.

Very apparent is the huge palatal root of tooth 26.



Before

952 Surgical View
Soft tissue flap reflection following initial therapy and root planing of the molars. It is clear that none of the 3 roots of the second molar (tooth 27) can be retained.

On the first molar, widely open furcations demand that the buccal roots be resected. Only the palatal root of the first molar can be maintained as an abutment for a more permanent restoration.



Courtesy *R. Metzger*

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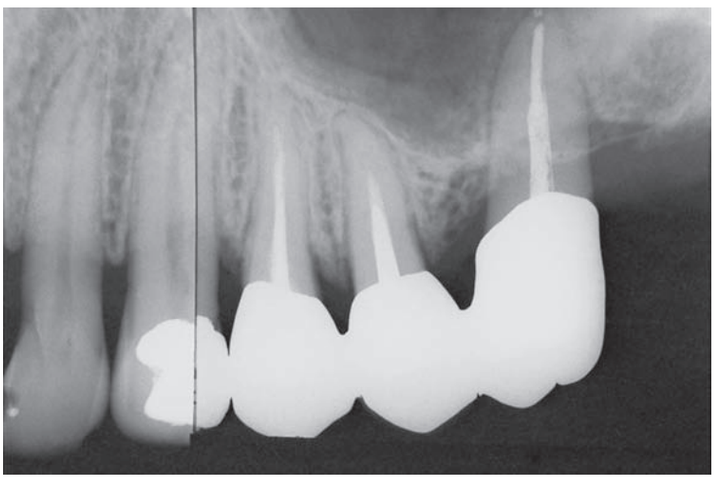
953 Six Weeks after Surgical Resection
Teeth 24, 25 and the palatal portion of tooth 26 have been prepared to receive a temporary bridge. On the buccal surface of tooth 26, one observes the AH-26 root canal filling material in the previous pulp chamber.

For the definitive bridgework, this palatal root will be strengthened using a pin build-up.



954 Definitive Reconstruction
Eight months after the original surgical procedure, teeth 24, 25 and the palatal root of 26 were definitively prepared to receive full crowns. Because of the remaining high tooth mobility (grade 3), the root of tooth 26 and its unfavorable palatal position, the 3 individual crowns were splinted (patient comfort). The broadly open interdental areas permit interdental hygiene.

After



955 Radiographic Appearance, 8 Months Post-Surgically
The crowns on teeth 24 and 25 are splinted. Because of its palatal and tipped position, tooth 26 is attached to the premolars via a precision attachment.

Tooth 25 exhibits a less than optimum root canal filling.



956 Clinical View 6 Years later
Despite the palatal position of the root of tooth 26, it was possible to achieve an optically straight-line and harmonic dental arch reconstruction.

Note: This reconstruction remained *in situ* for 10 years, and then tooth 24 was lost because of root fracture. A completely new fixed bridge construction included the canine (tooth 23) as well as the palatal root of tooth 26.

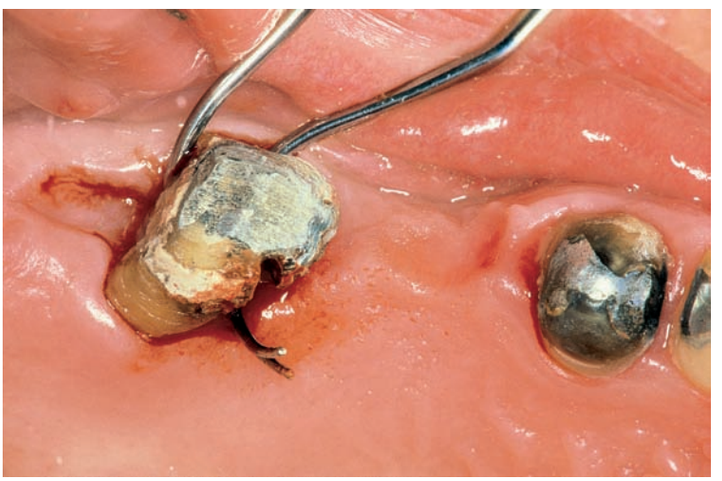
Courtesy *R. Metzger*

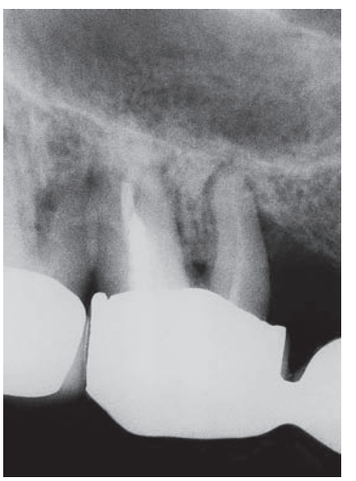
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Maxillary Furcation Involvement, F3—Trisection with Maintenance of all Roots

In cases of severe maxillary furcation involvement (grade F3, subclasses B and C; cf. p. 383) the question of which roots to extract and which to maintain can frequently only be made following *complete separation* of the tooth segments. Following that procedure, each individual root can be probed around its circumference, and its degree of mobility determined (cf. Fig. 957).

In the case presented here, a 45-year-old female desired to maintain tooth 16 after the hopeless tooth 17 had to be extracted, in order to avoid a free-end saddle situation or a dental implant. After removal of the defective bridge between 16 and 14 and separation of the roots of 16, it became obvious that all of the roots were equally mobile, with only 2–3 mm of remaining attachment. The decision was made to *attempt* to treat all three roots endodontically and periodontally and then to insert a temporary bridge. Thanks to the patient's optimum oral hygiene, six months later it was possible to seat a definitive restoration (3-unit bridge), which at the time of this writing has been in place and functional for 20 years.





957 Severe Furcation involvement, F3—Situation after Removal of the Old Bridge
The two Nabers probes demonstrate on tooth 16 the horizontal through-and-through involvement from distal and buccal toward the mesial.

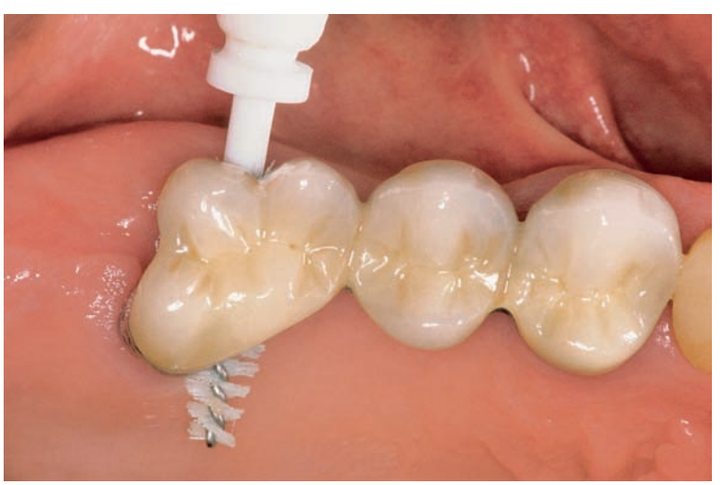
Right: Initial situation before extraction of 17. Etiology: Massive open crown margin, periodontal pockets and furcation involvement, incomplete root canal filling (Combined endo-perio problem, cf. p. 445).

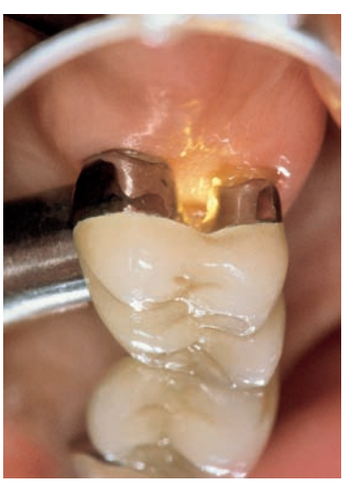




958 "Trisection" of Molar 16—Temporary and Definitive Bridges
Using a temporary bridge, a "waiting period" was observed in order to see if the patient could maintain all three roots plaque-free. Only after six months was the definitive bridge seated, and then only temporarily at first.

Right: Occlusal view of the three roots of the first molar.





959 Definitive Bridge Construction *In Situ*—Oral Hygiene
Premolar 14 and pontic 15 exhibit normal crown form and occlusal surfaces; the castings for coverage of the roots of 16 were fabricated with minimal undercut areas. The furcation entrances of the crown are easily accessible for cleaning with superfloss or interdental brushes.

Right: Distal furcation. The crown margins are supragingival.

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Mucogingival, Plastic Surgery

The indications for mucogingival, "plastic" periodontal surgery have increased in significance in recent years. Nevertheless, such procedures maintain a secondary place in terms of maintenance of periodontal health. However, patients' esthetic demands as well as morphologic/anatomic circumstances may require that dentists and periodontists devote more effort to the mucogingival arena. The goals and the various clinical procedures are summarized below, and will be described in more detail in the pages that follow:

1. Halting gingival recession
2. Increasing the width of attached gingiva
3. Deepening the vestibulum
4. Covering areas of gingival recession

The goals for 1–3 can be achieved using the following surgical procedures:

- Frenotomy
- Free "epithelialized" gingival/mucosal graft (FGG)

These surgical procedures do not change the architecture of the gingival margin. However, with the existence of gingival recession, a "creeping attachment" is possible (p. 412) by eliminating the "pull" on the mobile oral mucosa by frena.

In addition to halting gingival recession, today it has also become important for esthetic reasons to *cover the recession* (4). This can be achieved by the following treatment methods:

- Pedicle flaps
- Direct coverage with a free gingival graft (FGG)
- Connective tissue graft (CTG)
- Guided tissue regeneration (GTR)
- Growth factors and enamel matrix proteins

These types of esthetic periodontal procedures are targeted toward returning the gingival margin to its original location at or near the cementoenamel junction. Furthermore, ridge augmentation using connective tissue or bone may be considered part of mucogingival surgery (see Perio-Prosthetics 2, p. 489, and Implantology, p. 511).

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Mucogingival Problems ...

The major antecedent to the occurrence of mucogingival problems is almost always a genetically determined *morphologic* peculiarity. These include:

- A thin or totally lacking facial osseous lamella—buccoversion of the roots within the alveolar bone; dehiscence, fenestration
- The width and thickness (phenotype) of the attached gingiva
- Coronally attached frena in the absence of attached gingiva
- The vestibular depth

In the presence of such "natural" anatomic situations, especially if the patient practices inappropriate and excessive oral hygiene, lesions/injuries to the gingival margin can occur, as well as Stillman clefts and ultimately also gingival recession and wedge-shaped defects on the tooth roots.

Whether or not, and to what degree, functional disharmonies (bruxism/clenching) with abfractions resulting from stress on the teeth may increase the severity of soft tissue defects remains controversial today.

960 Delicate, "Thin" Gingiva
This 27-year-old female exhibits primarily healthy periodontal conditions. The gingivae appear to be somewhat thin, however, and appear "glassy" and without stippling. This anatomic/morphologic combination of the gingival phenotype may be an indicator for subsequent gingival recession, particularly if an improper and traumatizing oral hygiene regimen is practiced.
Mild gingival recession is already visible on teeth 33 and 34.



Predisposing Factor

- "Thin" Gingiva

961 Distinct Root Prominence
In this 19-year-old female, the mandibular anterior teeth were positioned quite anteriorally in the arch. The clinical picture anticipates that the coronal portions of the roots are not covered by bone (dehiscences). This condition would enhance the development and progression of gingival recession. On tooth 31, recession has already progressed into the mobile mucosa, and is secondarily inflamed.



- "Thin" Gingiva
- "Prominent" Bone
- Osseous Dehiscence?

962 Gingival Recession, Miller Class 4
Pronounced gingival recession extending to the mobile mucosa, combined with interdental attachment loss.
This 64-year-old male had for decades employed a traumatic, horizontal, scrubbing toothbrushing technique. In addition to the gingival recession and wedge-shaped dental defects, one can also observe grooves on the root surfaces as evidence of toothbrush abrasion.



- Traumatic Oral Hygiene
- "Abfractions"

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… and Possibilities for Resolution

Conservative Approach—Modifying the Toothbrushing Technique

In order to spare patients from unnecessary surgical procedures ("over-treatment"!), and if there are no significant esthetic problems, the following approach is recommended:

1. Inform the patient about the etiology
2. Thorough tooth cleaning and polishing, and removal of gross functional occlusal disharmonies
3. *Modification of the patient's oral hygiene technique*: Use of a soft toothbrush and possibly a sonic toothbrush
4. Fabrication of study models and/or intraoral photographs; during subsequent recall appointments, these permit comparisons with the actual clinical situation and illustrate the stability or the progression of gingival recession
5. Initially, short-interval recall appointments

Only if the mucogingival problems are not successfully addressed by these measures, and after an observation period of months or years, mucogingival surgical intervention may be indicated.



963 Stillman Clefts and Recession
A 51-year-old female teacher presented with Stillman clefts and areas of gingival recession that could not be explained. Possible etiologies for her condition included "aggressive" oral hygiene and stress-related habits. Additional possible etiologic factors included functional disharmonies (right side cross-bite and mid-line shift); the mucosal defects were symmetrical.



964 Mucosal Defect above Tooth 13
The etiology of gingival recession and cleft formation extending into the mobile mucosa could not be determined despite repeated examination and inquiry. The exposed root exhibited a wedge-shaped defect. The first effort was conservative (see above): The patient was instructed in an oral hygiene technique that was gentle in the facial regions. The wedge-shaped defect, which was a plaque-retentive niche, was filled using composite resin.



965 Six Years after Conservative Therapy
Without any surgical intervention, tooth 13 as well as tooth 23 exhibited significant improvement over the years, during which the patient was in strict recall. The gingival margin on tooth 13 is harmonic, and the cleft closed spontaneously.
It must be pointed out, however, that in other, more critical, areas, e. g., on teeth 31 and 41, surgical intervention was necessary.

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Frenotomy—Frenectomy

The simplest mucogingival surgical procedures are the frenotomy and the frenectomy. Frena may exert excessive pull upon the gingival margin and the interdental papillae, resulting in local gingival recession. For this reason, labial and buccal frena that splay far toward the gingival margin should be eliminated. This can be accomplished by simple surgical separation (*frenotomy*) or by removal of the entire frenum (*frenectomy*). The triangular or rhomboid wound that exists after frenotomy/frenectomy may be covered using a "mini" free gingival graft. If *no* free gingival graft is employed, 20–50 % recurrence can be expected.

The case below is a 17-year-old female with healthy periodontal conditions. However, slight recession of the interdental papilla between teeth 11 and 12 occurred due to excessive pull by the labial frenum. The frenectomy in the maxilla is portrayed below.

Clinical findings:

PI: 14 % BOP: 18 % TM: 0

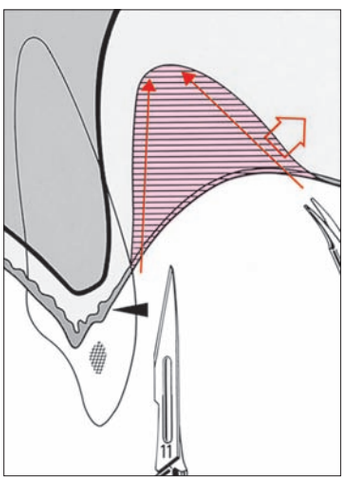
The figures below depict the clinical situation.

966 High Frenum Attachment between Teeth 11 and 21

The pull exerted by the extremely high frenum attachment caused severe retraction of the papilla tip between the central incisors (black arrows –Class 2 according to Jemt, p. 496).

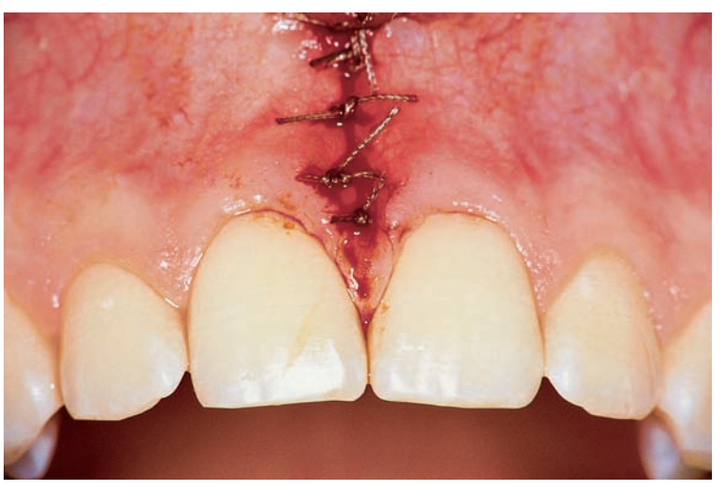
Right: The extent of this frenum becomes visible when the upper lip is reflected. The black arrow demonstrates the position of the receded papilla. The red arrows depict the incision.





967 Immediate Post-Surgical View

Excision of the frenum creates a rhomboid-shaped wound. A thin elevator or scissors is used to sever muscle and connective tissue fibers from the periosteum of the alveolar process, and the mucosal edges are approximated with sutures. It was not possible to close the wound completely at its coronal extent due to the immobility of the attached gingiva.

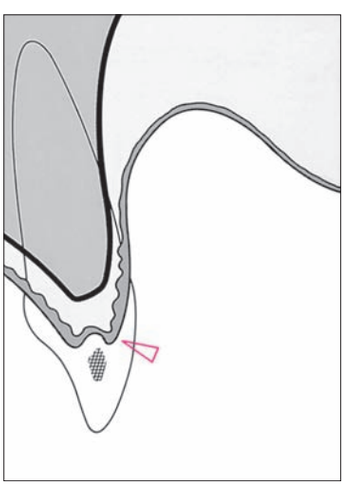


968 Two Years Post-Operative

Elimination of the frenum pull led to complete regeneration of the facial interdental papilla (arrows). The interdental space between the central incisors completely closed. (Class or Papilla Index Score 3/Jemt, p. 496.)

Right: The interdental papilla regenerated (open red arrow) and the vestibulum was slightly deepened.





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Free Gingival Graft—Autogenous Transplant—with Epithelium

Gingival extension by means of a free gingival graft (FGG) was first described by Björn (1963), and systematized by Sullivan & Atkins (1968a, b). The surgical procedure involves replacement or enhancement of the mobile, non-keratinized mucosa with "true" keratinized gingiva, usually obtained from the palate. Areas of recession are *not* covered. "Creeping attachment" is possible, but not predictable.

Indications

Many areas of *localized* recession can be completely halted simply by changing the patient's oral hygiene and elimination of damaging "habits," including elimination of occlusal trauma. If gingival recession continues despite such *local* efforts, there is an indication to halt the process, e.g., by widening the band of attached gingiva. An additional indication may be present when gingival recession extends to or beyond the mucogingival junction and renders oral hygiene difficult in such areas, especially near frenum attachments. Inflammation that is difficult to eliminate is always the consequence. There is constant movement of the oral mucosa at the gingival margin. Injuries to the gingival margin, e.g., by the toothbrush often occur due to such unfavorable morphologic/anatomic conditions.

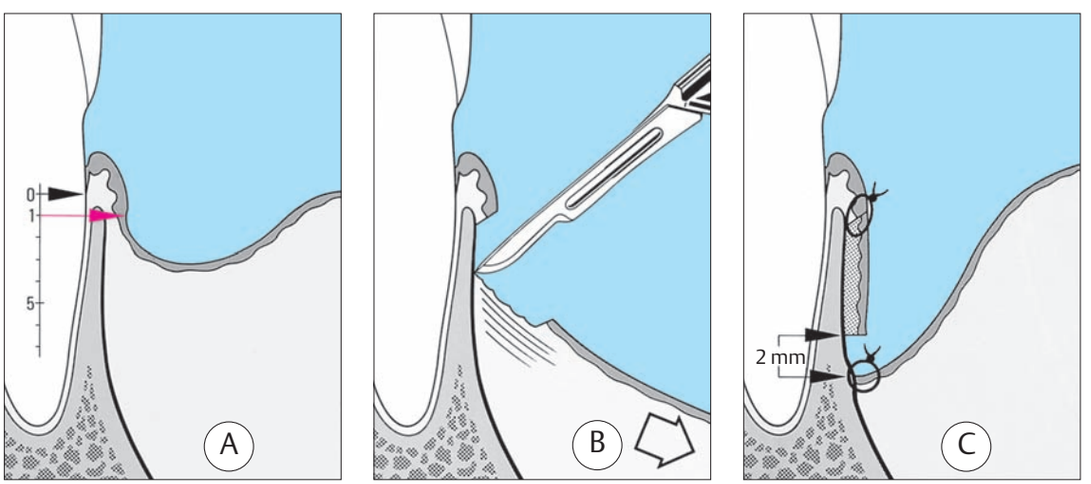
In such cases, the only treatment of choice is halting the progression of recession through use of a free gingival graft. Treatment of *generalized* recession by means free gingival grafting is possible, but is seldom indicated, because the quantity of transplant tissue from the palate is not unlimited.

Expansive areas of gingival recession may be successfully treated using the mesh graft technique or the Edlan-Mejchar procedure.

Contraindications

The FGG procedure is not indicated in areas of *stationary* recession that are accessible for oral hygiene, or do not exhibit signs of persistent inflammation, or do not present esthetic problems. The use of FGG surgery is today often also contraindicated when there is a true indication for *defect coverage* (p. 413).

The FGG is usually harvested from the palate. The whitish color of this highly keratinized mucosa is retained even after transplantation of the tissue. In the often visible maxillary anterior and canine regions, this may create an esthetic problem that must be considered p*re-operatively*!



969 Principles of FGG Surgery

A Initial situation. Hardly any (1 mm) remaining keratinized attached gingiva (zone between red and black arrows).
B The lip or cheek is stretched (arrow), and connective tissue and muscle fibers of the mucosa are carefully dissected from the subjacent periosteum.
C The FGG is sutured marginally to the attached gingiva and pressure is applied to adapt it to the subjacent periosteum.

Surgical Principles

The surgery is performed using block anesthesia into the vestibulum, with local infiltration at the surgical site.
The *first surgical phase* of the procedure involves preparation of the recipient bed apical to the recession area. A horizontal incision is made along the mucogingival line. If attached gingival is completely absent, the incision is made 1–2 mm apical to the gingival margin. The incision is made through the mucosa and submucosal tissues, but does not encroach upon the periosteum. Mucosa, submucosal connective tissue and muscle fibers are carefully separated from the underlying periosteum. In this way a *recipient bed covered with periosteum* is prepared to receive the free gingival graft. Caffesse and co-workers (1979a, b) were able to demonstrate that a FGG can also be successful if it is placed upon a "bleeding" osseous surface that is not covered by periosteum.

The *second surgical phase* is the removal of a ca. 1 mm thick graft, usually from the palate. The instruments for this procedure are illustrated on p. 402.

The *third surgical phase* involves adaptation of the FGG into the recipient bed, and its secure fixation to the gingival margin with sutures.

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Instruments for Harvesting the Transplant ...

The mucosa for a free gingival graft (FGG) is usually taken from the palate. Several special and easy-to-use surgical instruments are available for this procedure.

The *hand mucotome* can be used to harvest graft tissue of varying *width*. The graft *thickness* is not predetermined, and can also be varied.

The *motor-driven mucotome*, on the other hand, always provides a strip of graft tissue of identical width and thickness. The connective tissue undersurface of such a harvested strip is so smooth that it may be confused with the epithelial surface. For this reason, the epithelial surface should be marked before the surgery using a non-toxic, waterproof, felt-tipped pen. A tissue graft transplanted with the wrong side up cannot succeed!

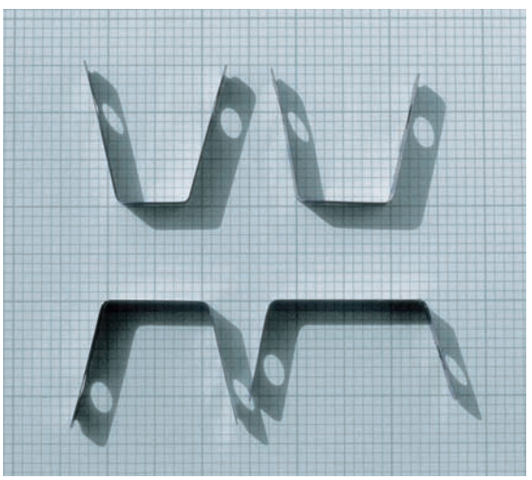
Scalpels and gingivectomy knives from the periodontal surgery instrumentarium can be used to harvest graft tissue of varying shape and thickness.

970 Hand Mucotomes
Left: These instruments (Deppeler) consist of a handle and a cutting head onto which disposable blades that are ideally sharpened are affixed. The handles are available straight (as shown here) or with an angle.

- **PR 1** 7 mm wide
- **PR 4** 9 mm wide
- **PR 2** 11 mm wide
- **PR M** 16 mm wide

Right: Disposable blades fabricated from razor blade steel.



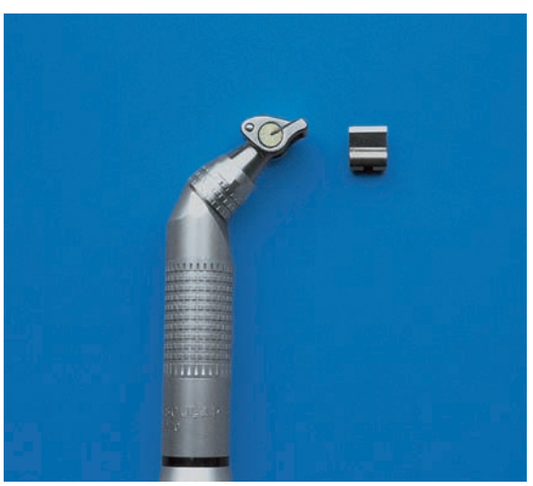


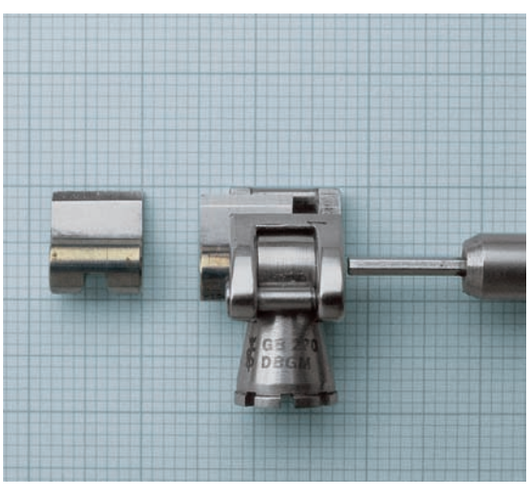
971 Motor-Driven Mucotome as Described by Mörmann
Left: This instrument (Aesculap Co.) makes it possible to harvest tissue grafts of uniform width and thickness.

- 6.5 mm width
- 0.75 mm thickness

The instrument head can be positioned as desired.

Right: Close-up of the instrument head; blades can be resharpened.





972 Scalpels, Gingivectomy Knife, Surgical Forceps, Mini-Hook
Left: A "customized" free gingival graft can be obtained using fine scalpels (No. 15 or 15 C), or the gingivectomy knife.
For lifting the graft out of the palate, the mini-hook (Gillis) is less traumatic to the tissue than a forceps; alternatively a suture can be used to lift the tissue graft.

Right: Magnification of the instruments' working tips.





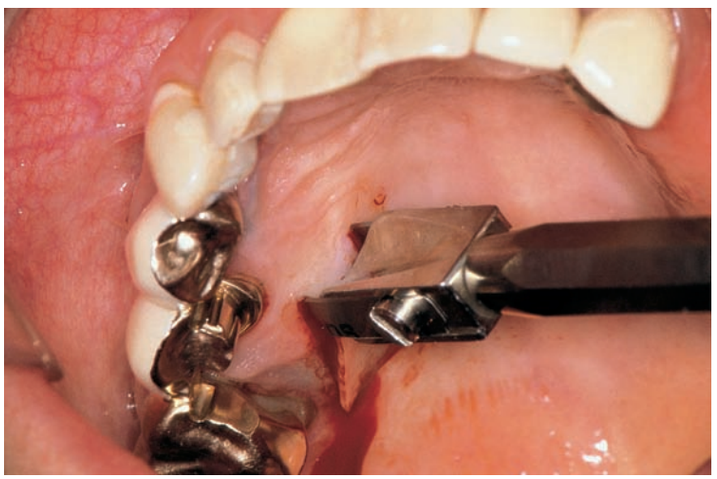
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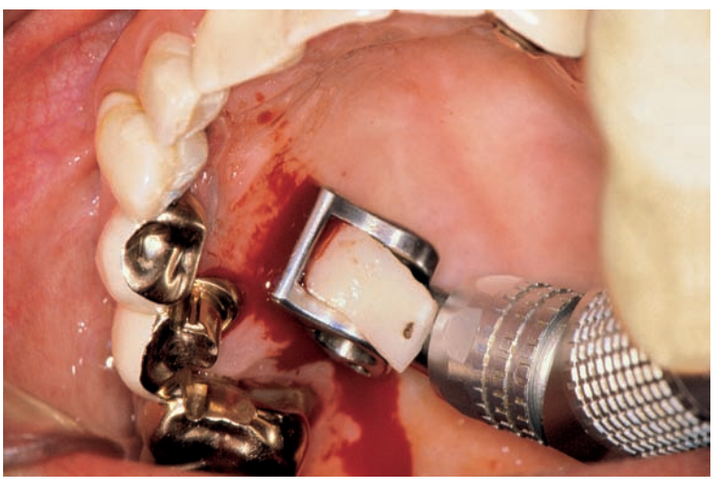
The hand or motor-driven mucotomes provide rectangular strips of graft tissue that must subsequently be trimmed to fit the prepared recipient bed.

If a perfectly fitting graft with a special shape is desired, it must be created using the scalpel and/or GV knife plus a pattern or template made of metal or plastic foil. This individual method for harvesting/trimming an individual graft is indicated especially when the recession to be treated is severe and must be covered with arcuate grafts. This is also true when a graft must be placed around a lone-standing tooth, where the graft must be adapted around the tooth, toward the alveolar ridge.

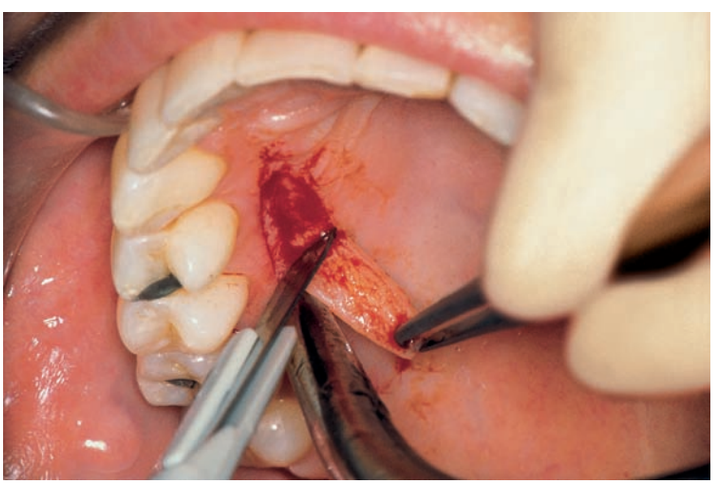
Removal of palatal tissue must be accomplished with profound local anesthesia. If anesthetic solution is injected with elevated pressure, the palatal mucosa will be lifted somewhat and may prove easier to remove after the incisions are completed. Incisions must not be too deep, in order to avoid injury to the greater palatine artery.



973 Harvesting a Graft from the Palate Using a Hand Mucotome PR 1 (7 mm)
The cutting head is moved slightly back and forth as the incision is made. To provide a precise cut, the index finger of the left hand may be used for guidance and the application of pressure. The ideal graft is uniform and maximally 1 mm thick.



974 Harvesting Palatal Graft Tissue Using a Motor-Driven Mucotome
The head of the instrument must be pressed lightly against the palatal mucosa so the blade engages the tissue completely, and a uniformly thick graft is harvested. Marking the epithelial surface precludes suturing the graft incorrectly into the recipient bed.



975 Harvesting a "Customized" Tissue Graft
Using a template prepared at the recipient site, palatal tissue is harvested using the scalpel. The first incision is made around the periphery of the graft, to a depth of 1 mm. The GV knife is then used (without the template) to undermine the graft margins, before teasing out the graft using the scalpel and forceps.

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Free Gingival Graft—Thickness and Shape

The structure of epithelium—keratinization, cell layers—is determined by the *subjacent connective tissue*! It is for this reason that every free graft must contain subepithelial connective tissue; this demands a graft that is at least 1 mm thick.

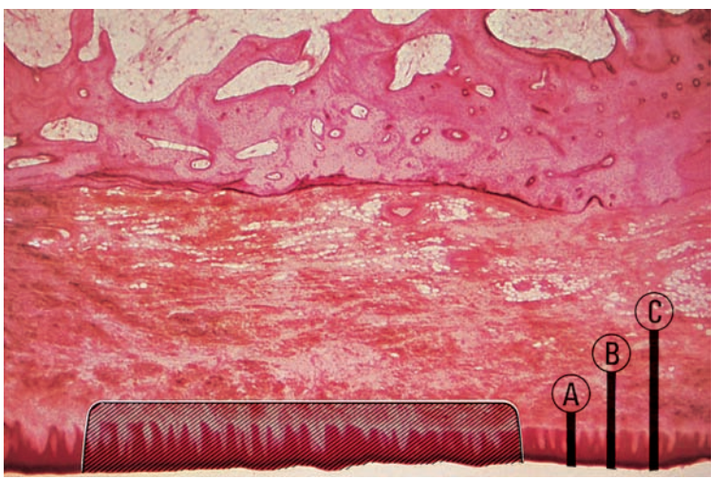
During the healing-in phase, the original graft epithelium is almost completely desquamated, while the subjacent connective tissue is "accepted" (see Re-vascularization studies, p. 411). New epithelialization occurs from the remaining basal cells and the surrounding mucosa (Bernimoulin & Lange 1972). Even epithelium that grows in from the non-keratinized mucosa above the transplant will differentiate into *keratinized* mucosa. Palatal rugae must not be transplanted, because they would be visible in the attached gingiva even after complete healing.

Harvesting a graft using the hand mucotome or a scalpel brings with it the danger of penetrating too deeply and severing branches of the greater palatine artery.

976 Histology of the Hard Palate
Soft tissue overlying the palatal bone is 3–6 mm thick. The ideal graft should be no more than 1 mm thick, containing both epithelium *and* subepithelial connective tissue. Shown here (hatched area) is a tissue graft ca. 0.8 mm thick and 6 mm wide.

A Thin graft
B Medium graft
C Thick graft

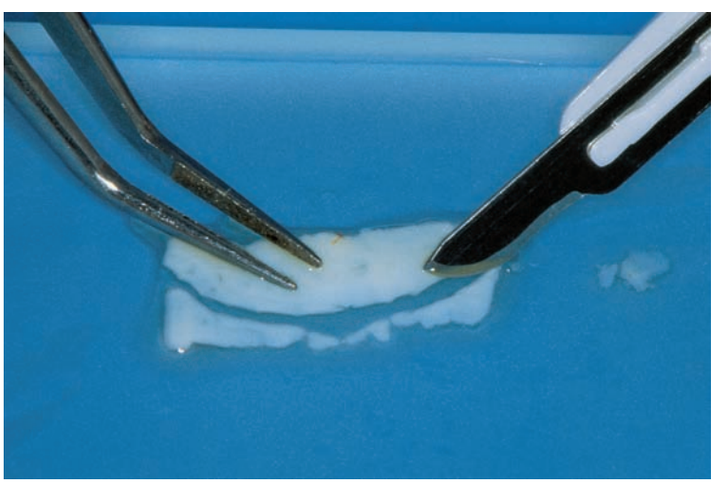
Courtesy *H. Schroeder*



977 Thinning an Excessively Thick Graft
Excessively thick grafts contain fatty tissue and glandular tissue. Following removal, such thick grafts must be thinned using the mucotome, a scalpel or scissors on a sterile glass slab under saline irrigation.



978 Trimming the Graft
Grafts harvested with mucotomes are rectangular in shape and often must be appropriately trimmed to fit the recipient bed. Such trimming is often not necessary if the graft is taken from the palate using a scalpel and a preformed template.



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Free Gingival Graft—Halting Recession

Surgical Procedure, Step-by-Step

Stopping the progression of gingival recession by means of FGG is indicated mainly in areas that are *not usually visible* when the patient speaks or smiles.

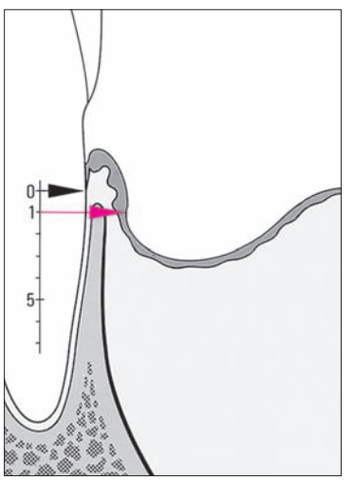
If there exist esthetic considerations in the *visible* anterior sextants, widening the attached gingiva is usually best accomplished in combination with *coverage of the recession* (p. 413). For decades, the FGG harvested from the palate has been an effective technique for extending the attached gingiva and therefore halting the progression of gingival recession.

This 44-year-old female exhibited the relatively rare combination of localized periodontitis with "classical" gingival recession. Her chief complaint was that her teeth appeared to be "getting longer." Her periodontitis was treated in the usual manner. The progressive recession, especially in quadrant 3, was treated by means of FGG.

Clinical conditions after periodontal therapy in quadrant 3:

PI: 11 % BOP: 14 % TM: 0–1

Clinical appearance, probing depths, recession, width of attached gingiva and radiographs are depicted below.



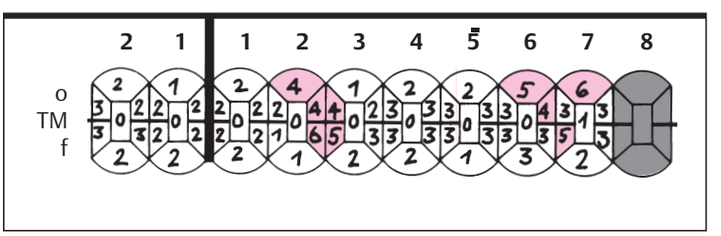


979 Initial Clinical View—Mandibular Left Sextant Following Periodontal Therapy

Gingival recession is apparent on teeth 32, 33 and 34. When performing the "roll test," with a periodontal probe, it is clear that tooth 34 has no attached gingiva.

Left: The diagram shows a section in the orofacial direction. There is hardly any attached gingiva (ca. 1 mm) present (zone between the red and black arrows).

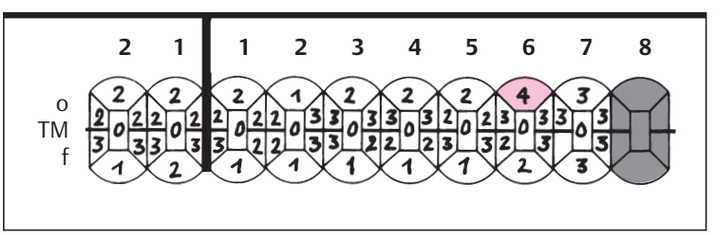
Initial Findings



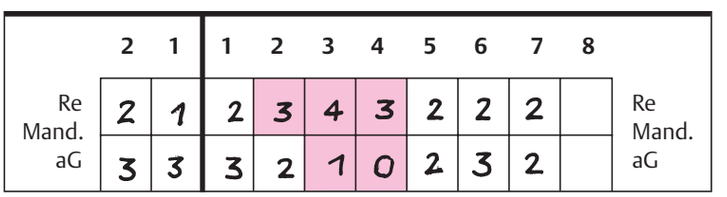
980 Probing Depths (PD) before and after Periodontal Therapy

Before periodontal treatment, the probing depths were 2–3 mm, with some localized probing depths up to 6 mm.

After Periodontal Treatment

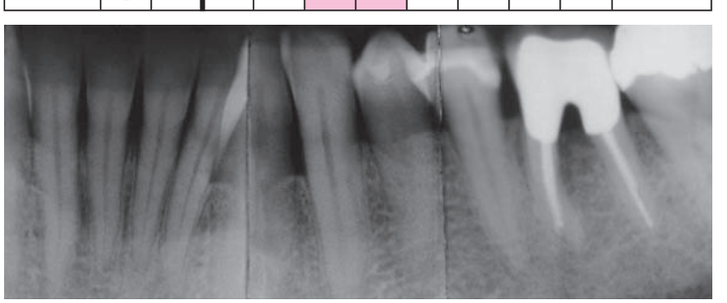


By means of purely closed periodontal treatment and before placing the FGG, the pocket probing depths were reduced to physiologic levels.



981 Recession (Re) and Width of the Attached Gingiva (aG) Following Periodontal Treatment

Pronounced gingival recession was obvious on teeth 32, 33 and 34 (pink). The attached gingiva on tooth 33 is 1 mm wide, and tooth 34 exhibits *no* attached gingiva.

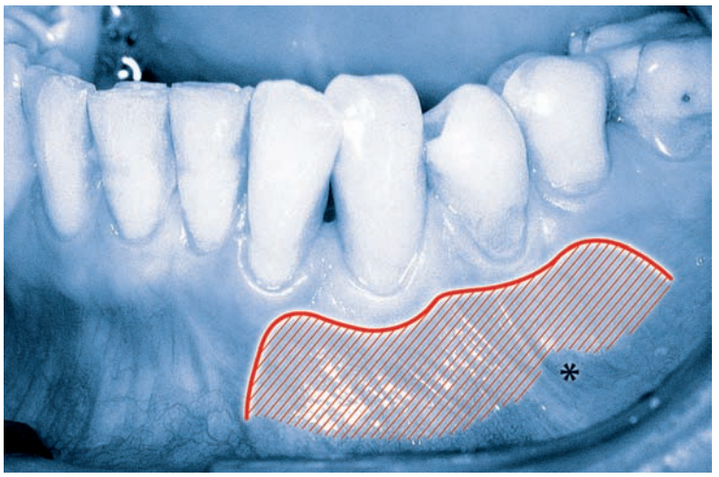


Radiographic Findings *before* periodontal treatment: Bone loss is obvious between teeth 32 and 33, and heavy calculus on the mesial of tooth 33.

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982 Surgical Planning
A horizontal incision is made along the mucogingival junction (MGJ); if *no* attached gingiva is present, this incision should be ca. 1–2 mm apical to the gingival margin. The incision ends both mesially and distally in an arcuate form targeted vertically toward the vestibulum in the region of the adjacent teeth where adequate attached gingiva is present.

Right: FGG, surgical protocol



Halting Gingival Recession, FGG: Surgical Protocol

1 Preparing the recipient site
- Horizontal incision along the MGJ
- Vertical incisions
- Preparation of split-thickness flaps

2 FGG—Removal of the *epithelialized* graft from the palate
- Hemorrhage control and covering the palatal wound

3 Trimming the FGG
- Fixation with sutures

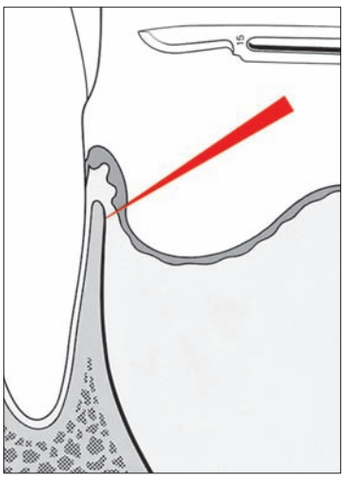
Post-operative instructions

First Surgical Phase: Extension—Creation of the Recipient Bed

983 Horizontal Incision
The assistant pulls on the lip and oral mucosa (retractor) then the No. 15 scalpel is used to make an incision along the MGJ, ca. 1 mm deep. If necessary, vertical releasing incisions can also be made. The periosteum should not be severed.

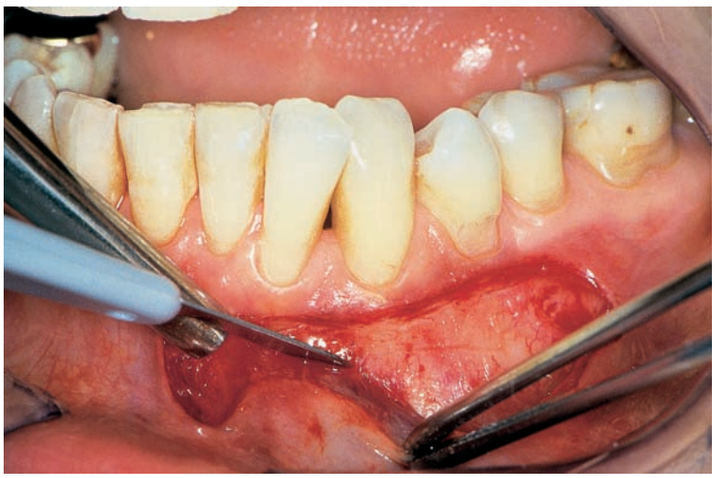
Right: Diagram of the incision at the approximate position of the MGJ.

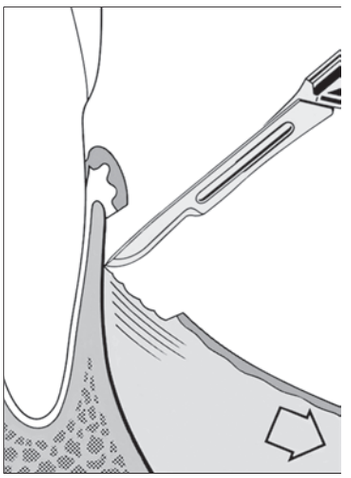




984 Preparation of the Recipient Bed
Connective tissue and muscle fibers are carefully dissected away from the periosteum. The scalpel blade must be maintained at an oblique angle to the periosteum. *Caution*: Mental nerve in the region of the premolar apices!

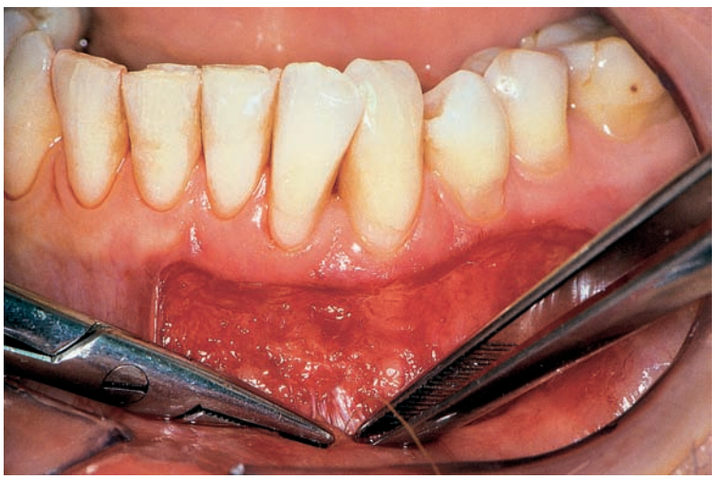
Right: Lip and cheek are reflected (arrow), opening the wound to permit clean preparation.

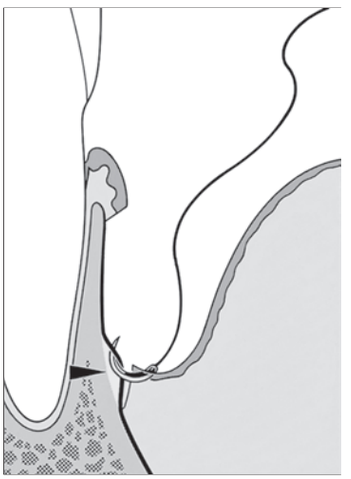




985 Apical Fixation of the Mucosa to the Periosteum (Optional)
The recipient bed should be wider at its apical extent than the planned graft. The free margin of the vestibular mucosa can be affixed to the periosteum, using resorbable sutures, but this is not obligatory.

Right: A small, curved, atraumatic needle traverses the mucosa and is guided beneath the intact periosteum (arrow).





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986 Template for the Free Gingival Graft
Sterile aluminum foil is used to create a "template" that precisely fits the recipient bed. The apical edge of this template should be about 2 mm short of the recipient bed margin.

If the graft is relatively straight and symmetrical (quadratic) a pattern or template may not be necessary, and the free gingival graft can be taken using a microtome (Fig. 977).





Second Surgical Phase: Harvesting a Custom Free Gingival Graft from the Palate

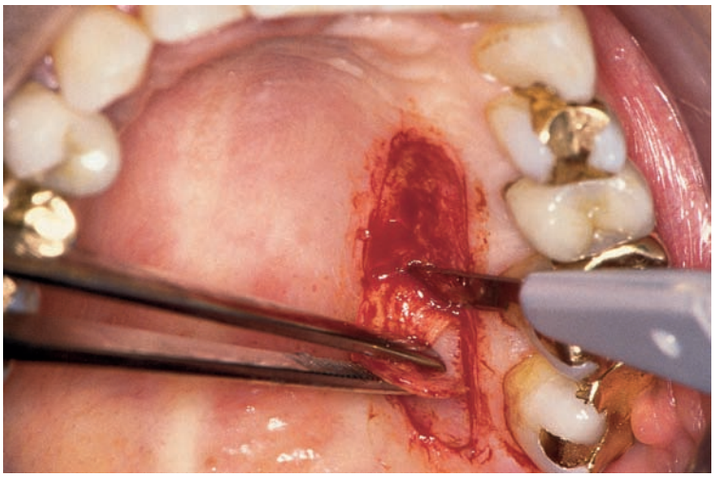
987 Aluminum Foil Template on the Palate
The foil is placed onto the palate, about 2–3 mm removed from the gingival margin. A scalpel is used to incise around the template to a depth of 1 mm.

Left: Incision line around the tissue graft on the left side of the palate (recipient bed is also on the left side: Patient *comfort*!).



988 Mobilization of the Graft Margins
A gingivectomy knife (e. g., GX 7, Deppeler) is used to undermine and reflect the margins of the graft tissue. A surgical forceps or the mini-hook (e. g., Gillis) can then be used to reflect and excise the palatal graft tissue.





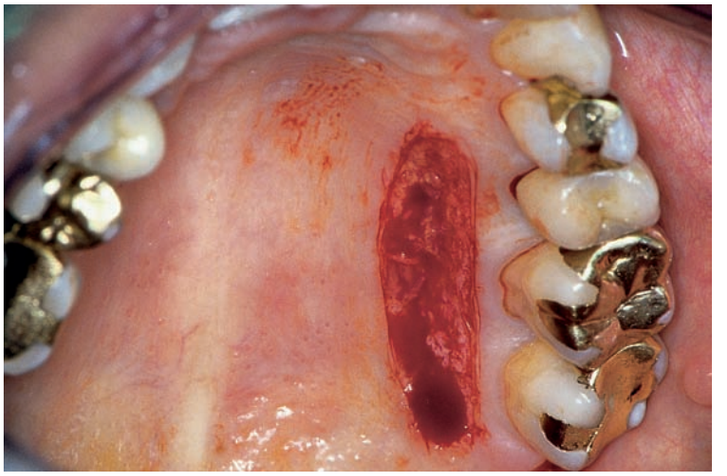
989 Removing the Graft Tissue
The No. 15 scalpel is used to free the graft from its underlying tissue. The Gillis hook or a suture can be used to apply sufficient tension for removal.

Left: The excised palatal tissue corresponds almost perfectly to the aluminum foil template.

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990 Treating the Palatal Wound
Hemorrhage is staunched with pressure on a gauze square. The wound can then be coated with cyanoacrylate (p. 310) or covered with styptic gauze. It is also recommended that the palatal donor site be covered with a mini-plast splint (compression protection against mechanical irritation/pain; patient comfort). The wound closes in 10–14 days.

Right: Transparent mini-plast splint on the plaster model.





Third Surgical Phase: Placing the Transplant (FGG)

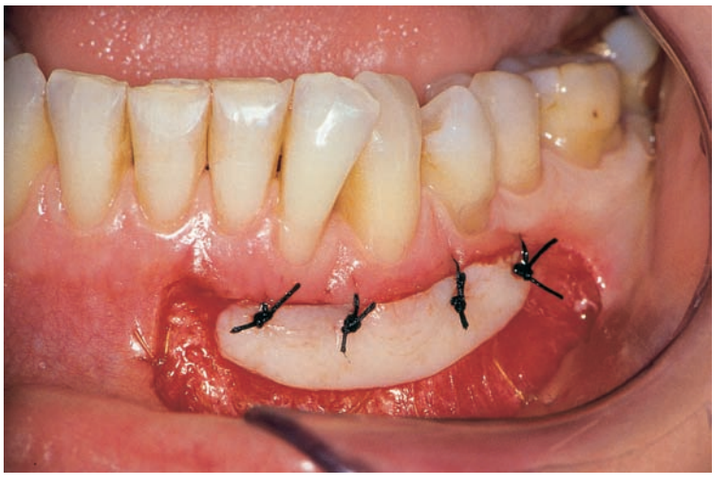
991 FGG "in Bed"
The border of the FGG approximates the incision line. Apically, the bed is ca. 2 mm wider than the FGG. During the first few post-operative days, this region must be completely immobilized to ensure healing of the graft.

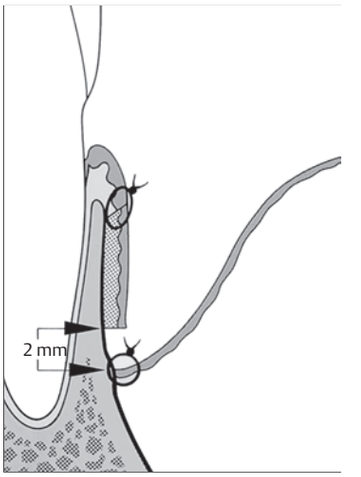
Caution: The graft must be placed with its connective tissue surface toward the periosteum! *Tip*: Always mark the *epithelial side* of the FGG using a felt-tipped pen!



992 FGG—Completing the Third Surgical Phase
The grafted tissue is secured using atraumatic sutures and a sharply curved needle, and then is held in place for several minutes with a moist gauze square. All mechanical plaque control in the surgical area is halted for ca. 14 days; instead, CHX rinses are prescribed.

Right: The FGG must be ca. 2 mm smaller than the recipient bed.

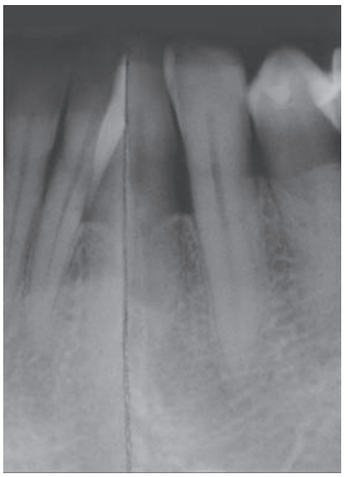




993 Six Weeks Post-Surgically
Healing is complete. The attached gingiva has been significantly broadened and the vestibule deepened. This ensures that gingival recession has been definitively halted (Rateitschak et al. 1979).

Right: The radiograph clearly shows that even the periodontitis has been successfully healed (clean root surfaces on the mesial of tooth 33 and new bone formation between teeth 32 and 33; cf. Fig. 981).





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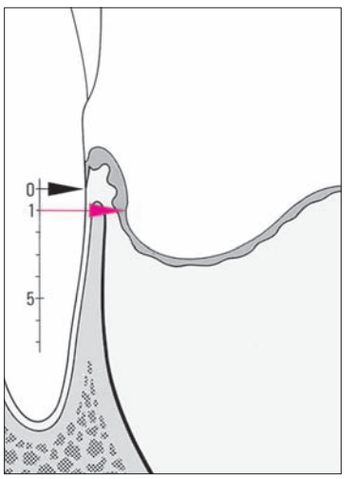
Free Gingival Graft—Halting Recession

Summary

"True" gingival recession is seldom a clinical manifestation of destructive periodontitis. In the case depicted here, however, 6 mm pockets were measured in the interdental regions of teeth 32 and 33. In the region of the facial recessions on these teeth, pocket probing depths were physiologic. In cases of combined periodontitis and gingival recession, it is the microbial infection (plaque and calculus removal) that must be treated first.

During periodontal healing (pocket reduction) the gingival recession must also be monitored. Despite modified oral hygiene (vertical—rotatory brushing), the areas of gingival recession continued slowly to progress, which led eventually to the complete loss of attached gingiva.

A mucogingival surgical procedure employing a *free gingival graft* (FGG) stabilized the situation. A new, 6–7 mm wide band of attached gingiva prevented any further progression of gingival recession and allowed the patient to resume an effective and non-traumatic oral hygiene routine.





994 Initial Clinical View Following Periodontal Treatment
The appearance of healthy gingiva between teeth 32 and 33 is apparent following initial periodontitis therapy. The papilla between these teeth appears to have receded. Gingival recession, however, was rapidly progressing.

Left: The diagram reveals the almost complete lack of attached gingiva (1 mm between black and red arrows) and the very *flat* vestibulum.

Before

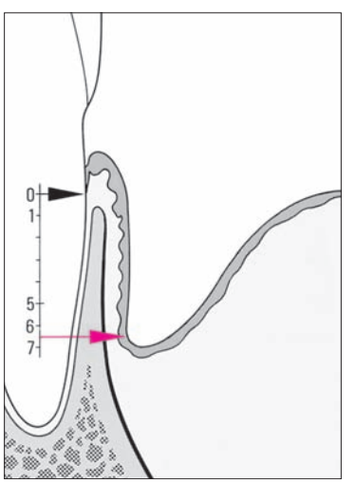
	2	1	1	2	3	4	5	6	7	8	
Re	2	1	2	3	4	3	2	2	2		Re
Mand. aG	3	3	3	2	1	0	2	3	2		Mand. aG

995 Facial Recession (Re) and Width of Attached Gingival (aG)
Before placing the FGG on teeth 32, 33 and 34, the gingival width was 0–2 mm, and the recession measured 3–4 mm.

After

	2	1	1	2	3	4	5	6	7	8	
Re	1	1	2	3	3	2	2	2	2		Re
Mand. aG	3	3	5	7	7	6	2	3	2		Mand. aG

Re and aG 6 Weeks later
In the surgical region, the width of attached gingiva was 6–7 mm and the progression of gingival recession had been completely halted. Note "creeping attachment" on teeth 33 and 34.





996 Six Weeks Post-Surgically
The FGG has completely healed in. Progression of gingival recession has been halted and the oral vestibulum is deepened. Clearly apparent, however, is the light color of the FGG: An FGG in the visible maxillary anterior segment may therefore be contraindicated for purely esthetic reasons.

Left: The attached gingiva has been extended to over 6 mm (distance between black and red arrows).

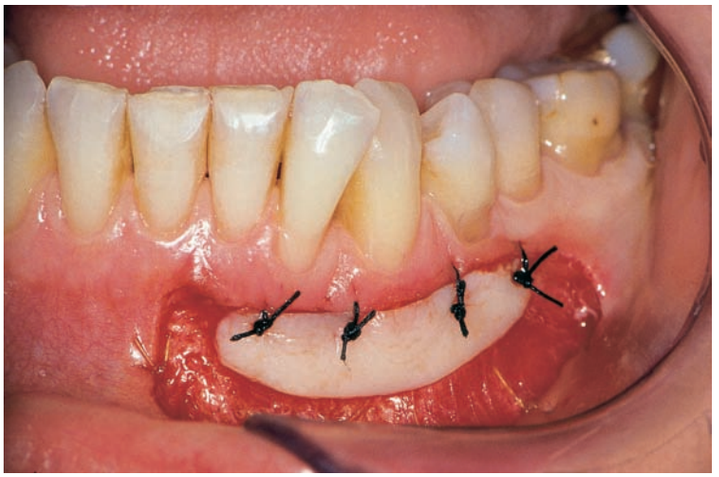
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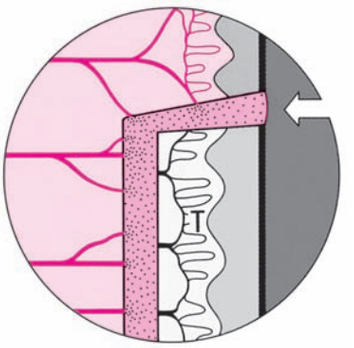
FGG—Wound Healing, Clinically ...

The healing of a FGG generally proceeds without complications if the graft (ca. 1 mm thick) contains portions of the lamina propria in addition to epithelium (maximum 0.5 mm). About two days after transplantation, portions of the epithelium desquamate. At this point the FGG is unsightly and appears necrotic. However, success of a FGG depends not upon the epithelium but upon the subepithelial connective tissue. New epithelialization occurs from the *gingival margin* of the graft bed. Even epithelium that grows over the FGG from the mucosa differentiates upon the transplanted connective tissue into a keratinized "palatal" epithelium (see Fig. 976). After one week, the graft has definitively healed into its recipient bed and is covered by a new epithelial layer. Complete keratinization is accomplished after ca. 4 weeks. Clinical failure is possible if the graft is placed with its epithelial surface apposed to the recipient bed, if a fixed coagulum forms between the graft and the bed, or if the graft is in some way mechanically dislodged within the first 2 days following transplantation (Sullivan & Atkins 1968a). Depending upon the expanse of the FGG, the palatal wound re-epithelializes in ca. 1–2 weeks.

997 FGG Immediately after Surgery
The graft is pale. It has not yet established circulation. Nutritive transfer occurs only via plasma that defuses from the periosteal bed into the FGG. It is important that the FGG not be dislodged or disturbed during the first few days.

Right: A blood coagulum exists between the transplant (**T**) and the surrounding tissues. This coagulum should be as thin as possible (white arrow).

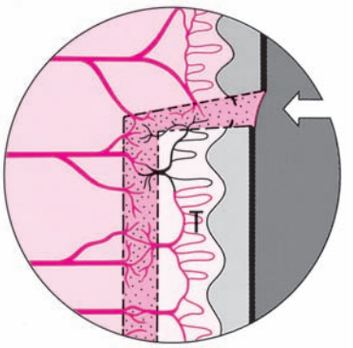




998 Three Days after Surgery
The surgical field generally appears "unpleasant" several days after the operation. The superficial cell layers of the FGG remain light in color, become necrotic and are slowly exfoliated. The wound surface that is not covered by the FGG becomes covered with fibrin (overextension).

Right: New vessels begin their penetration into the coagulum and the transplanted tissue. The FGG, at this point, has "taken."

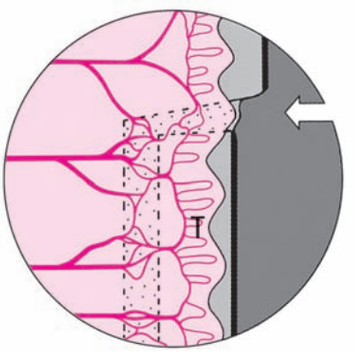




999 Seven Days Post-Surgery
The wound area is very carefully cleaned using 3 % H_2O_2 and the teeth are professionally cleaned with rubber cup and prophy paste. Sutures are removed. By this time, the grafted tissue is tightly bonded with the subjacent tissues via initial collagen fibers and vasculature.

Right: Vascular elements from the recipient bed have made contact with the vascular system of the graft. The circulatory continuity of the graft is again intact.





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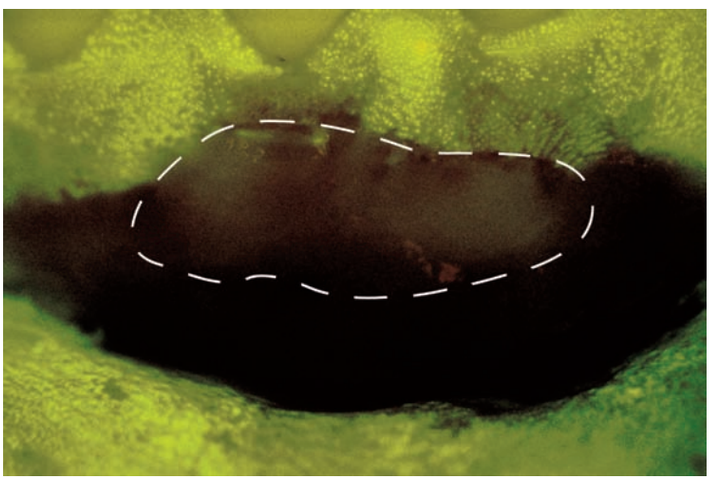
... and via Fluorescence Angiography

Fluorescence angiography is a technique that can be used to visualize the re-vascularization and healing of a free gingival graft (FGG, Mörmann et al. 1975): 2 ml Na-fluorescein (20 %) is injected intravenously into an arm vein. Only 15 seconds later, the substance can be visualized in the capillary circulation of the periodontium using UV light.

Immediately after transplantation, the FGG remains vital thanks only to tissue fluid. The actual "healing-in" (vascularization) begins on about the second post-operative day.

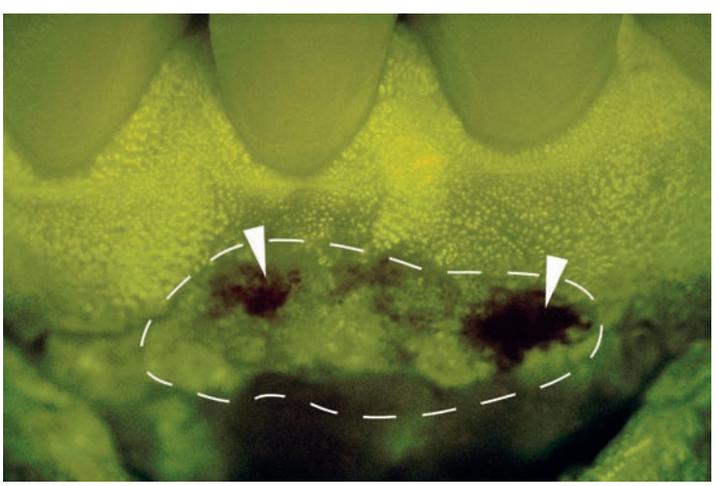
One week later, the healing process is almost complete. Apical to the graft, in the region of overextension, regeneration is not yet complete. A narrow zone is not yet re-vascularized.

Only after ca. 4 weeks does the graft exhibit the typical pale pink color of palatal mucosa.

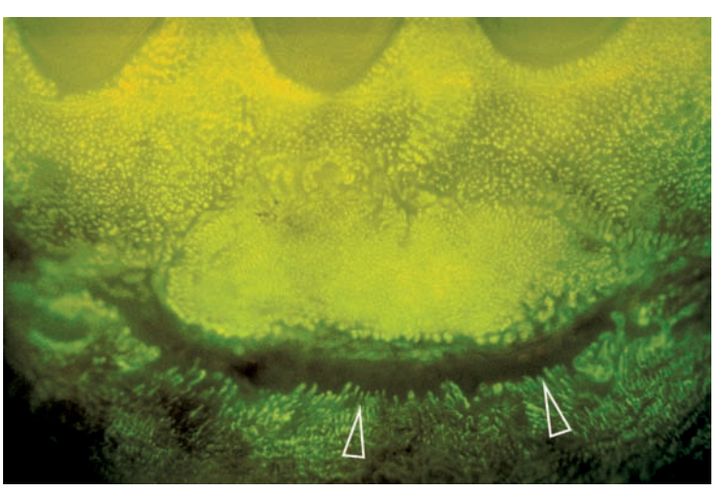


FGG—Fluorescence Angiographic Study

1000 Immediately after Surgery
The entire extension area is devoid of blood circulation (no fluorescence). Nutritive supply to the free gingival graft occurs only by diffusion of tissue fluids from the recipient bed.
The dashed line reveals the margin of the FGG, ca. 6 mm in expanse.



1001 Three Days Post-Operative
Initial vascularization of the free graft is noted, but several areas of ischemia persist (arrows). Extensive areas of the periosteum in the recipient bed apical to the free graft have not yet vascularized, while the marginal gingiva exhibits normal blood circulation.



1002 Seven Days Post-Operative
The free gingival graft is completely "accepted" and fully vascularized.
The apical region of the extension wound (recipient bed; not covered by the graft) appears to be closing slowly with a narrow band still devoid of vasculature (empty white arrows).

Courtesy *W. Mörmann*

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Advantages and Disadvantages of FGG

Advantages

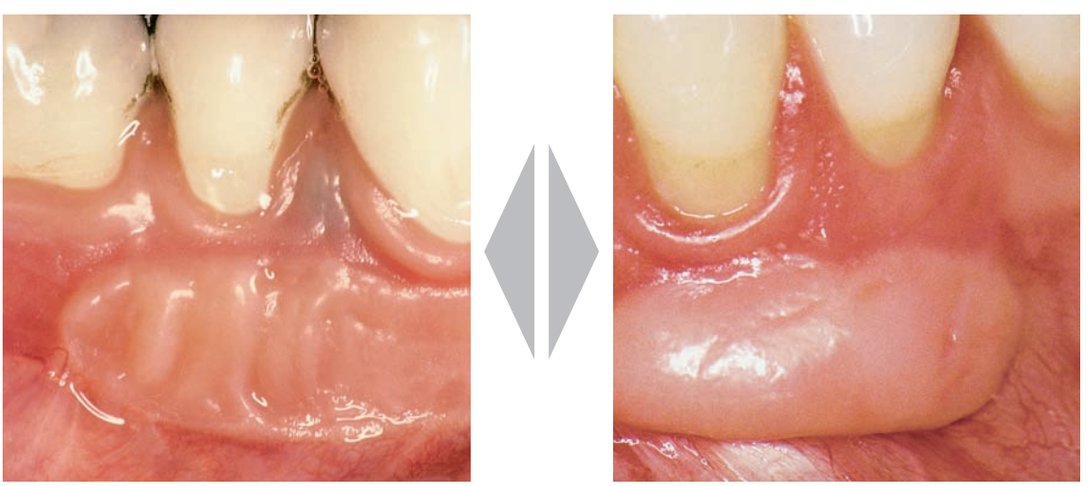
- Stopping the progression of gingival recession
- Deepening the vestibulum
- Widening the band of attached gingiva
- Simplicity of the surgical procedure

The "pull" of the mobile mucosa and frena on the gingival margin is one of the etiologic factors for *gingival recession*. This "pull" can be eliminated by a FGG. This serves to clarify why in some post-operative situations the so-called "creeping attachment" occurs with a coronal migration of the gingival margin.

If an esthetically compromising gingival recession occurs in a visible area—for example in the anterior maxilla—it is usually *covered* by means of mucoplastic surgical methods (p. 413). In many cases, however, the problem can be successfully resolved solely through correction of the patient's traumatic toothbrushing. This or the placement of a FGG apical to the area of recession can result in spontaneous inhibition of further progression of the gingival defect.



1003 Advantages of the FGG: "Creeping Attachment"
Gingival recession was more than 6 mm on the canine. It was possible to *halt* the recession by changing the patient's oral hygiene to a less traumatic sequence, using a soft toothbrush. A small FGG was placed in the canine region. There was improvement in the morphology of the gingival margin after three months. Nine years later (*right*): complete coverage of the previously denuded canine ("creeping attachment").



1004 Disadvantage of the FGG: Rugae—Exostoses
Left: The FGG transplanted some of the palatal morphology. The functional result was good but not the esthetics.

Right: Within two years after the placement of a relatively thick FGG, it was possible to clinically ascertain probable *exostosis*. This can be corrected via osteoplasty, but this is not obligatory. The exostoses are monitored at recall appointments.

Disadvantages

In its *structure and color*, every free gingival graft reflects its site of origin. Because most FGG are taken from the highly keratinized palate, especially thicker transplants exhibit a typical whitish colorization after transplantation, which is clearly visible in contradistinction to the non-keratinized mucosa, but also from the adjacent gingiva, a situation which persists over time.
If the FGG is harvested too anteriorally on the palate, *rugae* will also be harvested, and these will also persist at the transplant site.

In rare cases, there will be formation of *exostoses* beneath the transplanted tissue; these can be several millimeters thick. The etiology is likely some traumatization of the periosteum at the recipient bed during the transplant procedure, which leads to the formation of new bone (Czuskak et al. 1996, Otero-Cagide et al. 1996).

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Covering Areas of Recession

For decades, gingival recession and esthetic problems generally were of only very minor importance in the specialty of periodontology. Therefore, virtually no thought was given to how such esthetic problems might be treated. Although comparative epidemiologic studies don't exist, it appears that gingival recession has become more prevalent in recent decades. Already in 1986, Mierau reported that fully 20 % of 18–22 year-old Germans exhibited gingival recession.
People today have become more aware of their bodies, and oral health seems to play a very important role. Today the demand is for beauty, and that includes a perfect dentition. Such patient demands were readily accepted by the dental profession, and methods were developed to cover unesthetic gingival recession. In addition to esthetics, additional problems in the dental cervical region also require covering areas of recession. The following problems represent reasonable indications for such surgery:

- Esthetics—Patient's desire = important indication
- Hypersensitive tooth necks
- Wedge-shaped defects at the cervical area
- Risk of root caries

Covering areas of gingival recession can be accomplished using the following surgical methods:

- Pedicle flaps, repositioned coronally, laterally or rotated
- FGG—direct coverage with a free, epithelialized graft
- CTG—covering via the bilaminar technique—with flap and the free connective tissue graft, without epithelium
- GTR—coverage by means of a displaced flap incorporating a barrier membrane
- Additional new techniques, e. g., use of bioactive proteins

Mucogingival and plastic periodontal surgical procedures demand experience and clinical capability on the part of the therapist, and also compliance by the patient. The latter includes not only adherence to all post-operative instructions, especially with regard to oral hygiene, but also the elimination of risk factors, e. g., smoking (Patient selection, p. 297)

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Shape of the Defect—Selection of Surgical Technique

The four classes of mainly facial gingival recession described by P.D. Miller (1985) were described in the chapter "Forms of Periodontal Diseases" (p. 160). However, Miller did not provide information about the *etiology* of gingival recession.

While Classes I and II are usually the result of morphologic/anatomic conditions (thin or absent facial osseous lamellae) and improper, traumatic oral hygiene (scrubbing), Classes III and IV usually are the result of a long-standing periodontitis (gingival shrinkage) or periodontal treatment itself, particularly resective therapy. Tooth positional anomalies can also lead to Class III and IV gingival recession.

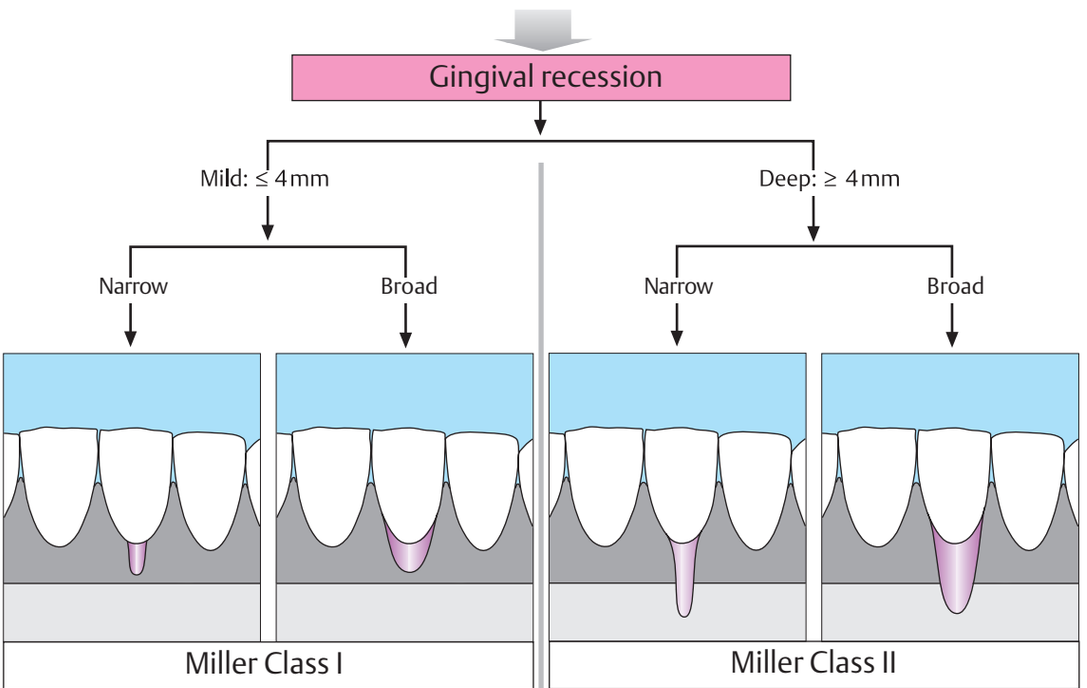
For these reasons, a "complete" coverage of areas of gingival recession can only be expected with Classes I and II.

Rebuilding or reconstructing lost papilla (interdental soft tissue; recession Classes III and IV) remains today a questionable undertaking.

1005 Pre-Surgical Consideration of the Gingival Defect—Aids for Determining the Appropriate Surgical Approach
The Miller Classes I and II and their further subclassification into "narrow" or "wide," determine the indicated surgical procedure. The method modified by DeSanctis and Zucchelli shows that any given type of recession may be treated with various surgical techniques.

● Recommended methods
○ Conditionally recommended methods

Modified from *M. DeSanctis & G. Zucchelli 1996*



	Miller Class I, Narrow	Miller Class I, Broad	Miller Class II, Narrow	Miller Class II, Broad
Pedicle, repositioned flap: Lateral, coronal, or semilunar	●	●	●	
Two-surgery technique/ Bernimoulin	○	●	●	○
FGG direct	●	●		
Bilaminar technique, connective tissue graft	●	●	●	●
GTR using various types of membranes			●	●

Therefore, the remainder of this chapter will deal with the treatment of Miller Classes I and II. Even with this limitation, however, not all surgical techniques are necessarily indicated for these types of gingival recession. In additional to Class I (defects within the attached gingiva) and II (defects extending into the mobile mucosa), differentiation must be made between *narrow* and *broad* areas of gingival recession.

In addition to clinical conditions as determining factors for the choice of a surgical approach, the preferences and proficiency of the clinician with diverse methods and their numerous modifications are also of importance.

The surgical techniques summarized in Figure 1005 (above) indicate that for Miller Class I recession, the pedicle flap, or direct coverage with a FGG or a connective tissue graft (CTG) is preferable. For Miller Class II (4 mm and deeper recession), the connective tissue graft or guided tissue regeneration (GTR) are indicated.

It will become clear that the free connective tissue graft is the preferred treatment (gold standard) and can be used for practically all types of gingival recession. Its only disadvantage is the second surgical wound in the palate (for autogenous grafts).

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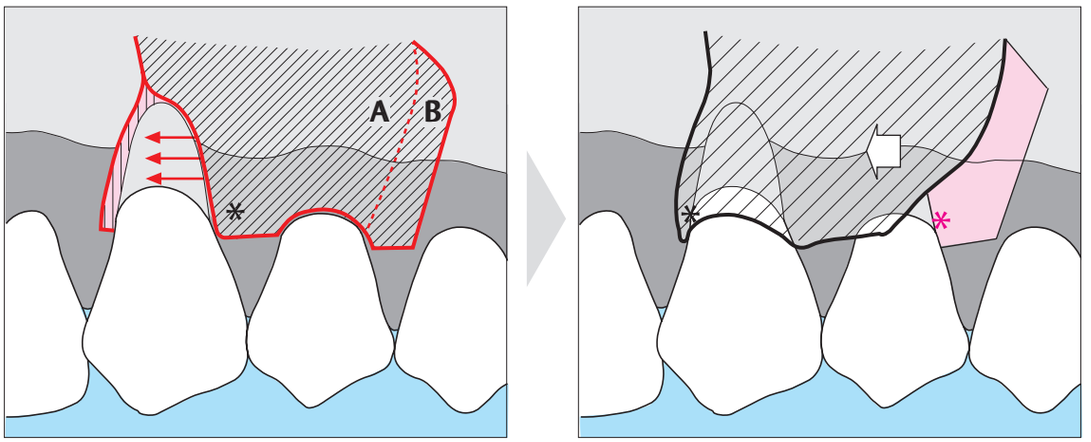
Pedicle Flap Techniques

Lateral—Coronal—Semilunar Repositioning

For more than 50 years, serious attempts have been made to not only halt the *progression* of gingival recession but also to *cover* it. Grupe & Warren (1956) studied the possibility of a *laterally* repositioned pedicle flap to cover localized gingival recession. The major disadvantage of the technique was that new gingival recession was "created" on the adjacent tooth from which the marginal gingiva was displaced. Modifications of the surgical technique were not generally accepted. Later, Allen & Miller (1989) proposed a "split flap" directly apical to the area of recession, and coronal repositioning. However, with excessive tension on a possible *excessively thin* flap, recurrence or necrosis occurred (Pini-Prato 2000). The method described by Tarnow (1986, Fig. 1008) brought with it the danger that the half-moon shaped incision in the mucosa over the recession site would expose a boneless dehiscence over the root surface.

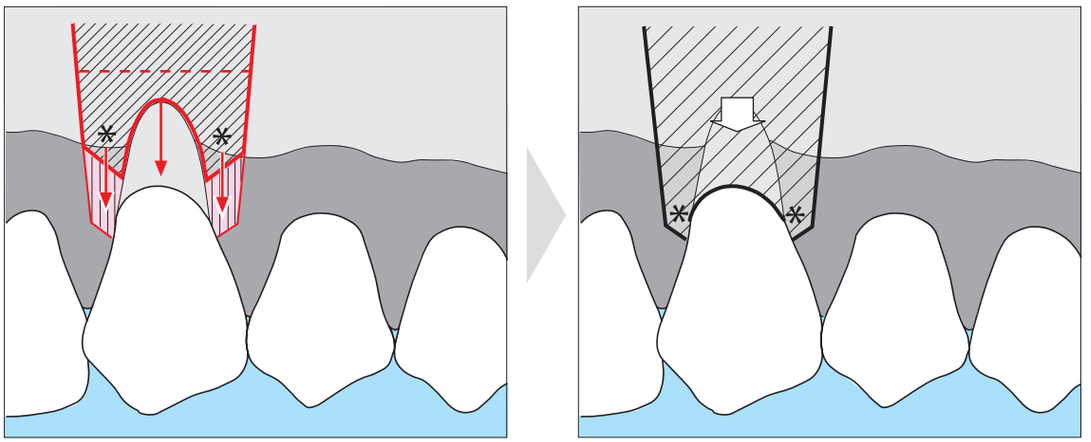
Despite these limitations, modified pedicle flap techniques are today enjoying a renaissance, for various reasons (p. 440).

1956 Grupe & Warren technique



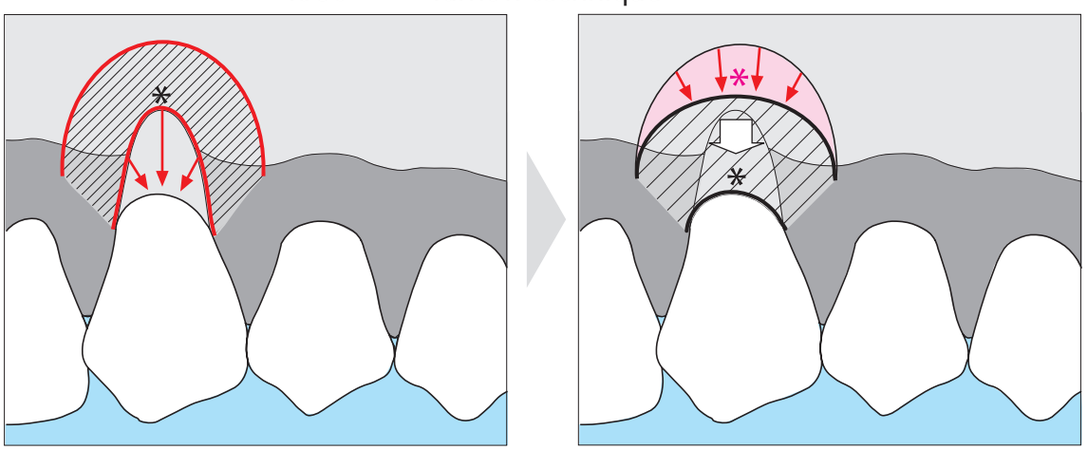
1006 Laterally Repositioned Flap (Grupe & Warren 1956)
After performing an incision around the area of recession on tooth 23, and a vertical incision distal to the adjacent tooth 24, a combined flap is prepared (**A** = full thickness flap; **B** = split-thickness flap). The flap is repositioned laterally over the area of recession (arrows, left) and sutured to place. This results in a situation in which tooth 23 is covered by the pedicle mucoperiosteal flap (**A**), while the distally exposed surface is covered by periosteum (red ✱).

1989 Allen & Miller technique



1007 Coronally Repositioned Flap (Allen & Miller 1989)
Using this surgical technique, a split-thickness flap is created following circumferential incisions around the defect, as well as vertical incisions. The papillae proximal to tooth 23 are "de-epithelialized" (very shallow gingivoplasty, arrow). After conditioning of the root surface, the flap is coronally repositioned to cover the denuded root surface without tension (arrows), and sutured to place. The surgical site may also be covered with a tissue adhesive (cyanoacrylate).

1986 Tarnow technique



1008 Half-Moon shaped, Coronally Repositioned Flap (Tarnow 1986)
This involves covering the recession area by creating a half-moon shaped, partial thickness flap, with pedicle blood supply bilaterally. Coronal to tooth 23, a marginal incision and a parallel incision in the mucosa are made. The split-thickness flap is repositioned coronally to cover the denuded root surface (arrows). The tissue is maintained in place under finger pressure for ca. 5 min, then covered with a dressing.

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Direct Coverage with a Free Gingival Graft

As patient demand for oral esthetics has increased, the covering of gingival recession, especially in the visible anterior regions, is increasingly performed. Even as early as 1968, Sullivan & Atkins, and then Holbrook & Ochsenbein (1983) described methods for covering gingival recession using a free gingival graft (FGG). Miller (1982, 1985, 1987) categorized the various types of gingival recession, and provided appropriate guidelines for the coverage of Class I and II defects (p. 161).

The case presented below is a 20-year-old female in which a Miller Class II gingival recession was covered directly with a FGG. The patient was a professional musician (wind instruments) and was therefore very concerned about her anterior teeth.

In this case, the goal was to cover the recession area, with simultaneous widening of the attached gingiva. Because function, not esthetics, was the priority, in this case the treatment plan included direct coverage using a free gingival graft.

1009 Initial Clinical View
This patient was referred from the orthodontist because following orthodontic treatment there existed a pronounced area of gingival recession on tooth 41. The patient exhibited good oral hygiene, but was not able to maintain the facial surface of tooth 41 in a plaque-free condition, and this led to localized inflammation.

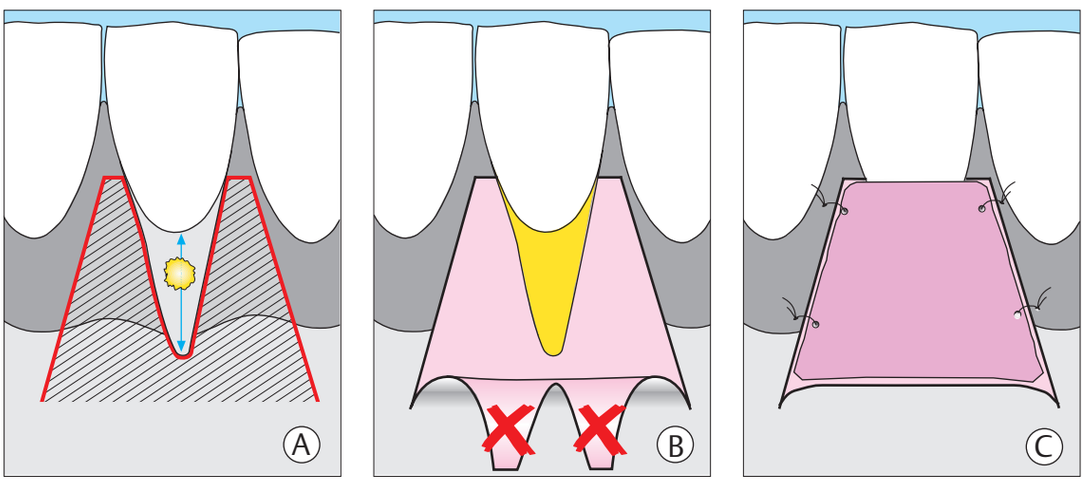


1010 Direct Coverage Using a FGG—Surgical Protocol

A
- "Conditioning" of the root surface
- Intrasulcular incision
- Horizontal incision at the cementoenamel junction
- Vertical incisions

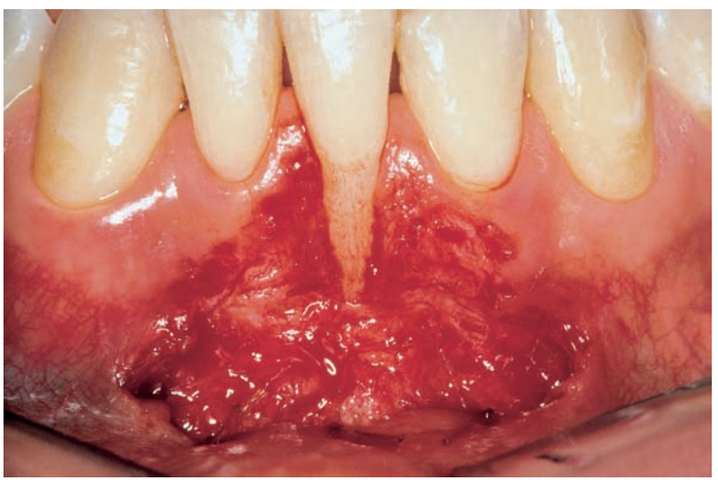
B
- Creation of a split-thickness flap, with shortening, p.r.n
- Evening the wound bed

C
- 2 mm thick FGG from the palate
- Trimming and adapting
- Fixation (sutures)



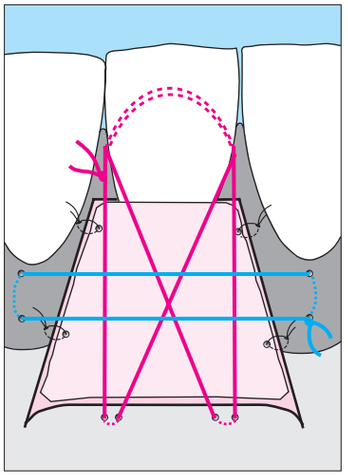
1011 Creating the Wound Bed
Intrasulcular, horizontal and vertical incisions are performed and the split-thickness flap is prepared. The quadratic incisions cut numerous blood vessels, so that following placement of the quadratic graft, there will be effective re-connection to the vascular system.

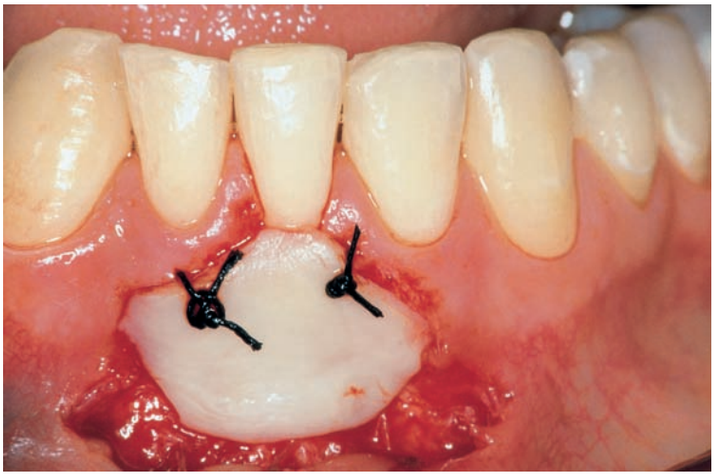
Right: "Biomodification" of the root surface using a tetracycline slurry (3 min).





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1012 FGG Sutured to Place
In contrast to other authors (Miller 1982, 1985; Holbrook & Ochsenbein 1983), only two sutures were used; however, the FGG was held *in situ* with pressure immediately post-surgically to assure adaptation to the root surface.

Left: Alternative FGG fixation

Black	Interrupted sutures
Red	Vertical and circumferential suture
Blue	Horizontal compression suture



1013 FGG Ten Days after Surgery
The sutures have been removed. The graft has satisfactorily "taken" (Re-vasculatization, p. 410). The superficial epithelial layers have sloughed.

The expanse of recession coverage cannot be determined at this time.



1014 FGG after Six Months
The FGG is fully re-epithelialized and exhibits (unfortunately!) the pale color of palatal mucosa. The previous gingival defect cannot be probed. The type of new attachment could only be determined histologically (long junctional epithelium?, connective tissue?).

Pasquinelli (1995) demonstrated in a single case, *regeneration* in the facial area of a formerly denuded root surface (bone, cementum and periodontal ligament).



1015 Eighteen Months after FGG
The area of recession is completely covered. Between the sixth and eighteenth month, "*creeping attachment*" occurred.
The clinical course of the marginal gingiva in the mandibular anterior region is harmonic and at the physiologic level. The probing depths are normal at ca. 1 mm. The light color of the FGG has intensified, but in this region it is not esthetically disturbing.

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Coronal Repositioning after Free Gingival Grafting—2-Stage Surgical Procedure

Bernimoulin (1973) described a *two-stage* method for covering gingival recession areas, designed to improve the relative unpredictability of earlier pedicle flap procedures:

- During an *initial surgical procedure*, a FGG is placed apical to the area of recession, as portrayed on p. 401.
  - If coverage of the area of recession is desired in addition to halting the *progression* of gingival recession, a few months later during a *second surgery*, coronal repositioning of the now much *thicker* tissue can be performed to cover the denuded root surface. Because of the requirement for a second surgical procedure, this procedure is seldom performed today.

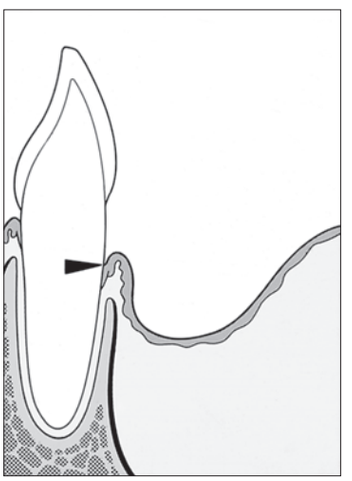
In the case presented here, a 26-year-old female was disturbed by a pronounced area of gingival recession on tooth 41; the tissue was secondarily inflamed. The tissue defect extended into the mobile oral mucosa. The patient's oral hygiene is good, but was difficult to perform on the affected tooth. Plaque, calculus and inflammation resulted. Lip movement caused tension on the gingival margin.

Progression of recession was halted with a FGG; but the patient desired to have the exposed root surface *covered*.

1016 Initial Clinical View
The anterior mandibular sextant exhibits mild, generalized gingival recession. On tooth 41, however, there is a significant area of gingival recession of 6 mm, which extends into the mobile mucosa (Miller, Class II). The marginal gingiva is inflamed.

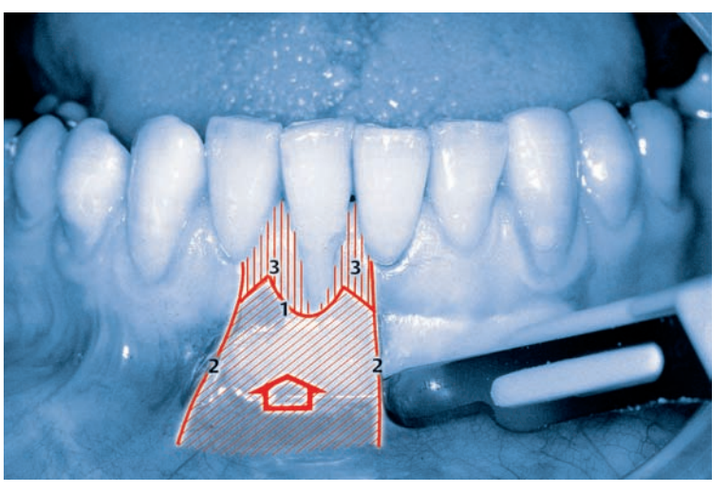
Right: Diagram of the initial situation in the orofacial section. Gingival recession without a periodontal pocket (black arrow).

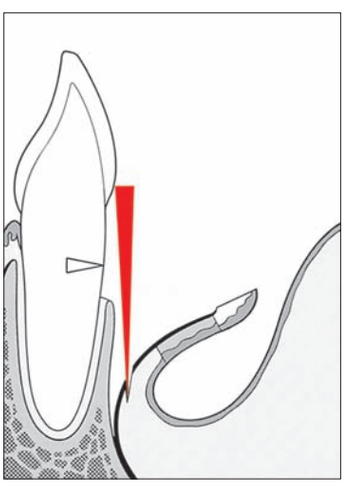




1017 Incisions for the Second Surgical Procedure
1 *Marginal:* On mandibular tooth 41, an arcuate incision is made. "New papillae" are created lateral to the initial incision.
2 *Vertical incisions* extending into the mucosa.
3 *Gingivectomy*: The papillae are de-epithelialized to create a recipient wound bed.

Right: Flap reflection with incision of the periosteum (large red arrow).

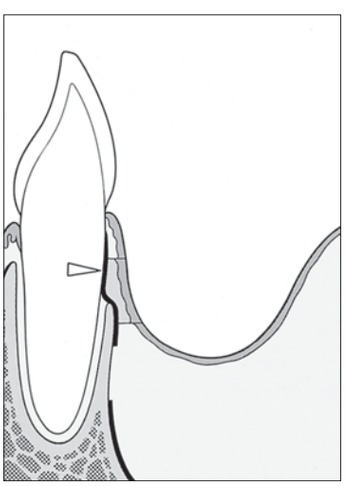




1018 Six Months after Coronal Repositioning
The gingival recession on tooth 41 has been successfully covered, and there is no periodontal pocket on the facial surface. The incision lines and spatial demarcations of the coronally repositioned FGG are clearly visible.

Right: The effect of repositioning the flap coronally is clearly depicted in the diagram. The open arrow depicts the position of the gingival margin pre-surgically.





Covering Areas of Recession with a Connective Tissue Graft

Covering areas of gingival recession with a *free gingival graft* (FGG) is usually successful (p. 416), but is associated with the major disadvantage that the pale coloration of the palatal mucosa is maintained and therefore the FGG method is often not indicated in visible areas for esthetic reasons. In an attempt to resolve the esthetic problem, during the 1980s alternative methods were developed, which involved the use of subepithelial connective tissue for covering areas of recession (Raetzke 1985, Langer & Langer 1985, Nelson 1987, and others).
These free *connective tissue grafts* (CTG) are partially or completely covered with gingiva and/or mucosa at the transplant site. The methods were subsequently modified (Harris 1992, Schädle 1993, Bruno 1994) and became routine in periodontal practice. The success of the procedure is generally predictable. On average, ca. 90% success in the esthetic coverage of areas of recession can be expected. CTG have therefore become the *gold standard* for covering areas of gingival recession.

As with a FGG, the "survival" of a CTG can only be ensured by an adequate blood supply. It is therefore safer to position the CTG in the recipient bed such that it receives nutritive blood supply from both sides ("bilaminar").

Care must be taken to prevent any damage to the connective tissue when it is harvested from the donor site on the palate. This procedure can be even more technically difficult that the creation of the recipient bed, and will therefore be described in detail on page 421.

Even before soft tissue flap reflection, the convexity of the root surface to be covered should be flattened somewhat using fine diamond burs. This will ensure tight contact of the tranplanted tissue to the tooth surface. Further, such odontoplasty creates a "smear layer," that can be removed via conditioning with biomodifiers (p. 351) such as citric acid (pH 1), tetracycline-HCl or EDTA. These agents mildly decalcify the root surface, exposing dentinal collagen fibers. This should provide a quicker and stronger attachment of the CTG with the root surface during the healing phase, via adhesins (surface molecules from activated fibroblasts) and their ligands.

Furthermore it is likely that the conditioning solutions kill bacteria that remain on the root surface or within cementum/dentin.

Following a successful connective tissue transplant, virtually no pocket depth can be probed at the gingival margin. Only histologic examination can reveal the type of connection between tooth and soft tissue. A long junctional epithelium in the coronal area is most likely (Harris 2001). In the apical regions of the former area of recession there may be actual connective tissue attachment (close approximation or even "new attachment").

As with all techniques for covering areas of recession, connective tissue grafts, depending upon the technique employed, lead to more or less pronounced widening of the band of attached gingiva (Bouchard et al. 2001).

On the next page, four techniques by periodontal pioneers in the "bilaminar" CTG technique are presented:

- CTG technique according to Langer & Langer (1985)
- CTG technique according to Nelson (1987)
- CTG technique according to Harris (1992)
- CTG technique according to Bruno (1994)

Beyond the cited authors, many other periodontists have offered modifications of the techniques.

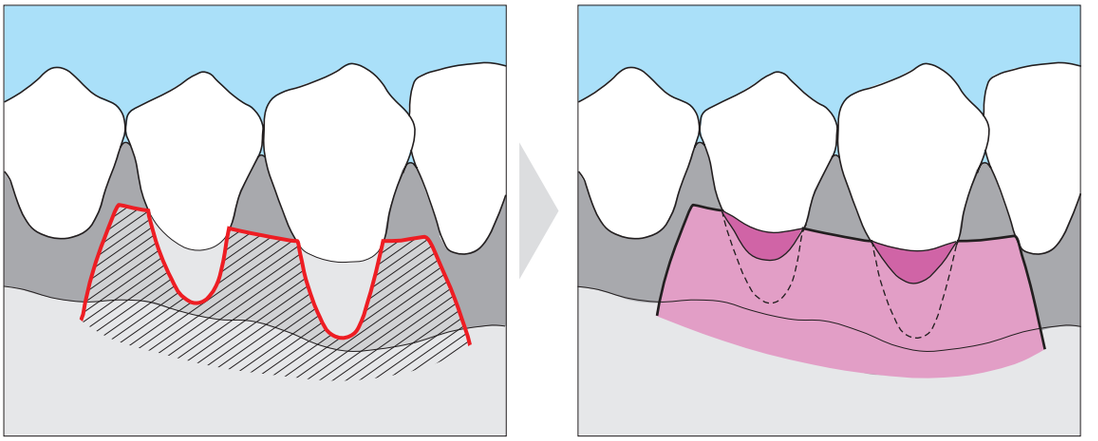
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Comparison of Four CTG Procedures

1985 Langer & Langer technique

1019 CTG—Langer & Langer: Coronal Flap Repositioning

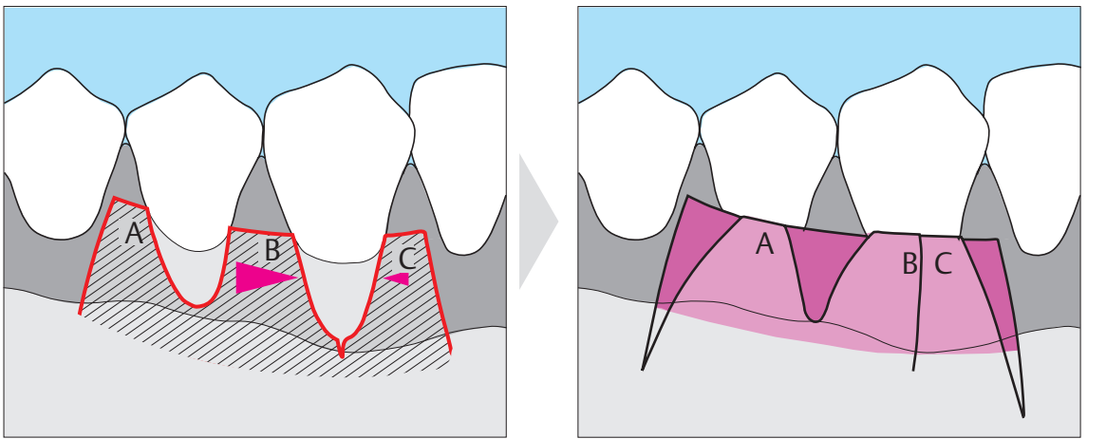
- Horizontal incision at the CEJ and gingival margin
- Vertical incisions beyond the mucogingival junction
- Split flap reflection
- Removal of the CTG from the palate
- Covering the recession area with the CTG up to the CEJ
- Partial covering of the CTG with the soft tissue flaps



1987 Nelson technique

1020 CTG—Nelson: Laterally Repositioned Flaps **(see also p. 425)**

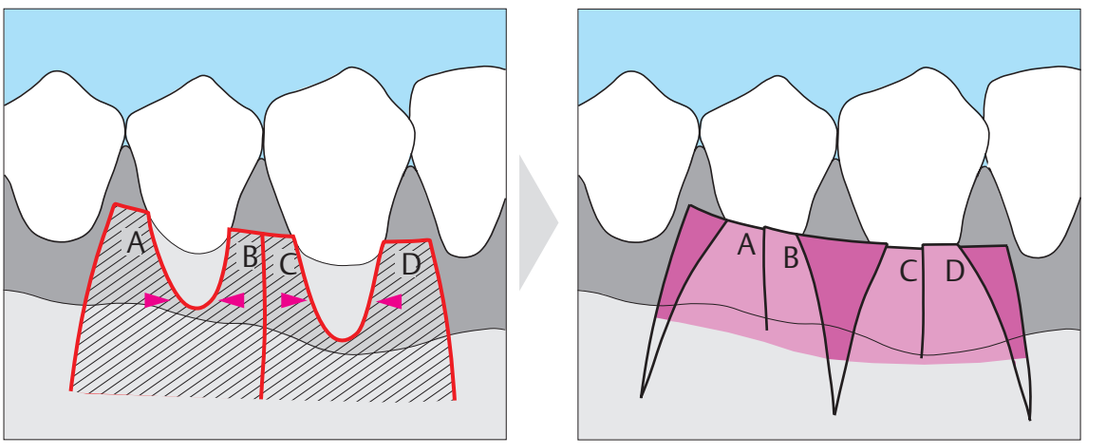
- Incisions
- "Freshening up" the adjacent soft tissues
- Reflection of full-thickness flaps
- Periosteal incisions, p.r.n.
- Removal of palatal CTG
- Covering the defect
- Covering the graft by laterally repositioning the flaps
- The CTG is nourished *bilaminarly*
  - From the soft tissue flaps
  - From interdental bone



1992 Harris technique

1021 CTG—Harris: "Lateral Double Papilla" Technique

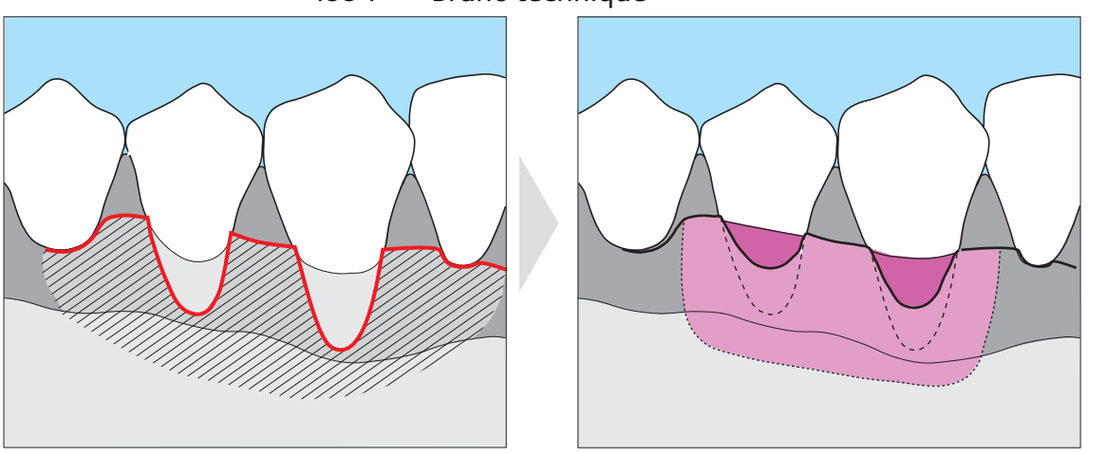
- Horizontal incisions
- Vertical incisions mesial and distal to the tooth being treated ("interradicular")
- Split-thickness flap reflection
- Root "conditioning"
- CTG from the palate
- Covering the recession with the CTG
- Joining the tips of the flaps over the area of recession
- Nutrition from the flaps and the periosteum



1994 Bruno technique

1022 CTG—Bruno "Envelope Technique" Horizontal Incision at the Level of the Cementoenamel Junction

- *No* vertical incisions
- Split-thickness flap reflection (subepithelial "pocket")
- CTG from the palate; technique, see p. 421
- Placing the CTG into the "envelope" created by the flap reflection
- Suturing of the CTG and the gingival flaps



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Harvesting Graft Tissue from the Palate

Free grafts are usually harvested from the palate. Such grafts may be epithelialized (FGG) or purely connective tissue (CTG). Both types of grafts may be harvested with varying thickness (Fig. 1024).

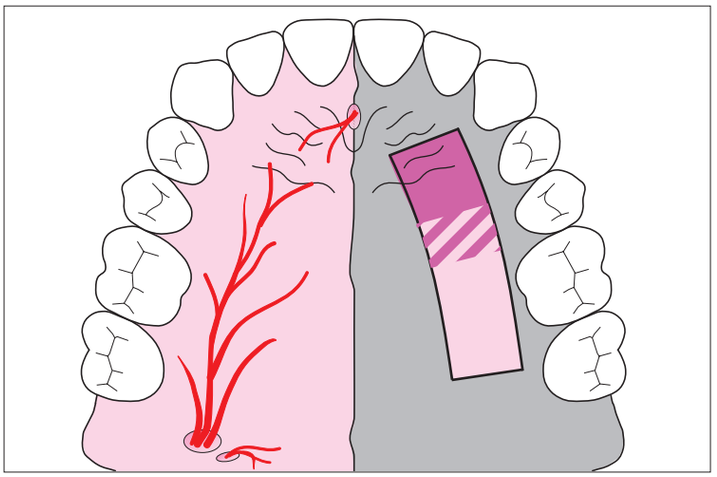
For direct *coverage of areas of recession*, relatively thick tissue segments are harvested ("full thickness" FGG; Miller 1982, Holbrook & Ochsenbein 1983). For treatment in areas where esthetics is of prime importance, thinner epithelialized grafts ("partial thickness") can be employed to *halt* the progression of gingival recession or *deepen* the vestibulum.

In the realm of esthetic, "plastic" periodontal surgery, the free connective tissue graft predominates. For a secure and resistant coverage of gingival recession, the CTG should be ca. 1.5–2 mm thick.

Massive pieces of connective tissue can also be used as free grafts for *soft tissue ridge augmentation*. Of particular importance esthetically, after this sort of ridge augmentation (p. 505) bridge pontics can be optimally integrated into the dental arch (ovate pontic, "emergence profile," papillae, p. 502).

CTG
Free connective tissue graft

FGG
Free gingival graft



1023 Donor sites for CTG and FGG—Vascular Considerations
Grafts must be harvested ca. 2 mm removed from the gingival margin in order to avoid subsequent palatal gingival recession. In addition, grafts should be harvested from the canine-premolar regions in order to avoid the greater palatine artery.

On the other hand, FGG must be harvested more distally in the *rugae-free* regions, because the thinner FGG transplants pose little danger of vascular damage.

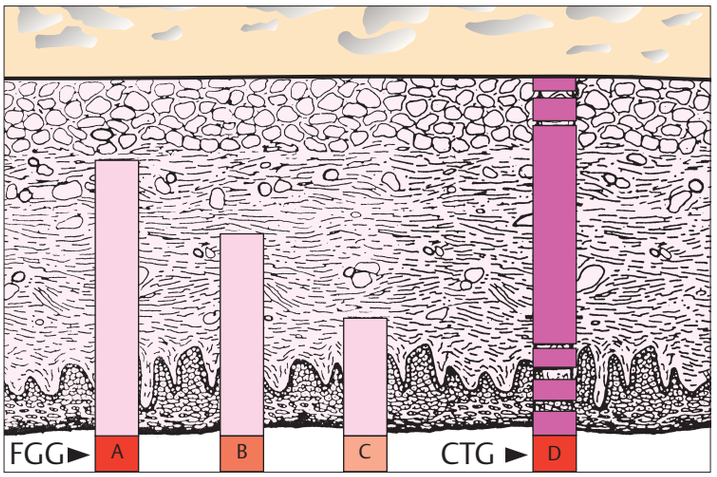
Bone

Glandular tissue
Fat

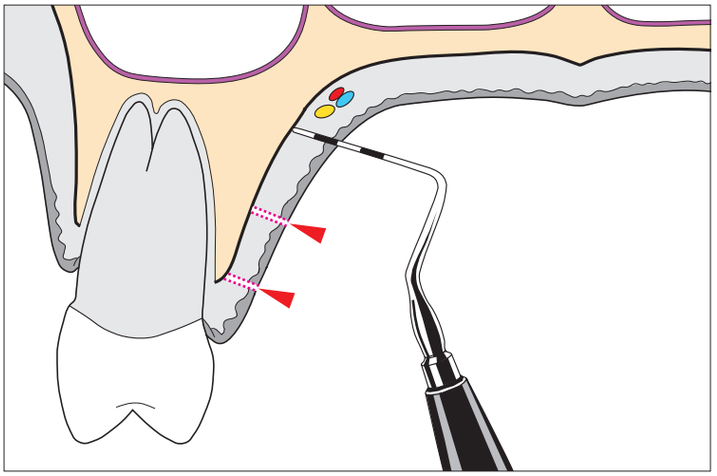
Connective tissue

Collagenous fiber structure

Epithelium



1024 Palatal Tissue—Comparison between the Histology of FGG and CTG: Free Gingival Graft
A "Full-thickness" graft for the direct coverage of recession areas
B, C Thinner grafts ("partial thickness") to halt progressive recession or to widen the band of attached gingiva
Free Connective Tissue Graft
D Varying thickness for the coverage of recession and correction of alveolar ridge defects.



1025 "Sounding"—Determining Palatal Areas Indicated for Graft Donor Site
The thickness of the palatal soft tissue should be at least 3–4 mm. This thickness is measured with a periodontal probe (e. g., CP 12) under local anesthesia.

With a very steep (high) palatal vault, the vessels may be up to 17 mm removed from the cementoenamel junction; with a flat palate, this distance may be only 7 mm (Reiser et al. 1996).

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Harvesting the Graft from the Palate—Schematic and Clinical

Various techniques for harvesting connective tissue from the palate will be demonstrated on the following pages. The authors of this *Atlas* prefer the "open door" technique shown in Figure 1033.

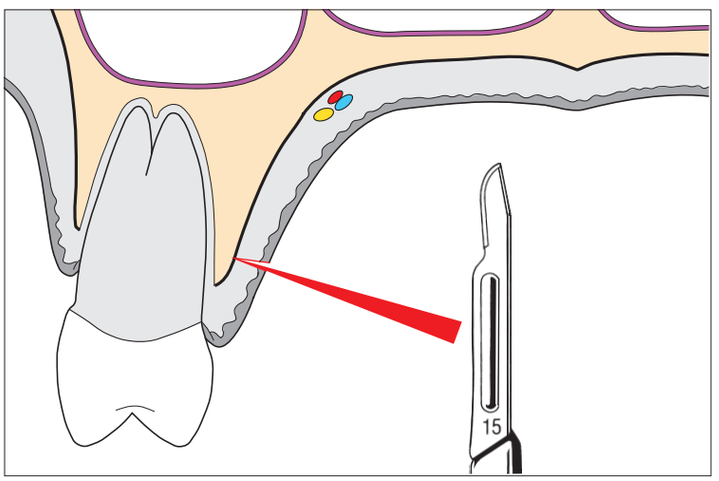
One significant complication during removal of a connective tissue graft can be severe hemorrhage. For this reason, the connective tissue graft should not be harvested too far distally on the palate. The presence of rugae is of less significance with the CTG than with an epithelialized FGG, which is more superficial and prepared much further distally in the region of the molars.

Bruno Technique

Bruno (1994) described harvesting the palatal graft without flap reflection, by means of two incisions. The first incision is *horizontal* and *paramarginal*. The length of this incision corresponds approximately to the length of the desired graft. Parallel to the first incision, a second, *vertical undermining* incision is then performed, which determines the width of the CTG. Following additional "internal" vertical incisions both mesially and distally, the wedge-shaped graft is released and removed using an elevator.

1026 Initial Incision—Horizontal

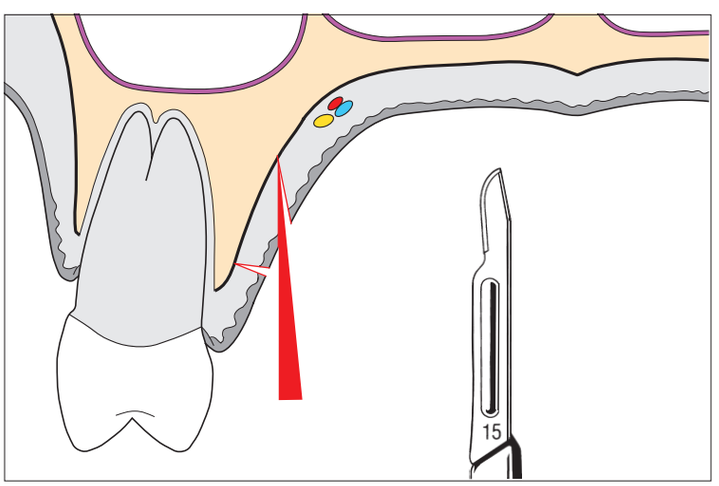
At the donor site, perpendicular to the long access of the tooth, the first incision is made down to the level of bone approximately 2–3 mm apical to the gingival margin. Before making this incision, the anesthetized palatal tissue must be evaluated for its suitability as a donor site: The soft tissue thickness must be at least 3–4 mm.



1027 Second Incision—Vertical

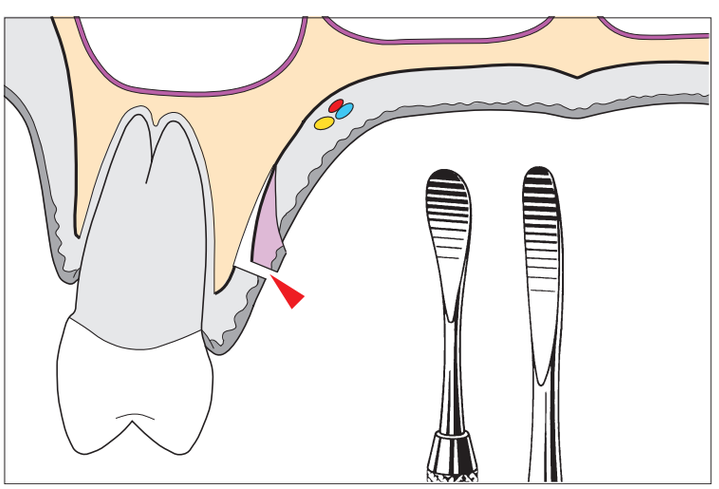
Approximately 2 mm apical to the initial incision line, the second incision is made. It should be almost parallel to the tooth long axis and, if possible, ca. 10 mm deep (the sharp portion of the No. 15 scalpel blade is a good measure). These incisions delineate the length and width of the CTG.

The greater palatine nerve and artery must be avoided!

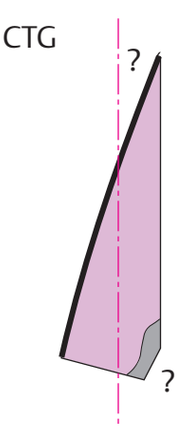


1028 Harvesting the Connective Tissue Graft

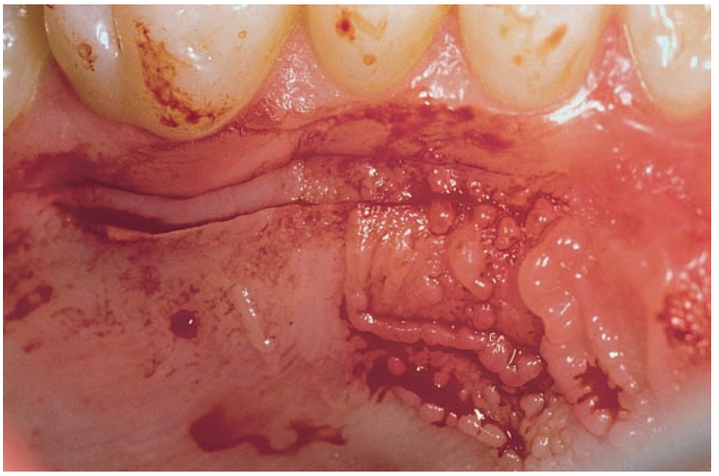
Using a fine elevator, the mesial and distal ends of the graft, delineated initially by the scalpel, are mobilized, and the graft is removed, including periosteum. Upon a sterile surface, the connective tissue graft must be trimmed before placement in the recipient bed (even thickness, removal of epithelium and fatty tissues etc.).



Right: CTG

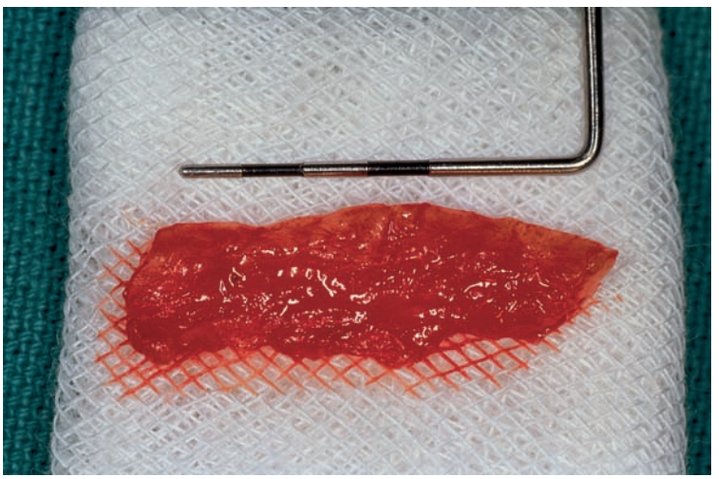


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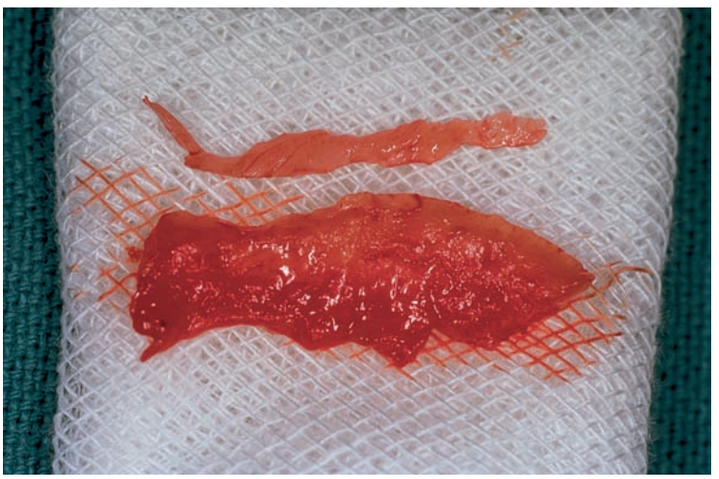


Practical Surgical Procedure for Harvesting a CTG from the Palate as described by Bruno

1029 Incisions
Here, the two necessary incisions have been made. They were made in the area of the two premolars (teeth 14, 15) and parallel up to 2 mm apart, extending to approximately the middle of the first molar (tooth 16).
The further tissue preparation is performed as shown diagrammatically in Fig. 1028.

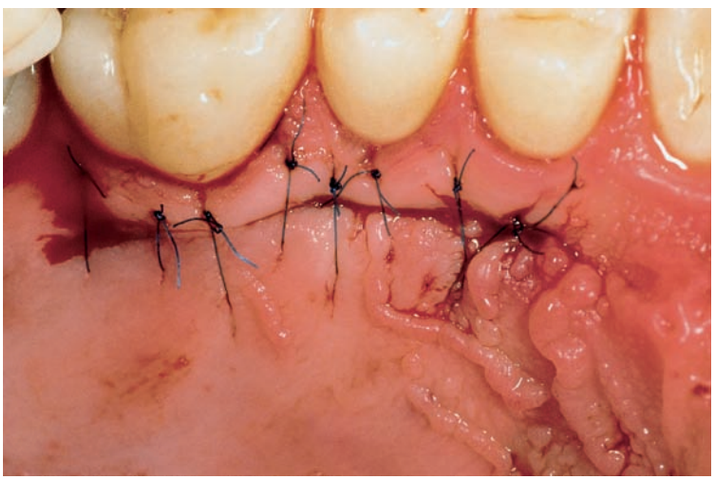


1030 Connective Tissue Graft
The CTG is 17 mm long. On its top surface there is an approximately 2 mm wide epithelialized strip that was created by the two incisions. The graft must not be permitted to dry out, nor should it be otherwise compromised.



1031 Removal of the Epithelium Surface—Yes or No?
For an esthetically optimum *coverage of the recession*, the layer of epithelium must be removed. The "new" CTG is positioned in the prepared "envelope" above the area of recession.

For the build-up of a *ridge defect* (e. g., inlay graft, p. 505) the epithelial layer should not be removed. It will reside in the crestal area of the built-up ridge.



1032 Suture Closure of the Donor Site
The palatal wound margins are usually only 2 mm apart, but it is not always possible to tightly approximate the wound margins (no tension!). But the remaining wound is generally minimal, and not uncomfortable for the patient.

Courtesy *C. Augustin*

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Additional Methods for Harvesting Connective Tissue Grafts

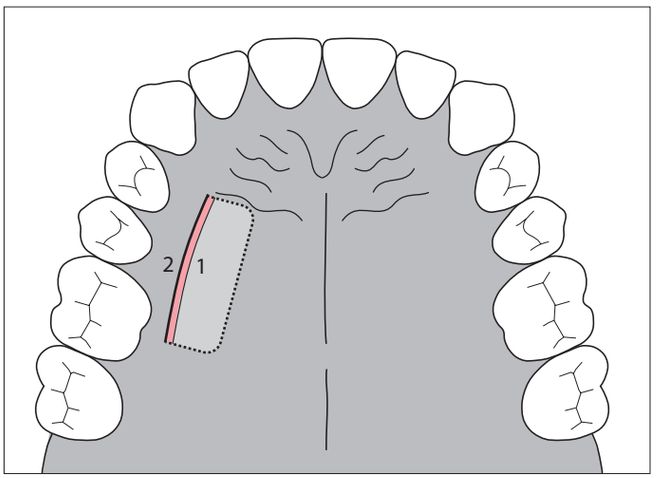
Two different techniques for harvesting a connective tissue graft can be differentiated:

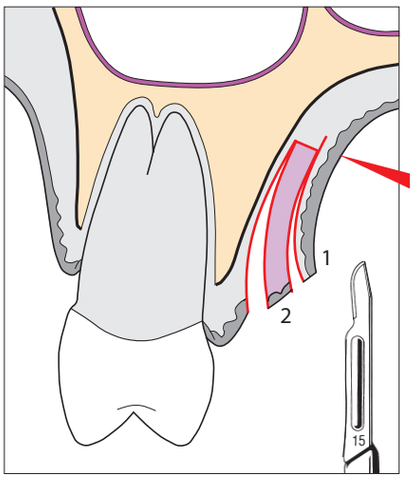
- The first technique creates pedicle "trap doors" consisting of epithelium and some subepithelial connective tissue. Following "opening of the door," a thicker CTG is removed using a scalpel or mucotome, and then the "door" is closed.
- The connective tissue is harvested using a scalpel with two blades (double incision, "double knife," Harris 1992), and both wound margins are easily sutured.

There have been many modifications of both of these techniques in terms of incisions and necessary armamentarium (see below). The ideal, complication-free harvesting technique has not yet been described.

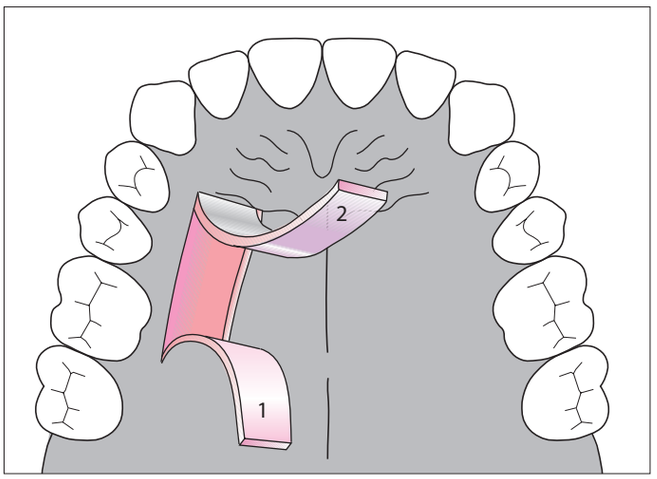
None of the recommended methods can completely eliminate difficulties or dangers. These include severe hemorrhage, disturbed wound healing (e.g., flap necrosis), as well as harvesting the CTG without direct vision (Bruno, Harris).

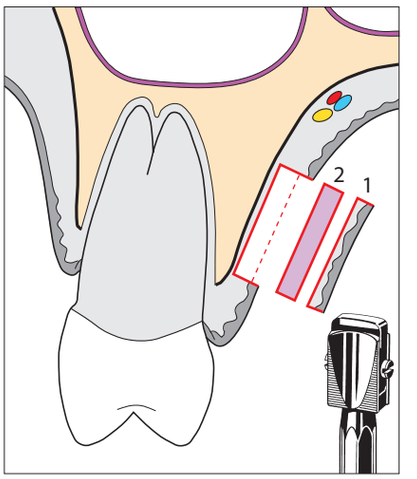
1033 The "Open Door" Method: Broad-Based Pedicle
A deep horizontal incision is made ca. 3–4 mm removed from the gingival margin. Two shorter incisions are made at either end of the initial incision, creating the "*door*." The door is undermined and opened (**1**). After sharp dissection, the CTG is harvested using an elevator.
If a strip of epithelium remains on the CTG, it can be removed by careful sharp dissection (**2**).





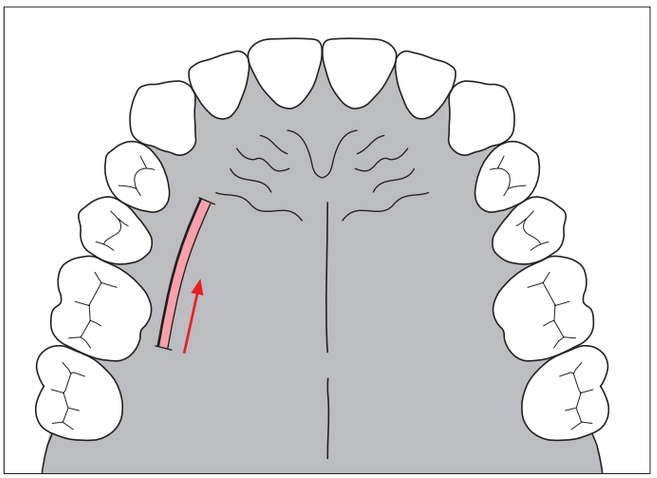
1034 "Open Door" Method with a Narrow Base
A palatal "door" (**1**) can also be created with a narrower and more distally located base. Preparation and the removal of connective tissue (**2**) can be performed with the scalpel or exclusively with a *mucotome* (Fig. 971). Using this procedure, the maintenance of a long strip of epithelium upon the CTG is not possible, and not even necessary in most cases for covering areas of recession.

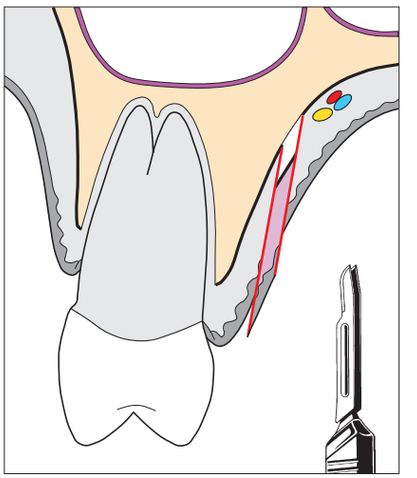




1035 Harvesting a CTG with the Double-Scalpel (Harris 1992)
This technique is similar to that of Bruno (p. 422), but is performed in a single procedure using a special double-scalpel with two parallel No. 15 scalpel blades. The distance between the two blades can be varied between 0.5 and 3 mm, depending upon the thickness of the desired CTG.

Harris double knife:
- H & H Company, Ontario, CA
- Harris Double-Bladed Scalpel, Miter Inc.





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Free C onnective Tissue Graft—Nelson Technique

Autogenous Connective Tissue Graft without Epithelium

Surgical Procedure, Step-by-Step

The diverse methods for covering areas of gingival recession with free connective tissue grafts (CTG) are similar. Nelson's (1987) simple technique involves covering the connective tissue graft at the area of recession with local mucosa (pedicle, laterally repositioned full-thickness flaps).

In the case presented here, a healthy 20-year-old female exhibited pronounced areas of recession on teeth 43 and 44, most likely due to improper oral hygiene and the pull of several frena. The cervical area of tooth 44 exhibits abrasion, probably due to toothbrushing. The anatomic/morphologic situation, not least the shallow vestibulum, effectively prevents adequate oral hygiene in this area (note BOP). In addition, the patient complained of sporadic hypersensitivity of the cervical areas of the teeth in this region. The remaining dentition exhibited no areas of recession and otherwise healthy conditions, including low plaque and bleeding indices.

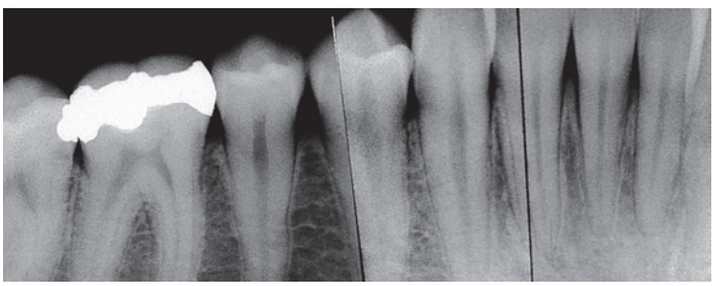


1036 Initial Clinical View: Gingival Recession on Teeth 44 and 43; Miller Classification I-II
Note the frena stretching from the gingival margins of these two teeth, the mild inflammation in the apical regions of the gingival margin, and wedge-shaped defects on the teeth. The papillae completely fill the interdental spaces.

Broad papillary tissue anticipates a good prognosis for CTG therapy (nutrition of the transplant).

	8	7	6	5	4	3	2	1	1	2	
Re Mand.			1	1	3	3	1	1			Re Mand.
aG			2	2	1	1	3	2			aG

1037 Recession (Re) and Width of Attached Gingiva (aG)
Only teeth 44 and 43 exhibit pronounced gingival recession of ca. 3 mm. Attached gingiva is completely absent on these teeth (< 1 mm).



Radiographic View
The interdental bone is at its normal height. Facial gingival recession is never visible in the radiograph.



1038 Initial View Following Disclosure
Following application of a disclosing solution, in the area of gingival recession it is clear that these regions are not plaque-free, even though the patient tries to maintain good oral hygiene. Potential problems include:

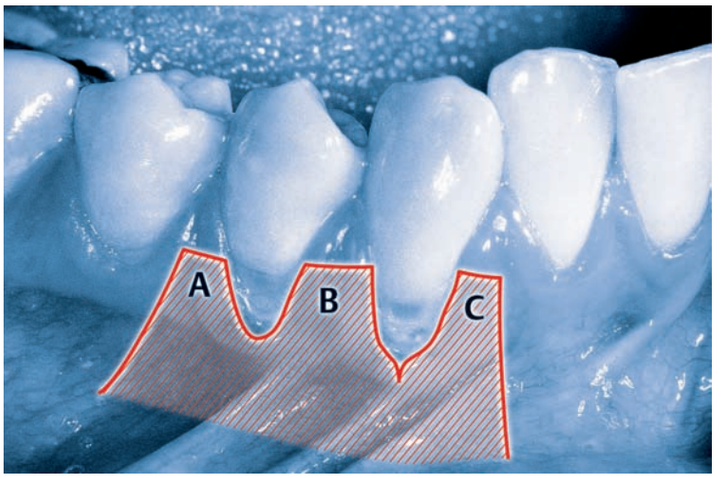
- Tooth neck sensitivity
- Root surface caries
- Gingival inflammation
- Progressive gingival recession
- True periodontal pocket formation

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1039 Planning the Incisions—Protocol

1 Horizontal incisions at the level of the proximal cementoenamel junctions. These should not involve adjacent teeth 45 or 42, which exhibit no recession, in order not to traumatize the attachment.
2 Vertical incisions into the mobile mucosa
3 Intrasulcular incisions on the teeth exhibiting recession.

Height of the papillary incisions **A, B, C**



Bilaminar Defect Coverage using CTG, Nelson Technique—Surgical Protocol:

- Horizontal incision
- Vertical incisions bilaterally beyond the MGJ
- Full flap reflection
- Incision of the periosteum
- "Conditioning" the root surface
- CTG removal from the palate
- Treatment of the palatal wound
- Placement of the CTG into the recipient bed (resorbable sutures)
- Flap repositioning and fixation
- Post-operative instructions

First Step: Creating the Recipient Bed

1040 Initial Incisions Completed

As per the plan (see above) the horizontal, vertical and intrasulcular incisions were made. On the gingival margin of the recession areas (teeth 43 and 44) the epithelium was maintained. The borders of the flaps **B** and **C** were connected during the surgical procedure by the CTG. The intent is for these tissues to grow together without disturbance.



1041 Reflecting the Mucoperiosteal Flap

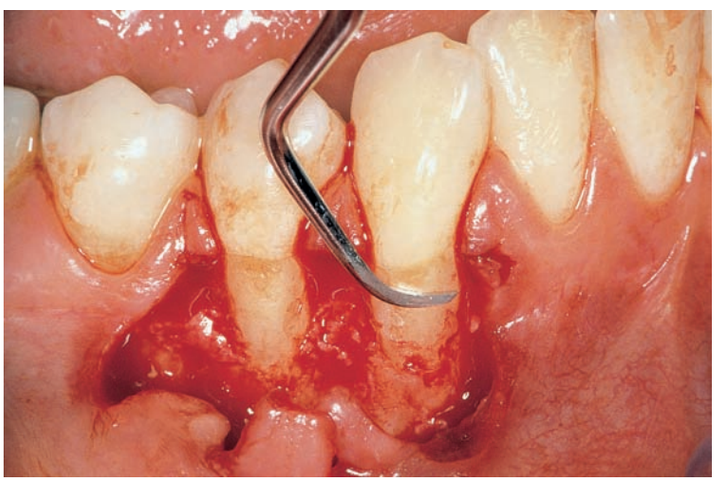
A *full-thickness flap* is reflected using the elevator. The exposed, compact bone does not exhibit bleeding points, but these must be created before placement of the CTG (to insure adequate nutrition for the transplanted connective tissue from the osseous side; Fig. 1043). At the depth of the mucosa, the periosteum is incised in order to free up flaps A-C in a tension-free manner to permit lateral repositioning and adaptation.



1042 Root Cleaning/Planing

Only in the region of gingival recession are the root surfaces cleaned and planed using hand instruments or fine diamond burs (15 µm). Attachment tissue must be preserved. Severely convex root surfaces must be flattened!

Right: A subsequent chemical conditioning of the exposed root surface is recommended ("biomodification," here with a slurry of tetracycline).





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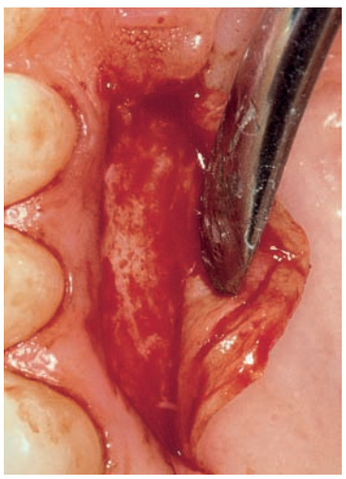


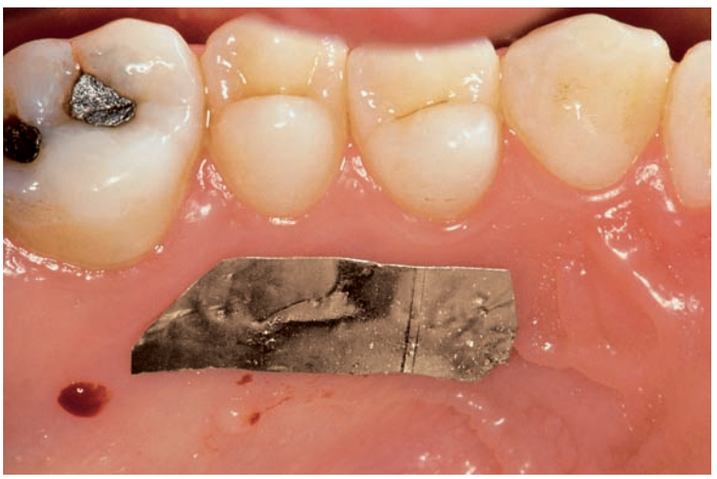
1043 Clean Root Surfaces
The cleaned and planed root surfaces are now prepared for the CTG transplant.
Small round burs are used to penetrate the compact bone in order to provide nutrition for the CTG (*Caution*: Roots, periodontal ligament and the mental nerve!).

Left: Sterile round burs and NaCl solution for penetrating the compact bone.



1044 "Template" for the Connective Tissue Graft
Similar to the procedure with FGG, the size and dimension of the CTG can be determined clinically using sterile aluminum foil. The CTG should extend slightly beyond the marginal osseous borders.



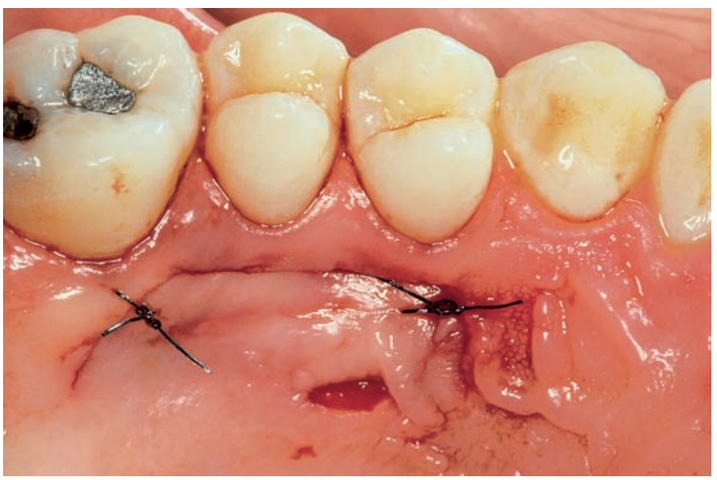


Second Step: Harvesting the Palatal Graft

1045 Removal Technique
The aluminum template is affixed to the palate using Vaseline. A broad-based yet thin "door opening" is created (epithelium and connective tissue).

Left: Medial opening toward the raphe. The subjacent connective tissue can now be excised and removed.





1046 Treatment of the Donor Site
After the CTG is removed, the "door" is closed, securely sutured, and finger pressure is applied to reduce hemorrhage and to minimize any coagulum formation.

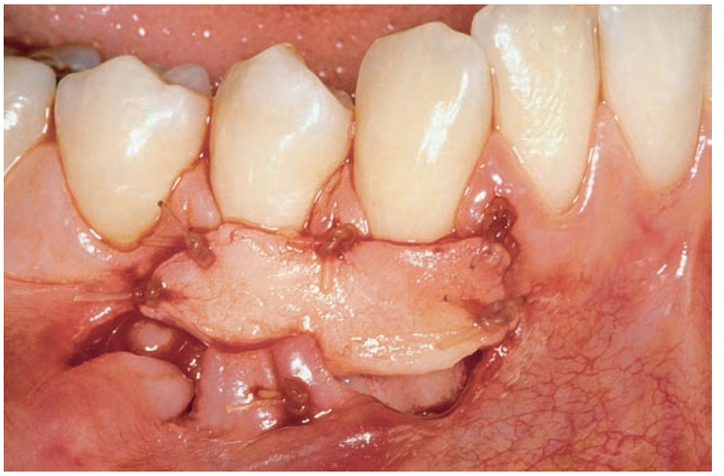
Left: Ca. 2 mm thick CTG. Following harvest, the graft must be protected from dessication. After any necessary trimming, it is immediately positioned into the recipient bed.

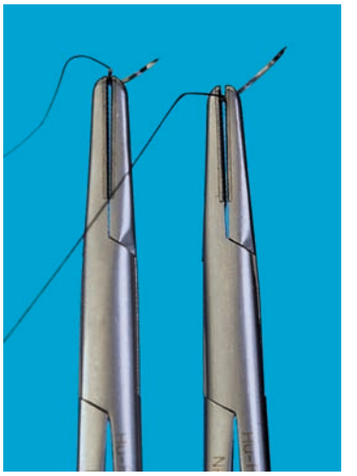
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Third Step: Placing the CTG

1047 CTG *In Situ*
The graft is affixed using several *resorbable* sutures.

Right: Resorbable suture material for fixation of the CTG (catgut; left), and *non-resorbable* needle-suture material (braided silk) for positioning and adapting the soft tissue flaps.





1048 Flap Adaptation/ Repositioning
As far as possible, the CTG should be covered by the repositioned soft tissue flaps, and as much as possible without any *tension*. Papilla tip A over the root of tooth 44 and the combined papilla tips B/C over tooth 43. The tips of the soft tissue flaps must be stabilized over the CTG.

Subsequent to the surgical procedure, *no* mechanical oral hygiene can be performed, only CHX rinsing.



Post-Operative View

1049 Fourteen Days after Surgery
The sutures have already been removed, but the clinical result at this point in time could have been better.
The small tissue tags (**A, B, C**) protrude at their coronal aspect and remain slightly mobile. The gingival recession on tooth 44 has not been 100 % covered.
At this time, mechanical oral hygiene in addition to CHX rinsing can be carefully re-instituted.



1050 Seven Months after the Surgical Procedure
One-half year after surgery the situation has consolidated and the results are much more favorable. The recession on tooth 43 has been almost completely covered and on tooth 44 recession is only about 1 mm.



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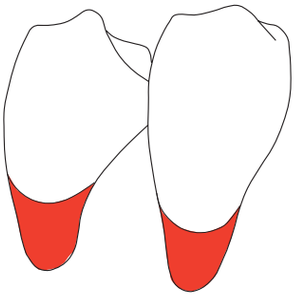
Free Connective Tissue Graft—Nelson Technique

Summary

As demonstrated in this case, the Nelson technique is a relatively simple and technically insensitive method for covering areas of gingival recession with predictable success. The 20-year-old female complained pre-operatively about mild pain at the gingival margin apparently caused by repeated slight injury during toothbrushing. Because of the total lack of attached gingiva, efficient oral hygiene in this area was hardly possible given the deep areas of recession and the shallow vestibulum.

The patient complained also of some tooth neck sensitivity, but she was mainly worried about the gingival recession and was afraid of tooth loss.

All of these problems were solved by the minor surgical procedure; the areas of gingival recession were almost completely covered. The high frenum attachments were displaced apically and the attached gingiva was effectively widened. This much-improved, healthy periodontal condition permitted effective and atraumatic plaque control.





1051 Initial Clinical View
All of the above-described problems are clearly visible and explain the patient's complaints and fears:
- Gingival recession on teeth 43 and 44 (depicted schematically at left)
- Virtually no attached gingiva
- High frenum attachments
- Mild inflammation resulting from plaque and possible toothbrush injuries
- Initial wedge-shaped defects on the buccal root surfaces

Before

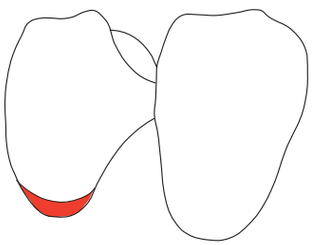
	8	7	6	5	4	3	2	1	1	2	
Re			1	1	3	3	1	1			Re
Mand. aG			2	2	1	1	3	2			Mand. aG

1052 Recession (Re) and Width of the Attached Gingiva (aG)
At the initial examination, gingival recession of 3 mm was detected on teeth 43 and 44. These teeth exhibited virtually no attached gingiva.

After

	8	7	6	5	4	3	2	1	1	2	
Re			1	1	1	0	0	1			Re
Mand. aG			2	2	2	2	3	2			Mand. aG

Re and aG Two Years Later
The gingival recession on tooth 43 was completely covered, but on tooth 44 the recession was covered "only" 2–3 mm ("66.6 %").





1053 Two Years after Surgery
In comparison to the clinical view after seven months (Fig. 1050), the clinical situation is significantly improved. On tooth 44, it appears that "*creeping attachment*" has occurred. Minimal recession of < 1 mm persists on this tooth. Most important esthetically is the significantly reduced area of gingival recession (see diagram, left) and not the percentage of coverage.

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Connective Tissue Graft … and Dealing with Complications

Surgical Procedure, Step-by-Step

The procedure for the "envelope technique" (Raetzke 1985, Bruno 1994) has already been depicted diagrammatically (p. 420).

In the clinical case presented here, a 29-year-old male presented in the clinic complaining of generalized and irregularly pronounced gingival recession, particularly in the right side of the maxilla. Various etiologic factors could explain this manifestation of gingival recession; the morphologic/anatomic conditions including buccoversion of the teeth and the resulting thin or lacking osseous lamella appear to have played a role in this case. In addition, the patient history revealed a traumatic toothbrushing technique (horizontal scrubbing) with a hard-bristle toothbrush and a very abrasive dentifrice. This would also explain the wedge-shaped defects and the hypersensitive tooth necks. The patient also frequently consumed acid-rich foodstuffs.

The patient's desire was to cover the most disturbing gingival recessions on teeth 13 and 12 as far as possible.

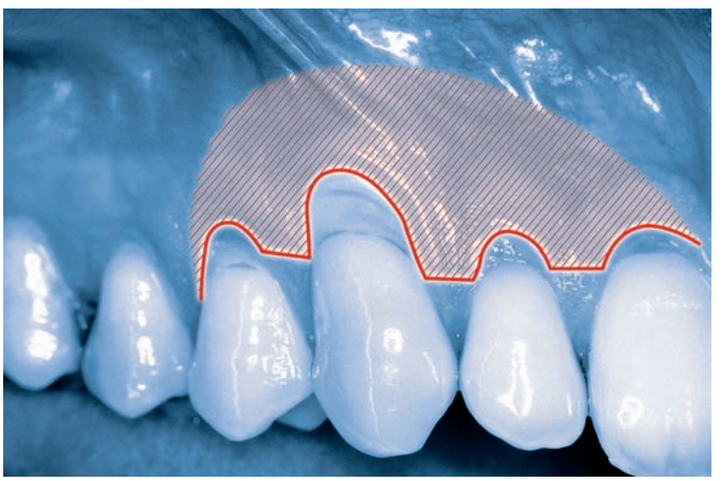
1054 Initial Clinical View
On teeth 11, 12, 14, and 15 one observes gingival recession of 1–2 mm, and on the *canine* almost 5 mm. The canine root also exhibits a pronounced wedge-shaped defect. As is the case with most gingival recession, the interdental papillae remain intact and fill the proximal spaces (Miller, Class I, wide).

Right: The radiograph reveals normal interdental osseous septa around the canine.



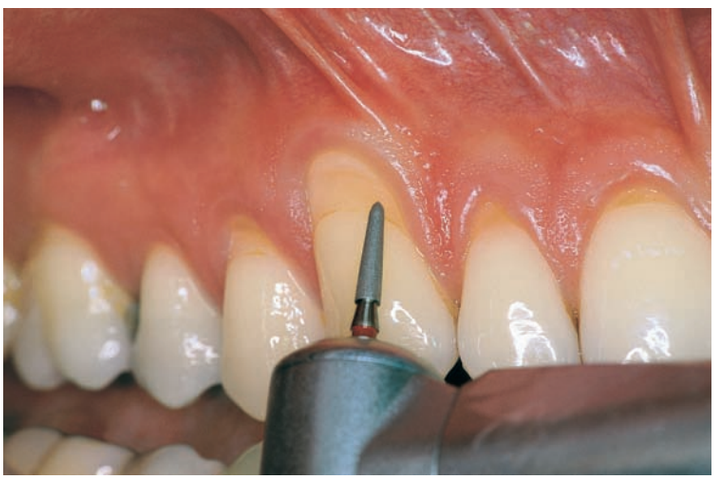


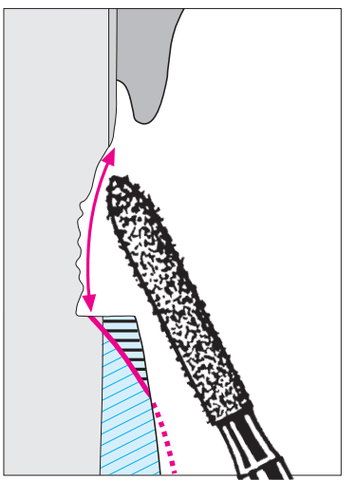
1055 "Envelope" Technique—Planning
- Marginal incision (red line) and the planned flap reflection beyond the mucogingival junction (hatched area).
- Mobilization/reflection of a full-thickness flap with incision of the periosteum (original method according to Bruno; split-thickness flap).



1056 Preparing the Recipient Bed—Minimal Odontoplasty
Wedge-shaped defects are almost always observed immediately apical to the cementoenamel junction on the root surfaces. Because the much harder enamel is scarcely abraded, a sharp border often persists. Before any soft tissue surgery, dental caries or class V restorations in this area should be removed.

Right: Odontoplasty at the CEJ using diamond burs.





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1057 "Conditioning" of the Root Surface
The root surface to be covered by the tissue graft is pre-conditioned using a slurry of tetracycline-HCl.

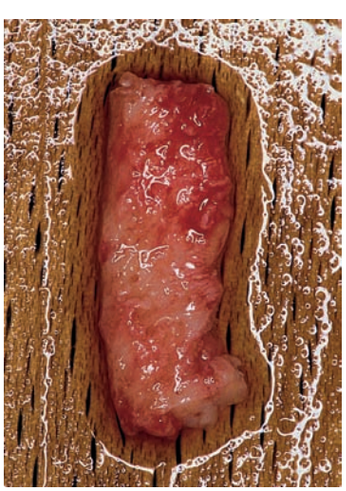
Left: Various possibilities for biomodification of the root surface.



1058 Incision to Create the "Envelope"
In contrast to the microsurgical incisions depicted in the previous case, incisions made with the normal scalpel (e. g., No. 15) are somewhat more traumatic, less precise and leave tissue tags behind.



1059 Soft Tissue Flap Reflection
Using an elevator, the mucoperiosteal flap is reflected beyond the mucogingival junction, and the periosteum is incised at the base. This is different from the original method describe by Bruno, in which a continuous split-thickness flap was described.





1060 CTG *In Situ*
The palatally derived CTG covers the recession on teeth 12 and 13 somewhat coronal to the CEJ. Final fixation of the surgical site is accomplished using resorbable sutures from the CTG to the palatal papillae.

Left: The CTG from the palate is minimally 2 mm thick.

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1061 Repositioning the Soft Tissue Flaps
The flap is affixed to the papillae between teeth 14/13, 13/12, and 12/11 using 4–0 synthetic sutures. The graft is relatively thick, and it is not completely covered by the flap in the region of the original recession.



Wound Healing and Problems ...

1062 Nine Days after Surgery
Sutures have been removed. The CTG has healed in, and its surface has begun to epithelialize. The tissue, especially above tooth 13, is thickened, and projects beyond the cementoenamel junction.



After 9 Days

1063 Four Weeks after Surgery
Normal wound healing is essentially complete, but the tissue in the region of the graft is hyperplastic. In addition, at the original margin of the CTG and the original papilla there is a distinct cleft (see summary of the case, p. 434). Normal mechanical oral hygiene can now be re-instituted.



After 4 Weeks

1064 Ten Weeks after CTG Surgery
The healing has progressed normally, but there is a persistence of the hyperplastic tissue as well as an unphysiologic course of the gingival margin around teeth 13 and 12.
The patient requested some thinning of the tissue, but this was delayed because in most cases such tissue enlargement recedes once regular plaque control (toothbrushing) is re-initiated. Probing depths around tooth 13 were 2 mm.



After 2½ Months

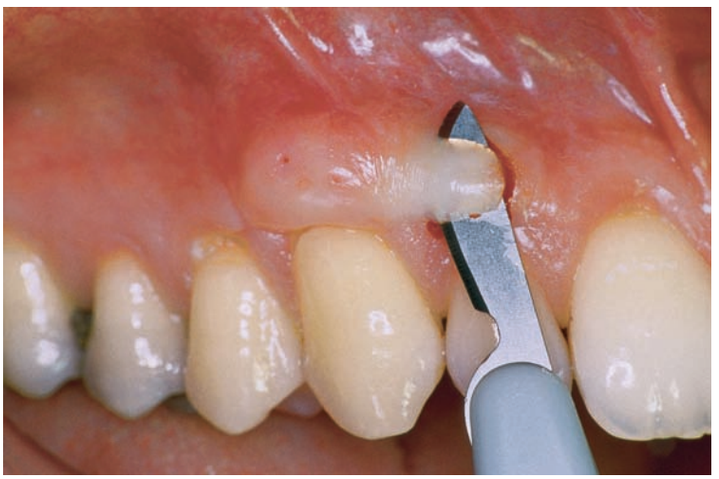
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After 4 Months



1065 Four Months after CTG
The esthetic and anatomic situation remains unsatisfactory. During "sounding" under anesthesia, it was clear that the tissue thickness was only soft tissue accumulation, not exostosis. Surgical contouring of the gingiva was therefore planned.





Minor Surgical Recontouring

1066 Gingivoplasty of the Hyperplastic Tissue
Four months after the initial surgery, a No. 15 scalpel was used to remove the redundant tissue.

Left: The thinning is performed with the scalpel; finer contouring can also be performed using coarse diamond burs.



1067 Immediately after Gingivoplasty
In this case, following removal of the redundant gingival tissue with the scalpel, the wound margins were smoothed using an electrosurgical loop. This also immediately stopped all hemorrhage from the site.
If the clinician has a laser device available, it can also be employed for this purpose.



1068 One Month after Gingivoplasty—Five Months after CTG
Although the bulk of the redundant gingival tissue was eliminated, only one month later there are signs of recurrence. The new tissue is relatively pale in color. The patient was placed on a regular recall schedule.

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Connective Tissue Grafting, with Complications

Summary

The probable causes for the gingival recession in this 29-year-old male were described previously. The chief complaint was progressing gingival recession, especially on tooth 13, which had a very sensitive tooth neck, and was esthetically objectionable. The treatment plan included coverage using a connective tissue graft. The surgical procedure was a minor modification of the guidelines described by Bruno (1994).

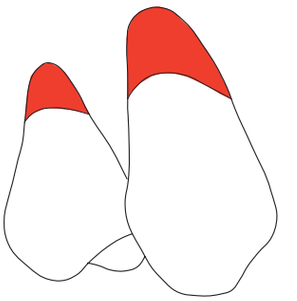
During the post-surgical healing process, complications ensued. The transplant was likely too thick and was situated upon residual periosteum which likely led to the hyperplasia-like alterations over the recession site. The cleft between the CTG and the interdental papilla appeared to be due to the in-growth of epithelium at this location (Bouchard et al. 2001).

Four months later, the gingiva was thinned and contoured. In the course of two years following the initial surgical procedure, the area of recession is optimally covered. The patient's complaints were completely eliminated.

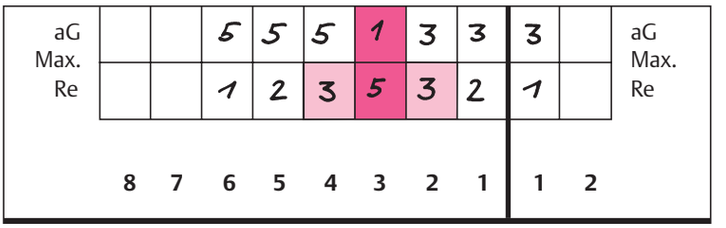
1069 Initial Clinical View—Class I Recession (Miller)
Gingival recession is obvious throughout quadrant 1. Recession on tooth 13 is especially pronounced at 4–5 mm. On this tooth, frena attach virtually at the gingival margin. Wedge-shaped defects are present on all of the involved teeth, especially on tooth 13.

Right: The diagram exhibits the areas of recession in red.



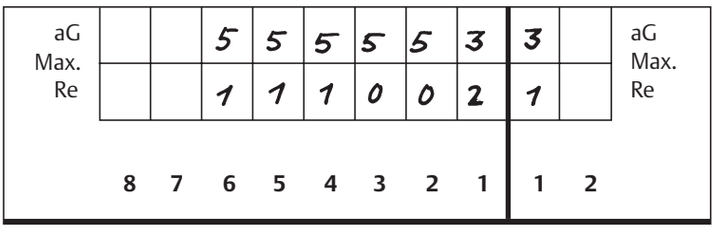


1070 Attached Gingiva (aG) and Recession (Re)
Initially, there was hardly any attached gingiva (aG) on tooth 13. Areas of gingival recession up to 5 mm were measured.



Before

The therapeutic procedures increased the width of the attached gingiva significantly (5 mm). The recession on tooth 13 was completely covered, and also for the most part on the adjacent tooth.

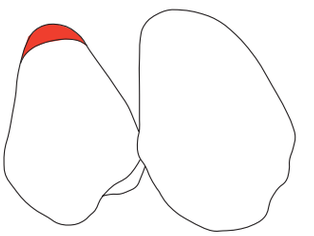


After

1071 Two Years later
After the areas of recession were covered, the clinical course of the gingival margin is much more harmonic. The gingivae exhibit some stippling. No elevated probing depths could be detected. In the area of the CTG, the gingiva appears paler and a bit thicker than the papillary region (esthetics?).

Right: Diagram of the treatment result on teeth 14 and 13. The coverage on 13 is 100 %. On tooth 14, only a small area of recession remains.





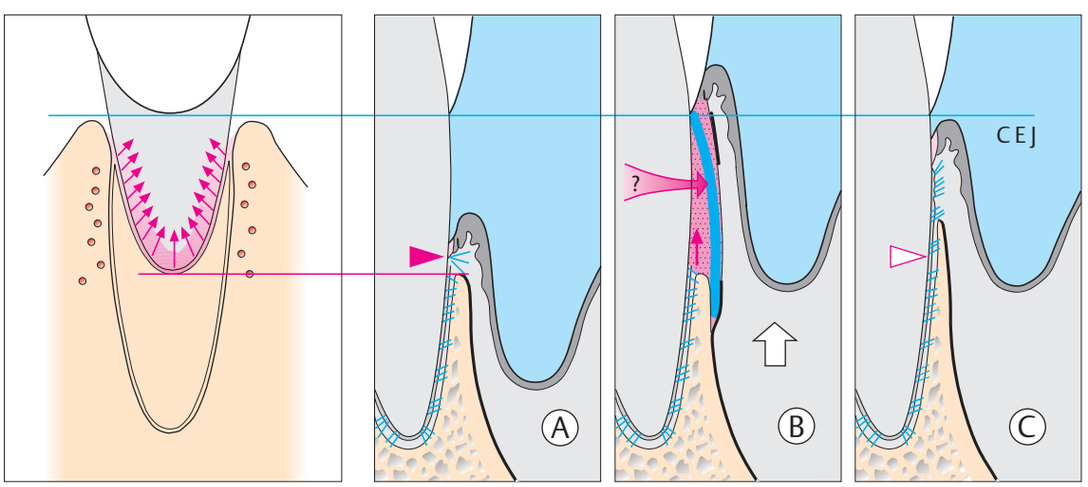
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Covering Areas of Recession via "Guided Tissue Regeneration"—GTR

Since the early 1990s, attempts have been made to cover areas of gingival recession using the GTR technique (Tinti et al. 1992, 1993; Pini-Prato et al. 1992; Coretllini et al. 1992; Tinti & Vincenti 1994; Trombelli 1999; Bouchard et al. 2001). The advantage in contrast to free gingival grafting is that no tissue has to be harvested from the palate as a s*econd surgical site*, and therefore related complications are avoided.

Both *non-resorbable* and *resorbable* membranes have been recommended, and *no* significant differences have been reported with regard to healing and success rate with, e. g., the GoreTex periodontal material (non-resorbable, titanium strengthened) and Guidor (resorbable; Roccuzzo et al. 1996). The decisive criterion is often the *patient* who, understandably, prefers the resorbable membrane method because this obviates the necessity for a second surgical procedure.

When attempting to cover areas of recession, however, it is less the choice between resorbable or non-resorbable membranes that is most critical; most important is the capability of *any* membrane to create sufficient space for precursor cells from adjacent periodontal tissues to migrate apically and laterally over the recession site (Fig. 1072, left).



1072 Covering Areas of Recession via GTR
The initial view (*left*) depicts a pronounced osseous dehiscence (without soft tissue). Regeneration must occur from precursor cells from the periodontal tissues surrounding the area of dehiscence (arrows).

A Orofacial section through the original gingival recession.
B The membrane creates space for PDL and bone cells to initiate tissue regeneration.
C Ideal final result.

Another important criterion for the success of recession coverage via GTR is a "thick" and well vascularized gingiva and mucosa to cover the membrane.

The patient must also participate to insure a good result. The surgical area must be stabilized because the formation of a resilient new attachment can take up to 3 months. While normal and optimum mechanical oral hygiene can be performed in the rest of the mouth, at the surgical site only chemical plaque control, e. g., with chlorhexidine, can be performed for ca. 2 months. If the marginal edge of a *resorbable* membrane is denuded during the healing phase it seldom has any effect on an optimal treatment result; however, if this occurs with *non-resorbable* membranes the consequences can be critical (infection, necrosis of the flap).

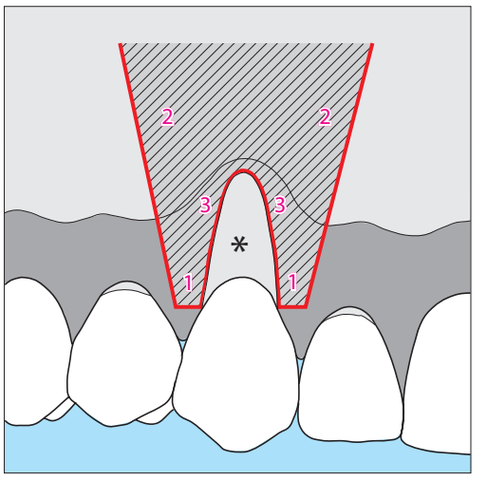
One must keep in mind that serious complications (flap necrosis, infection, extremely sensitive tooth necks etc.; Bouchard et al. 2001) when using the GTR technique are more difficult to correct than with other procedures.

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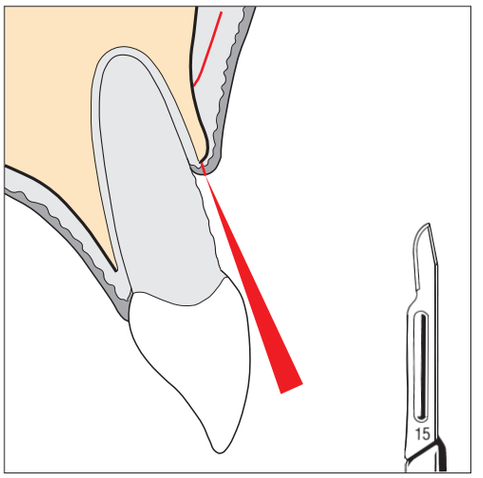
Covering Gingival Recession Areas via GTR—Principles of the Barrier Technique

1073 A—Incisions

1 The *horizontal incisions* follow the course of the cementoenamel junction.
2 The *vertical incisions* extend into the mobile mucosa.
3 With the *sulcular incision,* the sulcus epithelium is removed and the soft tissue flap is created.

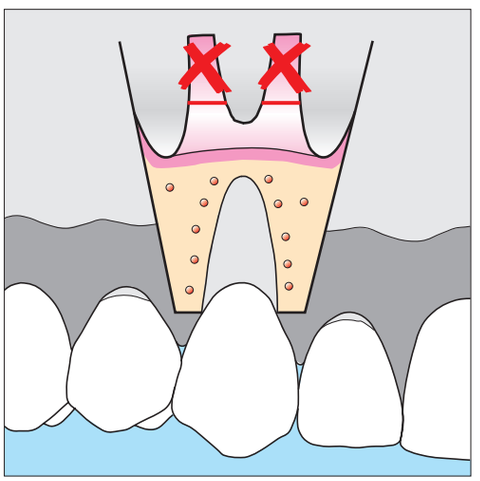


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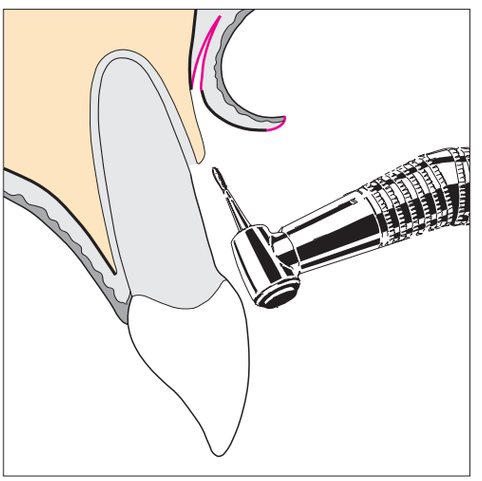


1074 B—Flap Reflection, Root Surface Treatment, Hemorrhagic Wound Bed

The soft tissue flap is reflected beyond the mucogingival junction; the periosteum is incised to form a split-thickness flap (*right*). Shortening of the papillary tissue flaps. Flattening of the convex root surface using fine diamonds (Perioset, *right*) and root surface conditioning.
Creating bur holes through the interdental cortical bone of the alveolar process to enhance blood flow.

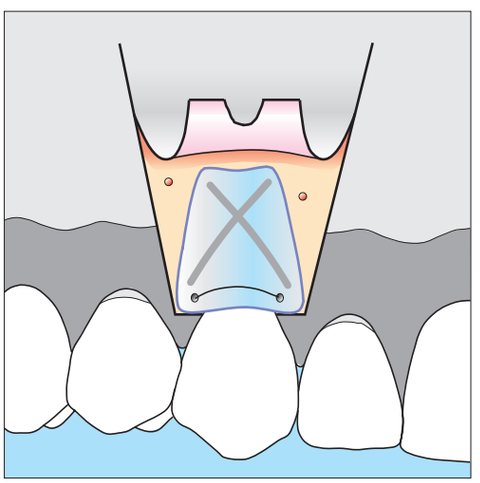


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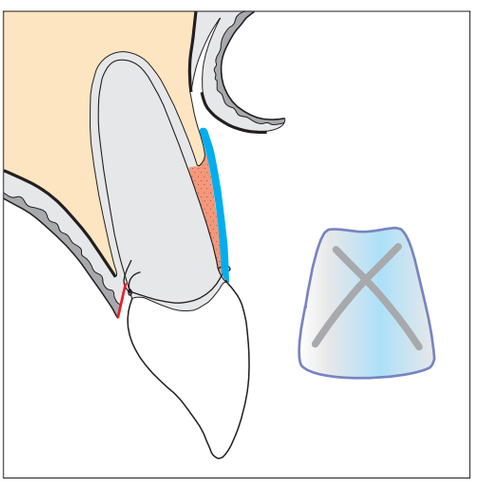


1075 C—Adaptation of the Membrane

A *titanium-reinforced GoreTex membrane* is secured with a surrounding suture. The palatal sulcus is deepened by means of a small wedge excision, permitting subgingival placement of the monofilament suture for 4–6 weeks (*right*). Thus the suture does not come into contact with the tongue and is less likely to become infected. Because the suture is subgingival, the patient is less likely to "play with it."



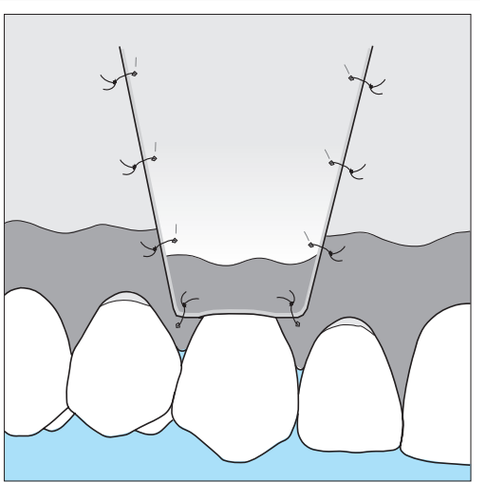
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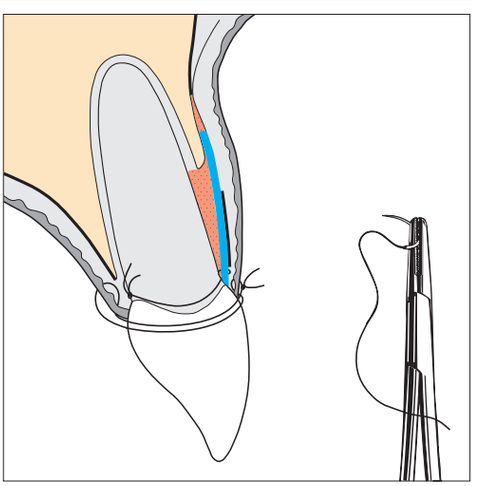
1076 D—Coronal Flap Repositioning—Sutures

The soft tissue flap is repositioned *without tension* somewhat coronal to the cementoenamel junction. Fixation is achieved via interrupted sutures (*left*), and possibly a supragingival encircling suture (*right*).
The sutures are removed after 10 days. The membrane and its sutures are removed 4–6 weeks later in a second surgical procedure.

Modified from *Jepsen & Jepsen 1995.*



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Covering Gingival Recession Using Resorbable Membrane

Treating Recession after Orthodontic Therapy

Following orthodontic therapy, especially when high levels of force have been employed, various complications may occur. Well known, for example, are post-orthodontic root resorption, occlusal interferences, but also mucogingival and related esthetic problems. The clinical morphology of the gingival margin may be irregular after orthodontic therapy. Teeth which appear "too short" or "too long" (gingival recession) may be treated by a *crown lengthening procedure* (p. 493) on the one hand, or by means of *recession coverage* on the other.

A 38-year-old male was referred from his general dentist for treatment of a submucosal abscess (root fragment, tooth 26). Radiographic examination further revealed an expansive follicular cyst around the deeply impacted canine (tooth 33). After treating the abscess, removing the root fragment and initial periodontal therapy, the decision was made to eliminate the cyst around 33 and to draw the impacted canine into its proper place in the arch. These attempts were successful, but after orthodontic therapy there existed a ca. 4–5 mm area of recession, which was treated using the GTR technique.



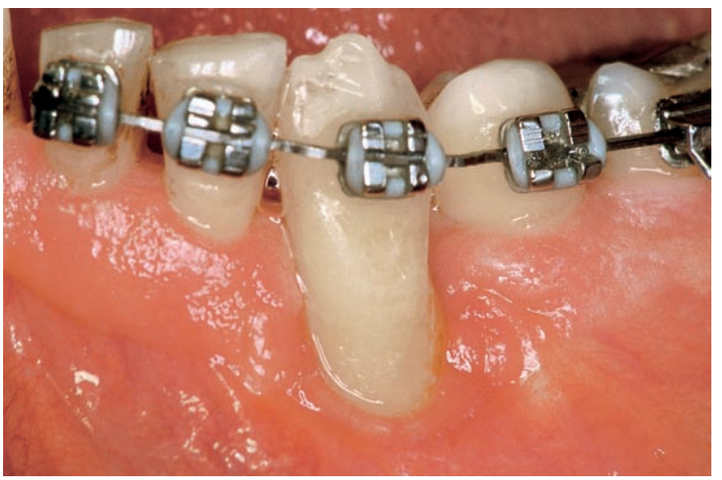


1077 Panoramic Radiograph
Initial radiograph reveals the root fragments of tooth 26 and the deeply impacted tooth 33, as well as an expansive follicular cyst exhibiting two chambers mesially and distally to the crown of the impacted canine. Teeth 32 and 34 are tipped distally and mesially, respectively. Tooth 34 will be extracted in order to create space for the canine.

Left: Tomogram (section) of the impacted canine within the cyst.



1078 Surgical View—Cystic Cavity Exhibiting the "Captured" Canine
Following cystostomy, the impacted tooth was affixed with adhesive brackets in order to move it slowly coronally. Chlorhexidine rinses have resulted in some dental staining. The treatment plan included extraction of tooth 34 to create space for the canine.



1079 At Completion of Orthodontic Therapy, 3 Years later
During the treatment there were two episodes of infection of the cystic cavity and this prolonged the orthodontic treatment.

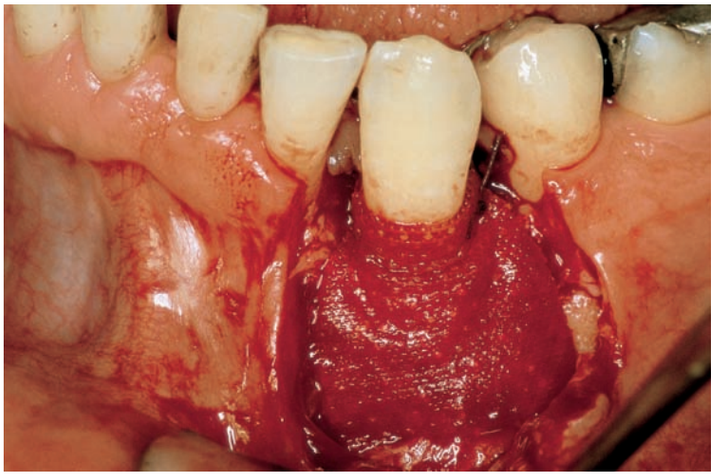
The exposed canine exhibits significant gingival recession, which was not unexpected (Miller, class III). The brackets and wires were in place for retention only. The patient's oral hygiene was moderate.

Courtesy of the *Basel Dental Clinic (Switzerland)*

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1080 Covering Gingival Recession via GTR—Resorbable Membrane
Even though the up-righted canine exhibited mesial and distal attachment loss, the attempt was made to cover the pronounced buccal recession using a resorbable membrane.
After flattening and conditioning the root, the membrane was adapted and affixed using a resorbable suture surrounding the tooth.

Right: Guidor membrane.





1081 Wound Closure
The gingival flap is repositioned and immobilized using interrupted and interdental sutures. The margin of the membrane is clearly visible, but this is of no consequence with the resorbable membrane (polylactic acid and citric acid ester) which, however, is no longer available on the commercial market.

The patient received systemic medication to reduce both pain and swelling, and used CHX exclusively for plaque control.



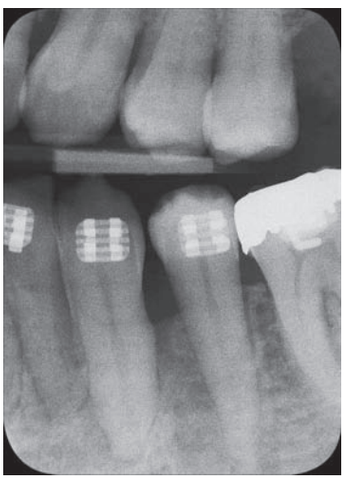
1082 One Month after the Operation
The non-resorbable, monofilament sutures that were used to stabilize the soft tissue flap were removed 10 days post-surgically. The coronal aspect of the membrane is clearly visible at the gingival margin, and the surrounding mucosal tissue is obviously erythematous.



1083 Three Months after Surgery
The area of recession has been partially covered. The soft tissues (gingiva and mucosa) have not completely consolidated (maturation phase). The patient was told to perform better oral hygiene.

Right: Radiographic view following orthodontic treatment.





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Covering Gingival Recession Using Resorbable Membrane

Summary—Treating Post-Orthodontic Gingival Recession

The 38-year-old male was referred to the clinic for treatment of an abscess surrounding the root fragment of tooth 26. To date, the patient was not bothered by the larger problem in the region of tooth 33, even though the canine was absent and a bluish livid swelling was visible in the vestibulum. After treatment of the acute situation around tooth 26 (opening the abscess and removal of the root fragment), the problem represented by the impacted canine was discussed with the patient and a treatment plan was proposed.

The plan included cystostomy (oral surgery) and subsequently orthodontic movement of tooth 33 into the dental arch. The patient accepted the treatment plan.

In some ways unfortunately, the synoptic and comprehensive treatment, which occurred in three different departments of the dental school, extended over three years; this was because following cystostomy and during orthodontic therapy complications occurred (infections) that had to be treated.



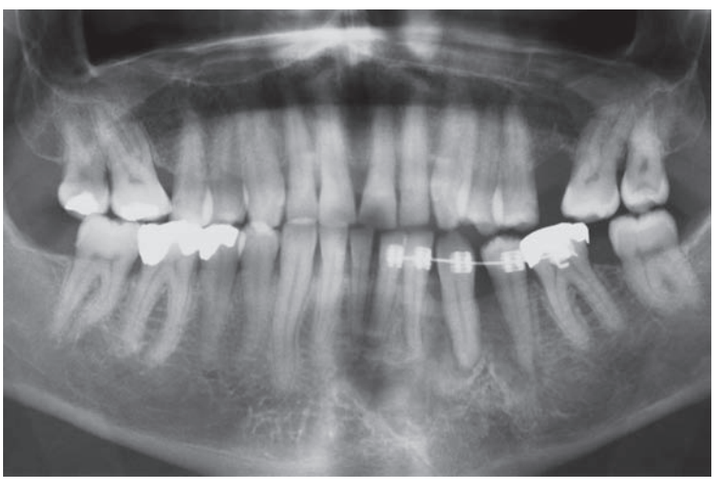


1084 Initial Findings
The panoramic radiograph reveals a deeply impacted canine and a two-chambered follicular cyst, as well as the root fragments of tooth 26, tipping of teeth 32 and 34. Teeth 16, 37, and 48 are missing.

Left: Clinical view following cystostomy and capturing of tooth 33. The brown stains resulted from CHX rinses.

Before





After

1085 Final Clinical and Radiographic Appearance
Radiograph following extraction of 26 and 34, as well as orthodontic repositioning of tooth 33. The latter is vital and exhibits an excellent osseous fundament.

Left: An adequate coverage of the massive gingival recession (Miller class III) was achieved. The lack of sufficient attached gingiva demands constant attention.

Perhaps as a consequence, the patient's compliance was not optimal during the extended phases of treatment, and the patient presented again and again with unsatisfactory oral hygiene. Despite these many obstacles, the complete treatment was brought to an end and the results were overall acceptable.

Following the orthodontic treatment, there remained a not unexpected area of gingival recession on tooth 33, and the orthodontist referred the patient to a periodontist for coverage of the recession area on the facial surface of the canine. This goal was partially achieved through use of a resorbable membrane.

Of particular importance in this case is not only the percentage or millimeters of the recession that was successfully covered, but also the fact that the large, two-chambered cyst healed completely and that tooth 33 is completely surrounded by new bone (Fig. 1085).

Finally, this case also demonstrates that in today's modern and highly specialized dentistry, an excellent collaboration among the various disciplines greatly benefited the patient. In this case, participating were the general dentist, the oral surgeon, the orthodontist and the periodontist.

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Covering Areas of Recession on Several Teeth—Possibilities

Patients today are placing higher and higher demands in the esthetic arena. Not only the dental specialties of orthodontics, prosthodontics and conservative dentistry must follow this trend, the periodontist must also participate. Especially in the visible maxillary anterior and premolar regions, with a high smile line, multiple and/or pronounced areas of gingival recession are no longer accepted ("long teeth"). Covering areas of recession that extend over 4–6 teeth will be demanded more and more in the future. The methods for covering areas of recession that have been described so far, using epithelialized free gingival grafts (FGG), connective tissue transplants (CTG) or the GTR technique are usually indicated only for treatment of a single tooth. In addition, most of these surgical methods require a second surgical site for the harvest of graft tissue, and this can lead also (if seldom) to complications (pain, hemorrhage, necrosis).

With the GTR technique, using membranes, a second surgical site is not necessary; however, membranes are rather costly, and the chances for success with GTR cannot yet be consistently predicted. The membrane techniques are also usually applied only to single teeth.

Today, the desires of the patient and also those of the clinician are therefore understandable: *New materials* and/or *simpler treatment techniques* for covering areas of recession over large segments of the dental arch in a single procedure without a second (donor) surgical site.

In the following pages, we will present three methods that may fulfill the above-delineated desires: The bridge-lap procedure (Romanos et al. 1993), papilla rotation technique (Zucchelli & DeSanctis 2000), as well as an attempt at "renaissance" of coronal repositioning of a pedicle flap (Allen 1993). The primary prerequisite for success of any of these procedures is thick gingival and mucosal tissue; only such tissue can guarantee adequate blood supply to the large flap and avoid the risk of necrosis. It is also obvious that the three methods are likely to be successful only with multiple areas of *mild* recession (2–3 mm).

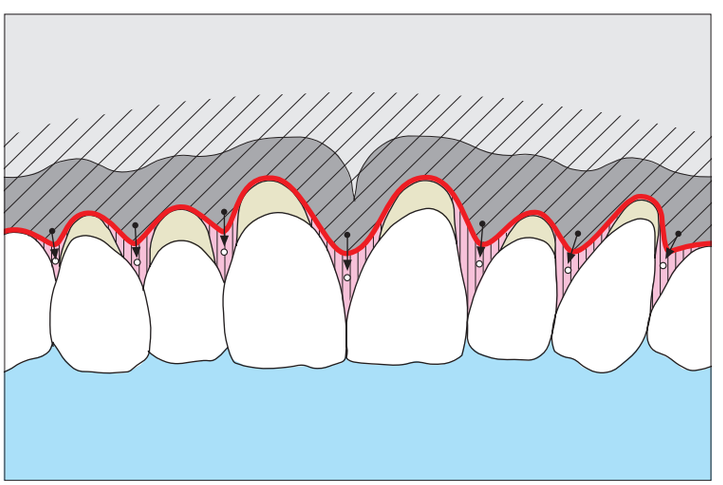
These extensive flap procedures will surely become more predictable in the future, when combined with matrix proteins or growth factors.

The surgical procedures described in the following pages are not only indicated and advantageous for covering or repairing gingival defects, but may also be used as *pretreatment* prior to seating expansive fixed bridgework, particularly in the maxillary anterior and canine regions.

Covering Expansive Areas of Gingival Recession (Allen 1993)

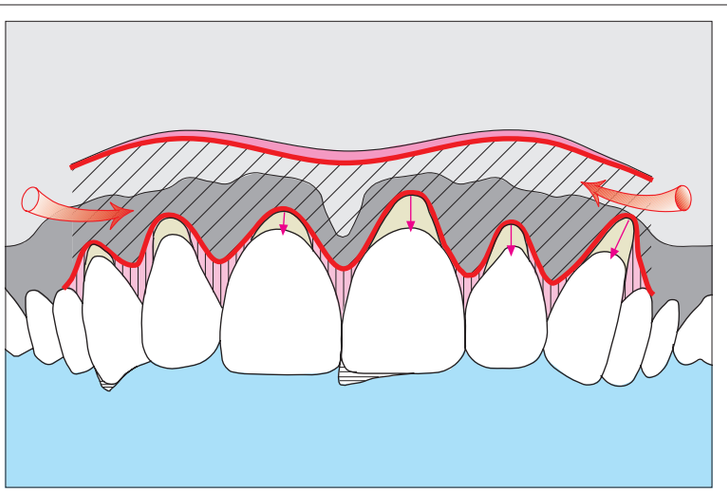
1086 Coronally Repositioned Pedicle Flap

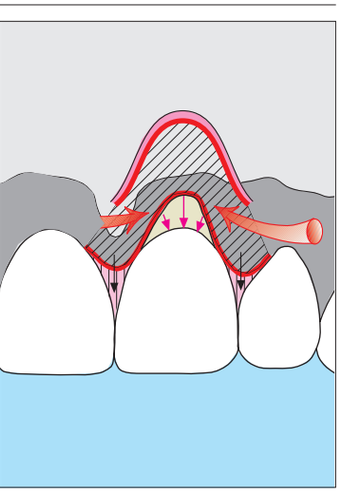
A scalloping horizontal incision creates the new papillae (black dots). A full-thickness mucoperiosteal flap is reflected beyond the mucogingival junction and beyond the deep epithelialized "old" papilla (small circles). In order to reposition the flap without tension, the periosteum must be incised apically (split-thickness flap).



1087 Laterally Replaced and Coronally Repositioned Pedicle "Bridge Flap" (Romanos et al. 1993)

A bridge-flap to cover the *single tooth gingival recession* was referred to as a "semilunar flap" by Tarnow (1986; *right* and p. 415). The technique can be employed to cover multiple areas of recession on adjacent teeth. The expanse of the flap is limited, because the "displaced" tissue can only receive nutrition from lateral tissues and from bone. Clinical failure brings grave consequences!





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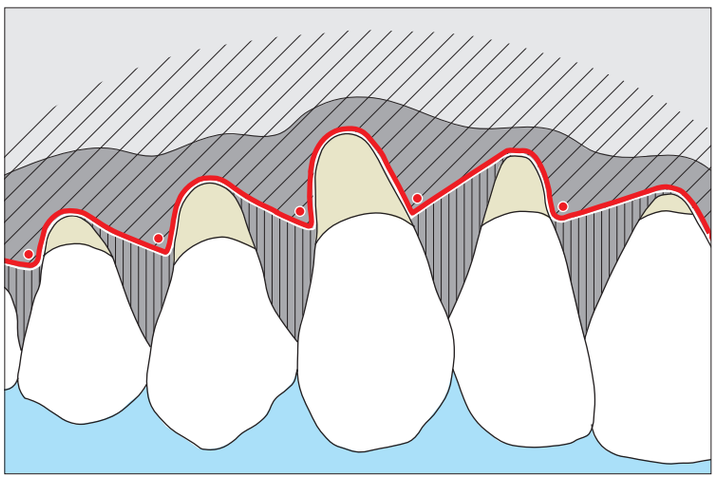
Coronally Repositioned Flaps and Papilla Rotation

Previously, we described two methods for covering areas of recession on several teeth simultaneously, using expansive *coronally* repositioned soft tissue flaps.

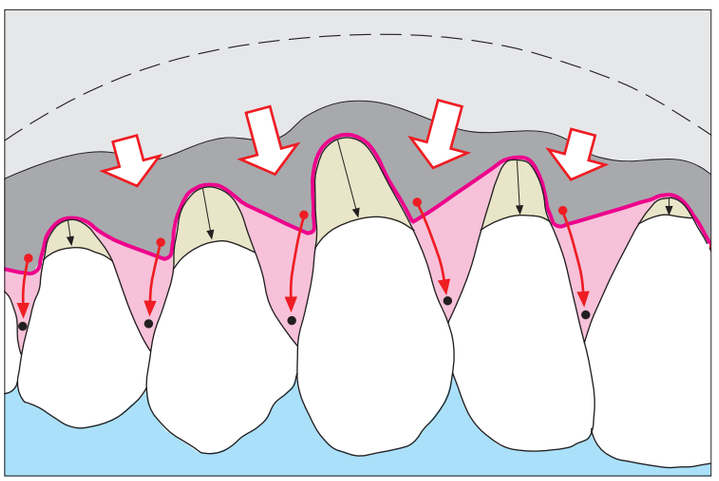
Zucchelli & DeSanctis (2000) have now proposed a more subtle method by which even deeper areas of recession can be covered by means of a limited coronal repositioning of the entire flap and also rotation of newly formed papillae. This technique permits esthetic correction of Miller class I and II areas of recession without harvesting connective tissue and therefore without a second surgical site.

This procedure has been successfully performed in 22 patients with esthetic problems and ranging in age from 18 to 34. As always, prior to the surgical procedure, professional prophylaxis and oral hygiene instruction/motivation was carried out.

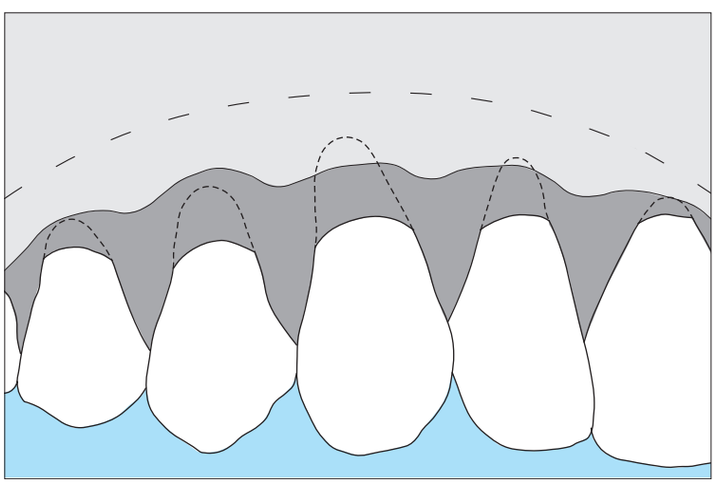
Follow-up monitoring appointments demonstrated that all areas of recession remained covered one year after the surgery. The practical surgical procedure is depicted schematically below.



1088 Incisions
The horizontal incision courses intrasulcularly around and above the areas of recession. Mesial and distal to the recession areas to be covered on the root surface, the new papillae (red dots) are pre-cut, and the "old" papillae are de-epithelialized. In the *coronal* region, a full-thickness flap is created. In the *apical* region a split-thickness flap is prepared in order to reposition the soft tissue without tension.
Note the geometry of the incision around the deepest recession area (canine).



1089 Coronal Repositioning of the Flap, and Rotation of the Papillae
The soft tissue flap is repositioned without tension coronally. Following subsequent rotation, the newly formed papillae completely cover the "old" de-epithelialized regions (black dots); through the combined flap replacement and repositioning of papillae, the areas of recession are completely covered. Sutures that encircle the individual teeth secure the "new papillae." Mattress sutures may be applied apically.



1090 Final Result
Complete coverage of all areas of recession using adjacent, repositioned soft tissues. The type of attachment onto the previous areas of recession can only be determined histologically (long junctional epithelium? connective tissue approximation?).
Advantageous prerequisites:
- Thick gingiva/mucosa
- Expansive papillary region
- Adequately deep vestibulum

Modified from *Zucchelli & DeSanctis 2000*

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Esthetic Improvement through Mucogingival Surgery and Prosthetic Restoration

In order to achieve optimum esthetic results, mucogingival surgical procedures can and in some cases must be combined with prosthodontic measures. Such *indications* exist, for example, in patients who have for years exercised improper oral hygiene that led to gingival recession and wedge-shaped defects at the tooth cervix; also in patients who have preferred acid-containing foodstuffs leading to erosion of enamel and dentin.

The 50-year-old male depicted below exhibited all of this intraoral damage: The wedge-shaped defects were partially filled. On the smooth surfaces of the teeth, the enamel is seriously dissolved and at the incisal edges the teeth have become so thin that teeth 11 and 12 had significantly broken down via attrition (shortened crowns). Esthetic improvements were also required because of this patient's very high smile line. The patient's oral hygiene was quite good.

Clinical Findings:

PI: 11% BOP: 14%

There was no interdental attachment loss.

1091 Initial Clinical View
Pronounced areas of gingival recession with wedge-shaped dental defects.
Note the erosions, and the short clinical crowns resulting from severe attrition of the incisal edges, especially teeth 11 and 21; this led to an inverse arch relationship. The treatment plan included initial therapy, as well as modified and refined oral hygiene.



1092 Step 1: Surgical Thickening of the Gingiva and Mucosa—2 Weeks Following Connective Tissue Graft (CTG)
The surgical result included bulbous papillae and a thickened gingival contour. This is a favorable result and anticipates success with the planned coronal repositioning of these tissues to cover the areas of recession in the region of the four incisors and the canines.

Right: Surgical protocol.



1093 Before Coronal Repositioning of the Soft Tissue
Several months following the connective tissue graft, and following maturation of that graft, as a preparatory measure for coronal repositioning of the soft tissue, the old composite restorations with the exception of tooth 12 were removed, all roughness was smoothed and the convexities of the root surfaces were flattened.

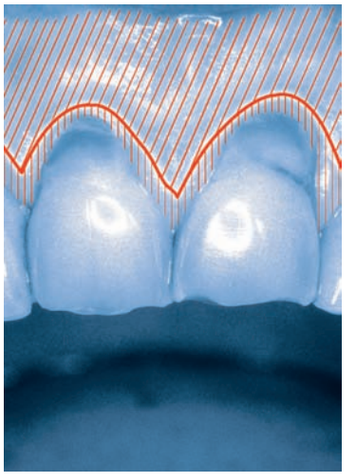
Case: Courtesy *M. Imoberdorf*

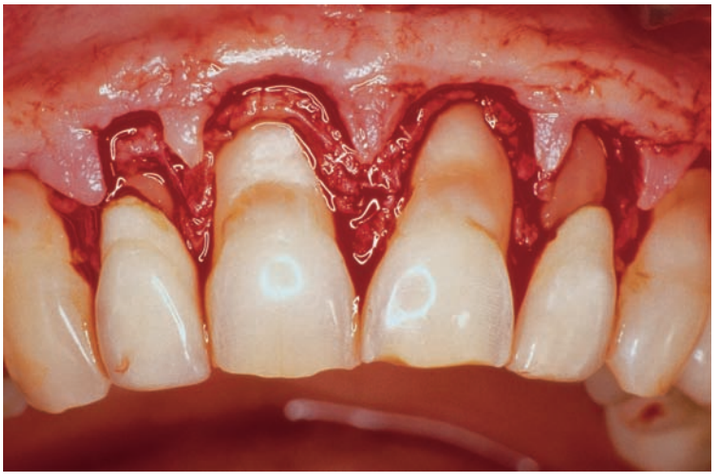


Protocol for the Esthetic Surgical Approach to the Maxillary Anterior Area

0	Initial therapy: – General periodontal therapy – Oral hygiene instruction
1	Connective tissue graft to thicken the tissue overlying teeth 13–23
2	– Covering the areas of recession: Coronal repositioning of the thickened flaps – Root surface conditioning
3	Long-term temporary: – Contours (gingival margin, interdental space)
4	Definitive restoration
5	Establishing the recall interval

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1094 Second Step: Coronal Repositioning of the Tissue Flap
The initial scalloping horizontal incision simultaneously creates "new" papillae (*left*).
A full-thickness flap is reflected up to the mucogingival junction, and further apically a split-thickness flap is prepared. The "old" papillae are *de-epithelialized*.
Before coronally repositioning the soft tissue flaps, the cleaned and planed root surfaces are biomodified (acids, EDTA), and possibly treated with Emdogain (amelogenic protein, p. 351).



1095 Coronally Repositioned Flaps
The flaps are repositioned coronally without tension and affixed using fine, monofilament sutures, 6–0.
The patient is advised to rinse the surgical site during the subsequent 2 post-surgical weeks with chlorhexidine.



1096 Two Weeks Post-Surgery
The initial healing phase in this surgical area is essentially complete and the tissue has begun to consolidate. At this point, the patient re-initiates careful *mechanical* oral hygiene and the chlorhexidine rinses can be reduced.
The teeth, themselves, have not yet been treated because the gingival margin is not yet stable and the maturation phase for the soft tissues will continue for several weeks.





1097 Steps 3 and 4: Dental Reconstruction
Five months after the second surgical procedure, the perfectly proportioned ceramic replacements are seated. The contours of the replacement crowns are such that the interdental papillae completely fill the interdental spaces (Class 3, Jemt, p. 496).

Left: Initial clinical view (the original cementoenamel junction is indicated).

Courtesy *M. Imoberdorf*

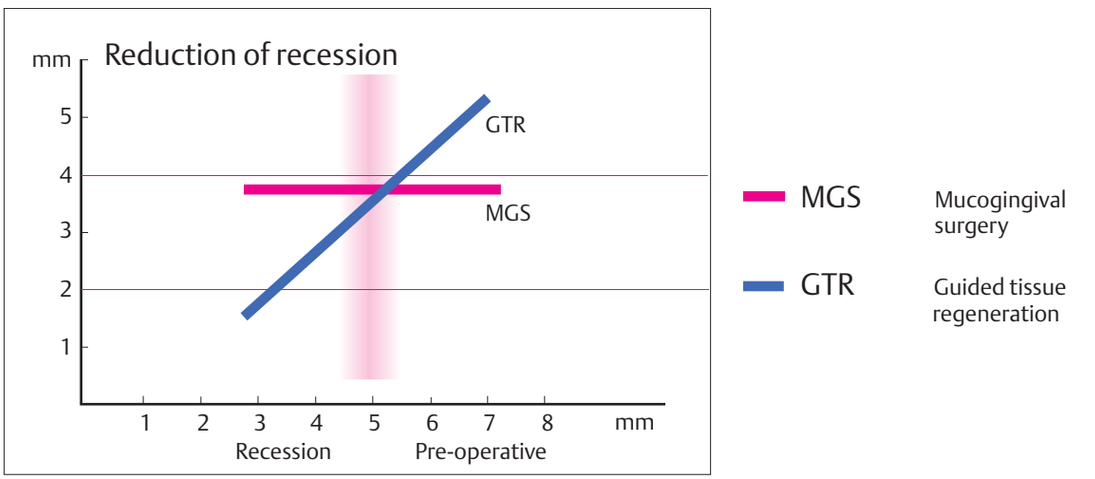
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Summary—Mucogingival, Plastic Surgery

In contrast to periodontitis, gingival recession appears to be increasing in industrialized nations. This may be a result of the increased awareness about oral hygiene; in addition, esthetic "demands" (white teeth, wellness) may be forcing patients toward *excessive* oral hygiene which, under certain morphologic/anatomic conditions could actually *elicit* gingival recession.

The most basic approach would be to *prevent* gingival recession by teaching young children an effective but *non-traumatic* plaque control method, and repeated follow-up examinations. Most of the traditional toothbrushing techniques don't last long! A paradigm shift is critically necessary both for the dentist and his/her patient: A true and proper and effective "prophylaxis" remains a utopic goal; The dentist and periodontist and dental hygienist will have to deal with the manifestations of gingival recession for many years to come.

When one considers *therapy* for gingiva recession, it is necessary to differentiate between *halting* the recession and *covering* the gingival defect, especially in the visible anterior segments (esthetics).



1098 Covering Areas of Recession by Means of Mucogingival Surgery or GTR Techniques
Upon comparison of mucogingival surgery techniques (Harvey 1970, Bernimoulin et al. 1973) with the GTR technique, the recommendation would be mucogingival surgery for areas of recession up to ca. 5 mm; with deeper gingival defects, the GTR technique appears preferable. Advantages and disadvantages of both methods must be considered.

Modified from *G. Pini-Prato 1992*

The Future of Mucogingival, "Plastic" Surgery

The goal of esthetic and predictable 100 % coverage of gingival recession, including multiple areas of recession, has not yet been achieved, despite a myriad of surgical methods (pedicle flap, free epithelialized and non-epithelialized grafts, GTR technique).

On the other hand, it will soon be time to revise the "poor image" of covering areas of gingival recession, despite normal pocket probing depths and the histologic evidence of "only" a long junctional epithelium. More and more dentists/scientists are espousing the concept that at least partial regeneration of all periodontal tissues (cementum, periodontal ligament, bone) will soon become possible, hopefully replacing the simple accumulation of connective tissue onto a root surface.

This vision of the future in periodontology and periodontal practice is being approached through ever-refined techniques, the use of new membranes, osseous filling materials, mediators (e. g., growth factors) as well as certain root dentin biomodifying agents such as amelogenins (Emdogain) or other adhesion proteins.

Today, however, these newest and most innovative treatment possibilities are dependent upon relatively expensive materials and therefore are very costly for our patients; the surgical approaches are also demanding, involving second and third stage procedures. Even if all of these efforts lead to a periodontal procedure that provides a 100 % esthetic result, questions will remain about its necessity, its usefulness, and its cost/benefit ratio.

A simple and inexpensive method to cover expansive areas of gingival recession does not appear feasible, even when constantly new methods, modifications and combinations are being published. For all of these reasons, there can be no doubt that the *early prevention* of gingival recession is the treatment of choice, yet today.

Additional mucogingival procedures:

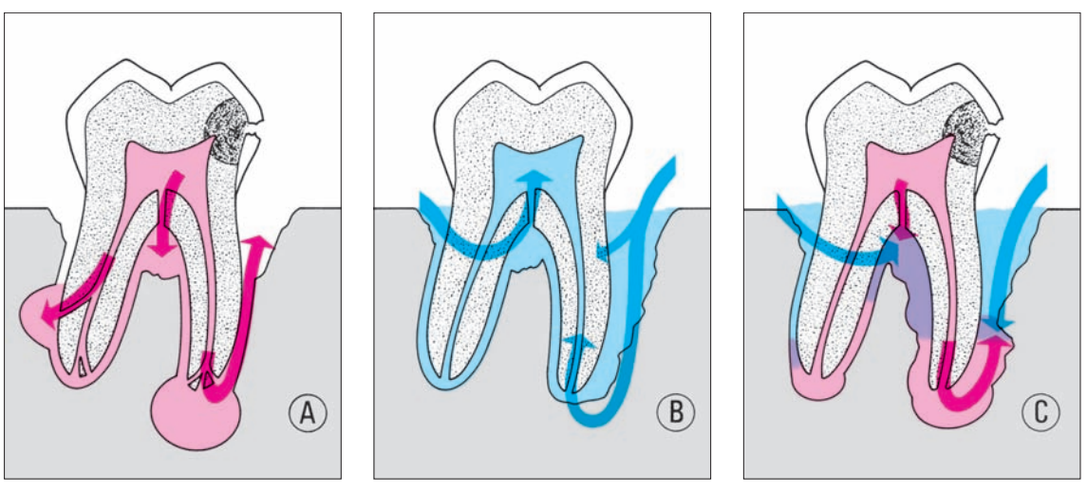
- Alveolar ridge enhancement (p. 505)
- Reconstruction of papillary defects? (p. 496)

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Periodontics—Endodontics

The periodontium and the endodontium have an intimate relationship. They are actually connected to each other at the apical foramen and often via lateral canals, especially in the furcation region. Thus, pulp pathology may directly affect the periodontal tissues. On the other hand, but less often, advanced periodontitis or even severe gingival recession may cause pulpal inflammation or necrosis by way of the apex, lateral canals or the furcation.
A *classification* of these relationships was purposed by Guldener & Langeland (1982). This classification was later summarized by Eickholz (2001):

- Class I: Primarily endodontic problems
- Class II: Primarily periodontal problems
- Class III: Combined endodontic and periodontal problems (endo-perio)



1099 Classification

A **Class I**—Primary endodontic problem: Infection via apex or accessory canals (furcation). Usually a very narrow and difficult to probe "drainage canal" exists near the gingival margin.
B **Class II**—Primary periodontal problem: Infection of the pulp via the periodontal pocket at the furcation, root apex, or lateral canals. A "wide" pocket can be probed clinically.
C **Class III**—Combined endodontic/periodontal problem

This classification includes infectious and inflammatory processes of the pulp and the periodontium as well as combined forms of these conditions. In a broader sense, endodontic/periodontal problems also include iatrogenically caused damage such as perforation and traumatic influences from accidents.

Even in a healthy periodontium, perforation will always lead to periodontal or interradicular lesions. Such lesions may communicate with a periodontal pocket, especially if the perforation is coronally located or if the periodontitis has already progressed far apically.

Accidental trauma can injure and contaminate the periodontium, and lead to infection or necrosis of the pulpal tissue.

Here we will discuss only the "classic," usually infectious, endo-perio problems. Frequently in such cases the first decision is whether the tooth can even be saved. Clinical data gathering in endo-perio problems is often complex: Special medical/dental history, vitality testing, pocket and furcation probing, tooth mobility determination as well as critical examination of radiographs (shape and number of roots or root canals). All of the findings must be synthesized to formulate a diagnosis and a rational treatment plan.

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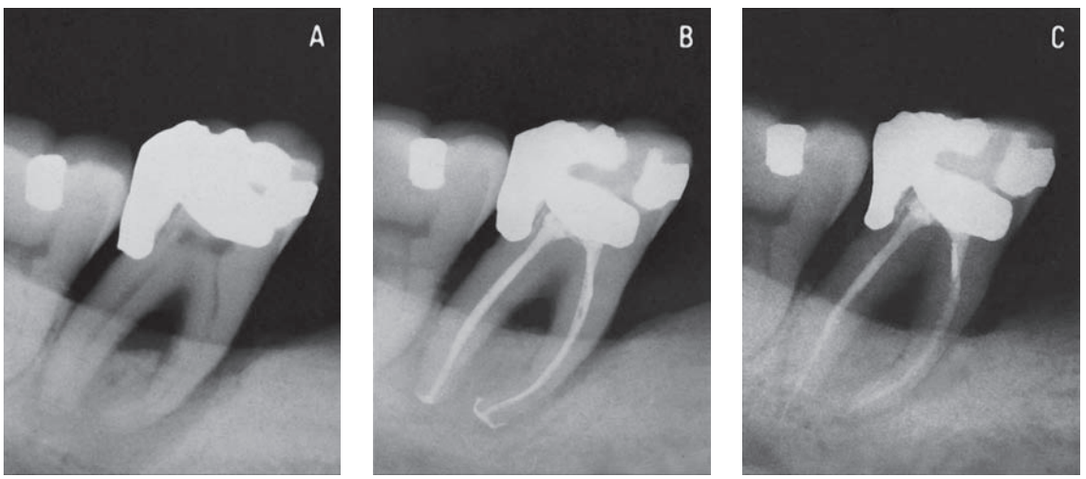
Class I—Primarily Endodontic Problem

The acute inflammatory manifestations in the case depicted below (molar tooth 46) are primarily of endodontic origin. There is also a pronounced loss of periodontal attachment. The pulp, beneath a large amalgam restoration, has become necrotic. The inflammatory process at the apex has expanded coronally, especially on the mesial root but also toward the furcation. The apical lesion communicates with a periodontal pocket.

In terms of *therapy*, there are several alternatives:

1 Extraction of the tooth; bridge or dental implant
2 Endodontic treatment of the distal root, hemisection, extraction of the mesial root, bridge construction
3 Attempt to retain the entire tooth (Fig. 1100 C)

For alternative 3, the endodontic treatment should be performed first. Remnants of the cementum and periodontal ligament fibers that have been affected by the endodontic pathology often regenerate and should not be destroyed by premature aggressive scaling. The periodontal lesion should be treated only after a waiting period of several months.



1100 Class I—Primary Endodontic Problem
A *Initial findings*: The mesial surface of the mesial root exhibits a radiolucency of endodontic origin to the apex; 1. Initial medicinal root canal filling.
B *Interim finding*: 2. Overfilled mesial canal.
C *Final view*: Three months after definitive root canal filling, the osseous defect on the mesial surface of the mesial root exhibits significant regeneration.

Courtesy *M. Meyer*

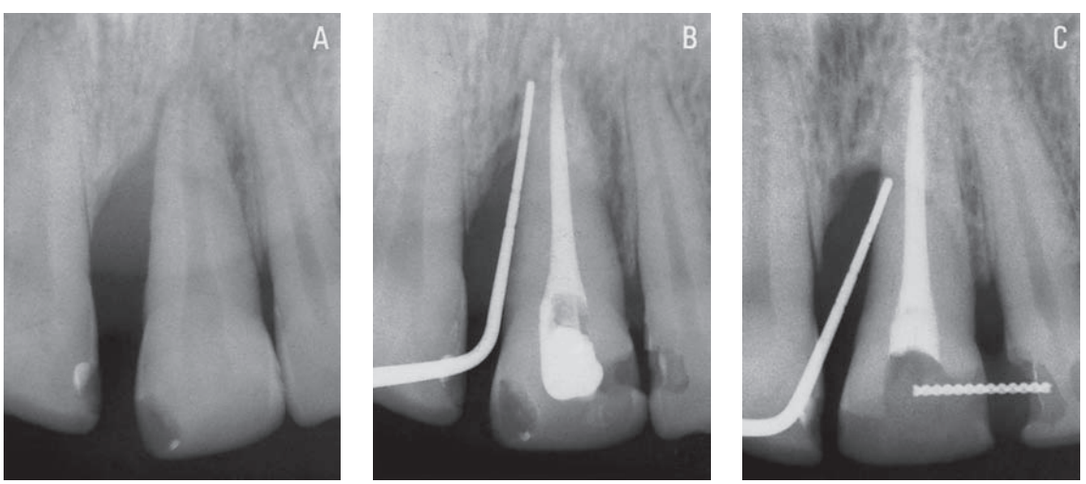
Class II—Primarily Periodontal Problem

Primary periodontal problems affect teeth with very deep pockets that reach the larger lateral canals or even the apex itself. This can lead to retrograde pulpitis or pulpal necrosis. Such teeth are often hopeless and should be extracted if they are not of special esthetic or functional significance for the overall treatment plan. In the case described below, involving tooth 21, the decision was made to attempt maintenance because extraction would necessitate a costly and complicated anterior bridge construction.

Initial examination revealed a deep and broad infra-alveolar pocket. Pus emanated upon probing. The radiograph revealed the typical, circumscribed periodontal bone loss (see p. 99, Fig. 195).

The treatment plan included endodontic procedures and subsequent periodontal treatment.

The prognosis is questionable in such severe periodontal involvement and a class II perio-endo lesion. In the case depicted here, the long-term success was good.



1101 Class II—Primary Periodontal Problem
A *Initial findings*: The 11 mm pocket on the mesial of tooth 21 has caused retrograde pulpitis.
B *Interim finding*: Following endodontic treatment, root planing was performed with a flap procedure and the mobile tooth was temporarily splinted (**C**).
C *Final view*: Twelve years later, new bone formation can be observed.

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Class III—Combined Perio-Endo Problems

A true combined perio-endo lesion results from confluence of endodontic and periodontal lesions that developed independently. Clinical examination generally reveals a more or less broad and continuous defect extending from the gingival margin to the root apex. As with Class II lesions, retention and maintenance of such teeth is questionable. It should only be undertaken if loss of the tooth would necessitate extensive and esthetically difficult reconstruction (bridge, cantilever, dental implant), or if the tooth is an important abutment for fixed or removable restorative work.

In the case depicted here, a 55-year-old female presented with untreated, chronic and severe periodontitis. If the questionable tooth, 32, were extracted, an extensive prosthetic reconstruction would be necessary.

The combined periodontal and endodontic *therapy* must be viewed as a heroic effort. The *prognosis* is generally questionable. In the case presented here, however, the long-term success was good. Expansive root canal enlargement (A) and "granuloma filling" (B) are seldom performed today.





1102 Class III—Initial Findings, Mandibular Anterior Area
Generalized, advanced periodontitis with gingival shrinkage. Heavy plaque accumulation. Almost 12 mm probing depth on the distofacial surface of non-vital tooth 32.

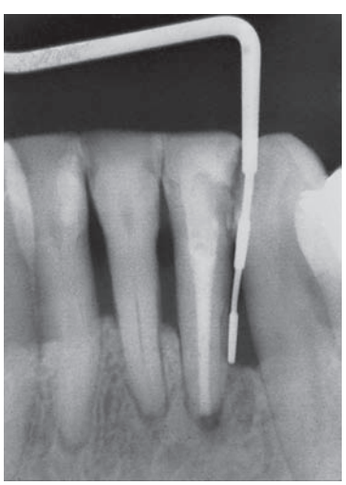
Left: Obvious large periapical lesion on tooth 32, communicating with the deep distofacial pocket (CP 12 probe). Advanced horizontal bone loss.







1103 Sequence of Endodontic Therapy
A Instrumentation of the canal with Hedstrom files.
B "Granuloma filling" with resorbable iodoform paste (caution: iodine allergy; see above). Scaling of only the *coronal* aspect of the root surface (periodontal pocket).
C Definitive root canal filling eight months after initial therapy; periodontal probing originally extended to the root apex (Fig. 1102, left), but now stops at the alveolar crest.

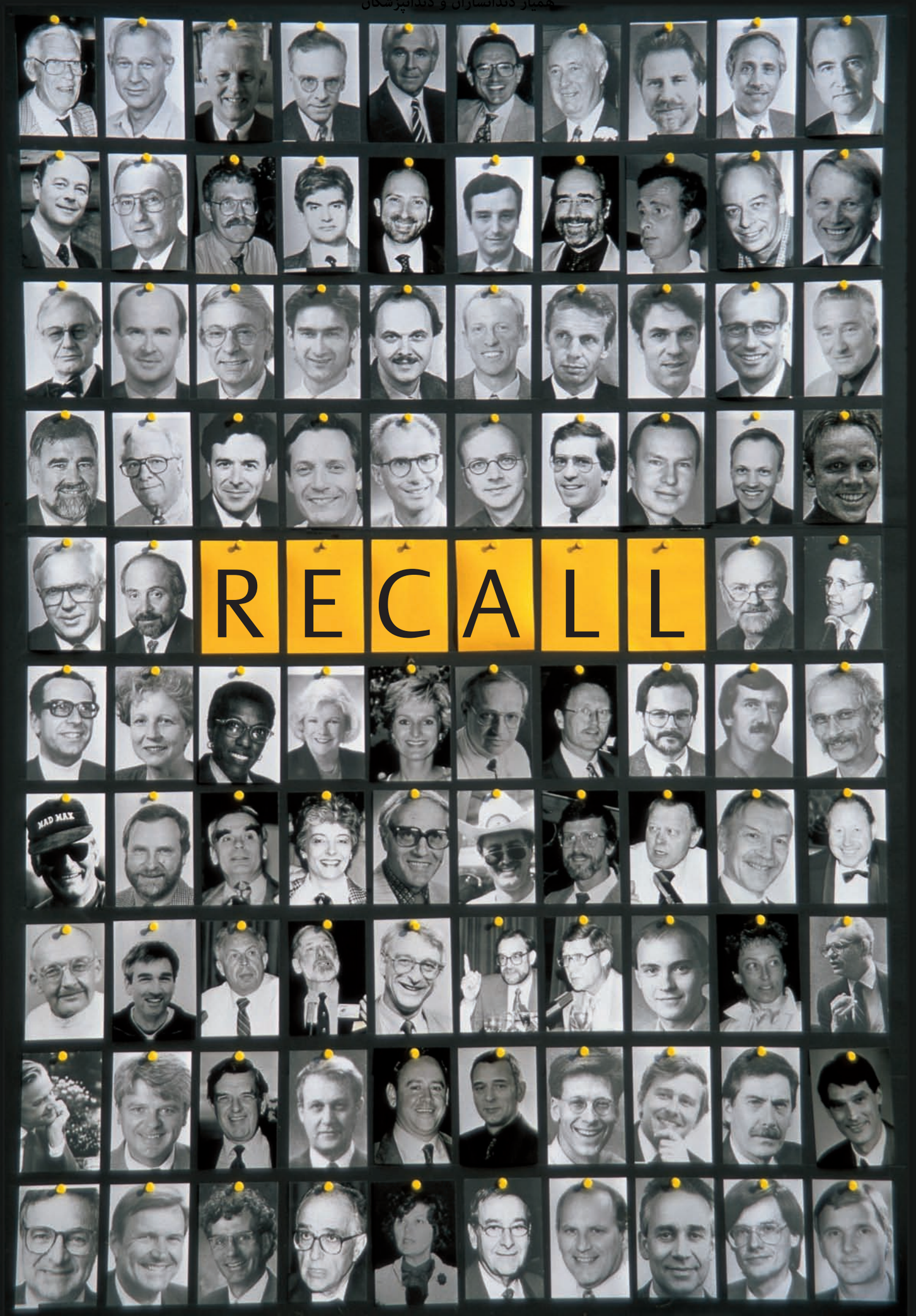




1104 Clinical Situation Three Years later
The pocket depth has been reduced (CP 12 periodontal probe). Since initiation of therapy the patient practiced virtually perfect (and perhaps excessive!) oral hygiene. Teeth 41, 31, and 32 were splinted using composite resin.

Left: Radiographic view 14 years later. The periapical problem and the drainage canal to the pocket appear to be almost completely resolved.

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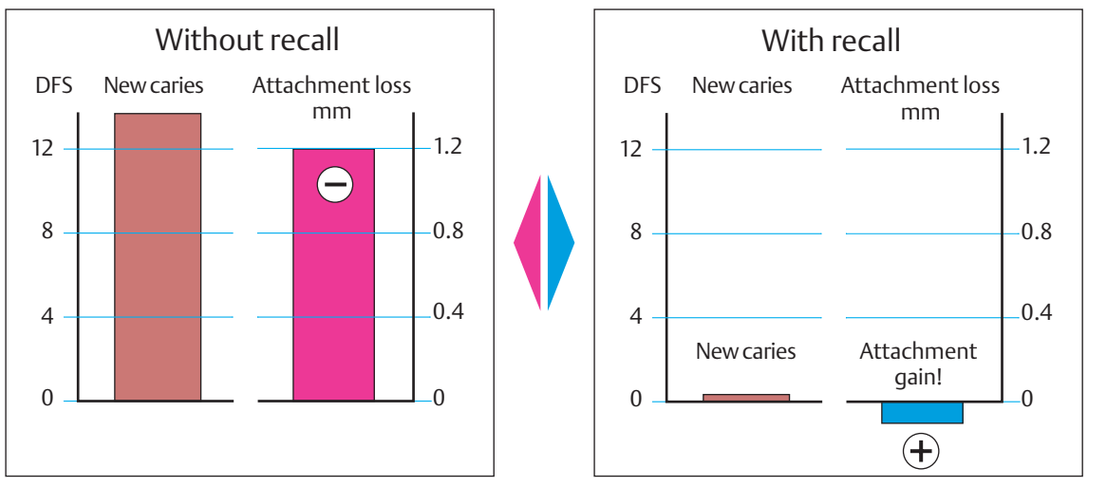


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Phase 3 Therapy
Periodontal Maintenance Therapy—Recall

The long-term success of periodontal therapy depends less on the manner in which the case was actively treated (Phase 1 and 2) than on rigorous follow-up of the wound healing process immediately after therapy and on how well the case is maintained in subsequent recall (Rosling et al. 1976, Nyman et al. 1977, Knowles et al. 1979, Ramfjord et al. 1982, Wilson 1996; Axelsson 2002, AAP 2003).

Clinical research by Axelsson & Lindhe (1981, Axelsson et al. 1991) demonstrated dramatically the effects of preventive measures during recall (Fig. 1105). This truly classic clinical study, which is on-going even today (!), continues to demonstrate that regular and short-interval (2–3 months) prophylaxis by the dental hygienist (!) results in virtually no new *caries* and not the slightest periodontal *attachment loss*. This landmark clinical study, originally planned for 6 years duration, casts some serious doubts about "classical" reparative dentistry!



1105 Axelsson Study: Caries and Attachment Loss with and without Recall
Left: Patients who received neither homecare motivation nor preventive measures during one dental visit per year had 14 new caries and progressive attachment loss over the 6-year period.

Right: Similar patients who received intensive professional prophylaxis every 2–3 months developed essentially no new carious lesions and actually exhibited some attachment gain!

The primary goals of maintenance therapy include:

- Maintenance of oral health (including cancer screening)
- Maintenance of chewing function, phonetics and esthetics
- Prevention of new infection (gingivitis, periodontitis)
- Prevention of re-infection of inactive residual pockets (periodontitis)
- Prevention of dental caries

These goals can be achieved through:

- Re-examination and re-evaluation
- Re-motivation and new information for the patient
- Re-instruction in oral hygiene and update of oral hygiene informative materials
- Supragingival plaque and calculus removal
- Subgingival debridement of pockets and root surfaces in areas exhibiting disease activity
- Topical fluoride application

Page 448: "Recall them all"
→ Defining legend, p. 523

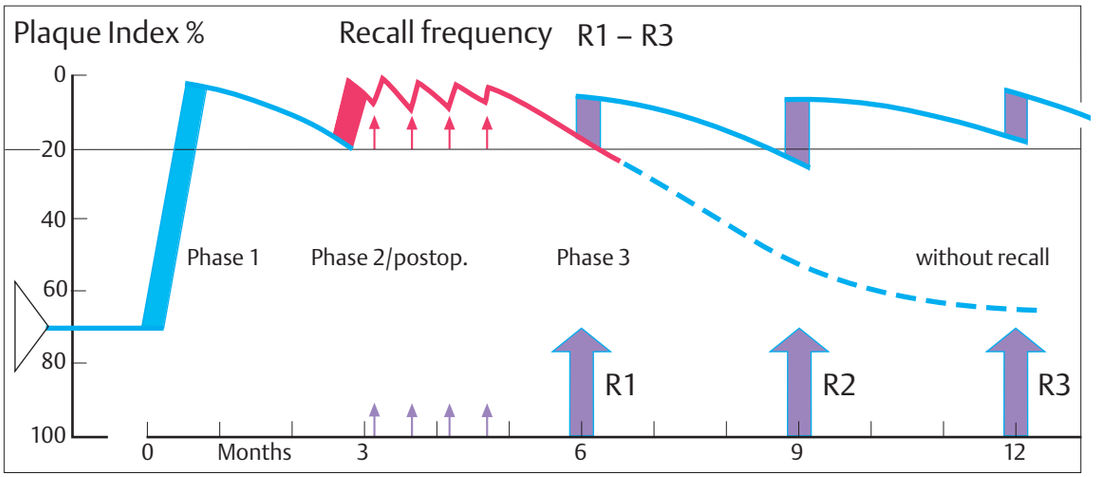
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Recall in the Dental Practice—Recall Effect

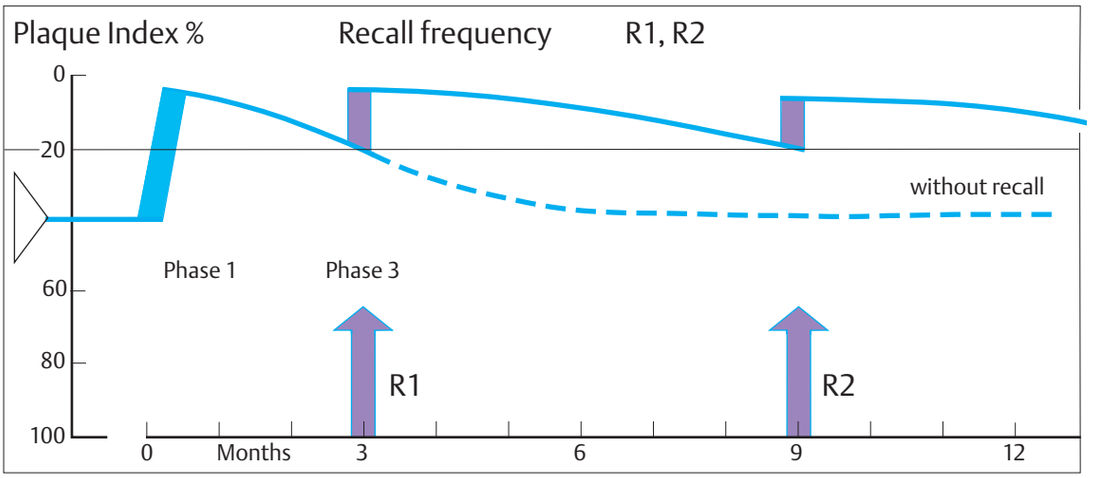
Recall—maintenance therapy—in every dental practice can only be accomplished using competent auxiliary personnel, usually a well-trained dental hygienist. In particular, every treated periodontal patient must remain in recall "for life," and be re-treated as necessary.

It is of course true that even the best recall system managed by competent practice personnel and supported by appropriate infrastructure will not be effective for *all* patients; there will always be patients who are poorly motivated, who do not accept personal responsibility for their oral health or who simply do not accept the recall schedule. Such patients, exhibiting non-compliance or "erratic" compliance (Wilson 1996) must be recognized before any systematic, active periodontal therapy is initiated.

But even patients who are highly motivated initially often, over the course of time, become less so. These individuals must be re-motivated during each and every recall appointment, and this is the highest calling for a truly prevention-oriented dental practice; *the dental hygienist is the key person!*



1106 Recall Effect I—Patients with a High Initial Plaque Index (PI = 70 %)—Schematic
With Phase 1 (blue curve) and Phase 2 therapy (red), healthy periodontal tissues result.
The plaque index is now below the 20 % mark; however, without re-motivation at recall appointments (**R1, R2, R3**) the PI rapidly sinks to the initial, poor level. With the relatively short recall interval of every 3 months, the periodontal therapeutic treatment result can be maintained over years.



1107 Recall Effect II—Patients with a Lower Initial Plaque Index (PI = 40 %)—Schematic
For patients with less severe periodontitis (only Phase 1 therapy) and better initial oral hygiene, a "motivation kick" 3 months after completion of active therapy (**R1**) and then every 6 months (**R2, R3**) was sufficient to eliminate any risk of disease recurrence.

Modified from *M. Leu 1977*

Recall Effect—Successful Prevention

The positive results of "prevention" have been demonstrated without exception for more than 20 years. Preventive dental medicine helps the patient not only to maintain healthy teeth in a healthy periodontium, but also inhibits the numerous potential negative effects of chronic inflammatory periodontitis on general systemic health (cardiovascular disease, diabetes, pulmonary problems) and pregnancy (risk of premature birth, low birth weight); such risks are greatly increased in the absence of periodontal therapy and without periodontal recall (Becker et al. 1984, Beck et al. 1996).
The recall interval will be different for each patient, depending upon her/his degree of motivation/compliance and manual dexterity. Depending upon the patient's success in personal oral hygiene and personal *daily "antimicrobial" homecare*, the recall interval may be established between 2 and 12 months.
The *time* necessary for a recall appointment seems always to be *underestimated*. The recall appointment is not just a brief "scraping" of the calculus in the mandibular anterior segment. The comprehensive diagnostic, prophylactic, and possibly also therapeutic procedures may require one hour or more, depending upon the case (p. 451).

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Recall—Continuous Risk Management

In the dental specialty of periodontology, the term "prevention" includes all measures targeted toward identifying the occurrence, progression or re-emergence of all forms of periodontitis—to prevent them or to identify them *early* in order to prevent further progression.

The very purpose of a recall appointment includes, in addition to purely clinical treatment, the obligation for risk-coordination in the sense of documentation of any changes that have occurred since the comprehensive initial diagnostic findings (cf. p. 193).

Early on it was recognized that the general patient history and clinical findings must be based upon a multilevel assessment of risks at three levels:

- At the patient level
- At the level of the individual tooth
- At the level of each surface of each tooth ("site")

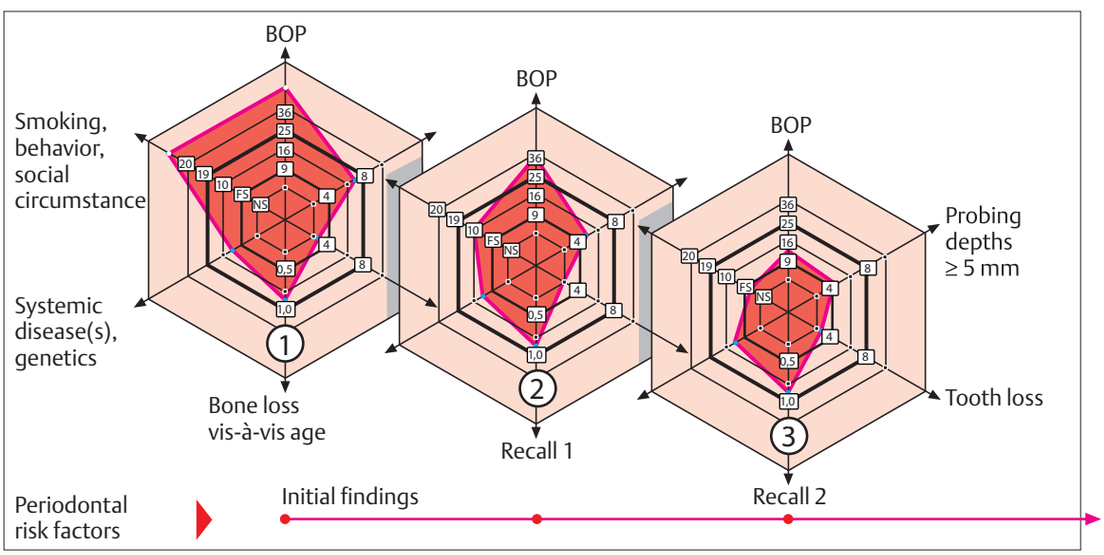
The general systemic medical history is without a doubt the most important (What is new since the last appointment? Cardiovascular problems? Metabolic disease/diabetes? Anticoagulant medication? Other medications?). These aspects of each patient must be up-dated at each recall appointment. Generally, every appointment begins with "How are you"?!

According to the Berne model (Lang & Tonetti 1996, p. 193), the individual risk profile for any given patient is determined by six *risk parameters*: Two are related to tooth surfaces (BOP and probing depths), and two are tooth-related (tooth loss and bone loss related to patient age); both of these patient-related factors determine the overall risk (alterable: smoking, life style; unalterable: systemic and genetic diseases).

What is missing, logically enough, is the participation of causal factors of periodontitis: The dental plaque "biofilm." However, it is important to understand the *reaction/response* of the susceptible host organism to the microbial colonization of the periodontal pocket. This is what determines the initiation, progression and course of disease.

For the dentist and above all the dental hygienist, who is in most cases responsible for maintenance therapy, the previously depicted model simplifies each individual case both diagnostically and prognostically.

The primary purpose of the schedule of recall appointments is to consistently follow the *long-term* health status of each patient's periodontal condition.



1108 Risk Assessment at Subsequent Recall Appointments
The "red area"—the graphically represented *total risk* of the patient—becomes less and less in this model case.

Unalterable risk factors (e. g., genetics) and the never-to-be-recovered bone loss leaves the depicted patient in a situation where he can no longer achieve the lowest level of risk.

Levels of Risk Assessment

Patient-related:
- Systemic diseases
- Environmental factors (smoking!)
- Compliance (oral hygiene)
- Attachment loss in relation to age
- Plaque accumulation in the oral cavity
- Percentage BOP on all tooth surfaces
- Number of pockets deeper than 4 mm

Dental arch relationships:
- Tooth tipping
- Tooth morphology (grooves, ridges)
- Tooth mobility
- Iatrogenic factors
- Remaining tooth support
- Furcation involvement

"Site" involvement:
- BOP
- Probing pocket depth
- Attachment loss
- Suppuration, pocket activity

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The "Recall Hour"—Practical Periodontal Maintenance Therapy

Clinical Findings

- *At every recall appointment*:
  - Gingival condition (BOP or PBI)
  - Plaque accumulation (PI/PCR or API; disclosing solution)
  - Activity of residual pockets
- *Additionally, every 6–24 months*:
  - Pocket probing depths, radiographs (?)
  - Occlusion, reconstructions (tooth vitality), caries
- *Additionally, every 3–4 years*:
  - Panoramic radiograph and individual PA films, p.r.n.

Clinical Procedures

Depending upon the findings, the following procedures should be performed:

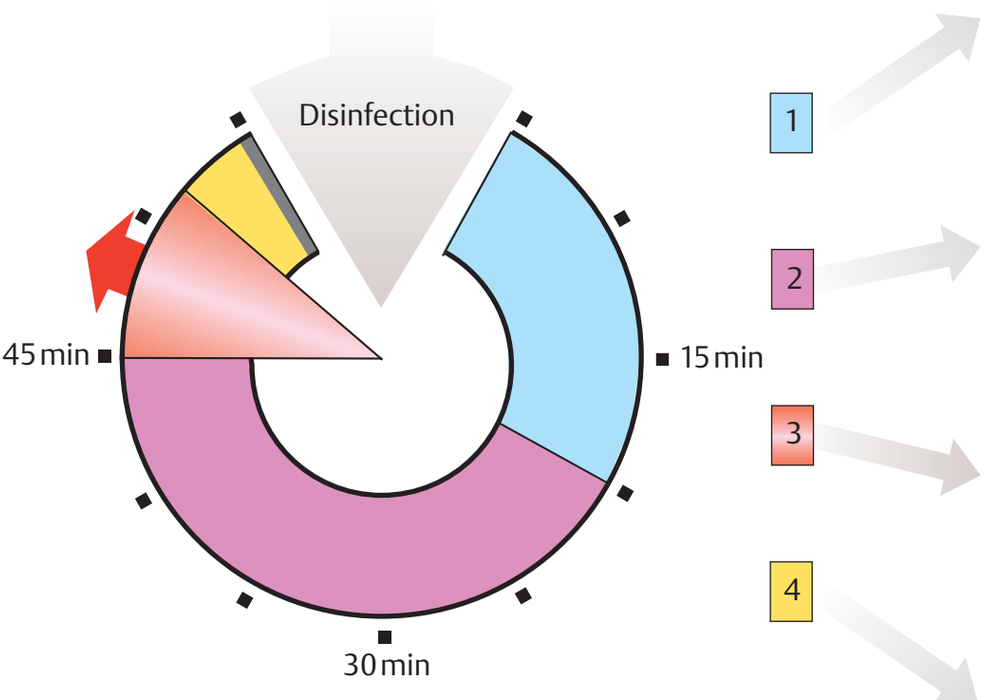
- *At every recall appointment* (*e.g., every 2–6 months*):
  - Medical history up-date
  - OHI and re-instruction
  - Re-motivation of patient compliance
  - Plaque and calculus removal *where indicated*!
  - Treatment of disease recurrence, p.r.n: debridement, topical meds (p. 291); additional appointments.

See also the *10-point program plus X*, below.

1109 The "Recall Hour"

The diagram portrays a rough approximation of a typical 1-hour recall visit. Rationalization of the instrumentarium (ultrasonic instruments) may compensate for the "lost time" required today for the thorough disinfection of the operatory. The fact is that the dental hygienist really has only about 50 minutes per patient.

The division of the "prophy hour" is in four most important segments; these are depicted on page 453:



1 Patient history, clinical data collection (ca. 15 minutes*)
2 Patient instruction, *instrumentation* (ca. 25 minutes*)
3 Treatment of active sites (ca. 5 minutes *)
4 Polishing, fluoride application (ca. 5 minutes*)

* The times required will vary from case to case.

Checklist: 10-Point Program Plus X

1	Medical history update	new systemic risks
2	Mucosal examination	oral cancer prevention
3	Evaluation of inflammation	motivation
4	Pocket probing depths	activity?
5	OHI	compliance
6	Oral hygiene	re-instruction
7	Calculus removal	targeted!
8	Biofilm removal	mild instrumentation
9	Polishing restorations	minimal abrasiveness
10	Fluoridation of the teeth	information about its effectiveness
X	Extra measures	radiographs, vitality, sensitivity, surgery etc.

Recall—"As Fine as Possible"

Of top priority at recall appointment is the instrumentation of *root surfaces* and *any exposed dentin surfaces*. Just as we advise patients to not use any abrasive dentifrice when cleaning interdental areas, the dental hygienist is also well advised to use only the *gentlest* machines and hand instruments as well as the least abrasive prophy pastes as she/he cleans and polishes tooth surfaces.

Goal: Clean, but not abrasive/destructive! Even after 100 recall appointments (20–40 years), there should be no gross evidence of excessive "scraping" on the teeth (cf. Fig. 1120)!

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Examination

Re-Evaluation

Diagnosis

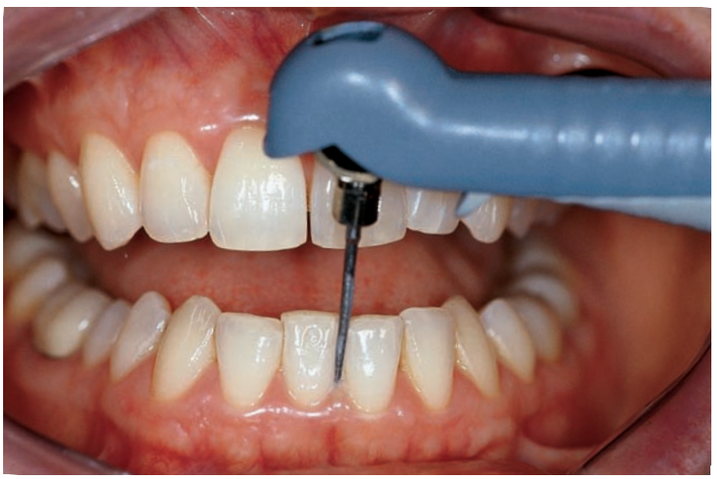


1110 Data Collection, Re-Evaluation, Diagnosis
The current status of the patient is evaluated.
The patient’s personal and even family difficulties are discussed as well as a follow-up of the general systemic health. Changes in medications are particularly important with older patients (stress?). Have any other risk factors changed (smoking)?
Note: Five minutes of attentive listening is much more important than dental instrumentation, even when time is of the essence.

Motivation

Re-Instruction

Instrumentation



1111 Motivation, Re-Instruction, Instrumentation
The latest scientific findings show us the way: Perform scaling only where calculus is present, and remove microbial biofilm using the most gentle of instruments. Evaluate new developments! For example, the Vector ultrasonic device (pictured here); water-powder spray devices with minimally abrasive powder (EMS-Handy 2; p. 282) etc.

Re-Treatment of Active Sites



1112 Treatment of Active Sites (Re-Infected or Newly Infected)
Individual active pockets can often be treated during the recall appointment (scaling; topical irrigation, e. g., betadine or controlled-release drugs). Pictured is an Atridox application into a “refractory pocket” (cf. p. 293).

Multiple active sites or true recurrence of periodontitis will require separate appointments for treatment (scaling or surgery, supported by medicaments, p.r.n).

Polishing

Fluoride Application

Re-scheduling



1113 Polishing, Fluoridation, Scheduling the Next Appointment ... Recall Interval
Before applying fluoride, all teeth are polished with the rubber cup and a fluoride-containing prophy paste.

Determination of the recall interval is made on the basis of risk assessment (type and severity of periodontitis, systemic and local risk factors, plaque control and patient compliance etc).

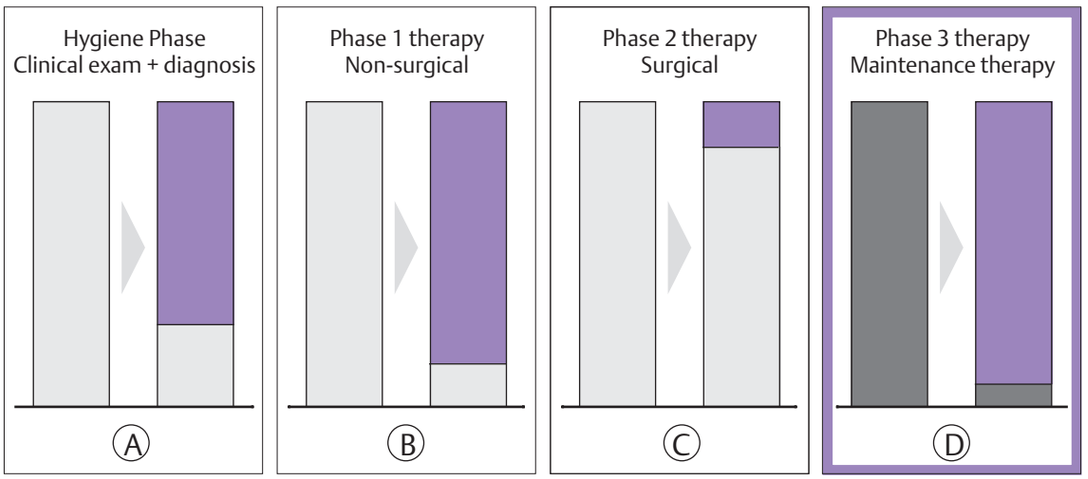
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Dentist and Dental Hygienist—The "Preventive Team"

Cost-effective and modern preventive measures—"prophylaxis"—would be impossible today without *highly qualified* auxiliary personnel such as the dental hygienist. The dental hygienist can actually *replace* the dentist in many procedures such as data gathering, prophylaxis, initial therapy and maintenance therapy (recall). A full time dental hygienist can provide care and maintenance for over 500 patients per year, based upon 3–4 one-hour appointments per year. Clinical care performed by the dental hygienist must be supervised and/or enhanced by the dentist.

The dental hygienist and other auxiliary personnel can also be of significant assistance to the dentist in the care and maintenance of all other dental patients, e.g., patients with all types of dental replacements (denture care, preventive measures on abutment teeth and fixed bridgework etc.).

1114 Relief of the Dentist (Gray) by the Dental Hygienist (Violet)
The dental hygienist (DH) is responsible for over 90 % of each recall appointment (**D**), but the DH can also assist the dentist in other areas.
For example during data gathering (**A**) and especially during *initial therapy* (**B**) the DH assumes more than 80 % of the work.
Even during the surgical phase of therapy (**C**) the DH can play a significant role in post-surgical wound management.



Auxiliary Personnel and Treatment Needs

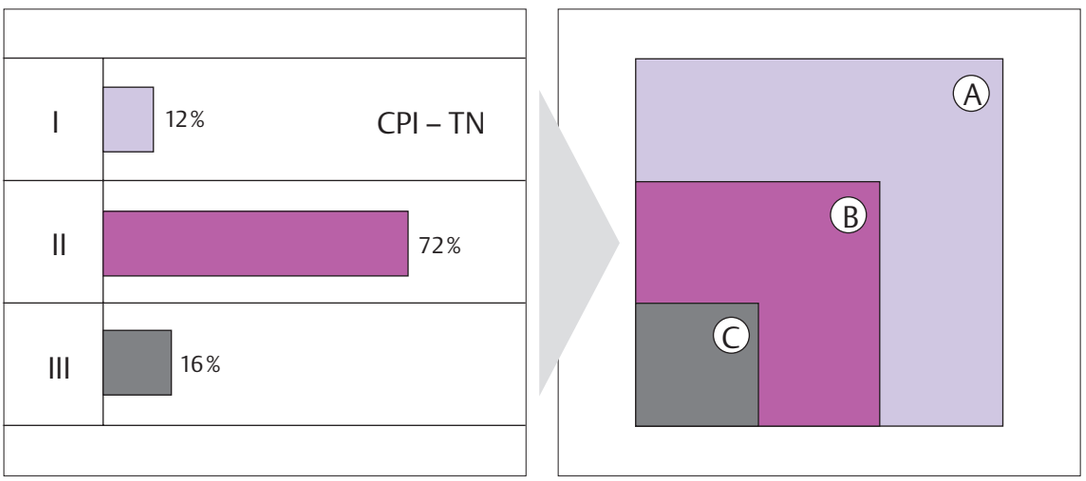
Hamburg Clinical Study, 1987—CPITN

Ninety-seven percent of individuals examined in the Hamburg study required some type of periodontal care (TN = "treatment need" of the CPITN; p. 72; Ahrens & Bublitz 1987). The type of treatment indicated varied dramatically:

- A total of 12 % (3 % of the healthy and 9 % of patients with plaque-induced bleeding on probing)—required repeated OHI.
- 72 % of those exhibiting calculus, and patients with probing depths to 5.5 mm required subgingival scaling.
- "Only" 16 % of patients exhibiting deeper pockets required more complex periodontal therapy (surgery).

Based on the study, 84 % of the population—the relatively healthy and those exhibiting gingivitis/periodontitis—could be treated prophylactically *and* therapeutically by auxiliary personnel: 12 % by trained dental assistants alone and fully 72 % by *highly qualified dental hygienists*.

1115 "Treatment Need/TN" in the Hamburg Study (1987)
TN I Oral hygiene instruction and *supragingival* plaque and calculus removal
TN II As above, plus *subgingival* debridement/scaling
TN III As above plus more complex therapy
A 100 % of prophylaxis (TN I) can be performed by a trained dental assistant or hygienist.
B 84 % of TN II can be performed by the dental hygienist
C For TN III, 16 % of cases require a dentist or a specialist.



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Failures—Lack of Periodontal Maintenance Therapy

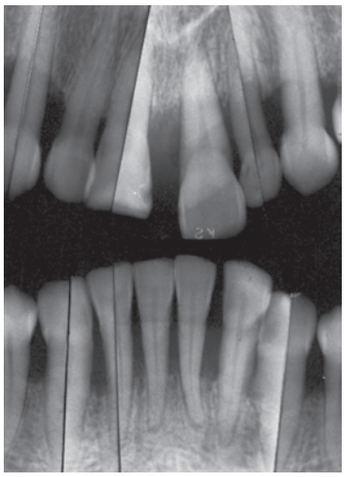
Success in Periodontal Therapy

- Inflammation (gingival bleeding) eliminated
- Pocket activity eliminated
- Probing depths reduced
- Attachment loss halted
- Tooth mobility stabilized or reduced

Failures of Periodontal Therapy

- Persistent bleeding
- Persistent pocket activity
- Increasing probing depths
- Advancing attachment loss
- Increasing tooth mobility

Most failures in the treatment of periodontitis can be explained. The most common causes of failure include: Incorrect dental and systemic medical history, incorrect diagnosis and/or prognosis, inappropriate treatment plan, inadequate therapy, *insufficient patient compliance* and *lack of maintenance* (Becker et al. 1984, Rateitschak 1985).





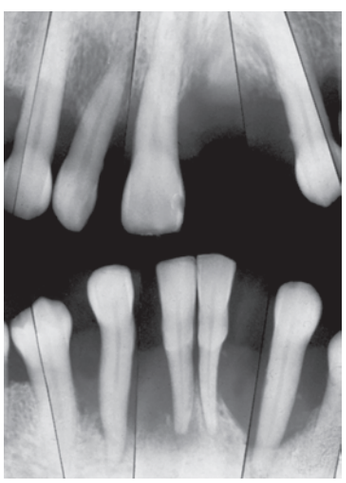
1116 Advanced, Rapidly Progressing Periodontitis
When this 38-year-old female presented, she had never been instructed in oral hygiene. She only wanted a dental check-up; she had no complaints!
Clinical findings:

PCR	100 %
PBI	3.8
PD	up to 8 mm, many active pockets
TM	very elevated, with tooth migration

Left: Radiographic appearance



1117 Following Initial Therapy
The clinical picture is significantly improved following thorough scaling and root planing. The patient's compliance with homecare instructions was inadequate from the beginning, and therefore no surgical procedures were performed. Shortly after initial therapy, plaque and calculus accumulation occurred (mandibular anterior area).
The patient refused to enter a regular maintenance recall schedule or to undergo orthodontic treatment.





1118 Five Years later, without Recall
The patient presented again, after teeth 22, 23, 32 and 42 had spontaneously exfoliated. The treating clinician elected not to attempt extensive periodontal therapy.

Therapy:
Maxilla: Complete denture
Mandible: Retain four premolars via periodontal therapy; removable partial denture.

Left: Radiographic findings five years later, without recall.

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Negative Results of Therapy

- Long teeth—exposed dentin
- Tooth mobility
- Phonetic problems
- Compromised esthetics (maxillary anterior region)

Depending upon the severity of the initial periodontitis, therapeutic measures (closed as well as surgical therapy) will lead to more or less pronounced gingival shrinkage and therefore to "lengthening" of the teeth. The patient must be warned of such possible consequences *before* any therapy is initiated!

Long teeth with their typically attendant dark interdental spaces represent the greatest problem, especially in patients with a high smile line (short upper lip). This situation not only compromises phonetics, but also esthetics, which has become ever more important today. These problems can be dealt with by means of very expensive prosthetics measures (p. 489), but can also be solved using simple and inexpensive treatments which, unfortunately, usually become "permanent temporary" solutions (p. 457).
(The problems of tooth neck hypersensitivity are discussed on page 458.)

Successful (?) Periodontal Therapy

1119 "Long Teeth"—Excessive "Compliance" by the Patient?
This 32-year-old female exhibited gingival recession even prior to surgery, and her esthetic appearance worsened after periodontal surgery, when her chief complaints included poor esthetics, phonetics, and tooth neck sensitivity.
She had not yet noticed the wedge-shaped defects on the posterior teeth.

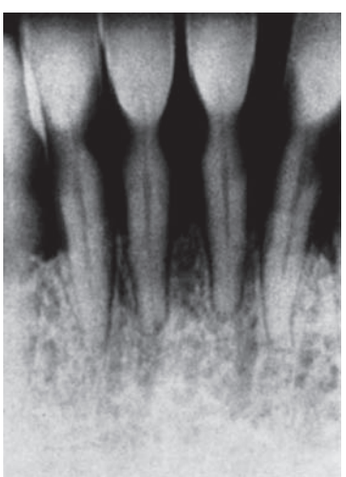


Results of therapy:

- Gingival recession
- Hypersensitive dentin
- Dark-appearing interdental spaces

1120 Severely Abraded Mandibular Anterior Teeth; Danger of Fracture
The condition could be due to overzealous homecare by the patient or to overaggressive scaling/root planing during recall appointments.
The patient must be instructed to use *no abrasive dentifrice* on the interdental brushes, rather only fluoride- or CHX-containing gel.
At recall appointments, the dental hygienist should use minimally aggressive instruments and prophy paste in the interdental region.





Oral hygiene "too good":

- Wedge-shaped defects
- Narrowing of the interdental tooth substance

1121 Cervical Caries
Note the deep wedge-shaped cervical defect with also a small but very deep carious lesion (*left*); removal of the caries led to pulp exposure: Root canal treatment was performed (*right*) without a rubber dam!
Note: The question remains how to perform gentle *yet thorough* treatment?
The regimen included once per day perfect plaque removal using a soft toothbrush, very little force, a gentle technique and a non-abrasive dentifrice.





"Inadequate oral hygiene":

- Cervical caries!
- Gingivitis
- Recurrence of periodontitis?

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Esthetic Improvement for Negative Therapeutic Results



1122 Clinical Appearance after Periodontal Therapy
The maxillary anterior teeth are unesthetically proportioned. The interdental spaces are large, appearing as "black triangles," which disturbed this young female patient. Dental reconstruction by means of veneers or crowns exceeded the patient's financial situation.
Planning: Restorations and a gingival mask.





1123 Gingival Mask Fabricated from Denture Base Acrylic Resin
Initially, interdental composite resin restorations were placed (acid etch technique). These were over-contoured to reduce the expanse of the interdental space. Using a rubber-elastic material, an impression was made for use in fabricating the color-matched gingival mask.

Left: Denture base acrylic resin in various shades, to blend perfectly with the patient's tissues.



1124 Inserting the Gingival Mask
The wedge-shaped interdental portions of the mask snap into place with minimum pressure, into the interdental areas.

In this case, the patient first seats the left side of the gingival mask, distal to tooth 23 and then with a rolling motion of the finger seats the gingival mask into the remaining interdental spaces.



1125 Much-Improved Esthetics
The margin of the gingival mask at the mucogingival junction is scarcely visible. This patient had a "low smile line" (p. 492) which led to an excellent esthetic (invisible) result.

Note: Because damage of such acrylic gingival masks is not uncommon, it is wise to prepare several masks for the patient.
Soft masks—on the other hand—become quickly discolored.

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Hypersensitive Dentin

Exposed tooth necks are doubly endangered:

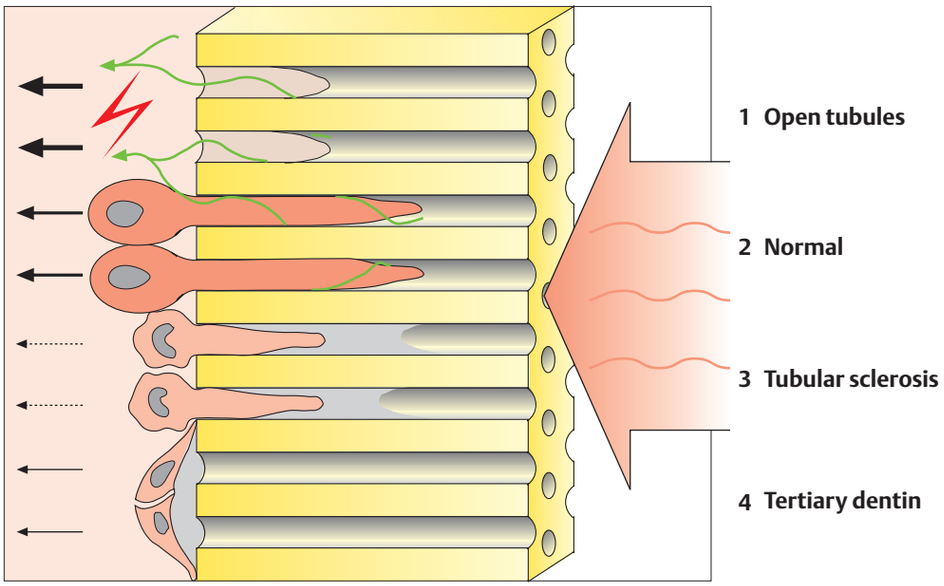
- Sensitive tooth necks (exposed dentin) render daily plaque removal questionable. Even lukewarm water will elicit *pain*, so that even a willing patient must overcome such discomfort in order to keep the tooth necks and the gingival sulcus plaque-free.
- With inadequate oral hygiene (insufficient compliance, incapacity, age etc.) and especially following radiation therapy in the head and neck region, the problem of *dentin caries* frequently emerges; a very difficult type of caries to treat.

Tooth neck pain is elicited by the irritation and rapid movement of dentinal tubule fluid, the so-called "hydrodynamic hypothesis" (Brännström 1963). Irritated pain receptors at the pulp-dentin junction lead the nerve impulses further (summary by Jensen 2003).

This *stimulus transfer* can be therapeutically reduced by tin and other fluoride, strontium and potassium salts (in dentifrices), non-filled resins, dentin bonding or laser closure etc.

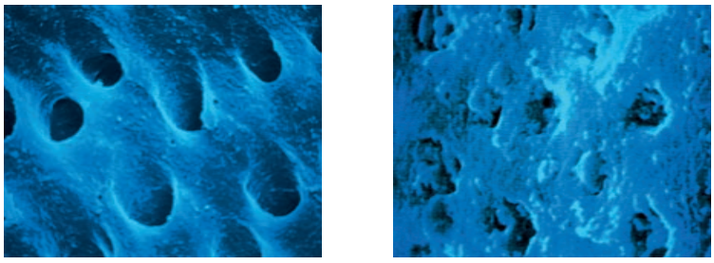
1126 Occurrence of Dentin Hypersensitivity—Possibilities
Root planing will lead to exposure of dentinal tubules. Four severities of reaction (black arrows) are depicted when an external irritation (wide red arrow, right) comes into contact with the dentin surface:

1 Open tubules, "dead tracks"
2 Normal: Open, vital odontoblasts with nerve endings
3 Tubules closed off; sclerosis
4 Tubules closed by tertiary dentin formation



1127 Dentin Tubules before and after Treatment with Fluoride
Left before, and *right* after treatment.

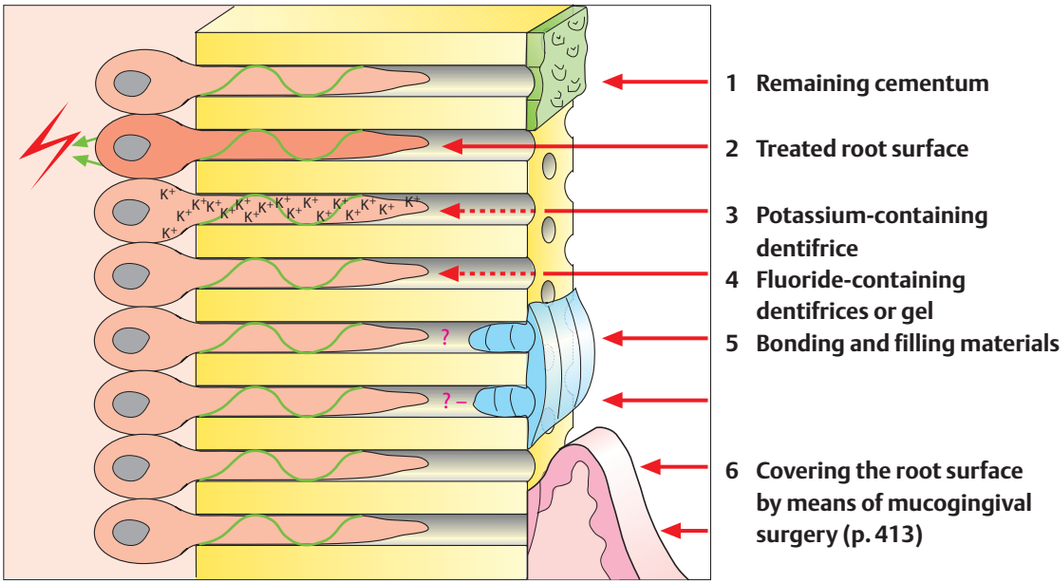
SEM courtesy *Gaba Co., Basel, Switzerland*



1128 Elimination or Reduction of Tooth Neck Hypersensitivity
The ideal situation is clean (endotoxin-free) autogenous cementum covering the dentin surface! The question is: What does "clean" mean and where does one find any cementum after root planing?

Principles of therapy:
- Tubule obliteration ("plugs")
- Tubule coverage
- Prevention of transmission of irritation

Depicted here: Tubules, odontoblasts, nerve fibers, potassium ions.



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Function—Functional Therapy

The masticatory system is composed of the jaws, the temporomandibular joints, the muscles of mastication, the nervous system, the teeth with their occlusal complexes, and the periodontal ligament. These components are physiologically and morphologically synchronized when function is normal.
Within certain tolerance limits, the masticatory system can adapt to deviations from the norm (compensation, adaptation). However, if severe alterations or diseases occur in any one of the component parts of the masticatory system, the other components may also be affected (de-compensation or regressive adaptation; Bumann & Lotzmann 1999, *Color Atlas of Dental Medicine: TMJ Disorders and Orofacial Pain)*.
Functional disturbances do not elicit periodontitis! Nevertheless, such disturbances must be recognized and treated because they elicit alterations in masticatory musculature and temporomandibular joint function as well as excessive amounts of tooth abrasion, increased tooth mobility and the progression of any existing periodontitis (Ramfjord & Ash 1983). This chapter will present:

- Normal function – Physiologic tooth mobility
- Disturbed function – Occlusal periodontal trauma
- Therapy for functional disturbances: Bite guards

Normal Function

The complexity of the stomatognathic system makes it difficult to define the term "normal function." It is only possible to describe physiologic data and norms for the individual components:

Force

Normal chewing force is dependent on the type of food consumed. When eating pudding or puree etc., chewing force is quite low as measured in Newtons (1 Newton = 1 kg x m/s^2 = ca. 100 p), while chewing tough meat, ca. 150 N (ca. 15 kp); even with very hard foodstuffs, chewing force seldom exceeds 200 N (Ammann 1980).

Duration

The "normal" temporal loading of the periodontium is short. The occlusal forces that are imposed upon the periodontium during chewing and swallowing are *intermittent* in character. The duration of a chewing maneuver is only about 0.1 – 0.4 seconds. During swallowing (even of saliva) the periodontium is loaded for only ca. 1 second. If these maneuvers are summed over a 24-hour period, it is revealed that only 15–20 minutes of peak loading occurs per day (Graf 1969).

Direction

The "normal" direction of forces that are imposed upon the periodontium during chewing is extremely variable. The ideal direction of loading would be vertical/axial because this would ensure that all periodontal fibers and the alveolar process would be loaded evenly. Such equal loading does not occur even in centric occlusion. During chewing, on the other hand, alternating and combined forces are exerted primarily from the horizontal/orofacial and vertical directions (Graf et al. 1974, Graf & Geering 1977).

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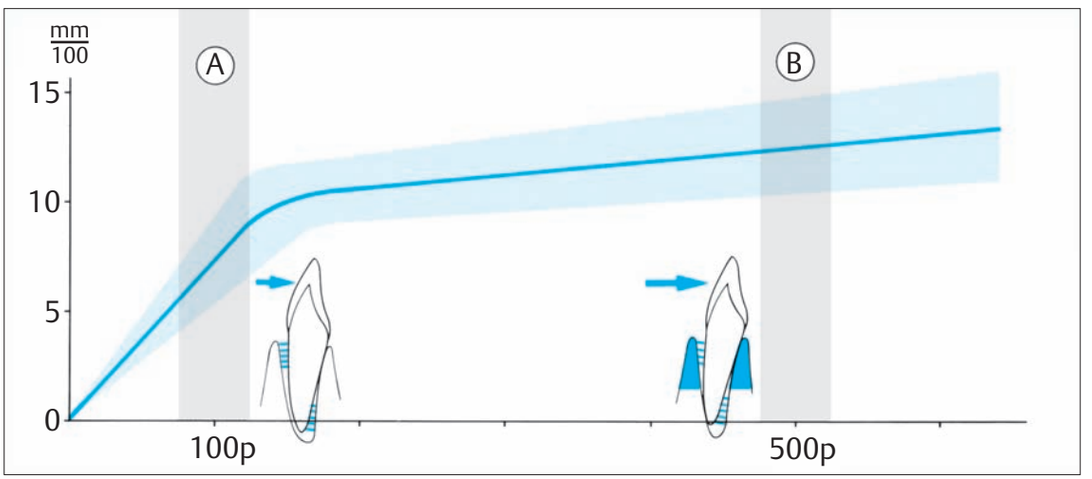
Physiologic Tooth Mobility

The "syndesmotic mechanism" by which the teeth are supported within their alveoli, and the elasticity of the entire alveolar process provide for a measurable physiologic tooth mobility in horizontal, vertical and rotational directions (Periodontometer, Mühlemann 1967; "Periotest," Schulte et al. 1983).

Tooth mobility varies in daily as well as larger cycles (biorhythm): Teeth are more mobile in the morning than in the evening (Himmel et al. 1957). In addition, each type of tooth exhibits characteristic mobility depending upon the *surface area* available for insertion of periodontal ligament fibers into cementum, and also according to root number, root length and root diameter (Fig. 1130).

Increased Tooth Mobility

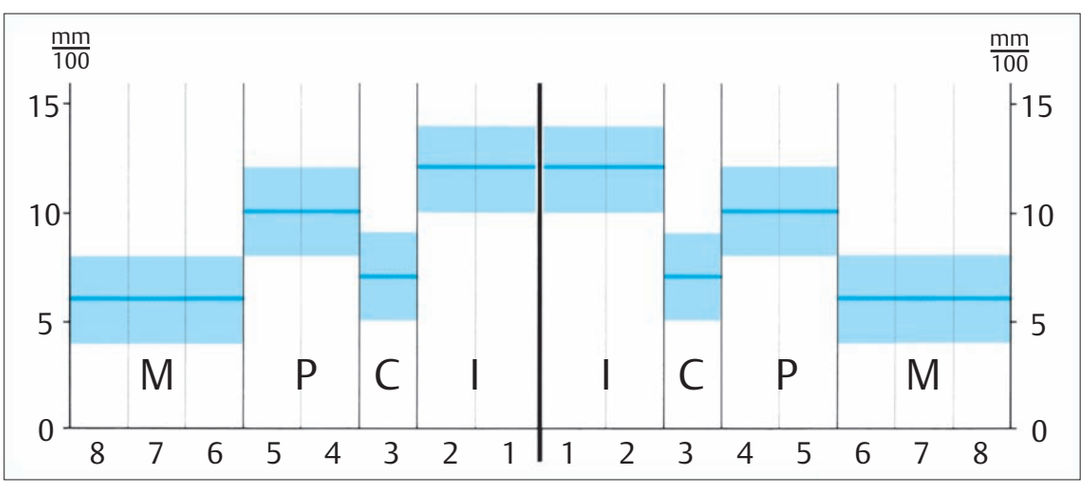
Elevated tooth mobility may result from occlusal trauma or quantitative bone loss due to periodontitis (Figs. 1131/1132). Increased tooth mobility *alone* is not a criterion of periodontal health or disease.



1129 Physiologic Tooth Mobility with Increasing Force
Force N = Newton, 1 N = ca. 100 p

A Initial Periodontal Tooth Mobility
Upon *light* loading in the orofacial direction with 1 N (ca. 100 p), only the collagen fibers of the periodontal ligament are stretched.

B Secondary Periodontal Tooth Mobility
Loading with 5 N (ca. 500 p), the entire alveolar process (blue) is elastically deformed.



1130 Healthy Periodontium: Mean Values of Tooth Mobility for Various Tooth Types
The values depicted here represent *mean mobility figures* following application of a constant heavy force in the orofacial direction (5 N, periodontal tooth mobility). These values would be TM = 0 in clinical charting (p. 174).

I Incisors
C Canines
P Premolars
M Molars

Initial Periodontal Tooth Mobility (A)

This is defined as the first phase of movement of a tooth after loading at 1 N (ca. 100 p) in the orofacial direction. The tooth moves relatively easily within its alveolus. Some periodontal ligament fiber bundles are stretched, others relaxed, but significant deformation of the alveolar process does not occur.

Initial tooth mobility is relatively high: It is a function of the width of the periodontal ligament space and the histologic structure of the periodontium; depending on the tooth type, it can range from 0.05 –0.10 mm.

Secondary Periodontal Tooth Mobility (B)

This is measured after application of 5 N in the orofacial direction. At this magnitude of force, delivered via the stretched periodontal ligament fibers, the entire alveolar process is deformed and this resists additional deflection of the tooth.

Variations in periodontal tooth mobility in a healthy periodontium depend upon the mass and quality of the alveolar bone. The normal periodontal tooth mobility ranges from 0.06 to 0.15 mm, depending upon tooth type.

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Occlusal Periodontal Trauma

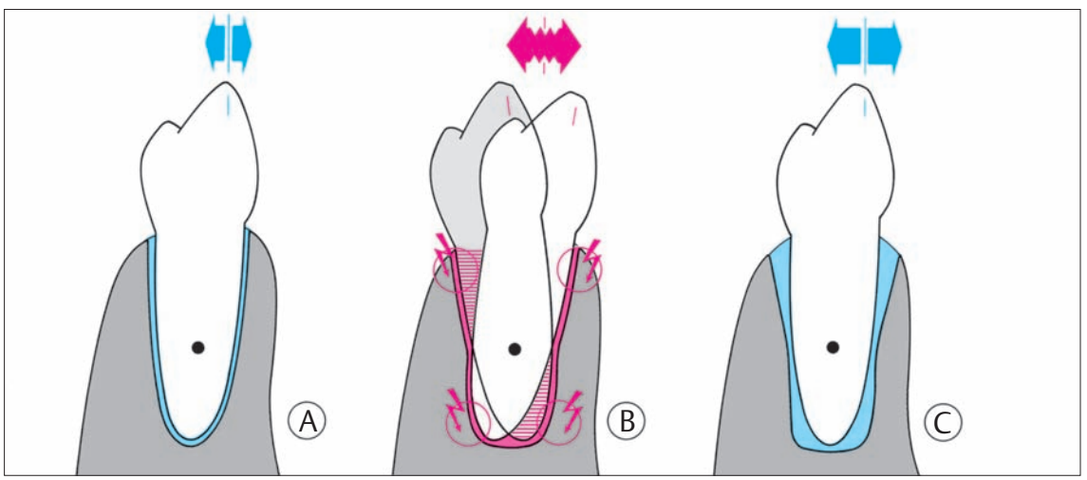
Definition

Occlusal trauma is defined as "microscopic alterations of periodontal structures in the area of the periodontal ligament, which become manifest clinically in (reversible) elevation of tooth mobility" (Mühlemann et al. 1956, Mühlemann & Herzog 1961).

That an excess of pro-inflammatory mediators could increase occlusal trauma is suspected, but has not yet been proven.

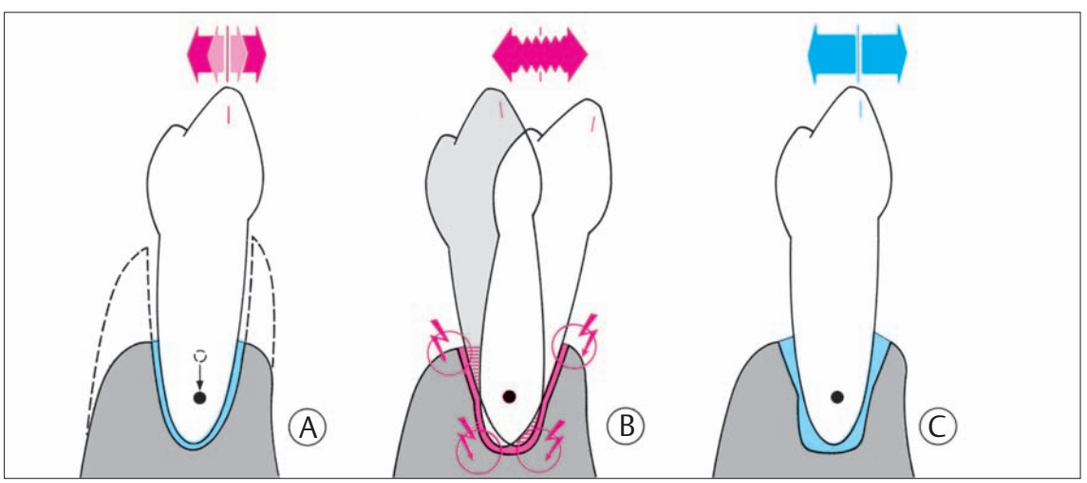
Consequences

Occlusal trauma causes *histologic alterations in the periodontium*: Circulatory disturbances, thrombosis of PDL vasculature, edematisation and hyalinazation of collagen fibers, inflammatory cell infiltration, nuclear pyknosis of osteoblasts, cementoblasts and fibroblasts, as well as increases in vasculature diameter (Svanberg & Lindhe 1974). The periodontal ligament space *adapts* by becoming wider ("hourglass shape"), and this is manifested clinically by elevated mobility and radiographic evidence such as triangulation.



1131 Normal Periodontium—Primary Occlusal Trauma

A Healthy periodontium.
B If this tooth is unphysiologically loaded (parafunctions—jiggling), histologic changes will occur in the periodontium (red) and the PDL space becomes wider in areas of pressure (red arrows).
C The periodontal tissues can *adapt* to improper loading. The PDL space assumes an hourglass shape, which *permits* elevated tooth mobility.



1132 Compromised Periodontium—Secondary Occlusal Trauma

A Elevated TM due to periodontitis.
B Even slight parafunctional loading will lead to additional elevation of tooth mobility ("secondary occlusal trauma"), leading to progressive tooth mobility.
C An adaptation with elevated tooth mobility may also occur.

Clinical experiments have proved that abnormal occlusal forces cause neither gingivitis nor periodontitis. However, the progression of an already existent periodontitis may be accelerated, especially during active stages of disease (Svanberg & Lindhe 1974; Polson et al. 1976a, b; Lindhe & Ericcson 1982; Pihlstrom 1986; Hanamura et al. 1987).

Adaptation to Unphysiologic Forces—Adaptive Phase

Even without treatment, the periodontium may adapt to long-term occlusal trauma. The PDL space remains wide, but retains its normal histologic appearance. *Tooth mobility* may remain elevated, but does not increase in severity (Nyman & Lindhe 1976).

Progressive Mobility with Unphysiologic Forces (Lack of Adaptation)—Progressive Phase

Heavy, continuous, abnormal occlusal forces may lead to progressively *increasing* tooth mobility, and function may be negatively influenced. The elimination of the *causes* of the trauma is then of primary importance.

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Occlusal Bite Guard—The Michigan Splint

When parafunctional habits (e.g., bruxism) result in occlusal periodontal trauma (increasing tooth mobility) or to progression of periodontitis, the dentition should undergo selective occlusal grinding and wear facets should be eliminated.

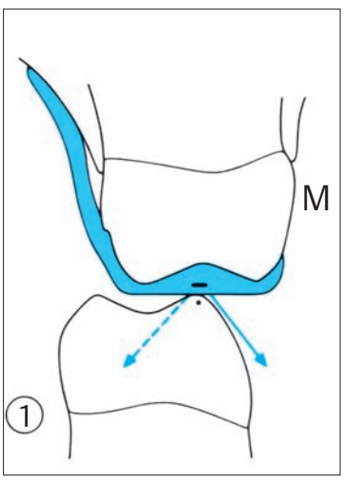
However, selective grinding is often impossible due to spasm in the masticatory musculature. Reciprocal functional relationships exist among occlusion, the periodontal ligament, TMJ, masticatory musculature, and the central nervous system (CNS). The activity of the CNS may also be significantly influenced by psychic factors (Graber 1985).

Such central nervous system hyperactivity is relieved through elevated tone within the masticatory musculature (clenching or possibly also bruxism). In such situations, if occlusal disharmonies are also present, a "vicious circle" is created which can best be broken through use of an occlusal bite guard, e.g., the Michigan splint (Ramfjord & Ash 1983). The result is that the occlusion is taken out of the "circle" and the masticatory musculature relaxes. In most cases, selective occlusal grinding can then be accomplished after only a few weeks (removal of premature contacts and/or elimination of balancing side interferences).

1133 Michigan Splint (Maxilla) in the Articulator
The removable bite guard is fabricated following registration of the occlusal relationships in an articulator; minimal bite elevation (splint thickness) is the goal.

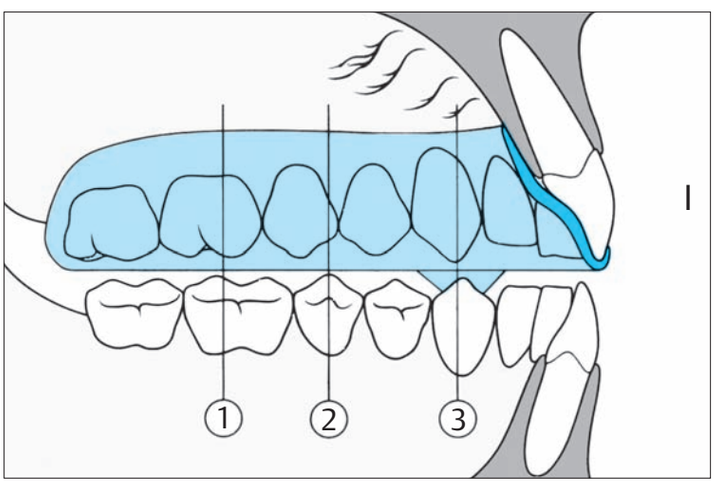
Right: Transverse section (**1**) in the molar region where the cusps are relatively flat (**M**). In this region, the splint can be flat. During lateral excursions (working and balance side, blue arrows) contact is lost almost immediately due to canine guidance.

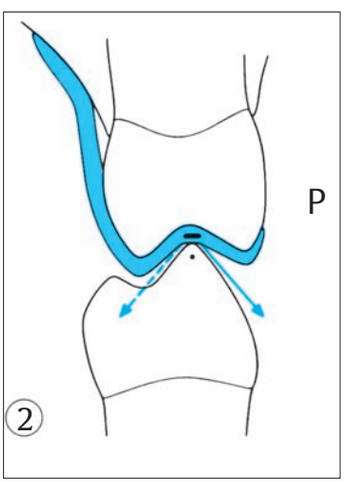




1134 Characteristics of the Michigan Splint
All buccal cusps of posterior teeth as well as *canines* and *incisors* (**I**) of the mandible occlude on the smooth acrylic surface of the splint. Transverse sections **1, 2, 3**.

Right: In section **2**, the steep cusps of the *premolars* (**P**), the profile of the splint is indented in order to prevent excessive bite elevation. Nevertheless, even here during lateral excursion (arrows) the working and balancing contacts must be immediately lost.

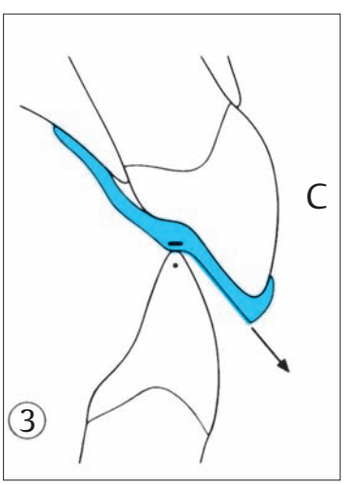




1135 Michigan Splint—Removable Bite Guard
The *occlusal contacts* between the splint and the buccal cusp tips of mandibular teeth are marked in red, and the *guidance pathways* for the guiding mandibular canines are shown in green.

Right: Section **3**. In the canine region (**C**) a "cuspid rise" is incorporated: This steep incline provides the sole guidance of the mandible during *lateral and protrusive movements*.





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Orthodontics

Periodontal and Esthetic Corrections

Anomalies of tooth position in a periodontally compromised dentition can be characterized as:

- Anomalies that have existed since tooth eruption
- Anomalies that have resulted from periodontitis (or functional disturbances)

A direct, causal relationship between existing dental malocclusion and periodontitis is difficult to document (Hug 1982, Zachrisson 1997, Ong et al. 1998, Thilander 1998). Dental crowding always makes plaque control more difficult. This leads to increased plaque retention, the consequence of which is increased gingival inflammation (Fig. 248).
But tooth migration must be differentiated from other anomalies; it only occurs over the course of time, and not seldom as a symptom/result of periodontitis. These sorts of intraoral manifestations frequently create esthetic problems for the patient, including:

- Protrusion of the maxillary anterior teeth
- Tooth elongation
- Torsion
- Tooth tipping

endo

Etiologies

The factors involved in the etiologic development of dental malocclusion following loss of periodontal support are not always obvious. Possible/probable causes include occlusal disharmonies (tooth tipping following tooth loss), oral parafunctions such as tongue thrust or lip biting, occupation-related peculiarities such as holding nails or pins between the teeth, the playing of wind instruments etc. It has also been shown that not only the granulation tissue present in a deep periodontal pocket may exert a force that causes drifting, but that intact supracrestal fibers on the "healthy" side of a tooth may exert a pull resulting in tooth migration.

Treatment planning

Periodontal therapy must be completed before any orthodontic treatment is begun. The advantages and disadvantages of orthodontics must be considered. Is the treatment motivated by functional or esthetic concerns? Could the problem be solved by other means, e. g., by functional, morphologic or esthetic odontoplasty, or through prosthetic means?

Apparatus

If the decision is made to proceed with orthodontic therapy, depending upon the diagnosis, goal of treatment and difficulty of the proposed therapy, the myriad of therapeutic possibilities include simple wire ligatures, removable apparatus and fixed brackets on the facial/buccal or even – almost invisibly – on the oral/lingual aspect of the dentition. The choice will depend in many cases on the desires of the (adult) patient. Patients exhibiting difficulties in adequate plaque control may also lead to compromises (e. g., removable devices).

Risks

In a dentition with periodontitis, orthodontic treatment always represents an additional trauma for the remaining tooth-supporting structures. Tooth mobility increases in virtually every orthodontic case. This is fully comparable to the situation following occlusal trauma. Following orthodontic therapy, long-term retention is always required in a periodontally compromised dentition, at least in the form of semi-permanent splinting (p. 473) or a night guard.

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Maxillary Anterior Segment Space Closure Following Periodontal Therapy

A 46-year-old female suffered from chronic and advanced periodontitis. In addition to bacterial infection (plaque), the etiology included severe psychic stress for family reasons, and the patient was overworked and weary.

The functional analysis revealed premature contacts between teeth 13, 14 and 45, 44, with a slight deviation from the left side anteriorally; this caused the maxillary anterior teeth to be mildly traumatized. Figures 1136 and 1138 depict the clinical view *after* initial periodontal therapy and subsequent surgical treatment.

Figure 1137 shows the probing depths and radiographic appearance before any periodontal treatment. Initially the clinical values were

PI: 70% BOP: 69%

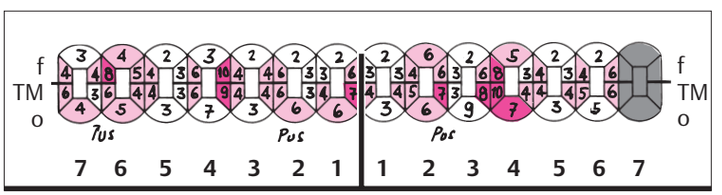
During the periodontal therapy, protrusion and diastemata occurred and these bothered the patient. She desired esthetic improvement of her situation. Orthodontic treatment and conservative restorative therapy were indicated.

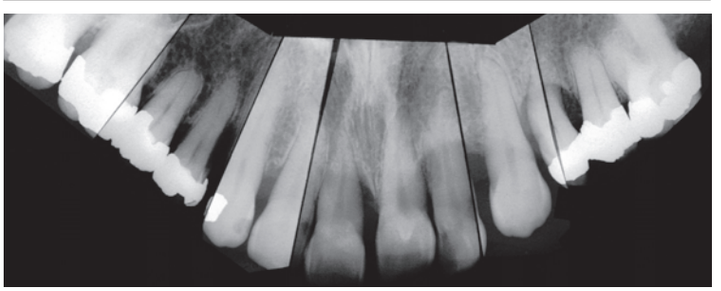






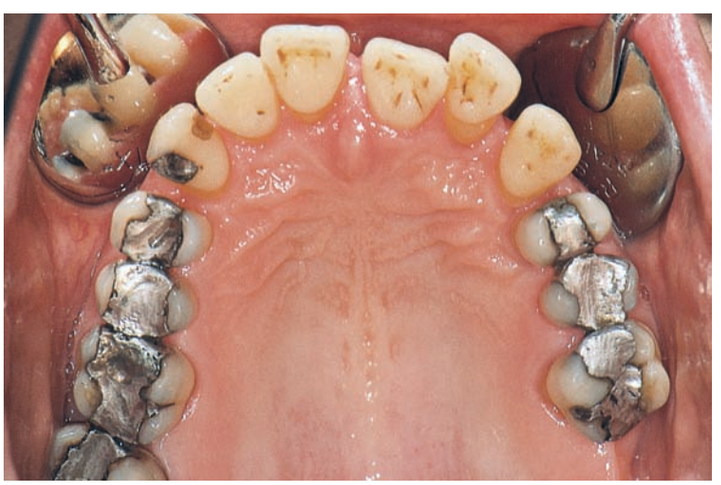
1136 Clinical View *after* Periodontitis Therapy (Above)
The periodontal treatment essentially eliminated the pockets (initial findings). However, morphology and texture of the gingiva remain less than optimal.





Before

1137 Probing Depths and Radiographic View *before* Periodontal Therapy (Right)
Probing depths in the maxilla were particularly deep in the interdental and palatal areas. Note deep pockets on teeth 14, 11, 22, 23 and 24.



1138 Clinical Picture of the Maxilla *after* Periodontitis Therapy, Occlusal View
The gingiva exhibits essentially healthy relationships. The unharmonic arch configuration and the old restorations will be subsequently corrected and replaced.

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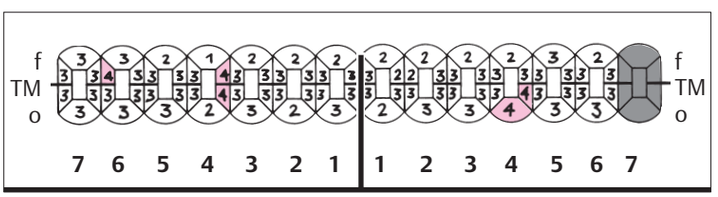
1139 Orthodontic Apparatus in the Maxilla
The treatment was performed using the segment-arch technique. At the time of this photograph, the treatment was nearing the final phase. The active orthodontic treatment required ca. 14 months.



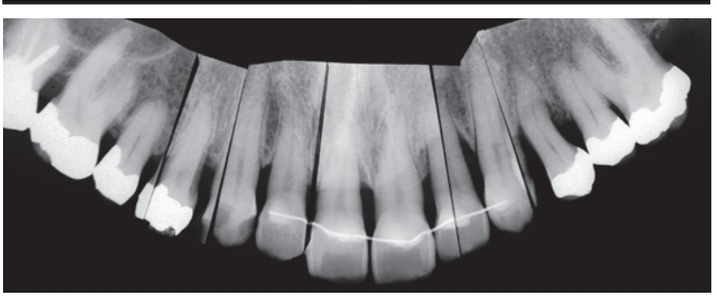




After



1140 Clinical View Two Years after Completion of Treatment (Above)
Periodontal health and optimum esthetics.



1141 Probing Depths and Radiograph Appearance Two Years after Treatment (Left)
At most sites, the probing depths were reduced to physiologic levels. Several residual pockets of 4 mm remained. The radiograph shows stable relationships; in some areas, new formation of cortical bone.



1142 Occlusal Clinical View of the Maxilla Two Years after Completion of Treatment
The maxillary anterior teeth were maintained with a long-term retentive splint. The total conservative treatment was competed on all teeth. The gingivae exhibit healthy conditions.

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Uprighting the Mandibular Second Molar

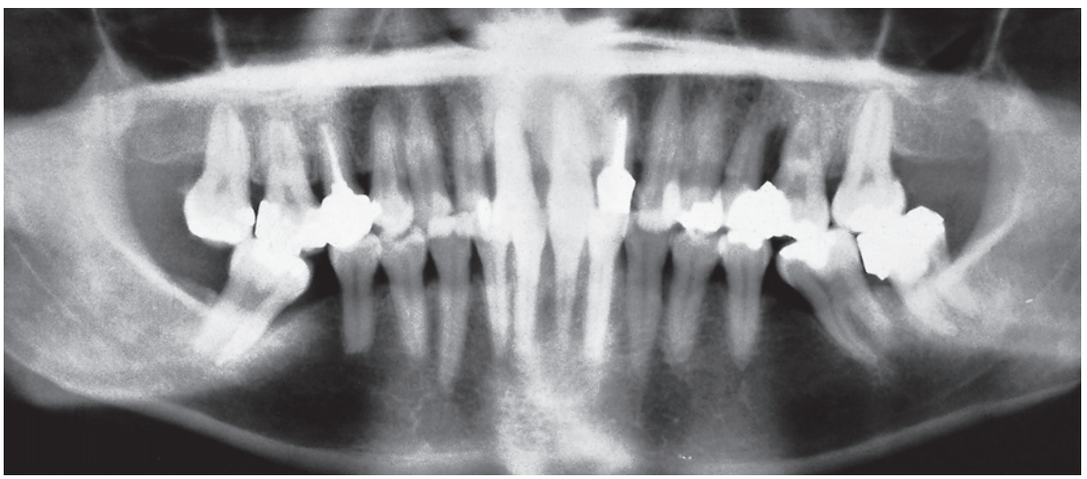
It is not necessary to prosthetically close every space in the dental arch. If the occlusion is stable and there is adequate periodontal/anatomic support, no drifting of the adjacent teeth should occur.

However, in a periodontally compromised dentition, tipping of a second molar into a first molar extraction site is common. The mesial tipping of the second molar leads to a plaque-retentive niche, which promotes the development of a deeper periodontal pocket (Ericsson 1986).

This was the case presented by a 48-year-old female with moderate periodontitis.

Tooth 38 was extracted during initial therapy, for endodontic reasons. Both mandibular second molars were uprighted during a 7-month period. The tooth movement was accomplished with an appliance incorporating a bite plane extending distally to the bicuspid area, a labial bow, and activatable uprighting springs for the molars.

Instead of the two fixed bridges, one might have considered dental implants to replace the missing first molars (Fig. 1145).



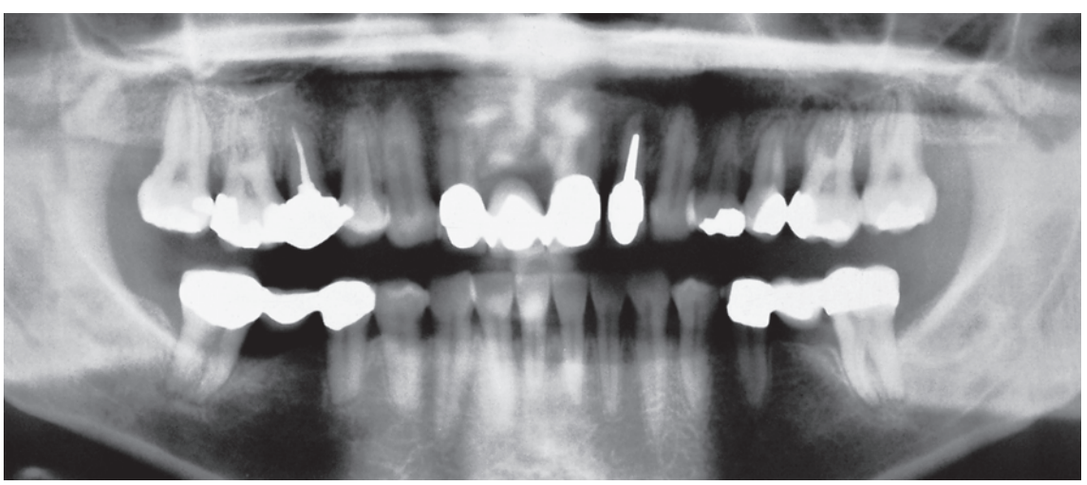
1143 Initial Panoramic Radiograph—Mesially Tipped Mandibular Molars
Following (justified?) extraction of the first molars years ago, the second molars had tipped severely into the spaces, forming treatment-resistant pockets (plaque-retentive niches). Whether or not dysfunctional occlusal loading of the tipped teeth is in any way responsible for the formation of mesial periodontal pockets has not been proven.
Tipped molar teeth often present balancing side interferences.





1144 Removable Appliance—Lateral Views
Mandibular appliance of clear acrylic incorporating an anterior bite plane, labial bow and uprighting springs.

Opening the bite was necessary to eliminate occlusal interferences on the molars during uprighting. In this case, it might have been possible to fabricate a somewhat less clumsy appliance!



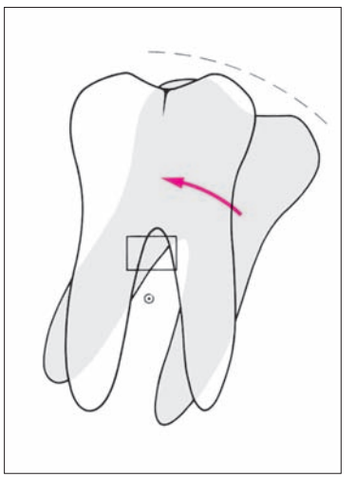
1145 Panoramic Radiograph after Treatment—Uprighted Mandibular Molars and 3-Unit Bridge Constructions Bilaterally
Tooth 38 was extracted and the two lower second molars were uprighted.
The two 3-unit bridges were seated immediately, making any additional orthodontic retention unnecessary. Definitive periodontitis treatment can now proceed. Effective plaque control by the patient is possible.

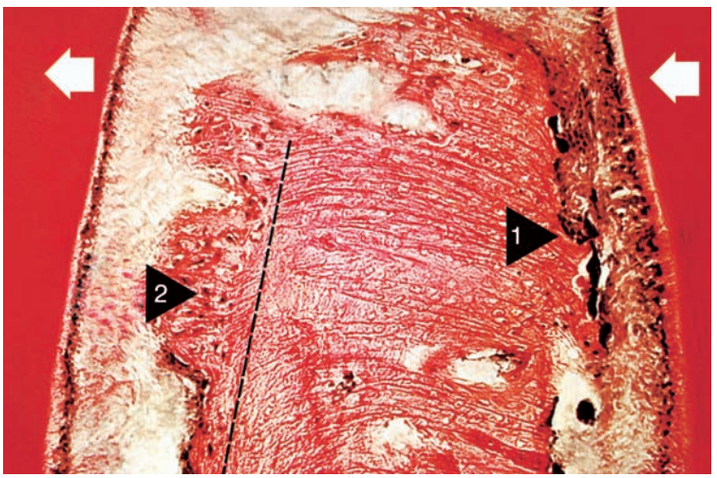
Courtesy *P. Stöckli*

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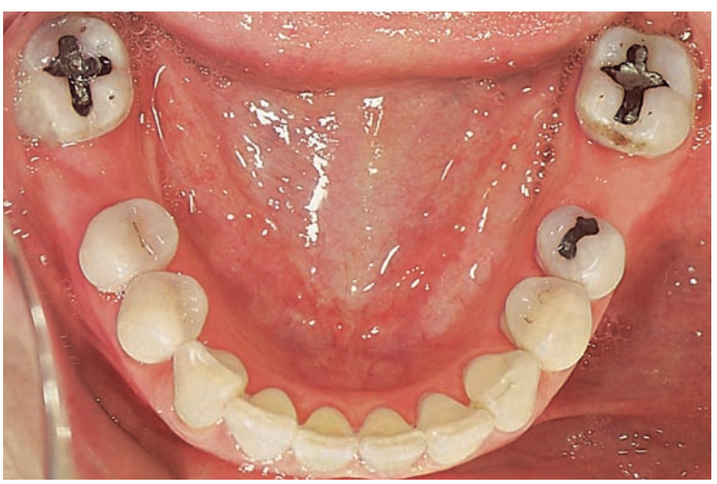
1146 Initial Occlusal View
The second molars (47 and 37) are severely tipped mesially. The tipping has narrowed the extraction sites, making adequate oral hygiene difficult and promoting plaque retention. Pockets exist mesial to both second molars.



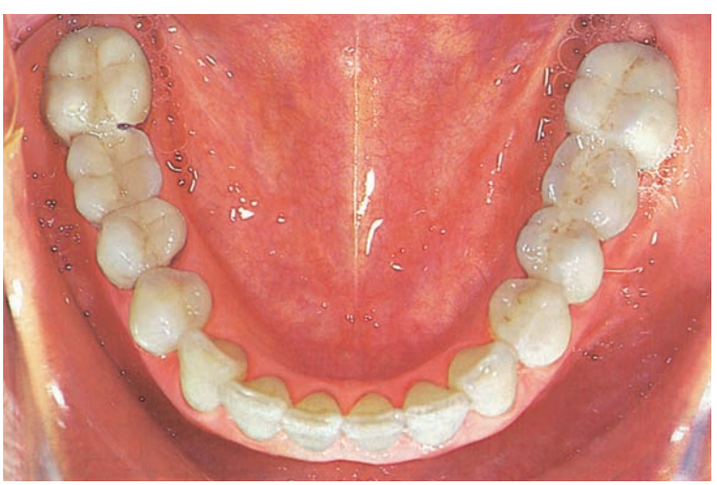


1147 Bone Deposition and Resorption During the Orthodontic Treatment
The histologic picture shows the region of the bifurcation (white arrows = direction of movement). In the zone of pressure (black arrow 1) one observes osteoclastic bone resorption, and in the tension zone (black arrow 2) bone apposition (van Gieson, 25x). Histology courtesy of *N. Lang*

Left: Molar uprighting occurs around a point of rotation between the roots.



1148 Uprighting of the Mandibular Second Molars, Complete
The widened interdental spaces are clearly obvious (cf. initial view, Fig. 1146).
The buccal wall of tooth 47 has fractured (not due to treatment). For this reason, bridgework rather than dental implants was selected to close these spaces.



1149 Space Closure—Retention
Fixed bridgework maintains the uprighted molars and affords ideal replacement of the missing teeth.
The interdental spaces of the abutment teeth and bridge pontics were contoured to permit and facilitate the use of interdental brushes by the patient. Optimum oral hygiene is possible.

Courtesy *P. Stöckli*

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Maxillary Anterior Esthetic Correction Following Periodontitis Treatment

A 56-year-old female complained of esthetic problems in the maxillary anterior segment (tooth migration and mild gingival recession, "long teeth"). A small diastema had been present even at age 20, but had become much wider during the previous ten years. This led the patient to seek possibilities for space closure and resultant esthetic improvement.

The patient was unaware of any other gingival problems. She smoked ca. 10 cigarettes per day, but stopped smoking during the treatment phase. Despite the relatively high plaque index, the gingiva did not exhibit significant inflammatory symptoms. In the maxillary anterior region, pocket probing depths up to 7 mm were detected. Tooth 21 exhibited significant attachment loss up to two-thirds of the root length.

PI: 68 % BOP: 58 % TM: see Fig. 1151

Following periodontal therapy, a "simple" orthodontic treatment was planned for the maxillary anterior region.

1150 Initial View before Periodontal Therapy
Moderate, chronic periodontitis with profound osseous defects and migration of teeth 11 and 12. Mild gingival recession on all maxillary incisors and both canines. The magnitude of plaque accumulation and gingival bleeding was not necessarily expected upon initial clinical observation.

Right: Pronounced attachment loss on tooth 11 and especially on tooth 21.



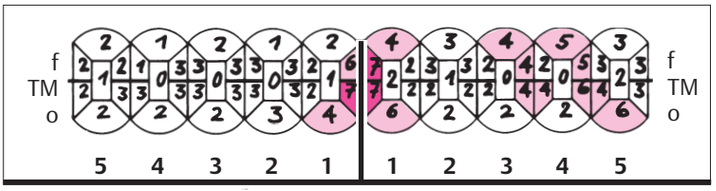


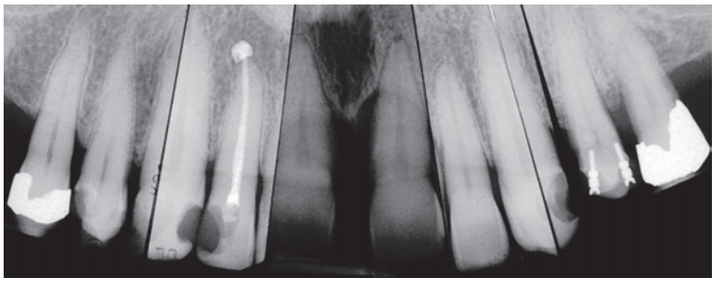
1151 Pocket Probing Depths and Radiographic View
Pockets of ≥ 6 mm were observed in the maxillary incisor, canine, and premolar regions only on teeth 11, 21, 24 and 25.

Radiographic view: The films clearly reveal the especially pronounced attachment loss between the two central incisors.

Additional findings:

- PI 68 %
- BOP 58 %





Initial Findings

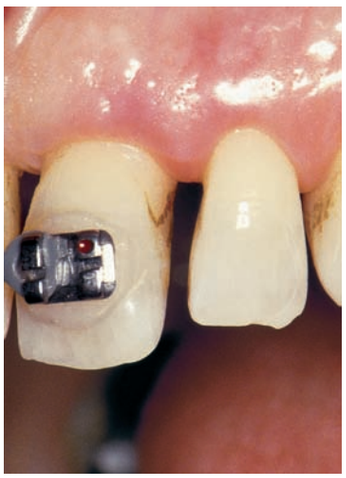
1152 After Initial Periodontal Therapy
Following supra- and subgingival debridement, there was shrinkage of the marginal gingiva, and in the depth of the pockets some "repair" (p. 206). The probing depths were reduced to 4 mm or less.

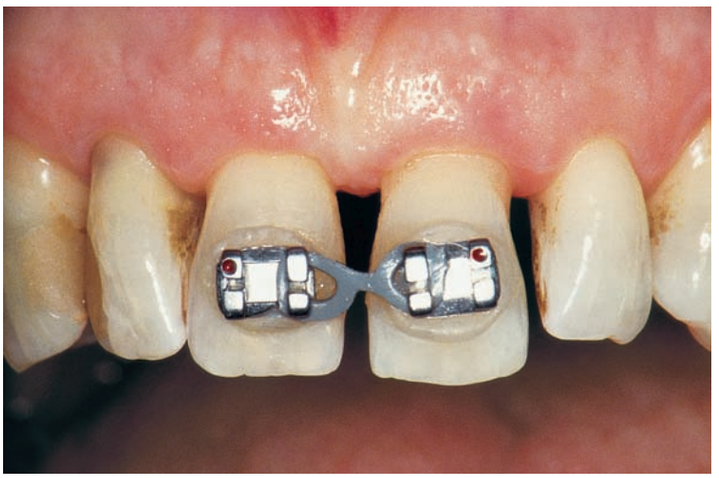
Right: Set-up of the treatment plan: Diastema—"distribution." Movement of tooth 21 mesially (1.5 mm). Closure of the remaining interdental gap by "additive" measures using composite filling materials.





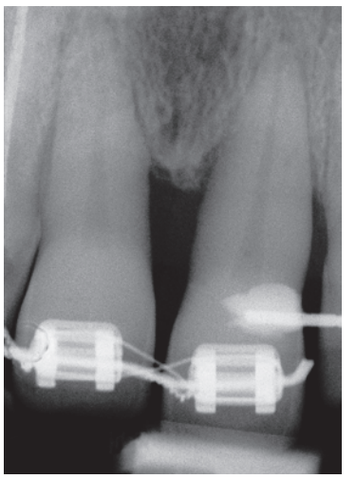
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1153 Active Phase of Orthodontic Tooth Movement
The ca. 3 mm wide diastema between teeth 11 and 21 was reduced to ca. 1.5 mm. This also created a similar 1.5 mm wide diastema between teeth 21 and 22.

Left: Note the new diastema between teeth 21 and 22 occasioned by the orthodontic mesial movement of the central incisors.

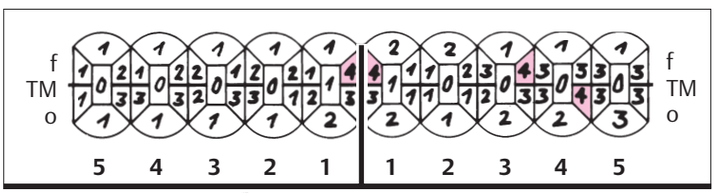


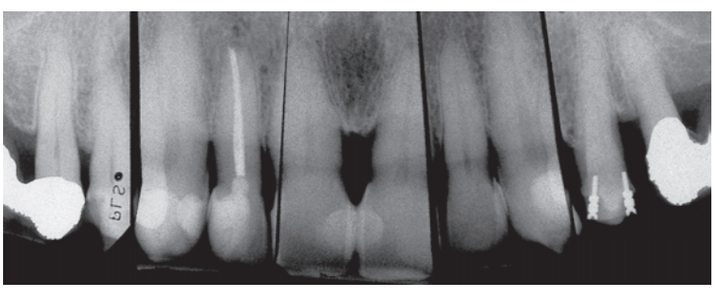


1154 Retention Phase—5 Months after active Orthodontic Treatment
Teeth 21, 22 and 23 are retained in their new positions using a simple arch wire affixed with composite resin. Tooth 11 is "supported" against tooth 12 using composite resin. Orthodontic retention of teeth 11 and 21.

Left: Radiographic taken during the retention phase.
In the apical region of the defect between teeth 11 and 21, new bone formation can be observed.

Final Results





1155 Probing Depths and Radiographic Findings 7 Years (!) after Treatment
The patient is seen in recall every 6 months. The situation is stable. Only a few pockets exhibit probing depths of 4 mm.

Radiographic view: After consolidation of the attachment niveau post-therapy, stable relationships have been maintained over the years.

- PI < 10 %
- BOP < 10 %





1156 Clinical View 7 Years later
Composite resin was used to splint 11 and 21 while simultaneously widening them. Tooth 21 was shortened. "Creeping attachment" alleviated the recession somewhat. The papillae between teeth 12 and 11 as well as between 21 and 22 regenerated.
Left: After removal of the brackets 7 years previously and before shortening of tooth 21 and composite resin treatment of the central incisors.

Courtesy *J. Hermann*

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Treatment of the Malpositioned Canine

A 14-year-old female was referred because of extremely severe gingivitis and malpositioning of the maxillary right canine, which caused a plaque-retentive niche. This young girl's oral hygiene was miserable. The patient proved extremely difficult to motivate. The erythematous, edematous, swollen gingiva bled at the slightest touch. Following initial periodontal therapy, a removable orthodontic appliance was selected in favor of a fixed appliance because of the unreliable patient compliance. Fixed orthodontic apparatus collects plaque biofilm readily and demand a higher level of oral hygiene from the patient.

A removable appliance can easily be cleaned when removed from the mouth, and also permits improved oral hygiene of the teeth. Financial considerations may also be a factor in the selection of fixed or removable orthodontic appliances.

PI: 87 % BOP: 80 %
Pseudopockets up to 7 mm

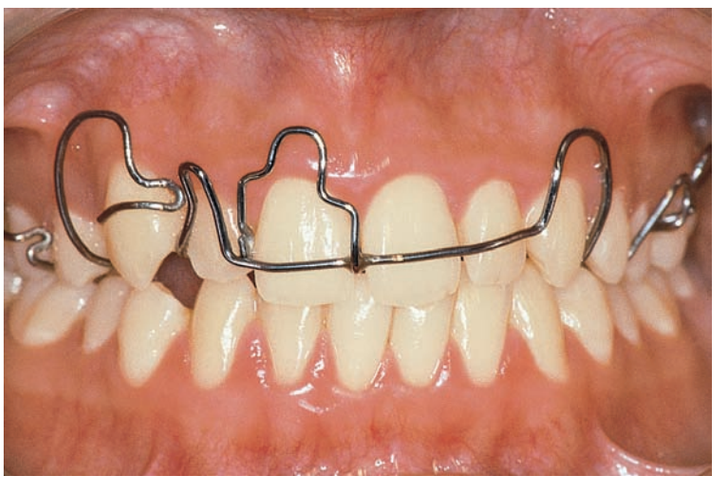
During orthodontic therapy, the patient's oral hygiene improved following repeated OHI and motivation.





1157 Initial View—Malpositioned Canine, Severest Gingivitis
The plaque-disclosing solution revealed the enormous amount of plaque on all teeth. The severely malpositioned canine in combination with crowding created numerous plaque-retentive niches. Gingivitis was severe and pseudopockets were ubiquitous.

Right: Detail of 13, 12. When the gingiva was reflected, the massive subgingival plaque accumulations were visible.



1158 Removable Orthodontic Appliance
Following initial periodontal therapy, a palatal plate with a labial arch wire, guidance stops for tooth 11 and elastic facial springs for pulling the canine (13) into alignment.
The alignment of the canine could only be achieved following extraction of the premolar (14). The orthodontic treatment lasted about 1 year.





1159 Final Result 3 Years after Initiation of Therapy
The orthodontic result is acceptable. The patient (now 17 years old) became much more compliant and performed homecare effectively. Disclosing solution reveals only minor plaque accumulations. Bleeding on probing was virtually absent (BOP 5 %).

Right: Bringing the maxillary right canine into line made interdental hygiene much easier (use of dental floss is depicted).

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Splinting—Stabilization

The significance, value and indication for immobilizing mobile teeth by means of permanent splinting remain controversial as a periodontal therapeutic technique. In order to clarify whether there exist any true indications for splinting, one must first consider the causes of tooth mobility:

- *Quantitative* loss of tooth-supporting structures due to periodontitis
- *Qualitative* alterations of tooth-supporting structures resulting from occlusal trauma, above all parafunctions
- Short-term trauma to the periodontium due to treatment for periodontitis
- Tooth mobility caused by accidental trauma
- Combinations of the above

Mobile teeth whose degree of mobility is not increasing (*elevated* versus *progressing* tooth mobility), generally do not require splinting. Exceptions to this general rule include accidental trauma, and the elimination of severe parafunctions using temporary *bite plates* (p. 462, "Function—Functional therapy"). Tooth mobility caused by occlusal trauma should be treated primarily by selective occlusal adjustment (Vollmer & Rateitschak 1975), i. e., premature contacts and disturbances in lateral or protrusive excursions of the mandible should be diagnosed and eliminated.

Following *selective occlusal grinding* to remove parafunctions, bruxism such as clenching and improperly targeted loading, physiologic forces are distributed onto the entire dentition. Selective occlusal odontoplasty is targeted to remove the etiologic factors of parafunctions, the "trigger" factors, e. g., premature contacts, balancing side interferences etc.

In addition to purely occlusal disharmonies, psychic components may also be partially responsible for parafunctions and therefore also for tooth mobility. A combination of local treatment such as selective grinding combined with splinting may possibly be supported by psychopharmacologic medications, but only after consultation with the patient's physician.

In recent years, splinting/stabilization of individual teeth or entire dental arches has become much more common because of the increased use of regenerative treatment methods. If the goal of treatment is not only new bone formation but also especially "new attachment," i. e., the regeneration of all periodontal structures after treatment, this will most likely occur through some minimal *temporary* stabilization of the treated (mobile) teeth (to prevent dislodgment of the coagulum from the root surface).

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Tooth Mobility and Healing

Following all types of pocket treatment, the pocket fills with blood. The fibrin net of the coagulum closely approximates the conditioned root surface. The organization of this coagulum, the formation of new periodontal structures (by periodontal ligament fibroblasts) requires several weeks. If a tooth is constantly mobile during this healing phase, the attachment (adhesins!) to the root surface will be significantly disturbed.
The demand for "stabilization" of the treated teeth is especially important after the use of regenerative techniques such as the implantation of bone and bone-replacement materials, and the GTR technique, as well as the use of growth factors and matrix proteins, and even combinations of these methods. Additional indications are illustrated below.

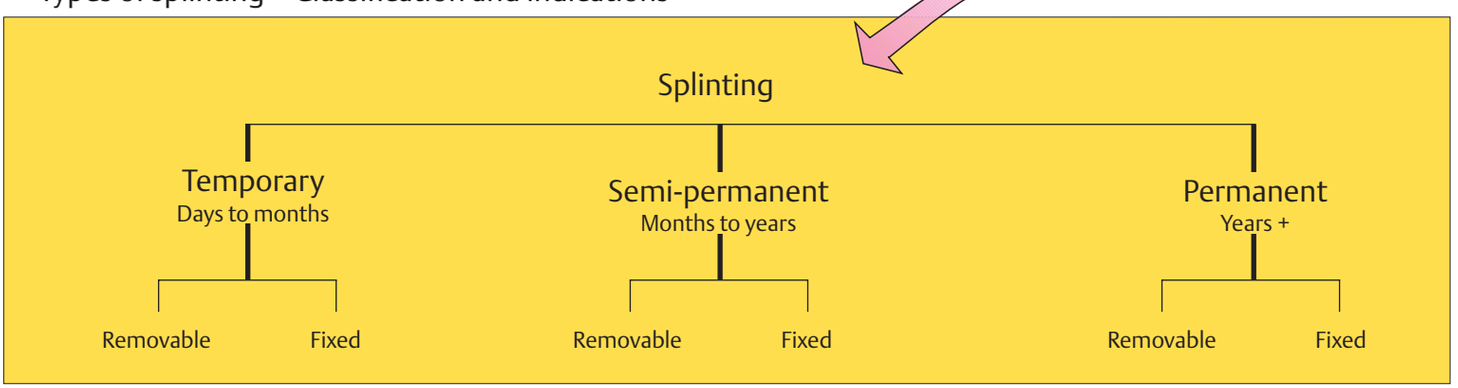
Elevated Tooth Mobility—Etiology, Treatment

The table below (modified from Flemmig 1993) attempts to define the causes and the necessary therapies for elevated tooth mobility; discussions in this regard are also dependent upon changes in the radiographic appearance of the periodontal ligament space (occlusal trauma) and clinical attachment loss.

Elevated tooth mobility (TM) – Etiology and treatment

TM Tooth mobility	PDL space	Alveolar ridge height	Etiology	Therapy
Elevated	Widened	Normal	Occlusal trauma	Occlusal adjustment
Elevated	Widened	Reduced	Attachment loss and occlusal trauma	Periodontitis therapy and occlusal adjustment
Elevated	Normal	Severely reduced	Attachment loss	Periodontitis therapy and possible splinting
Increasing severity	Increasingly widening	Reduced	Attachment loss and occlusal trauma	Periodontitis therapy + occlusal adjustment + splinting

Types of splinting – Classification and indications



Indications for Various Types of Splinting

- *Temporary or semi-permanent*: These protect against further trauma caused by occlusal and oral parafunctions (e.g., tongue pressing, "sucking"). It can be used as an emergency procedure with extremely mobile teeth. It also can serve to reduce trauma—mechanical, instrumental—during periodontitis therapy.

- *Semi-permanent/permanent*: This can enhance masticatory comfort when teeth are highly mobile; also to stabilize the teeth during periodontal healing phases, especially following regenerative therapies.
  Awaiting the long-term prognosis.
  Retention following orthodontic treatment.

- *Permanent*: Used for complex oral rehabilitation where abutments are highly mobile or where only few abutments must support the reconstruction, particularly when such abutment teeth have minimal periodontal support.
  Distribution of occlusal forces when parafunctions cannot be eliminated. In such cases, without splinting, there is a danger of progressively increasing tooth mobility and tooth migration (Nyman & Lindhe 1979).

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Temporary Splinting

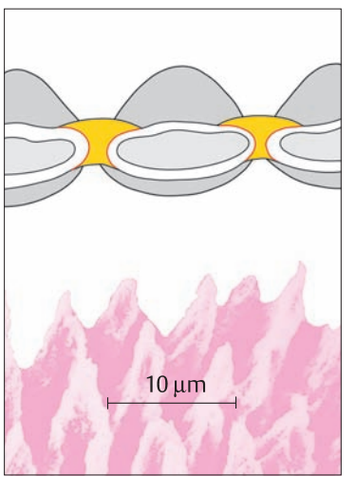
The simple wire ligature may serve as a *fixed* splint for a few days to several weeks. Wire ligatures are seldom used today, however, primarily because of the esthetic considerations. A more commonly used fixed splint is the acid-etch composite resin splint *without* tooth preparation, and sometimes with cavity preparation. Such a splint can be quickly applied with a rubber dam in place; however, it is a temporary measure because adhesion of the resin to the tooth structure is not strong enough without additional mechanical retention (cavity preparation, grooves etc.). Fracture of such splints is common if more than 3–4 teeth are included.

A *removable*, temporary splint can be made of acrylic resin pulled under vacuum, used only for a short period of retention for individual teeth.

Such splints were formerly used as a "bite guard" in the treatment of oral parafunctions. This approach proved not to be particularly effective and today the "Michigan splint" fabricated in a professional dental laboratory predominates.



1160 Wire Splint
Soft steel wire (0.4 mm diameter) is wrapped around the facial and oral surfaces of the teeth to be splinted, and the ligature/splint is tightened by twisting the ends. Stabilization of individual teeth is accomplished by application of interdental ligatures. Acid-etch resin "stops" may be applied to the labial surface to prevent the wire from sliding apically. The wire ligature/splint is used intra- and post-operatively, in most cases in combination with a wound dressing.



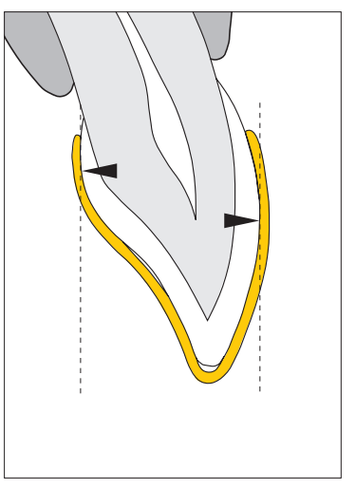


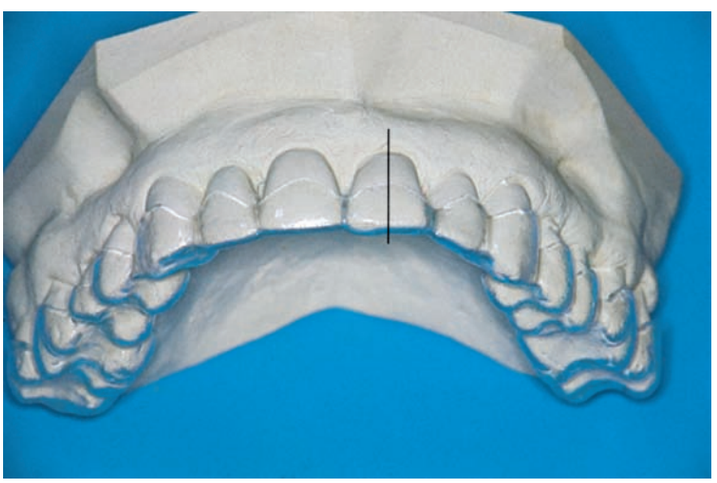
1161 Composite Resin Splint, no Tooth Preparation
Following thorough cleaning of the teeth, and under the rubber dam, the proximal surfaces are acid-etched and resin is applied. The apical region must be left open for oral hygiene.

Left: Above, incisal view of the resin splint *in situ* (red = etched enamel, yellow = resin).

Below: Etched enamel surface.

SEM courtesy *F. Lutz*





1162 Vacuum-Formed Removable Acrylic Splint
This splint can be used for short-term retention or stabilization of teeth.

Left: The border of the splint must extend beyond the height of contour of each tooth, orally and facially (black arrows) to provide secure retention.

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Semi-Permanent Splinting—Anterior Area

The most frequently employed *fixed*, semi-permanent splint in the *anterior region*, which can remain *in situ* for months or even years, is splinting via composite resin (acid-etch technique) in combination with tooth preparation. It is often possible to simply remove all of the old anterior tooth restorations and subsequently incorporate the composite resin splint. The practical procedure is identical to the placement of composite restorations with the acid-etch technique.

The splint absolutely must be prepared with the rubber dam in place: Even tiny amounts of moisture after etching the enamel and during application of bonding and composite material will weaken the splint. Light-polymerized resin is recommended for this type of splint because of its long working time.

Removable semi-permanent splints may be fabricated as cast chrome-cobalt alloy frameworks incorporating typical partial denture clasps. This type of splint is indicated today only for wearing at night, often as a retentive appliance following orthodontic or surgical therapy.

1163 Composite Resin Splint with Tooth (Cavity) Preparation
This 38-year-old female was adamant that her almost hopelessly involved maxillary anterior teeth be retained.
Following initial therapy and up until the time that the long-term prognosis for the entire dentition could be established, the mobile teeth 11, 21 and 22 had to be stabilized. Proximal (Class III) restorations were removed.

Right: Radiographic view. Pronounced bone loss.





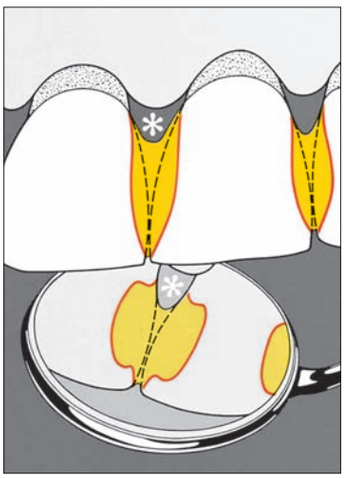
1164 Splint Application with the Rubber Dam in Place
After etching the minimally prepared enamel margins with phosphoric acid, the cavity preparations and the coronal portions of the interdental spaces are filled with light-cured composite resin, which is permitted to set and is then definitively polished.



1165 Three Years later
The interdental spaces were left open beneath the contact areas; this permitted good interdental hygiene using toothpicks and interdental brushes.

Right: Schematic depiction of the splint. Etched enamel (red), composite resin (yellow), combination of micro- (etching) and macro-retention (cavities). The asterisk (*) denotes the open interdental space (hygiene).





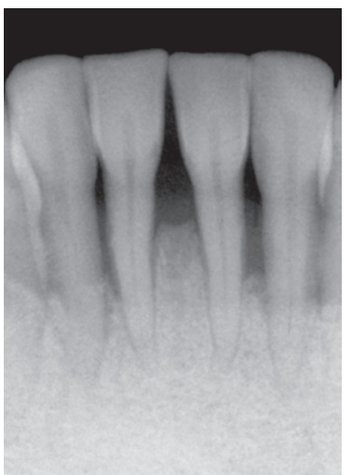
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Permanent Splinting—Adhesive Technique

Soon after introduction of the acid-etch technique for anterior restorations, the so-called adhesive bridges and adhesive splints were propagated (Rochette 1973).

In recent years, this technique had been refined. In particular, various methods for enamel preparation have been developed, that ensure adequate retention of such reconstructions following only very *conservative* tooth preparation (Marinello et al. 1987, 1988). When splinting in the anterior segments, the proximal surfaces of the teeth are rendered parallel, and fine grooves are prepared. For occlusal support, shallow, peg-shaped depressions at the margin and above the lingual tubercle are created. The incisal edges are not included, for esthetic reasons.

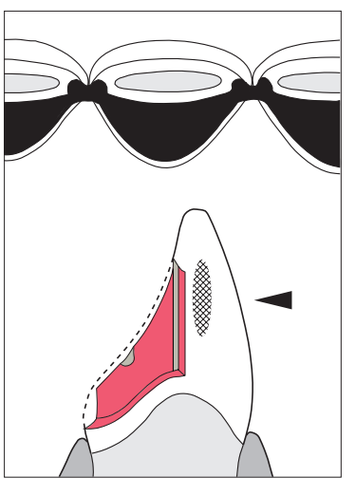
In contrast to semi-permanent splints that simply bond adjacent teeth with acid-etch resin, bridges and splints prepared with the acid-etch technique can serve for definitive immobilization of mobile teeth (cf. p. 479).

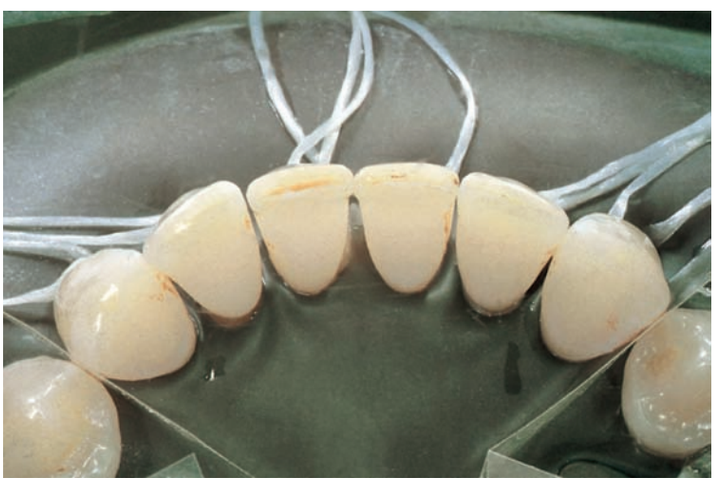




1166 Mandibular Anterior Area Following Initial Periodontal Therapy
The high degree of mobility of teeth 31 and 41 was particularly disturbing to the patient (insecurity, fear of tooth loss). To minimize the trauma of the impending periodontal therapy, and to improve patient comfort, a cast and cemented splint was planned.

Left: Radiographic findings: Particularly pronounced attachment loss between 31 and 32 and between 41 and 42.





1167 Before Seating the Splint
A rubber dam must be placed in the area of operation before acid-etching and subsequent seating of the splint. The elegant preparations (only in enamel!) are scarcely visible in this clinical picture.

Left: Preparation and splint form. The occlusal and lateral aspects depict the minimal tooth preparation. Enamel must be maintained, otherwise adhesion following acid-etching is not guaranteed, because only *bonding to enamel* is secure.





1168 After Seating the Splint with Composite Resin
Periodontitis therapy (subgingival scaling and possible surgical intervention) can be continued without compromise (time, poor temporaries etc.). The healing process can be observed over months or even years (long-term prognosis).

Left: Clinical picture three months after subgingival scaling.

Courtesy *W. Iselin*

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Prosthetic Stabilization—Long-Term Temporary

In cases of advanced, aggressive periodontitis, the stability of the periodontal treatment result must be observed over an extended period of time before seating a definitive, fixed replacement. In order to avoid any trauma to highly mobile teeth during this consolidation phase, the use of a fixed long-term temporary may be warranted (p. 480).

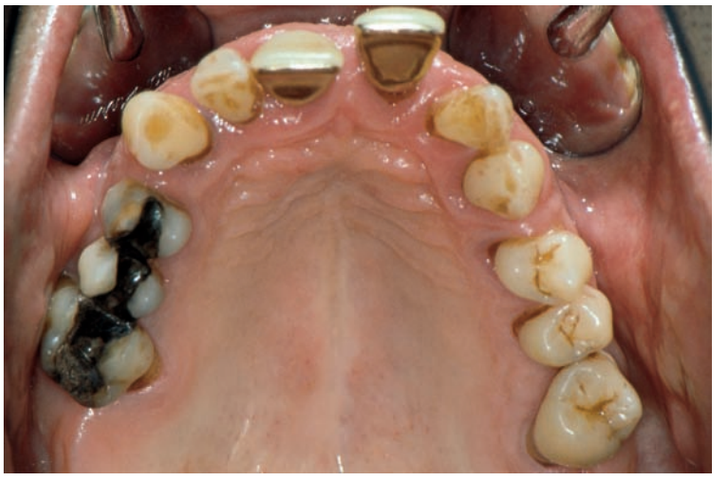
Because of dental fear, this 48-year-old male had not visited the dentist for many years. Severe tooth mobility and migration, especially in the maxillary anterior segment, and spontaneous tooth exfoliation in the mandible finally led him to our clinic. The patient was a heavy smoker and was experiencing psychological problems (divorce). In the mandible, it was necessary initially to fabricate a complete denture (dental implants later?). In the maxilla, all of his teeth were retained, with the exception of tooth 27. Following initial periodontal therapy, open root cleaning/planing was performed (in some cases with temporary wire ligature retention) and thereafter minor orthodontic corrections:

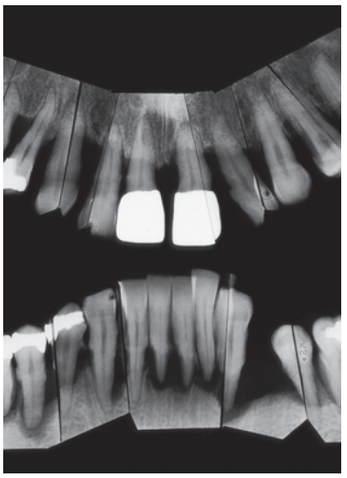
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The clinical view and radiographs are depicted. When treatment was initiated, the patient was *Aa* positive.

1169 Initial Clinical View
Inflamed gingiva, pocket probing depths up to 10 mm, irregular tooth position within the dental arch, severe tooth migration particularly of tooth 21, old and corroded amalgam restorations.

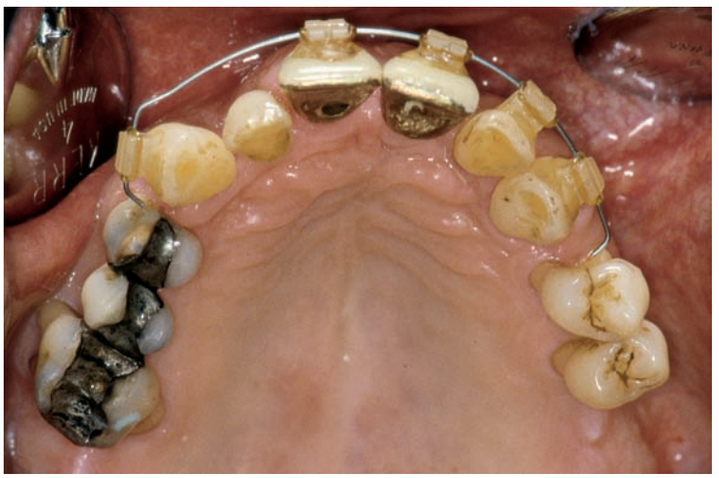
Right: Radiographic view. In the maxilla, the bone loss approached two-thirds of the root length. In the mandible, the alveolar bone was actually apical to the root tips. Tooth 34 exfoliated spontaneously.





1170 Orthodontic Treatment
The planned fixed bridgework reconstruction required a harmonic dental arch and therefore demanded minor orthodontic procedures despite advanced attachment loss; in such cases these are easily performed.

Right: During open periodontal therapy wire ligature splints were applied to reduce trauma during the surgical procedure. The patient was prescribed amoxicillin and metronidazole as systemic supportive therapy.





1171 Long-Term Temporary Bridge
Following periodontal therapy, a metal-reinforced long-term temporary bridge was seated from 13 to 23 (unsure prognosis!). The patient had stopped smoking. He was so satisfied with the "temporary," that he has not yet returned (after nine years!) to receive his "definitive" bridge.

Right: The visible metal crown margins were not an esthetic problem for the patient (who had a long upper lip and a mustache).





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Perio-Prosthetics 1 Standard Techniques

In advanced periodontitis cases it is frequently not possible to maintain all of the teeth. During the treatment planning stage, teeth that are hopeless must be identified and extracted early on, because these teeth represent bacterial reservoirs. Such teeth can enhance the microbial re-colonization of other teeth (esthetics, temporaries?).
During initial therapy or at the very latest during surgical interventions it must be determined whether additional teeth must be sacrificed. In addition to the extent of periodontal attachment loss, other factors also play a role in the decision-making process:

- Must the lost tooth actually be replaced?
- Might an exclusively premolar occlusion be satisfactory? (Käyser 1981; De Boever 1978, 1984)
- Must every missing tooth absolutely be replaced, e.g., missing first molar and stable occlusion?
- How important is the tooth as a strategic abutment for dental reconstruction?
- Are there significant endodontic problems?
- Are there inherited or genetic risk factors?
- What is the overall prognosis for the entire dentition?

Last but not least, the type of comprehensive dental reconstruction is less important than the ultimate maintenance or rebuilding of masticatory function, speech and (more and more today) esthetics; in short, the oral/dental satisfaction of each individual patient.

This chapter, *Perio-prosthetics 1/Standard Techniques* presents the following themes:

- Temporaries—removable or fixed?
- Adhesive bridges as long-term temporaries
- Fixed temporaries—problem zones
- Fixed acrylic temporaries—practicality
- Fixed, metal-reinforced, long-term temporaries—case report
- Definitive fixed reconstruction—complete bridgework
- Molar loss—what now?
- Telescope constructions in a highly compromised remaining dentition
- Cast metal prostheses—“social solution”

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Temporaries—Removable or Fixed?

If teeth are missing, or if *during* periodontal therapy hopeless teeth must be extracted, it is often necessary even during the treatment, especially for esthetic and/or functional reasons, to provide temporary replacement of the missing teeth. This type of temporary replacement must be "periodontally friendly." The temporary must in no way negatively influence the periodontal healing processes, and must not traumatize the remaining adjacent teeth.

Removable and fixed temporaries are quite different, but the latter should be selected whenever possible. These temporaries must sometimes be worn for long periods of time (weeks, months). During such time, the periodontal healing and consolidation of the treated remaining teeth occurs, and their value as abutment teeth for a definitive restoration can be determined.

Following comprehensive periodontal treatment, during the tissue maturation phase, changes in the morphology of the gingival margin are common. This can be very important and of *significance for the preparation of an abutment tooth* for a fixed bridge or a telescope construction (supra-/subgingival location of the crown margins; esthetics).

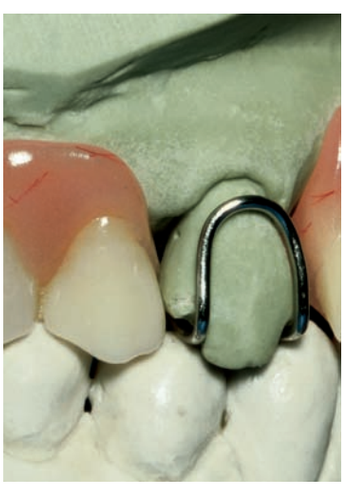
The following cases and attendant difficulties will be presented:

- Removable immediate temporary
- Adhesive bridge in the mandible—"long-term temporary"
- Fixed temporary—problem zones
- Fixed acrylic temporary
- Fixed, metal-reinforced, long-term temporary

Removable Immediate Temporary

In a 45-year-old female with advanced periodontitis, teeth 17, 15, 14, 12, 21 and 25 had to be extracted. The remaining five teeth (16, 13, 22, 23 and 24) were simultaneously treated periodontally. A removable immediate temporary was prepared and seated, and ca. 10 days later, following suture removal, the temporary denture was relined.





1172 Temporary Partial Denture, after Relining
After periodontal surgery, which included tooth extraction and flap procedures, the palatal plate temporary prosthesis was adapted intraorally and relined using a permanently soft material (Kerr-Fit). The soft relining material also served as a wound dressing.

Right: The wrought wire clasp on tooth 13 prevents apical displacement of the temporary denture. As shown, the clasp is at not yet at its definitively seated position.





1173 Relined Temporary Maxillary Denture
The clasp design for the canine teeth was functionally optimal but esthetically less satisfactory.

Right: Detail of the clasp on tooth 13, which prevents apical displacement of the denture.

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Adhesive Bridge in the Mandible—Long-Term Temporary

Mandibular anterior teeth often exhibit severe but irregular periodontal attachment loss of unknown etiology. Therefore, often a single anterior tooth is lost, but its adjacent teeth are periodontally treatable. Clinical treatment for this situation today might include the use of a single-tooth dental implant, but only when the spatial relationships are appropriate (alveolar ridge width, distance to the "neighbors").

A much simpler solution is the adhesive bridge.

Both methods avoid the more classical bridge construction and therefore the hard substance of the adjacent teeth is not compromised. In addition, the adhesive bridge requires considerably less time to fabricate and is therefore less expensive for the patient.

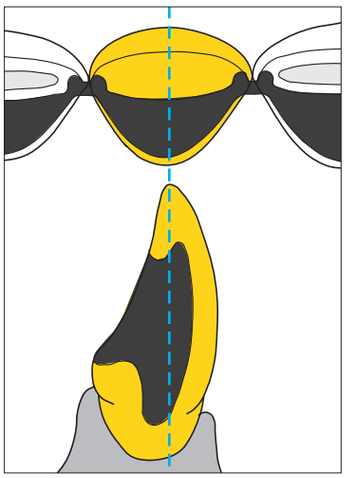
A 41-year-old female presented with tooth 31 exhibiting pronounced attachment loss and severe mobility. The tooth was deemed hopeless. After extensive discussions with the patient about treatment options, the adhesive bridge was selected.





1174 Extraction of Tooth 31
Hopeless tooth 31 is extracted and the adjacent teeth and the entire remaining dentition are treated periodontally. The mandibular anterior teeth 42, 41 and 32 are caries-free.

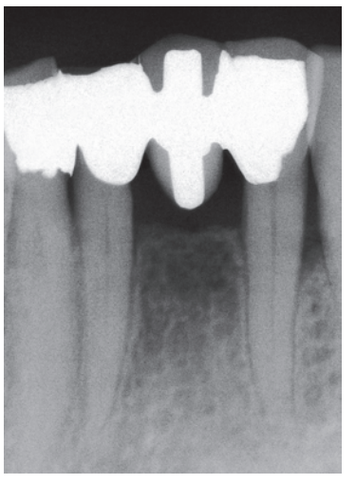
Left: The radiograph depicts a Williams probe in the 10 mm pocket.





1175 Try-In of the Bridge Framework
The bridge framework spans all four anterior teeth. The "hooks" over teeth 41 and 32 guarantee precise positioning during the try-in. The hooks are removed before definitive seating.

Left: Schematic depiction of the pontic, which contacts the soft tissue (caution: hygiene!).
Above: Occlusal view.
Below: Orofacial section.





1176 Seated Adhesive Bridge
Before cementation, e. g., during framework try-in, the color and shape of the pontic are checked. Here, it is too wide and a bit too long apically (red marks). After etching of the lingual surfaces of the three remaining anterior teeth, under the rubber dam, the bridge is seated using chemical and light-polymerized composite resin ("dual-cement").

Left: "Maryland bridge," 17 years following extraction of tooth 31.

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Fixed Temporary—Problem Zones

- Gingival margin—crown margins
- Interdental area—papillae
- Pontic—ridge lap
- Occlusion—occlusal surfaces

If, during the course of periodontal treatment, extraction of un-maintainable teeth and/or if teeth are already missing, in most cases a temporary replacement is indicated. An exception can be the extraction of molars (no antagonist; esthetically non-sensitive areas).

If a temporary must be fabricated and seated *during* periodontal therapy, the temporary must be adapted to the new morphologic relationships following completion of active therapy because of functional and/or esthetics reasons, or the temporary must be completely renewed.
If fixed, definitive bridgework is planned, it is wise to always use a fixed temporary replacement. Such temporary bridges must withstand very high biological and esthetic demands. Within the limitations of laboratory fabrication, any temporary bridge should anticipate the definitive bridge; this is true particularly for occlusion, shape, color, length of the abutment teeth and pontics as well as the shape of interdental spaces.

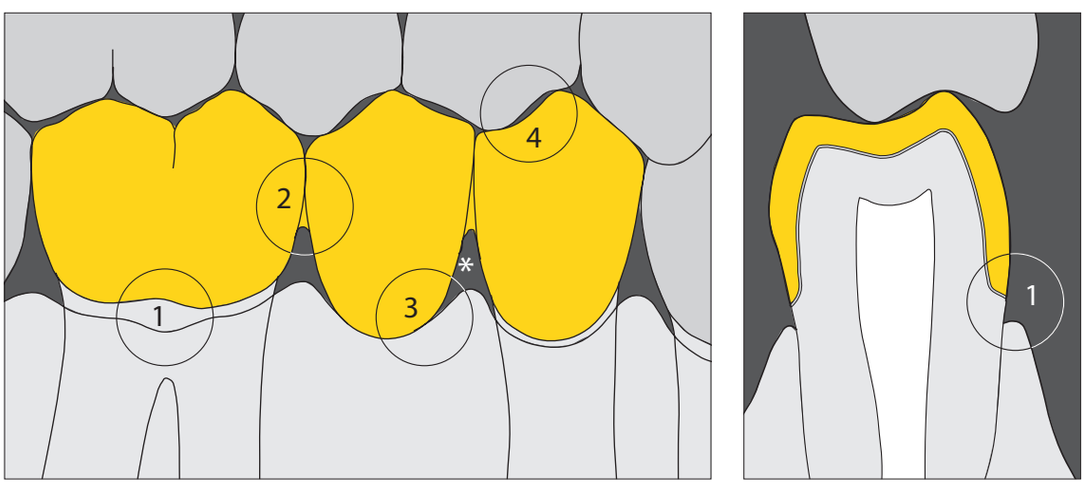
It is also important that any temporary not disturb or inhibit long-term healing after periodontal therapy.

The crown margins of the temporary replacement must be precise and must be *supragingival*.
The morphologic situation can change significantly during the months following periodontal therapy. The gingival margin around the treated abutment teeth may shrink apically or—seldom—move coronally ("creeping attachment," e.g., following surgical crown lengthening procedures). Osseous resorption may occur in the region of the extracted teeth. Any resulting unharmonic clinical morphology of the gingival margin around abutment teeth and in the edentulous alveolar segments can be observed while the temporary is *in situ*, and necessary corrections can be made:

- Excessively short teeth often have to be lengthened (gingivectomy or flap surgery with ostectomy; caution: biologic width [BW], p. 493).
- Excessively long teeth can often be shortened during the reconstructive phase or by selective odontoplasty (caution: dental pulp morphology).
- Ridge augmentation is also possible if there is significant shrinkage of the tissue in the area of extracted teeth.

1177 Periodontal Problem Zones with Fixed Replacements—Posterior Segment

1 Crown margin: Supragingival, precise marginal adaptation
2 Interdental area: Open for oral hygiene
3 Pontic: Only slight contact with the alveolar ridge (hygiene)
4 Occlusion/Articulation: No premature contacts



Right: Buccolingual section.

Because the extent and expanse of tissue changes cannot be precisely predicted, a fixed temporary should be worn for as long as possible (months) and should be optimized as necessary over time.

As previously noted, the temporary bridge should mimic as far as possible the definitive bridge (the temporary can provide a model for the dental laboratory technician to copy!). In addition to differences in the materials employed, differences between the temporary and definitive bridge will exist in terms of the position of the crown margins, which will be slightly subgingival in visible regions of the mouth and supragingival in the posterior segments. Additional differences may include the form and ridge adaptation of the pontics.

All of the fabrication and design characteristics for a temporary bridge must provide for optimum oral and dental hygiene, a factor that is just as important as the anatomic/morphologic construction of the temporary. The toothbrush will be effective on smooth surfaces and the facial and oral cervical regions (1), spiral brushes for larger interdental spaces in the posterior segments (2) and "superfloss" for cleaning between the abutment teeth and above all in the esthetically important maxillary anterior region below the pontic (3).

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Fixed Acrylic Temporary—Practicality

The extraction of anterior teeth always requires the immediate insertion of a temporary. Such temporaries can be fabricated:

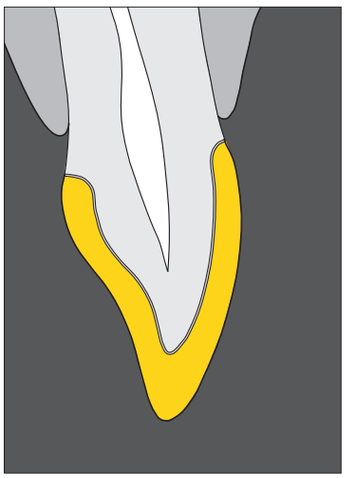
- Immediately in the dental office after taking an impression, or
- In a dental technical laboratory.

Immediate temporaries fabricated within the dental office are usually only necessary in emergency situations where unanticipated tooth extraction or crown loss has occurred. In most other cases, temporaries fabricated in a dental laboratory using articulated study models are prepared; this procedure permits the correction of any tooth positional anomalies (migrations, diastema etc.). In addition to their requirements (see below), esthetically pleasing temporaries provide motivational value for the patient; temporaries also serve as an example for the dental technician with regard to tooth form, arrangement and color (Frank 2000; Lang et al. 2002; Bücking 2002; Czerny 2003).

The case depicted below is a 42-year-old female with advanced chronic periodontitis, in whom a fixed temporary, fabricated in the dental laboratory, was seated.



1178 Clinical View Following Initial Therapy
Migration of the maxillary anterior teeth. Deep pockets, especially on the palatal surfaces of teeth 12, 21 and 22.
The initiation of treatment includes the extraction of these teeth. At the same time, the adjacent teeth 13, 11 and 23 were treated for pocket elimination. During an extended healing phase, a fixed acrylic temporary was indicated.





1179 Immediately after Surgery
Extraction and revision of the periodontal condition on the prepared abutment teeth have been completed. A frenotomy was also performed.

Left: Diagram of the crown (yellow) with clearly *supragingival* margins.

Requirements for a Temporary Bridge

- Stabilization of the abutments and function of the replaced teeth
- Protection of dentin and pulp
- Contouring of the gingiva—"emergence profile" (of the pontic, p. 502), "support" for new papillae (p. 496)
- Possibility for oral hygiene, especially in the interdental areas
- Motivation for the patient
- A template for the definitive bridge



1180 Temporary Acrylic Resin Bridge
The bridge was prepared in a dental laboratory and was adjusted and seated in the clinic. All crown margins are clearly supragingival. Definitive tooth preparation and construction and seating of the final bridgework can begin only after complete healing of the periodontal surgical areas and the extraction wounds; this is generally a matter of months.

Left: General requirements for a temporary bridge.

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Fixed, Metal-Reinforced Long-Term Temporary

In advanced cases, when the periodontal prognosis is not predictable or the patient wishes to maintain even "hopeless" teeth, the indication is for a stable, long-term temporary. The temporary will serve to *splint* the highly mobile teeth during the healing process, permits optimum oral hygiene and provides additional time for definitive treatment planning.

This type of metal-reinforced long-term temporary bridge, fabricated in a dental laboratory, may serve in some cases (see below) for years, and is termed a "temporary definitive restoration."

A 50-year-old male presented with very advanced and chronic periodontitis. Possible treatment plans included multiple extractions and dental implants, which were originally (1988) declined by the patient. He wanted to maintain all of his teeth, especially the anterior teeth.

Treatment plan for the maxilla: Extraction of teeth 14 and 26, amputation of the distobuccal and palatal roots of tooth 16, periodontal initial therapy with temporary acrylic bridge splinting of the maxillary anterior segment. Open (surgical) periodontal therapy and a *long-term temporary*.

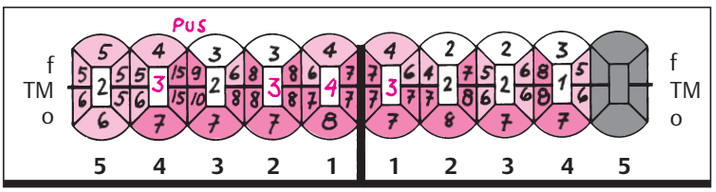
1181 Initial Clinical View
Severe periodontitis with pronounced gingivitis. The patient complained of tooth mobility and halitosis.

Right: Initial abscess formation above teeth 21, 22 and 23, as well as tooth 31.

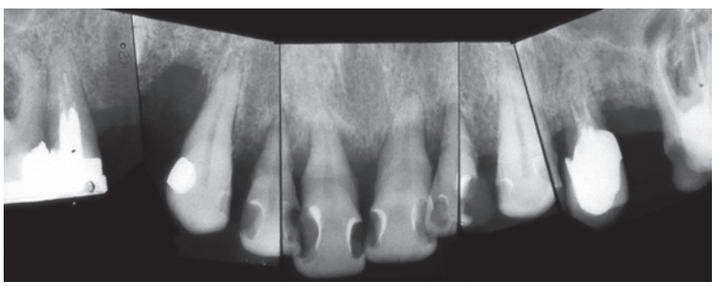




1182 Pocket Probing Depths and Tooth Mobility of Teeth 15 through 24
The initial examination revealed pocket depths up to 15 mm and tooth mobility (TM) to grade 4.



Radiographic Initial Findings, Maxilla
Generalized, pronounced osseous defects.
Inadequate restorations and endodontic therapy.
Note: Tooth 14 had to be immediately extracted on an emergency basis.



Before

1183 Anterior Maxillary Segment During Surgery
During "open" root planing, the deep interproximal osseous defects were obvious (cf. radiographs and clinical probing). On the facial surface, tooth-supporting bone is still in evidence, and this must be preserved during the surgery. In order to prevent therapeutic trauma, the severely mobile teeth were temporarily splinted using composite materials.

The patient suffered from bruxism.



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1184 Clinical Appearance four Months after Surgical Treatment
Four months later, the healing of the soft tissues is generally acceptable, but the morphologic situation in the interdental areas has not yet completely consolidated.
Interdental hygiene in these areas was intensified using spiral brushes.

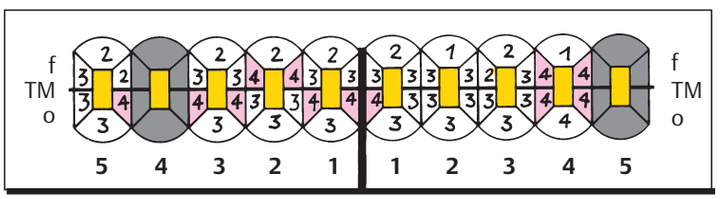


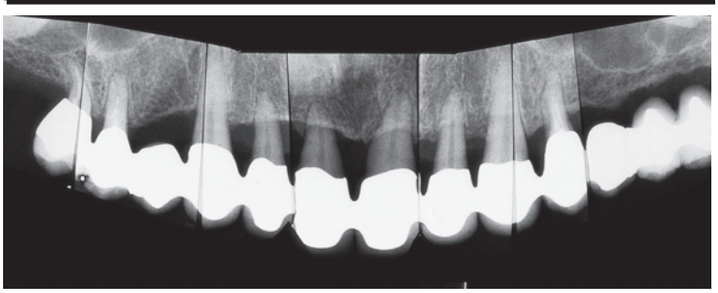


1185 Maxillary "Temporary/ Definitive" Bridge
Six months after conclusion of periodontal treatment, a complete and metal-reinforced bridge was seated as a long-term temporary. The crown margins were all supragingival in order to enhance marginal gingival maturation (esthetics, see Fig. 1187).

Left: Definitive tooth preparation on teeth 21, 22 and 23. Interdental papillae have been lost due to the lack of subjacent osseous support.

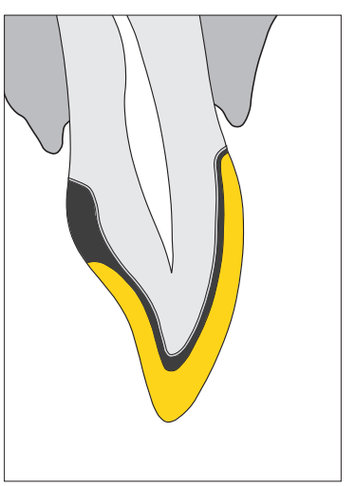
After





1186 Probing Depths 3 Years later
The periodontal situation is stable. In interdental areas one notes maximally 3–4 mm probing depths. Tooth mobility of the individual teeth is not measurable because all of the crowns were splinted to each other (yellow areas).

Radiographic View, 3 Years later
Incorporated bridge. The osseous fundament has consolidated and exhibits some compact bone formation (cf. initial radiographic view, Fig. 1182).





1187 "Smile Line"—Esthetics
Thanks to the upper lip level, the metal margins of the crowns were completely covered. From a purely esthetic point of view, the patient was so satisfied with this temporary bridge that he wore it for 12 years (!). Only when the bridge fractured was a new, implant-born reconstruction performed.

Left: PFM abutment. The metal framework is massively supported on the palatal surface and continued to the preparation margin (metal margin).

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Definitive Fixed Reconstruction—Total Bridgework

The transition period between a "long-term temporary" and a definitive dental replacement is not always distinct. Not only the local, intraoral situation, the diagnosis and prognosis, but also the patient's own desires, his/her ethnic origin, personal desires and financial possibilities for the treatment plan are of extreme importance. One must differentiate between long-term temporary, simple and removable prosthetic treatment, or more complex (and expensive) fixed reconstructions.

In the case depicted here, a long-term temporary was in place for 12 years! In this case, with a similar initial situation and based upon the patient's own desires, a radical treatment plan was initiated and after wearing an acrylic temporary for a very short period of time, a complex and fixed porcelain-fused-to-metal (PFM) bridge was seated.

A 46-year-old Asian male suffered from advanced and chronic periodontitis. He sought periodontal treatment, and a fixed bridge reconstruction in the maxilla.

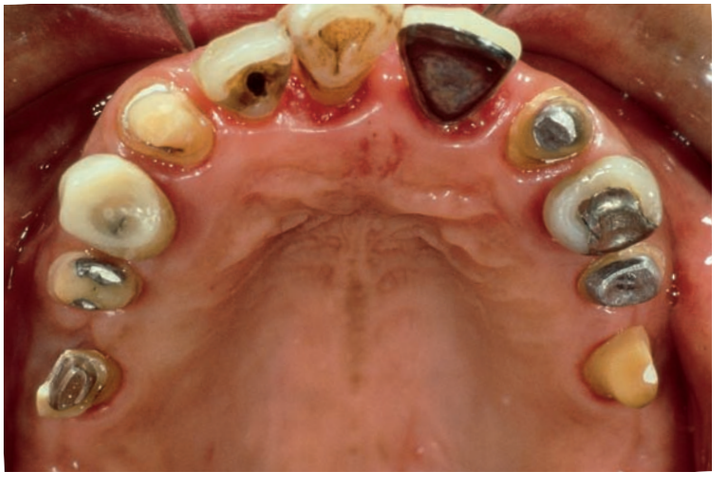
The treatment plan included maintenance of the palatal roots of molars 16 and 26 as well as teeth 15, 13, 23 and 25.

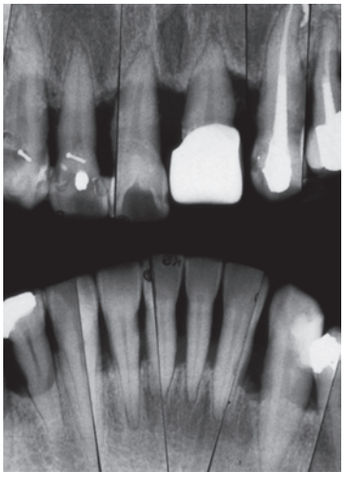
1188 Initial Clinical View
Absolutely terrible conditions pervade the entire dentition: Severe periodontitis with gingival inflammation, gingival recession, tooth malpositioning and an irregular and esthetically objectionable gingival margin in the maxillary anterior segment.
Poor restorative dentistry, secondary caries, tooth 22 missing, insufficient occlusion and other clinical findings result in a totally unpleasant appearance that was disturbing for the patient.



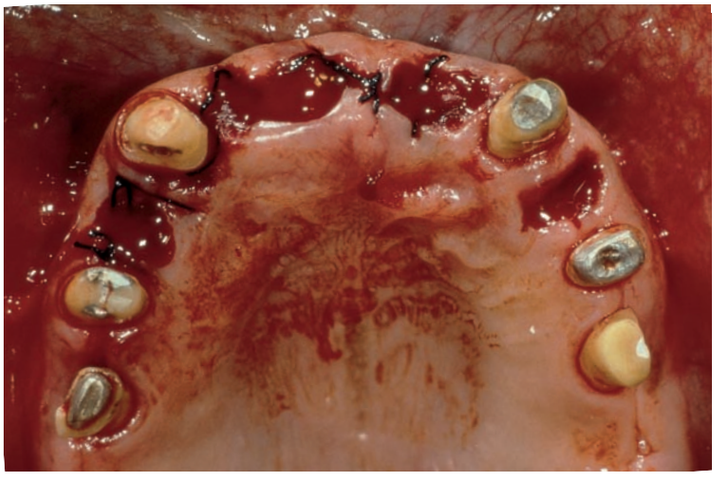
1189 Occlusal View
Closed Phase-1 therapy was performed on teeth 15, 13, 23 and 25, as well as on teeth 16, 26 which were determined to be maintainable. These teeth were then provisionally prepared. Teeth 14, 12, 11, 21 and 24 were subsequently extracted.

Right: Radiographic initial view. In addition to the pronounced bone loss in the maxillary anterior area, it is clear that at least ⅔ of the tooth-supportive bone has been lost in the mandible.





1190 Post-Extraction Clinical View
Removal of the teeth was performed carefully, and wherever possible maintaining the facial osseous lamella. Sutures were placed only loosely. Consideration of a ridge-maintaining implantation of bone replacement material would be reasonable.



Courtesy *A. Adler*

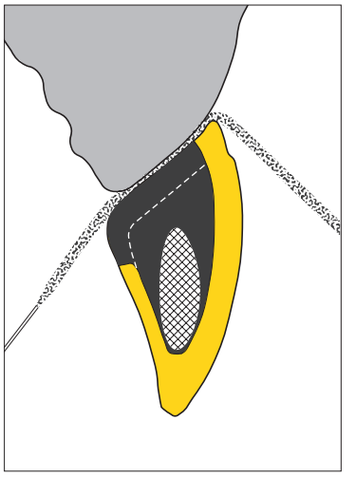
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1191 Temporary Bridge In Situ
The acrylic temporary bridge, fabricated in a dental laboratory, was seated immediately after the tooth extractions. With regard to esthetics it should be virtually identical to the subsequent definitive restoration.
Note: Spontaneous space closure between 32 and 33.

Left: "Set-up" of the anterior teeth on the laboratory model. The missing tooth 22 was included, and a slight overbite was incorporated on the left side.





1192 Definitive Maxillary Anterior Bridge In Situ
The pontics replacing the incisors lie flush against the alveolar ridge (modified ridge lap). These pontics give the impression of longer natural, periodontally treated teeth in the absence of interdental papillae, but with elongated interdental contact areas.
Left: In this cross-sectional schematic through an anterior pontic, it is clear that the expansive contact with the ridge must be established so that superfloss can be used to clean effectively.





1193 "Smile Line"
When speaking or *smiling*, the patient exposes about 1/3 of the teeth in the maxilla, and about 2/3 in the mandible. The esthetics in the maxilla is therefore more than satisfactory.
The open interdental spaces in the mandible are a bit esthetically disturbing.

Left: Final radiographic view of maxilla and mandible, anterior areas. Severely compromised but nevertheless healthy and consolidated osseous fundament.



1194 Lingual View of the Mandibular Anterior Segment
The patient performs excellent oral hygiene. The remaining periodontal support is quite far apical, but exhibits healthy gingival condition. Note the spontaneous space closure between 32 and 33 (in comparison to the initial view, Fig. 1188).

Fortunately, there was no difficulty with tooth neck sensitivity.

Courtesy *A. Adler*

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Molar Loss—What Now?

If end-standing molars are lost (with the exception of wisdom teeth, which are usually not replaced), it is necessary to first consider the situation in the entire dentition before any discussion of molar tooth replacement (or *not*).

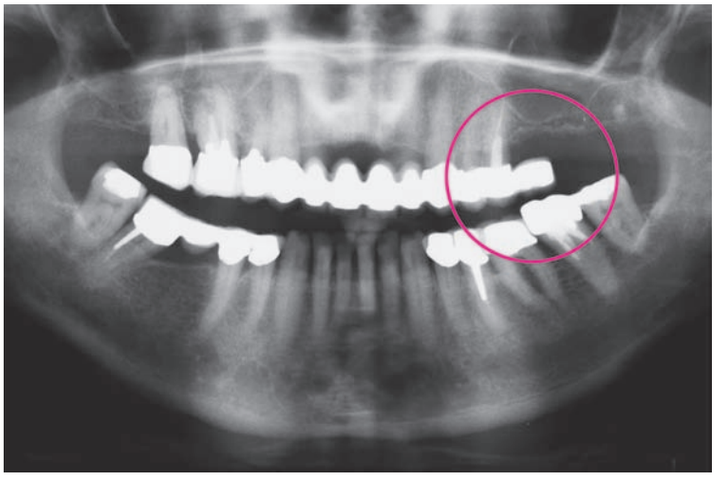
The following factors may be determinant:
- Functional situation in the entire dentition
- Remaining dentition in other areas, especially in the antagonist region of the opposing arch
- The patient's own demands with regard to masticatory ability and comfort
- Patient compliance (OH)
- Hereditary and genetic risk factors

The following treatment possibilities exist:
- A premolar occlusion (no replacement of molar teeth)
- A free-end saddle partial denture
- Dental implants
- Cantilever bridgework

Three examples of cantilever bridges are depicted below.

1195 Maxillary Left Bridge Construction with a Cantilever Pontic
This 51-year-old woman wore a removable partial denture for 10 years, which replaced the anterior teeth and the molars on her left side. As she enjoyed a more favorable financial circumstance, she desired a fixed reconstruction. The case was treated by means of a bridge with a cantilever in the region of 26 (red circle).

Right: Cleaning the cantilever pontic with superfloss.

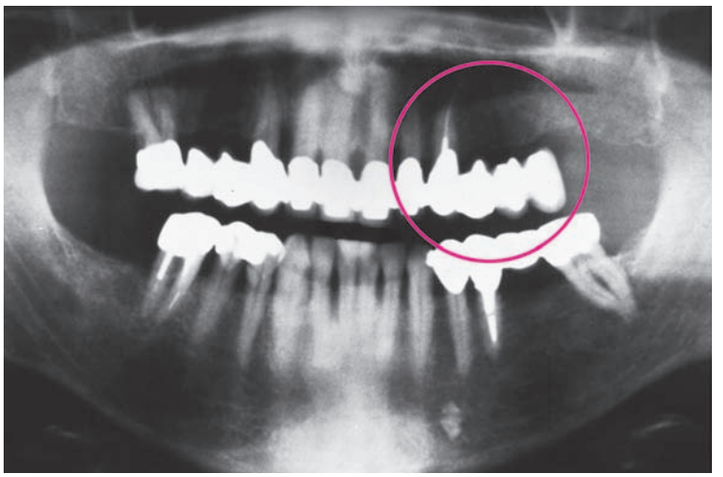


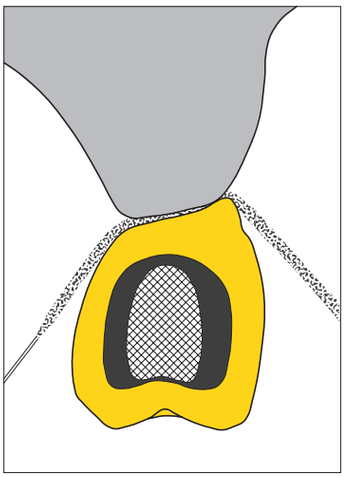


1196 Fixed Bridge with Three Cantilevered Pontics
This 54-year-old male refused a removable prosthesis following periodontal therapy in the maxilla. The problems include:
- Free-end edentulous situation
- Furcation involvement (F2) on tooth 16
- Only moderate oral hygiene

Treatment plan: Total fixed bridge
Critical area (red circle): Three-unit cantilevered extension.

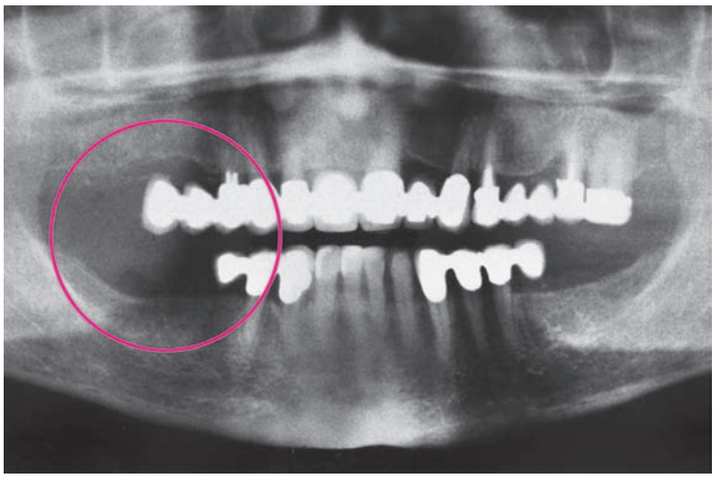
Right: Shape of the cantilevered unit in cross-section.

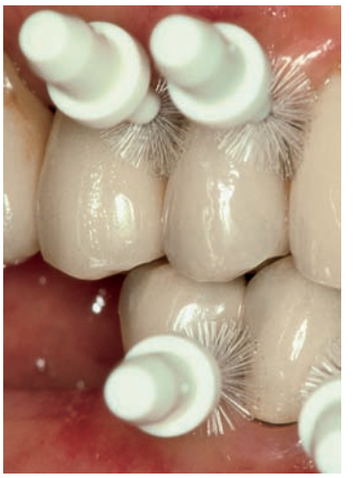




1197 Cantilevered Bridges in Both Maxilla and Mandible
In this 66-year-old female, several hopeless premolars and molars were lost due to advanced furcation involvement. She again refused any removable partial denture. Therefore, bilaterally in the mandible and in the maxillary right segment, cantilevered fixed bridges were seated, which have "lasted" for 14 years!

Right: Cleaning of the open cantilevered interdental spaces using spiral brushes.





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Telescope ("Coping") Bridgework with Few Remaining Teeth

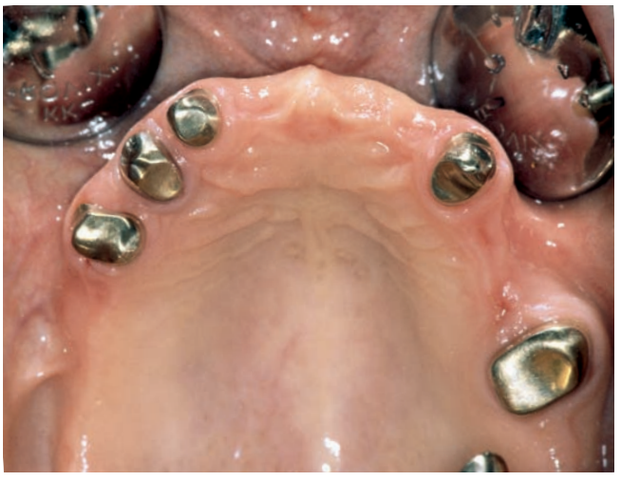
Following comprehensive treatment of advanced periodontitis, often few abutment teeth remain; in such cases a telescope construction ("copings") may be indicated instead of fixed bridgework, a cantilevered bridge or dental implants. Thanks to the removable superstructure, the remaining abutment teeth are easy to clean, and any remaining alveolar ridge defects can be dealt with prosthetically.

Depending upon the number of remaining abutments, the superstructure may be in the form of a bridge or prosthesis, possibly with incorporation of a transpalatal arch.

In a 45-year-old female with severe and aggressive periodontitis, after periodontal therapy and extractions only five abutments remained.

The treatment plan involved a removable telescope bridge. After discussion with the patient, an "experiment" was planned in which a superstructure of a *removable bridge* and a *partial denture* with a *palatal arch* was fabricated. After extended use of both treatment modalities, it was up to the patient to decide which restoration to use over the long term.





1198 Remaining Teeth as Primary Coping Abutments
Maxillary teeth 14, 13, 12, 23 and 26 received "telescope" caps 6 months after periodontal therapy; these teeth were the primary abutments.
The second phase of treatment (see below) can be a removable bridge (**A**) or a removable partial denture (**B**).

Left: Detail of the "coping" crowns. The margins of the copings were intentionally located *supragingivally*.





1199 A—Telescoping Bridge
This removable replacement resembles a total fixed bridge. The palate is left completely exposed.

Left: Detail of the bridge construction. The patient objected to the long central incisors and the open interdental spaces ("black triangles") in the esthetically sensitive anterior region.





1200 B—Telescope Partial Denture
The excellent coloration of the anterior tooth replacements made it possible to improve the spatial proportions of the anterior teeth and close the interdental spaces using "artificial" acrylic papillae. Interestingly, the patient (not the dentist!) preferred this partial denture: Esthetics and chewing comfort were better (more secure hold due to the palatal arch).

Left: Detail of the removable partial denture superstructure.

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Cast Framework Prostheses—"Social Solution"

In many cases, primarily due to financial considerations and the lack of universal dental health care insurance, the ages-old cast-metal removable partial denture may be the treatment of choice. According to Bergmann et al. (1982) this type of partial denture may last for up to 10 years in patients who have undergone comprehensive periodontal therapy.

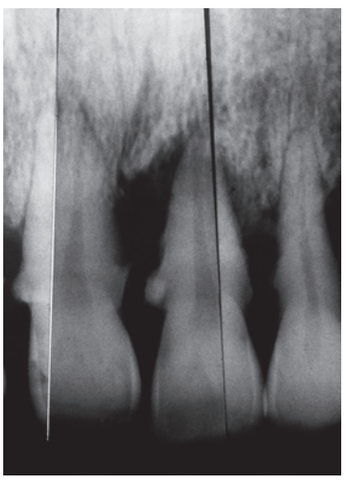
Advantages
- Minimal cost
- Minimal time for fabrication
- Maximum possibility for subsequent modifications with acrylic resin (gingiva, papillae, ridge defects)
- Compensation for or maintenance of diastemata
- Possibility for subsequent, comprehensive reconstructive therapy (fixed bridgework, dental implants etc.)
- Simple oral hygiene for the prosthesis and remaining teeth

Disadvantages
- Traumatization of the clasped teeth
- Not optimum precision
- Elevated risk of caries
- Psychological problems: "something removable."

1201 Initial Clinical View
A 40-year-old female suffered with generalized, moderate, chronic periodontitis, with profound defects in the maxillary anterior segment. Note pronounced gingival inflammation and inadequate amalgam restorations. Teeth 12, 11, 21 and 22 were deemed hopeless.

Right: Radiograph of the extremely poor support in the maxillary anterior segment. Note also heavy calculus accumulation.





1202 After Periodontal Treatment and Restorative Measures
For purely financial reasons, the replacement of the maxillary anterior teeth had to be performed using a cast framework partial denture. Defective amalgam restorations were replaced; in preparation for the removable partial denture, the teeth were selectively modified (occlusal rests, space for the clasps, parallelism etc.).

Right: Several oral hygiene aids were employed: Toothbrush, interdental brush, denture brush.





1203 Cast Framework Partial Denture In Situ
The periodontium of the remaining teeth is protected by the construction of the clasps and palatal arch. The partial denture does not create new plaque-retentive niches. The clasp arms and the palatal arch are located removed from the gingiva as far as possible.

Right: With the denture *in situ*, the clasps are supragingivally located and the interdental spaces are easily rinsed and open for cleansing.





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Perio-Prosthetics 2
Supplemental Measures, Esthetics

Prosthetics in general and perio-prosthetics especially have undergone remarkable development and transition in industrialized countries in recent years, especially in the areas of dental materials and technologies. It is no more simply of a question of functionally and phonetically acceptable replacement of lost teeth but also optimum esthetics in the visible areas of the oral cavity, and even beyond that to a positive self-image for the entire individual. Today it is true that to a certain extent social and work-place success may also be tempered by one's overall external appearance.
For these reasons, every dentist must be fully aware that the patient's esthetic demands, and the fulfillment of those demands, are not necessarily or immediately related to general systemic health. The individual's psyche may be enhanced by purely esthetic measures, and this may have some significance in systemic health.

Goals, Problems and Solutions in Esthetic Perio-Prosthetics

The primary and underlying goal of esthetic perio-prosthetics goes beyond the creation of an ideal situation with regard to tooth color, shape and arrangement as well as gingival margin morphology. Today one must consider that every dental reconstruction has to be adapted to the individual patient and his/her individual desires (Chen & Schärer 1995, Garber & Salama 1996, Schmidseder 1999; Rüfenacht 2000).

Here we present the following esthetic perio-prosthetics problems, and possible solutions:

- Crown margins—biologic width, dentogingival complex
- Esthetic width—umbrella effect
- Smile line—red-white proportions
- Surgical crown lengthening—principles and case report
- Papilla loss—classification, surgery and/or prosthetics?
- Papilla loss—prosthetic treatment with veneers
- Papilla loss—prosthetic treatment using crowns
- Edentulous arch segments—pontics
- Ridge defects—classifications
- Ridge defects—prosthetic correction
- Ridge defects—principles of surgical correction
- Ridge defect—surgical augmentation of a Class 3 case

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Crown Margins—Biologic Width—Dentogingival Complex (DGC)

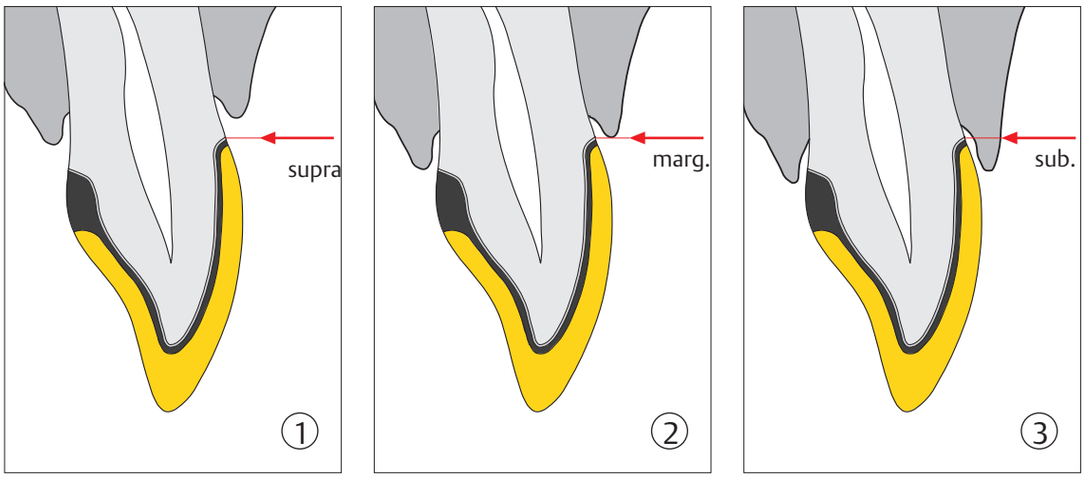
In keeping with time-honored prosthetic principles, in the *non*-visible areas crown margins should be located supragingivally in order to make optimum oral hygiene and plaque control possible.

In visible anterior segments, crown margins may be placed intrasulcularly along the gingival margin. However, even subgingivally located crown margins must not cause injury to periodontal tissues. This demands knowledge of the expanse and spatial relationships of the supracrestal structures, i.e., the so-called *biologic width* and the dentogingival complex (Gargiulo et al. 1961, Nevins & Skurow 1984). It is critical to understand that these parameters are not the same in all patients. For example, the height of the alveolar ridge crest is normally about 3 mm below the gingival margin, but may be more or less than 3 mm. This can only be determined under local anesthesia ("bone sounding"). Not only the height of the alveolar bone but also the thickness of the bone and the gingiva (phenotype, p. 8) are of significance for the definitive placement of crown margins (Fig. 1206).

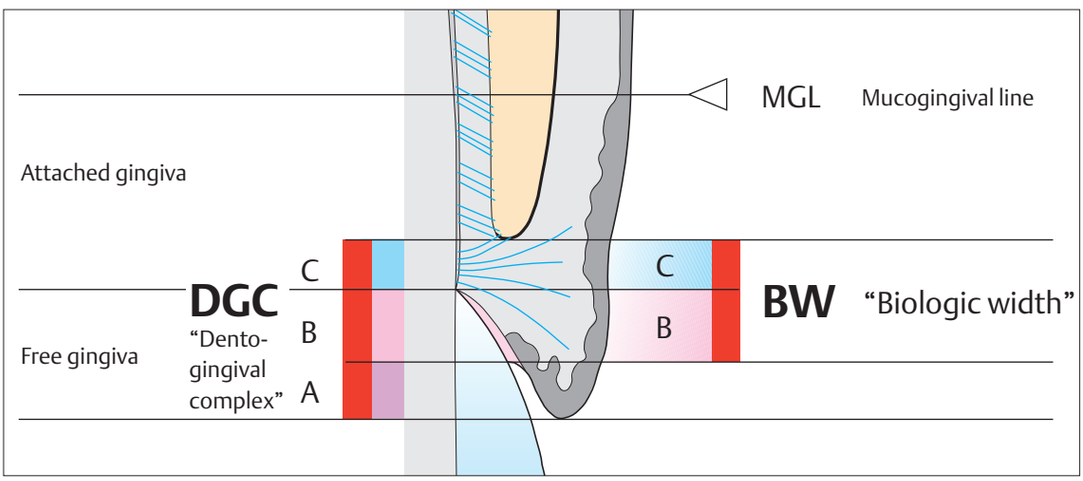
1204 Location of the Crown Margin—Example: PFM Crown

1 Supragingival
2 Epigingival (marginal)
3 Intrasulcular (subgingival)

1 and **2** are periodontally "friendly," but do not fulfill today's high esthetic demands. In the visible anterior region, intrasulcular (subgingival) positioning of the crown margin is mandatory, which demands skill and precision during tooth preparation and also reliable patient compliance.



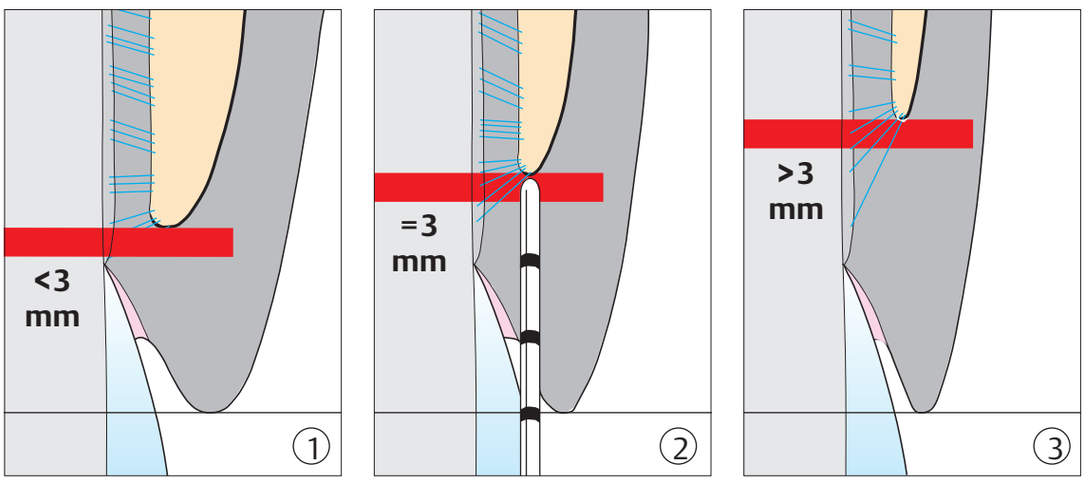
1205 Biologic Width and Dentogingival Complex
A Gingival sulcus ca. 1 mm
B Epithelial attachment, junctional epithelium ca. 1 mm
C Connective tissue attachment ca. 1 mm
The values of **A, B, C** vary from tooth to tooth and individually. The minimal space requirement of these tissues is the "biologic width" = **B + C**. This must not be exceeded. Most important is the *dentogingival complex (DGC)*.



1206 Varying Course of the Alveolar Bony Crest—Height and Thickness—Gingival Phenotype

1 High alveolar crest, short dentogingival complex. Caution advised: The preparation margin must not extend > 0.5 mm into the sulcus!
2 Normal alveolar crest height ("bone sounding").
3 Deep alveolar ridge, long DGC

The placement of any crown margin depends primarily upon the DGC.



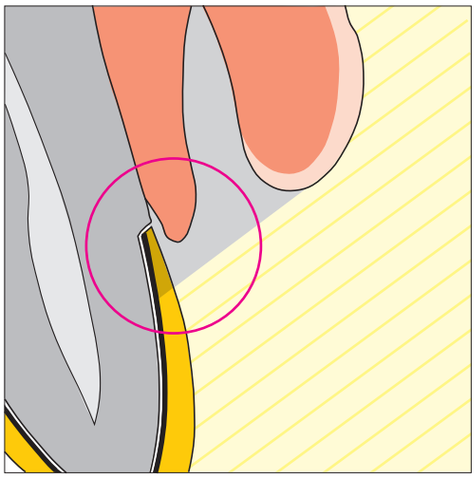
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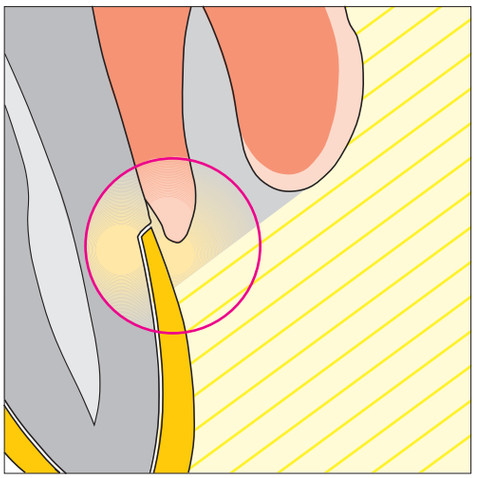
Esthetic Width—Umbrella Effect—Transparency

With crown and bridge constructions, esthetic aspects are becoming ever more important (anterior segments!). Expanding from the concept of biologic width, Magne and coworkers (1999) introduced the term "esthetic width," and summarized how each of the individual treatment results could be achieved in terms of mimicking natural structures. *Umbrella effect*: *Non-transparent* crown margins (PFM etc.) inhibit any diffuse illumination of the marginal gingiva from the labial aspect if the upper lip shadows this area: Interdental papillae and the gingival margin at the crown margin appear bluish and dark.

Illuminated *transparent* crowns conduct light; the gingival segments appear brighter from the labial aspect: They exhibit the normal pink color.

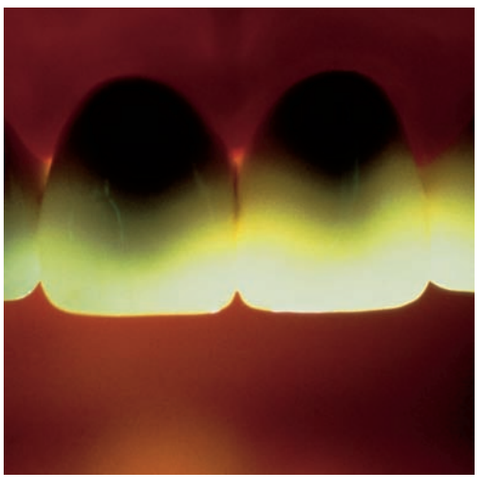
Transparency: Reflection, deflection and transmission of light depend upon the materials used in the dental construction, including transparent crowns and veneers but also the tooth preparation (discoloration, build-up, post) and even the cement used to seat the restoration.

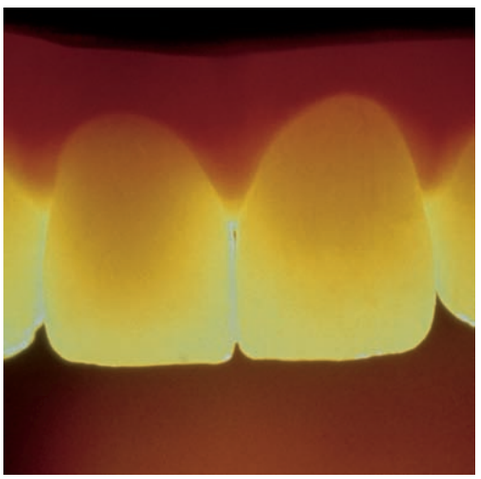




1207 Umbrella Effect
Left: Light coming in obliquely from above (yellow; e. g., normal room lighting) does not illuminate the gingiva because it is shaded by the upper lip. The PFM crown also inhibits any illumination from behind: Therefore, the gingiva appears dark, "dead."

Right: Semi-transparent materials (crowns, cement, dentin) disperse incoming light. Therefore the marginal gingiva exhibits its natural color.





Opaque vs. Transparent Crowns and Veneers

1208 In Transparent Light . . .
Left: Despite pleasing transparency in the incisal region, the body of the crown and the entire gingival margin appear dark (PFM).

Right: Transparent materials provide an even transition to the semi-transparent tooth separation.
Also here, dispersed light illuminates gingiva and interdental papillae.





1209 ... and in Daylight
In direct light—*as the dentist sees his/her reconstructive efforts*—the above-mentioned effects are less pronounced with both crown techniques (PFM *left*, ceramic crown *right*). Nevertheless, at the gingival margin, the differences are obvious. The mother of pearl effect is similar to a natural tooth.
Fact: For a completely satisfactory esthetic result, the appearance of the gingiva must also receive due consideration.

Courtesy *D. Edelhoff*

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Smile Line—Red-White Proportions

The esthetics of the human face have been considered and described for millennia.

Even at its highest level, esthetics is *subjective*, because "*beauty lies in the eye of the beholder*" (and also in the mirror!). Nevertheless, there are certain *objective* and generally accepted guidelines for the analysis and possible treatment of esthetic compromises in the dentition (Belser 1982: "The esthetic checklist"). But the question always arises concerning whether and to what degree esthetic corrections are even necessary (Müller 1996, Zuhr 2001). Such questions must be answered primarily by the patient him/herself.

In the dental/periodontal field, facial beauty is often analyzed and considered according the following points (Schmidseder 1998, Rifkin 2000, Rüfenacht 2000):

- Symmetry of the face and the dentition
- Lip shape and "smile line"
- Visible red-white relationship (e. g., "gummy smile")
- Tooth proportions, shape and texture; tooth color
- Papilla loss; defects of the alveolar ridge

In the following pages, primarily periodontal esthetic problems and possible solutions will be depicted.

1210 High Smile Line—Score 3
A large expanse of gingival tissue is visible when the patient smiles (see below): "Gummy smile."

Clinical finding: Short upper lip, hyperplastic gingiva, periodontal pockets, poorly adapted crown margins, mouth breathing.

Treatment: Periodontal therapy! Temporary crown replacements. (Improve nasal breathing.) Subsequent decision concerning whether the "gummy smile" should be further treated.



1211 Smile Line—Classes, Scores 0–3
Jensen and coworkers (1999) classified the position of the upper lip in relationship to the maxillary gingiva during "smiling" (to assist in treatment planning if any correction of the red-white relationship is desired!).

Test: Un-forced smiling should be practiced; the dynamic of the upper lip should be momentarily stopped in order to observe the lip line with the *teeth closed* (rest position).

Classification of the "smile line"	Upper lip – Interdental and marginal gingiva	
Class	Type – Description	Evaluation: **IDG** interdental gingiva **M** gingival margin
Score 0	"low smile line"	IDG less than 25% visible M not visible, teeth masked
Score 1	"average/ideal smile line"	IDG 25–75% visible M visible on individual teeth
Score 2	"high smile line"	IDG >75% visible M <3 mm visible (overall)
Score 3	"very high smile line"	IDG completely visible M <3 mm wide maxillary band of gingiva visible, beyond the MGL –"gummy smile"

1212 Very High Smile Line
Despite a normal lip length, during smiling much more than 3 mm of gingiva is visible (score 3). The anterior teeth are too short and deviate from the normal proportions.
Causes: Probing reveals inflammation-free pseudopockets resulting from *incomplete passive tooth eruption*; thick gingival phenotype (cf. p. 8).

"Therapy": If the patient so desires, facial crown lengthening via *gingivectomy* can be performed.



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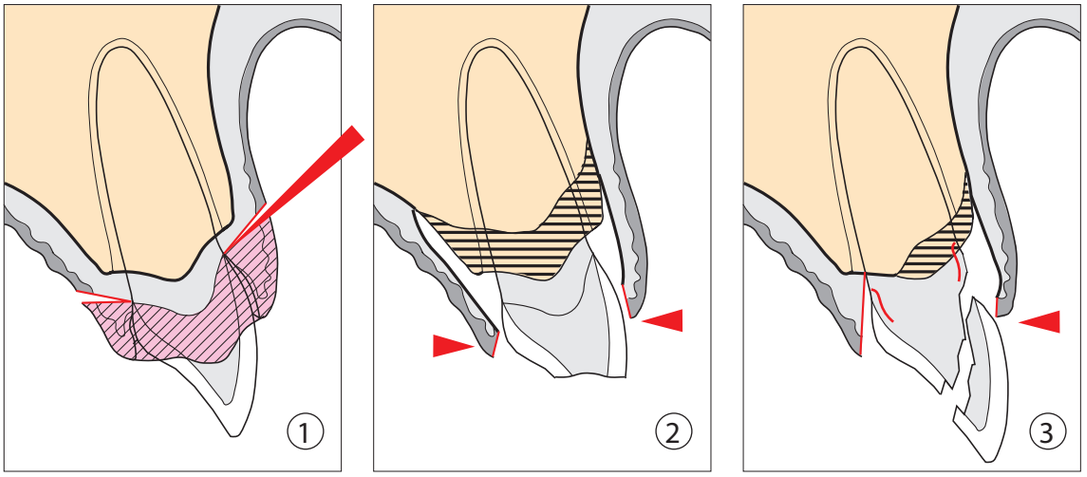
Crown Lengthening—Principles

Depending upon the indication, the clinical crown can be lengthened prosthetically, surgically or in a combined procedure. There are many surgical possibilities (Brägger & Lang 1991, Lauchenauer et al. 1991, Jorgensen & Nowzari 2001) and the approach will depend upon the individual situation. Possible interventions include:

- Gingivectomy/gingivoplasty (GV/GP)
- Flap reflection with partial ostectomy ("ramping")
- Flap reflection with circumferential ostectomy

The *gingivectomy* is often used in cases where the natural tooth has not erupted completely, or with gingival hyperplasia.

Ostectomy (partial or circumferential) may be indicated if the necessary distance to the margin of the planned artificial crown does not exceed a minimum of 2–3 mm (biologic width), or if the abutment tooth is too short to provide sufficient retention for the prosthetic replacement (defects due to caries or fracture, abraded teeth due to bruxism etc.). Esthetic problems such as asymmetry of tooth length and a "gummy smile" (Fig. 1210) lengthen this list. The following pages depict an appropriate clinical case.



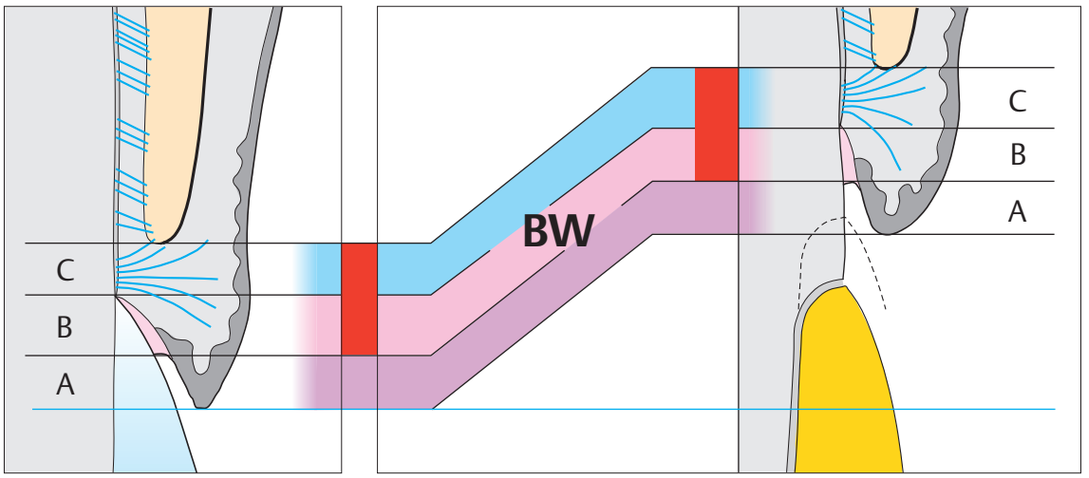
1213 Surgical Crown Lengthening—Schematic
Papillae and gingival mass must be maintained! Red arrow point: Thinned flaps.

1 **Gingivectomy/gingivoplasty**
e. g., to reduce gingival hyperplasia
2 **Total circumferential ostectomy**
e. g., abraded tooth
3 **Partial ostectomy "ramping"**
e. g., oblique tooth fracture



1214 Crown Lengthening—Case Presentation
In this case of a 59-year-old female (see next pages), the initial and final clinical results are depicted.

Purpose for the surgery:
- Attrition/abrasion
- Large restorations with caries
- Discoloration (vital tooth)
- Generally: Esthetics of the maxillary anterior segment



1215 Biologic Width During Crown Lengthening by Means of Ostectomy
Maintenance of the biologic width (**B + C**) as well as the individual dentogingival complex (**DGC**) must be maintained during treatment planning and crown lengthening.

For example, in order to "lengthen" a tooth by 2 mm, the *alveolar crest* must reside > 5 mm deeper (more apical) than the "old" *gingival margin*!

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Surgical Crown Lengthening—Step-by-Step

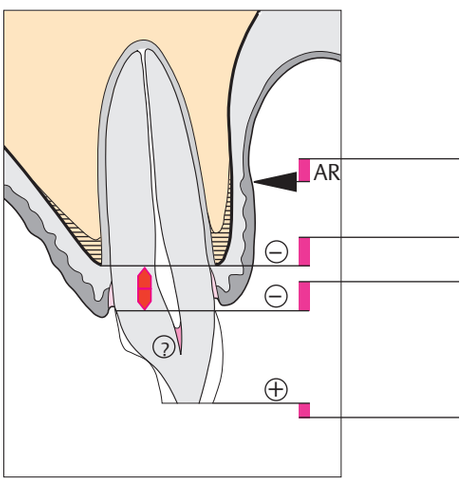
The surgery is performed according to the following protocol:

- Pre-preparation of the abutments, circular chamfer at about the level of the gingival margin
- Scalloping horizontal incisions, facially and orally, vertical releasing incisions, mobilization of the full-thickness flap
- *Ostectomy* around the anterior teeth maintaining an even distance—here ca. 5 mm—from the circular step of the tooth preparation margin
- *Osteoplasty* of the facial bone: Creation of embrasures and/or sluiceways
- Scaling and/or removal of cementum on the exposed root surfaces: *Caution*: "creeping attachment" (p. 412)
- Apical repositioning of the tissue flaps, with complete coverage of the alveolar bone (goal: "0 mm pockets")
- Suture closure; final tooth preparation; margin at least 1–2 mm supragingival; adapt the temporary restoration
- Modification of oral hygiene: In the surgical site, only chlorhexidine rinses initially, and then very gently mechanical plaque control
- Seating of the definitive crown only after complete healing/maturation of both soft and hard tissues

1216 Marking the Initial Preparation—Steps at the Original Gingival Margin
The first of the five teeth 13, 12, 11, 21, 22 has already been prepared (incisal edge and margin).

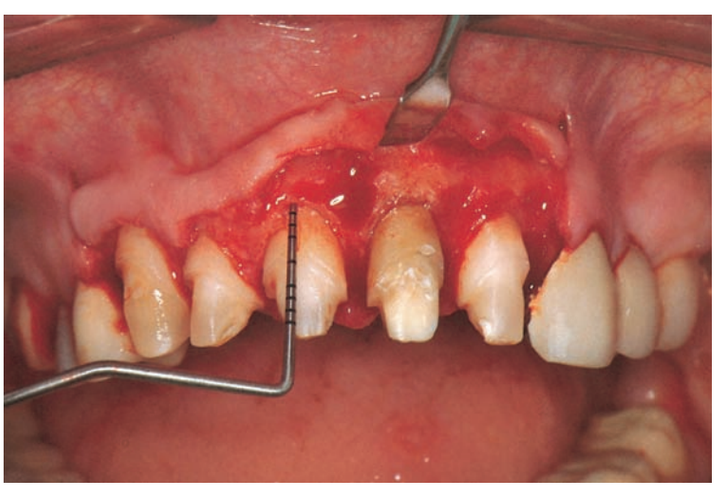
Right: Planning. At the incisal edge, the crowns should be only minimally lengthened (+). The osseous crest is reduced apically ca. 3 mm (hatched area). The DGC (red double arrow) must be maintained; crown preparations must be lengthened by ca. 2–3 mm (cf. p. 495).





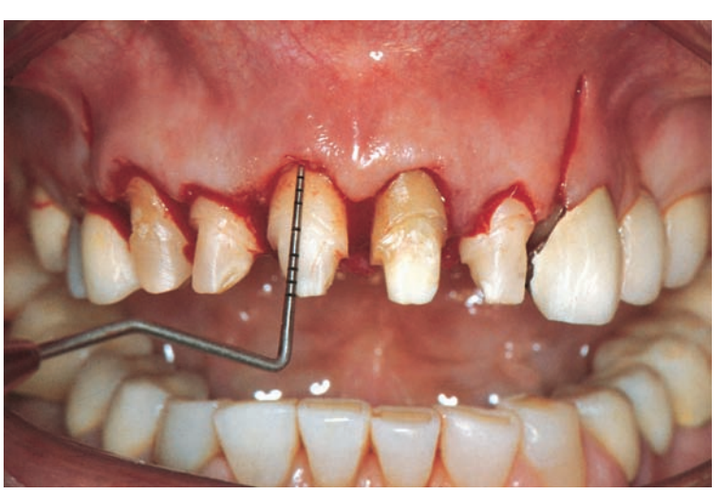
1217 Flap Reflection and Ostectomy—Maintenance of the Dentogingival Complex
The preparation margin of tooth 11 (probe) depicts the pre-operative gingival margin. In order to lengthen the crown preparation by 3 mm, the alveolar crest must be displaced apically by at least this amount (see *right* of the probe).

Right: Hand instruments and machine-driven instruments for ostectomy and scaling.

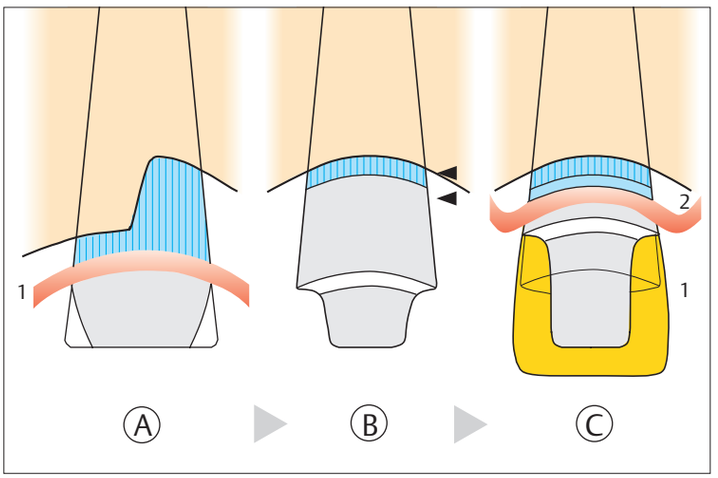




1218 Checking the Extent of Ostectomy
For a temporary evaluation, following osseous resection, the *thinned* and if necessary shortened soft tissue flap is replaced slightly apically. It is clear that the necessary ca. 3 mm crown lengthening has been achieved, which will guarantee retention of the replacement crown (double red arrow in Fig. 1220, left).



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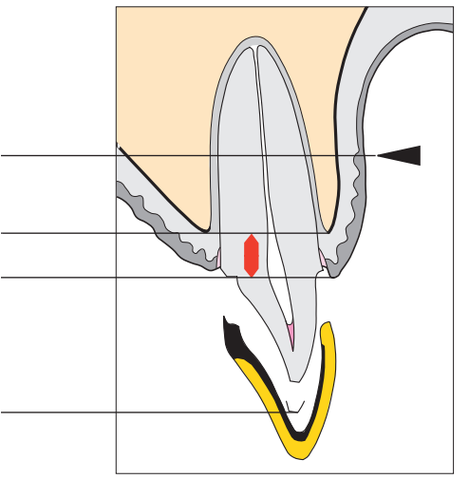


1219 Diagram of the Surgical Procedure—Hard Tissue

A Tooth preparation down to dentin. Gingival margin (red, **1**). Ostectomy 50 % completed. Root surface (periodontal ligament) blue.

B Ostectomy complete. Blue hatched area = reduced fiber apparatus. Between the black lines is the necessary biologic width.

C Bone and gingival morphology (**2**), as originally (**1**) and with the tooth preparation margins.





1220 Final Tooth Preparation—"Elongation" of the Abutment Teeth

The facial soft tissue flap is repositioned and securely sutured. Now the circular step preparation of the abutment teeth is lower apically but not nearer than 1 mm to the new gingival margin.

Left: The diagram depicts the minimal apical *flap repositioning* (AR) as well as the crown lengthening (red double arrow) and the minimal incisal *crown lengthening*.





1221 Second Temporary

Four weeks later, for functional reasons (severe parafunctions) a splinted long-term temporary was seated. The narrow interdental spaces were created to enhance "new formation" of papillae between the relatively widely spaced teeth.

Left: Surgical site 7 days postoperative. Healing has not progressed satisfactorily, and interdental papillae are lacking.





1222 Definitive Reconstruction—6 Months after Surgery

The definitive and somewhat massive PFM crowns were seated 3 months after the surgery. The tooth shapes are much more natural (triangular) in form. In addition, during the healing period the interdental papillae filled out nicely within the proximal spaces. Because of the patient's wishes, the bridge 23–26 was also replaced.

Left: The radiograph clearly depicts the wide interdental osseous septa.

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Papilla Loss—Classification, Rules

Mucogingival, "plastic" surgery today offers many possibilities for the improvement of oral/dental esthetics. Such surgical procedures can be employed to cover areas of gingival recession, build-up alveolar ridge defects, correct an irregular gingival margin morphology, and even reduce a "gummy smile." The loss of interdental papillae and the resultant "black triangle," presents one of the greatest problems in esthetic dentistry. The causes of papilla loss include:

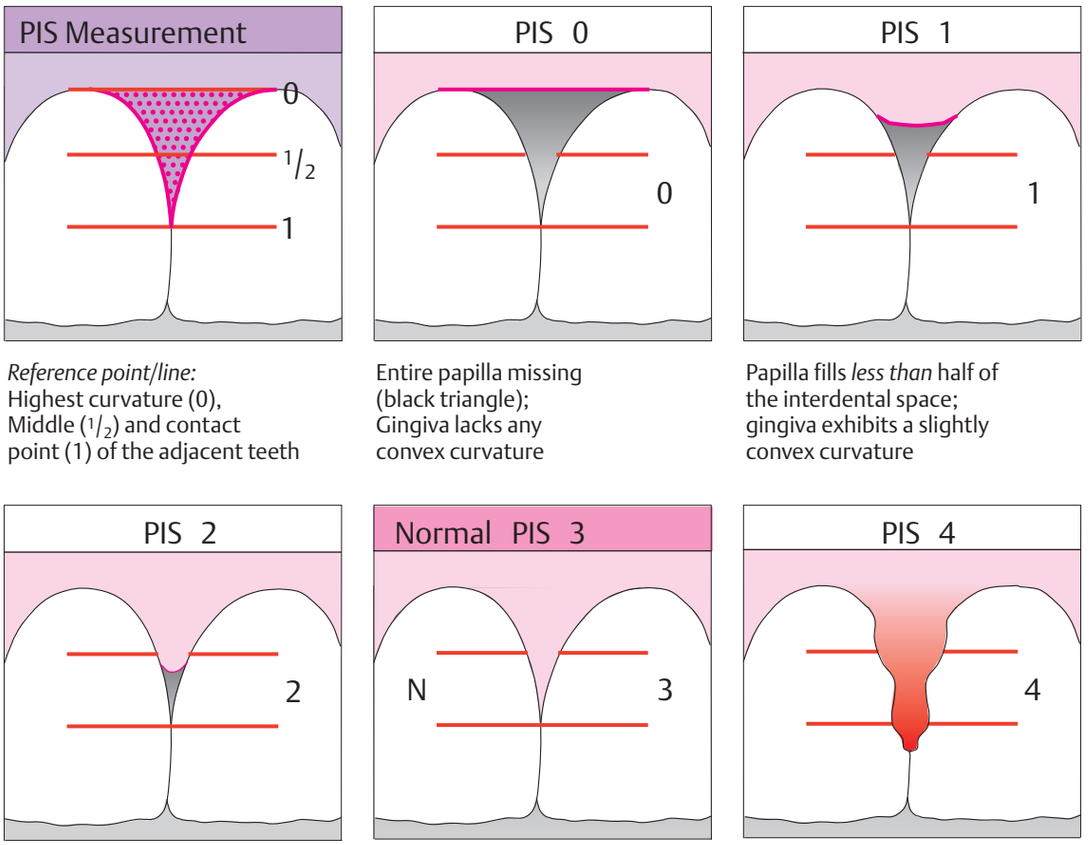
- Interproximal bone loss caused by periodontitis; loss of alveolar bone support
- Gingival shrinkage following classical closed or surgical periodontitis treatment
- Tooth position anomalies; missing teeth adjacent to natural teeth or dental implants

Even today, the reconstruction of interdental papilla remains a very difficult surgical endeavor (Miller's recession classifications 3 and 4; p. 163). From the purely prosthetic approach, however, the maintenance or rebuilding of an interdental papilla is possible if the distance between proximal bone levels and interdental contact surfaces is below ca. 5 mm (Tarnow et al. 1992).

1223 Papilla Index—PIS
In 1997 Jemt published a classification of interdental papilla loss, the so-called "papilla index score" (PIS), with five grades (0–4), intended primarily for implant crowns and adjacent natural teeth. The index discriminates three reference lines for the measurement of papillae (red lines: 0, ½, 1- above left).

Grade 0 PIS 0—Complete absence of the interdental papilla (large black triangle)
Grade 1 More than half the papilla missing
Grade 2 At least half or more of the papilla is present
Grade 3 Optimum soft tissue contour, the papilla fills the entire interdental space
Grade 4 Hyperplastic and inflamed papillae, irregular soft tissue contours

Modified from *Jemt 1997*



Reference point/line: Highest curvature (0), Middle (1/2) and contact point (1) of the adjacent teeth

Entire papilla missing (black triangle); Gingiva lacks any convex curvature

Papilla fills *less than* half of the interdental space; gingiva exhibits a slightly convex curvature

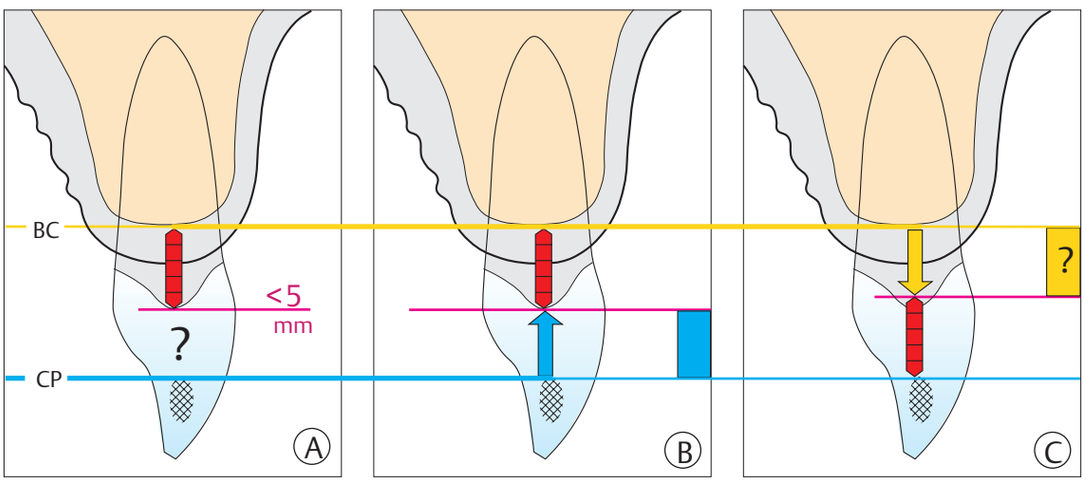
Partial papilla fills the interdental space half or more, but not completely

Papilla fills the entire interdental space: Ideal tissue contour and tissue harmony

Hyperplastic papilla covering too much tooth structure; irregular tissue contour and texture, possible color changes

1224 Possibilities and Limitations of Papilla Reconstruction
A Interdental bone loss. The distance between the osseous ridge (**BC**) and contact points/surfaces (**CP**) must be *less than 5 mm* (red double arrow), in order to insure papillary "regeneration."
B *Prosthetics*: By means of apical repositioning of the contact points
C *Surgery*: Attempt to elicit interradicular bone regeneration?

Modified from *Tarnow et al. 1992*



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Papilla Loss—Prosthetic Rehabilitation with Veneers

In the attempt to solve severe esthetic dental problems, there must often be a combination between and among mucogingival and plastic oral surgery, as well as prosthetic intervention in addition to orthodontic measures. In such cases the goal to be achieved must be precisely defined in order to insure appropriate treatment planning. The patient must be informed about the often enormous costs of such combined therapy, and the time likely to be required.

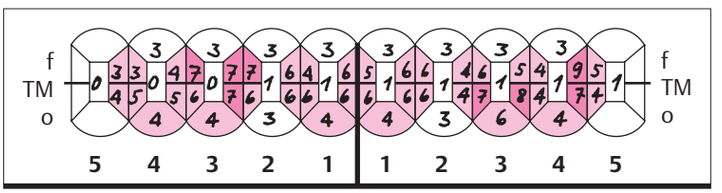
Optimum results will only be achieved by the *collaboration* among dental *specialists* in the appropriate disciplines.

A 38-year-old female with aggressive periodontitis presented with her desire for esthetic relief in the maxillary anterior segment (long and wandering teeth; irregular diastemata and large interdental spaces). Treatment plan: 3-part—

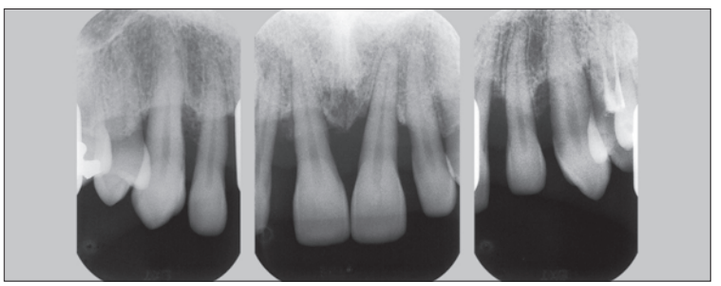
1 *Periodontitis therapy* to reduce probing depths
2 *Orthodontics*: Adjusting the anterior teeth axes to esthetic proportions and creating esthetic interdental relationships
3 *Prosthetics*: Widening of the anterior teeth and closure of the interdental spaces by means of veneers (*papilla replacement* via "long" contact areas).



1225 Initial Clinical View
When she smiled, the patient was bothered by her long, narrow teeth and the large, wide and irregular "dark spaces" (negative spaces) between her anterior teeth. The course of the incisal line was also unharmonic. These esthetic defects created a tremendous psychological problem for the patient, to the point that she tried never to even open her mouth or laugh in public.



1226 Initial Pocket Probing Depths and Tooth Mobility
Interdental probing depths in the esthetically sensitive area of teeth 14–24 were measured to 9 mm. Surprisingly, these teeth were not highly mobile (TM grade 1).



Initial Radiographic View
Horizontal and vertical interdental osseous defects, in some cases extending beyond ½ of the root length.



1227 Following Initial Periodontal Therapy
Following Phase 1 and Phase 2 initial periodontal therapy the condition was stable. Risk factors (genetic, hereditary) are not known. The patient exercised good oral hygiene, which was monitored during initial periodontal maintenance therapy. The next treatment phase is the closure of interdental spaces via orthodontics.

Courtesy *P. Magne, B. Dubrez*

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1228 Following Stage 2—the Orthodontic Treatment
Using fixed orthodontic apparatus, the maxillary anterior teeth were moved such that the interdental spaces were identical in dimension.
In this present clinical condition it will be possible to treat the unproportional teeth using naturally formed and ideally proportioned veneers, which will also close the interdental spaces.

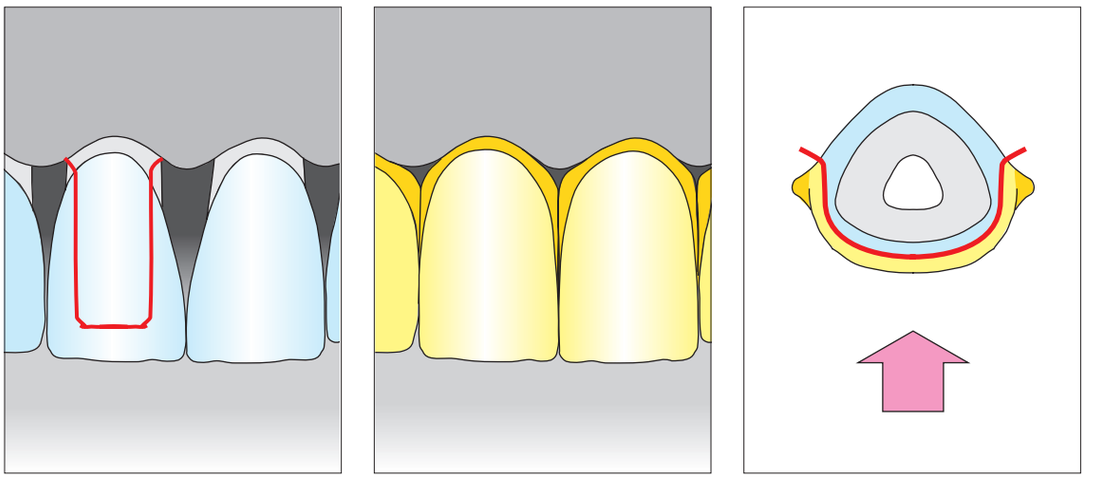
Orthodontic therapy courtesy *A. Darendeliler*



1229 Diagram of Treatment Planning for the Specially Shaped Veneers (Laminates)
Left: Preparation margins, path of insertion from the *facial*!

Middle: Seated, specially fabricated veneers with natural tooth shape (yellow): Darkened "wings" (orange) form the interdental contact *surfaces* and prosthetically fill the dark interdental triangles!

Right: Prepared tooth and veneer with "wings" (orange), arrow: Path of insertion.



1230 Stage 3: Prosthetic Preparation, Clinically
Teeth 13–23 have been prepared to receive the veneers. The incisal edges have been significantly shortened, the preparations proximally extend relatively far palatinally. The facial margins are near to the gingival margin.

Right: Special diamond burs to create a precise preparation margin at various levels.

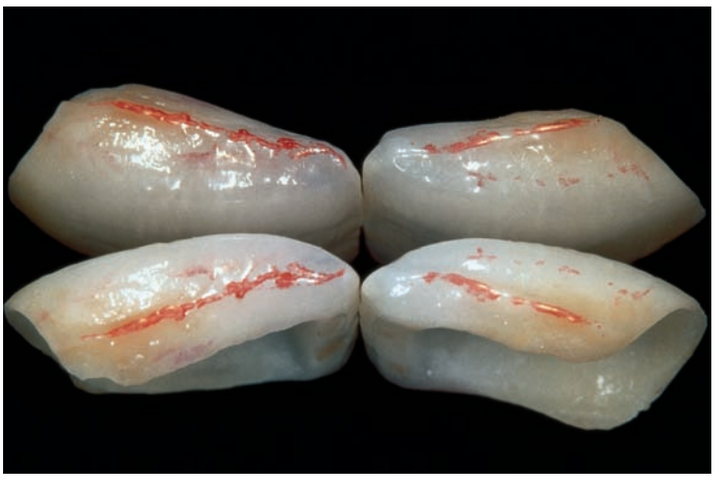


1231 Cervical View of the Laminates for Teeth 11 and 21, in Mirror View
This detail photograph clearly shows the pronounced widening of the teeth and also the approximal "wings" (to eliminate the visual effect of the dark interdental regions).
Clearly visible also is the long contact line, which extends from the incisal edge almost to the gingival margin, and then appears to form a flat "papilla" (cf. Fig. 1233).

Courtesy *P. Magne*



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1232 Proximal View of the Laminates for Teeth 11 and 21, Mirror Image
The red markings clearly show the long proximal contact lines. The most cervical segment of this line approaches the maximum distance of 4–5 mm from bone, as described by Tarnow (Fig. 1224). This innovative prosthetic measure virtually guarantees filling of the interdental space with a "permanent" gingival papilla.





1233 Laminates *In Situ*
Esthetics is clearly optimum. Of special note is the harmonic course of the gingival margin and the "renewed" interdental papillae ("red esthetics").

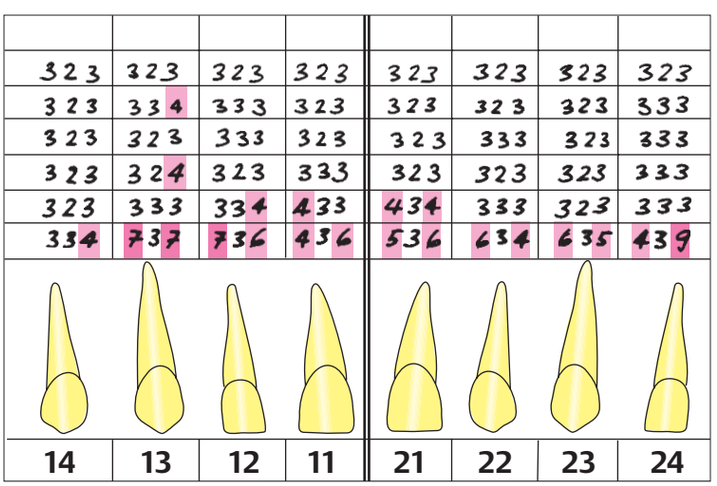
Left: Radiograph of teeth 11 and 21. The osseous fundament has consolidated.
Because of the previous orthodontic treatment, a thin palatal retention wire was incorporated onto the natural enamel, to be worn for an extended period of time.





1234 Final Clinical View—"Smile Line"
The "*white esthetic*" has also been optimally reconstructed: Note the harmonic course of the incisal line, normal tooth proportions, closed interdental spaces. This sensitive woman was now ready to re-enter social society!

Left: The success of this treatment effort is so clearly obvious in comparison to the initial clinical view (Fig. 1225).



1235 Changes in Pocket Probing Depths on Teeth 14–24 during Treatment and in Subsequent Recall
The facial and facial-proximal measurements of probing depths over the course of 9 years reveal a *reduction* of the physiologic values.

Courtesy *P. Magne*

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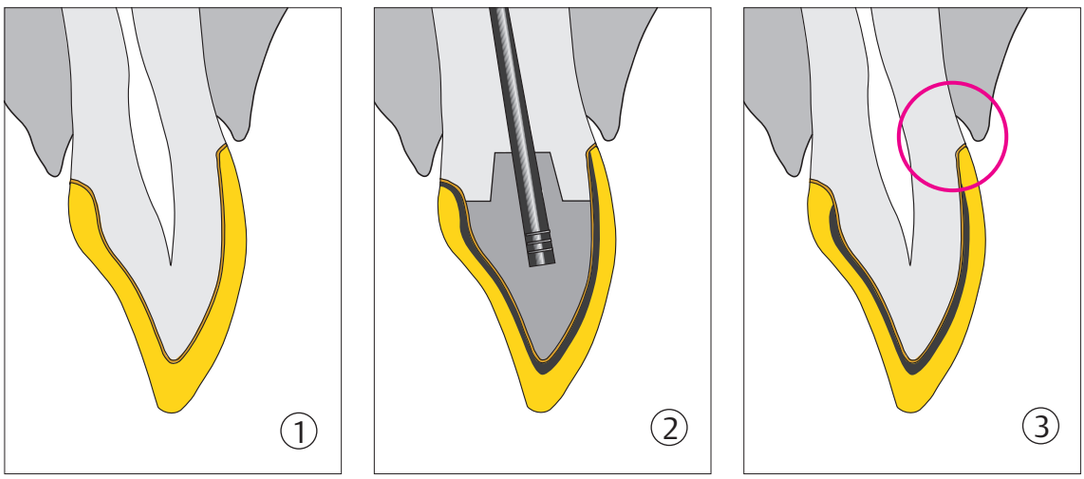
Papilla Loss—Prosthetic Treatment Using Crowns

The classical treatment for severely defective *anterior teeth* remains the crown. Both types—the full ceramic crown and the metal-reinforced crown—have their indications (Edelhoff et al. 1998). The semitransparent *porcelain crown* most resembles the nature crown, but several prerequisites must be considered: The natural tooth must not be discolored; it must not have any opaque restorations or opaque cement! The *porcelain-fused-to-metal* crown is indicated for use on *severely stained* teeth or on build-ups (screws, pins), especially in patients with *bruxism*.

Porcelain-fused-to-metal and other more opaque crowns (Al- or Zr-oxides) often exhibit the "umbrella effect," a dark coloration of the marginal gingiva (p. 491). In the anterior area where PFM crowns are unavoidable, it is possible to soften this negative effect by modifying the morphology (e. g., in cases of bruxism). The metal ends 2–3 mm before the preparation margin. This is stabilized with pure ceramic, without any opaquing substance, and using a translucent cement (Magne et al. 1999, Magne 2002).
Case: PFM crown with "wings," in a patient with papilla loss.

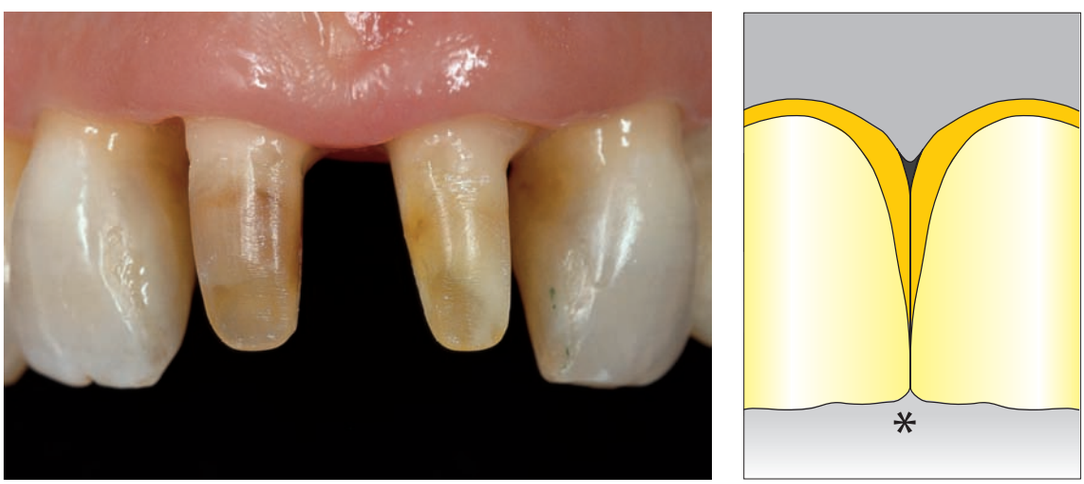
1236 Esthetically Different Anterior Crown Constructions

1 Full porcelain crown with slightly subgingival margins: Optimum esthetics.
2 PFM upon a non-vital tooth; metal pin build-up; the metal framework ends at the preparation margin: "Umbrella effect."
3 PFM upon a vital and non-discolored tooth; metal framework ends ca. 2 mm before the preparation margin; porcelain without opaquing substance at that level, and transparent cement: *No* "umbrella effect." No dark gingival margin.



1237 Prepared Teeth 11 and 21
In a 40-year-old female with parafunctions, the massively discolored yet vital anterior teeth are to be crowned following periodontal treatment. An additional goal is to improve the esthetics by masking the black triangles and missing interdental papillae.

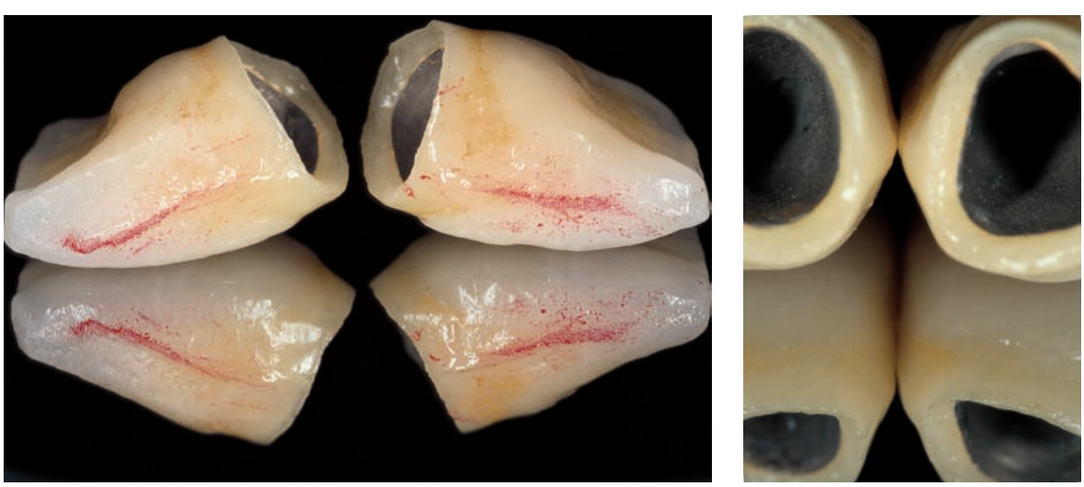
Right: As in the previous case (restoration using veneers, p. 497), the interdental spaces were not eliminated by "prosthetic papillae," but rather with long contact areas between the teeth.



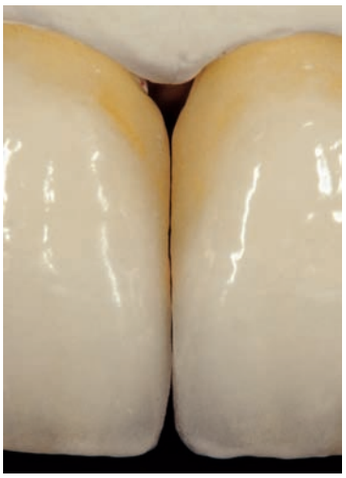
1238 Modified PFM Crowns—Mirror View
The metal portion of the crown does not extend to the preparation margin. The ceramic crown in this "esthetic region" is fully translucent. The red markings delineate the axial contact lines.

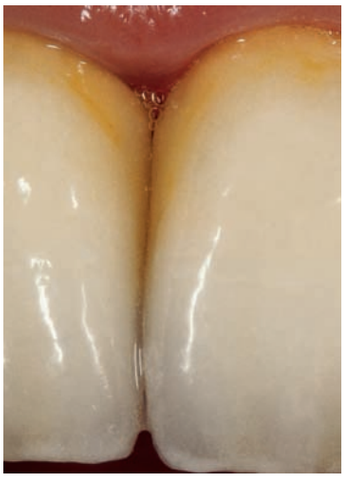
Right: Apical view of the crowns. The reduction of the interdental space results through small "wings" of porcelain (see also Fig. 1229).

Courtesy *P. Magne*



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1239 Contact Lines and Interdental Papilla on the Model (Left) and In Vivo (Right)
Due to the modified shape of the crowns, the interdental space has been narrowed, and the esthetics is not disturbed by the absence of a papilla (see initial view, Fig. 1236). Not only has the esthetic situation significantly improved, phonetics was also improved.



1240 Tooth Shape and Gingival Margin Morphology
Despite the width at the cervical region and the length of the contact zone between the replacement teeth, the prosthetic teeth appear esthetically normal vis-à-vis the remaining natural teeth. Note the translucence in the incisal regions and the coloration, which corresponds optimally to the remaining teeth. The tooth shape remains somewhat triangular and slim, and the somewhat darker interproximal segments are scarcely visible.



1241 Anterior and Canine Regions During Mandibular Protrusion
The restored central incisors articulate with teeth 42, 41 and 31. The patient is no longer even aware of the previous parafunctions.
Note also the healthy periodontal conditions and the tooth morphology in the mandible.





1242 Harmonic Course of the Incisal Line and the Lower Lip
The patient's initial desires for improved esthetics were for the most part fulfilled. The PFM crowns cannot be distinguished from the adjacent natural teeth.

Left: Plaque control must be efficient. It can be performed using regular dental floss, dental tape or "Superfloss."

Courtesy *P. Magne/Quintessence Publishers 1999*

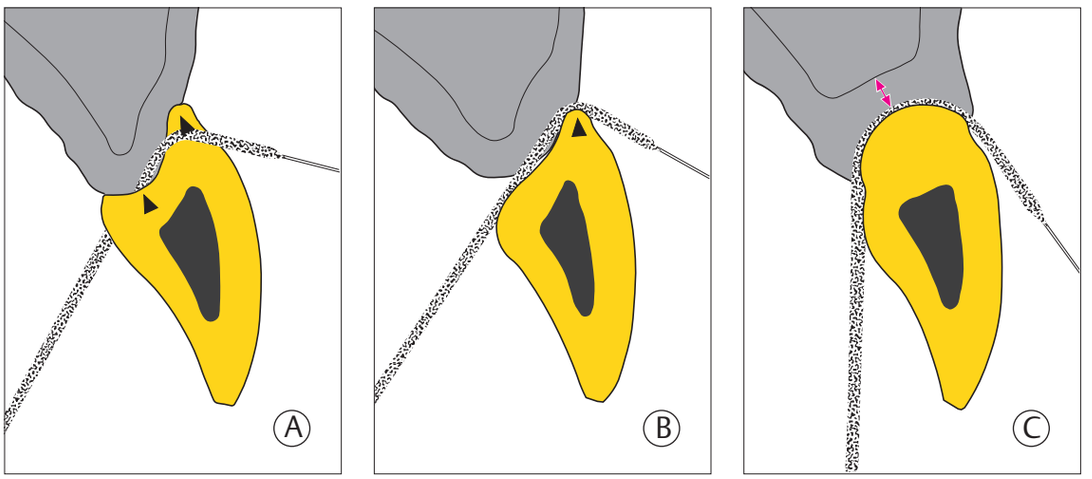
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Edentulous Arch Segments—Pontics

Pontic Shape in the Visible Area

Previously it was always stated that pontics must only have point or line contact with the alveolar ridge for purely hygienic reasons. With today's elevated level of esthetic demand, particularly in the visible anterior segments, this rule has been modified with the presumption that the patient will perform adequate oral hygiene *beneath* the pontic.

Saddle-shaped pontics, "total ridge lap" (Fig. 1243, A), can be applied to almost any alveolar ridge shape. The most difficult plaque control (the undersurface) can be improved by incorporation by the "modified ridge lap" (B; Stein 1966), and dark interdental spaces (papilla loss) can be avoided. The "ovate" pontic (C; Abrams 1981) is "embedded" into the contoured soft tissue, at least 2–3 mm; this solves most of the problems ("emergence profile," hygiene, phonetics etc).

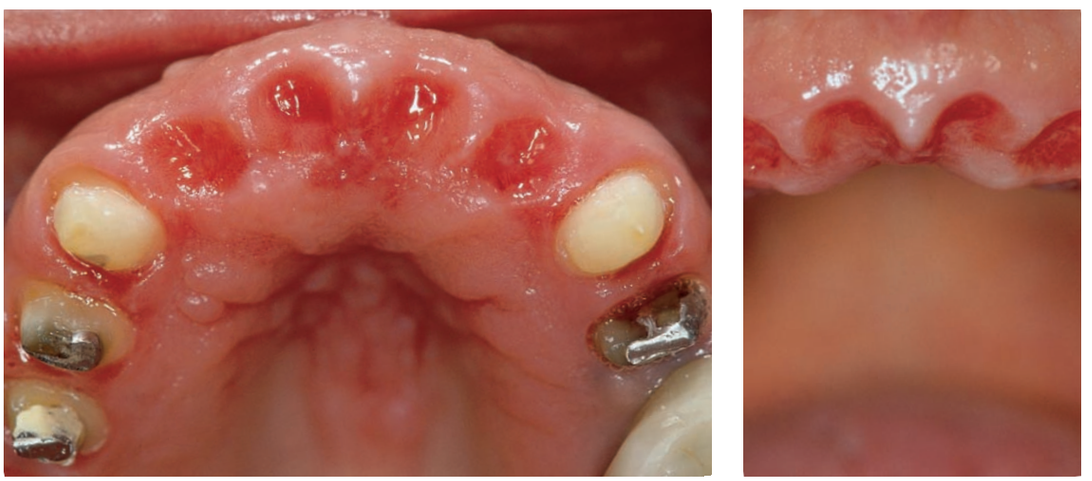


1243 Pontic Shape in the Anterior Region

A "Total ridge lap" is difficult to clean (diagonal use of superfloss).

B Modified "ridge lap" is easier to clean but tends to provide a retentive area for food particles on the palatal aspect.

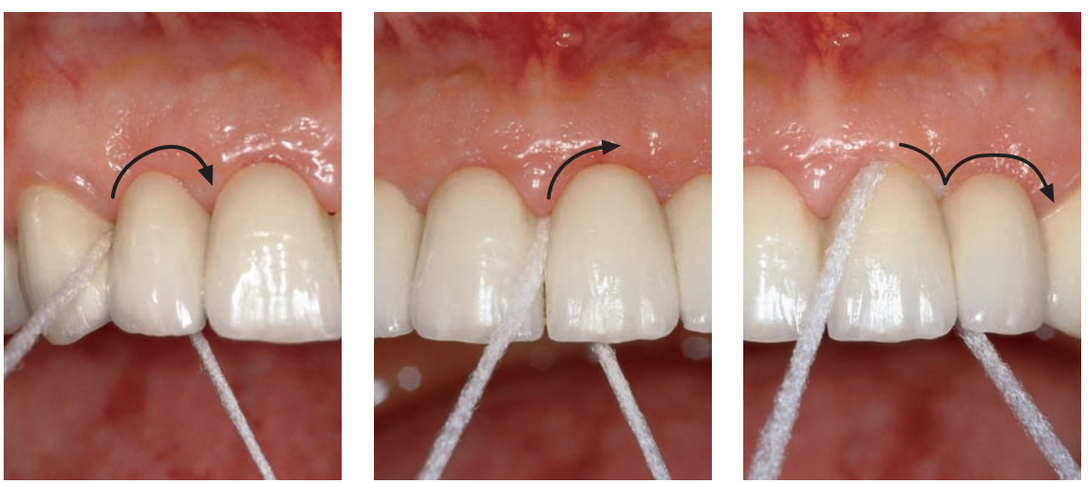
C Ovate can be cleaned optimally and naturally because of the "emergence profile." Ovate pontics, however, require a wide alveolar ridge with mucosa that is at least 2 mm thick (red arrow).



1244 Clinical View immediately after Removal of a Temporary Bridge with four Ovate Pontics in the Anterior Segment

By using a temporary bridge, the ovoid depressions in the soft tissue of the alveolar ridge consolidated. This type of pontic formation of "interdental" new papillae is effective (see also *right*). The pontics appear to protrude naturally from the mucosa ("emergence profile").

Today, this type of pontic is the best functional and esthetic solution.



1245 Plaque Control with Ovate Pontics

Same patient as above with the new and definitive PFM anterior bridge reconstruction.

In such cases superfloss is the ideal method for oral hygiene. Once inserted between abutments 13 and pontic 12 (*left*), the interdental spaces and the ovoid fundus of the pontic can be easily and optimally cleaned (*middle*, between teeth 11 and 21; *right*, beneath pontic 21).

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Alveolar Ridge Defects—Classifications

Alveolar ridge defects that result from trauma, tooth extraction or periodontal disease often require periodontal surgical correction *before* prosthetic treatment. If dental implants (p. 511) or fixed bridgework are planned, such ridge defects must be carefully evaluated and classified, and corrected if indicated. A classification of alveolar ridge defects has been proposed (Seibert 1983, Allen et al. 1985, Wang & Al-Shammari 2002):

- *Horizontal defects* H
- *Vertical defects* V
- *Combination defects* C

Of course, smaller defects, especially in the less esthetically sensitive posterior segment, can remain untreated and/or be covered by a *removable prosthesis*.

Especially in the esthetically important anterior arch segments, any surgical endeavor will require *implants* (osseous build-up) or fixed bridge constructions (soft tissue build-up; p. 505) for the placement of *pontics*. Any failures or mistakes in any of these procedures will lead to unacceptable structural, functional and above all esthetic compromises.

Alveolar ridge defects – qualitative classification		*Seibert* nomenclature, 1983	*Allen* nomenclature, 1985
Horizontal	Labiolingual defect with normal ridge height	Class I	Type B
Vertical	Coronoapical defect with normal ridge width	Class II	Type A
Combination	Horizontal + vertical ridge defect	Class III	Type C

Alveolar Ridge Defects

1246 Qualitative Classifications Based on Expanse of the Defect
Summarized from Seibert (1983) and Allen et al. (1985):

- **Class I (type B)** 33 %: Cases
- **Class II (type A)** 3 %
- **Class III (type C)** 56 %
- no defect 9 %

Modified from *Chen & Schärer 1995*

Alveolar ridge defect – semiquantitative classification			
Horizontal degree of severity		Vertical degree of severity	
Measurement vis-à-vis arch curvature	Nomenclature	Measurement vis-à-vis adjacent papilla tips	Nomenclature
< 3 mm	Mild	< 3 mm	Mild
3–6 mm	Moderate	3–6 mm	Moderate
> 6 mm	Severe	> 6 mm	Severe

1247 Semi-Quantitative Classifications
Studer (1996) classified alveolar ridge defects according to their severity with relationship to *dental arch curvature* and the *adjacent papilla tips*.
Using this classification, the volume of the defect and the necessary augmentation can be determined, which is not possible with the classification above (Fig. 1246).

Details of prognosis			
A Defect classification	Prognosis	B Expanse of the defect	Prognosis
Class I	++	Single-tooth defect	++
Class II	+	Two-tooth defect	+
Class III	+/–	Three-tooth defect	+/–
		Four-tooth defect	–
C Horizontal defect	Prognosis	D Vertical defect	Prognosis
Mild	++	None	++
Moderate	+	Mild	+
Severe	+/–	Moderate	+/–
		Pronounced	–

1248 Expected Success with Soft Tissue Augmentation
Based upon the various criteria—dimension, expanse of the horizontal and vertical tissue loss—it is possible to establish a prognosis; the mildest and simplest ridge defects are attended by the highest predictability of success.
The more expansive the ridge defect (number of missing teeth), the less is the chance of success using *soft tissue augmentation* (Fig. 1255).

Modified from *S. Studer et al. 1996*

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Alveolar Ridge Defect—Prosthetic Solution

As previously stated, the simplest approach to compensating for a ridge defect is the preparation of a cast-metal framework partial denture. More difficult and costly but esthetically superior are fixed or fixed/removable bridge constructions, e.g., upon copings or bars.

It is also worthy of mention that surgical ridge augmentation (soft tissue) and prosthetic approaches can be combined when, for example, the surgical attempt at ridge augmentation does not lead to the expected level of success.

In the 38-year-old patient depicted below, the esthetic problem (missing teeth 11 and 21) was solved with a *removable prosthesis* following insufficient soft tissue augmentation of the ridge:

- Abutment teeth 12 and 22, with minimally invasive, adhesive and precision attachments for the pontic
- Central incisors as pontics with natural length and width
- "Gingival mask" made of porcelain, a customized and color-coordinated separate prosthesis

The entire construction is removable for optimum hygiene, and the visual esthetics were optimum.



1249 Defect and Replacement
The missing maxillary central incisors were replaced as a double pontic, using precision attachments to the two lateral incisors. The esthetic result was lacking (missing interdental papilla between 11 and 21; dark triangle); phonetic difficulties also made this simple restoration unacceptable.



1250 Double Pontic with Separate Porcelain Gingival "Mask"
The gingival mask was formed from two removable portions, to fill the interdental space between the two pontics and the ridge defect. An optimum result with minimal expense; a simple solution but requiring careful and vigorous plaque control (removable portions).



1251 The Combined Reconstruction *In Situ*
The final result gives no visible clue that a prosthesis is in place. The central soft tissue defect ("black triangle") is completely covered by the gingival mask, and even the spaces between both centrals and laterals are covered. The course of the "gingival margin" over the central incisors corresponds perfectly to the esthetic principles (Belser et al. 1981); the gingival mask corresponds harmonically to the natural gingiva. Courtesy *C. Augustin*

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Alveolar Ridge Defects—Surgical Corrections—Methods

Alveolar ridge defects in the anterior regions can be augmented with *soft tissue* to better accommodate bridge pontics.

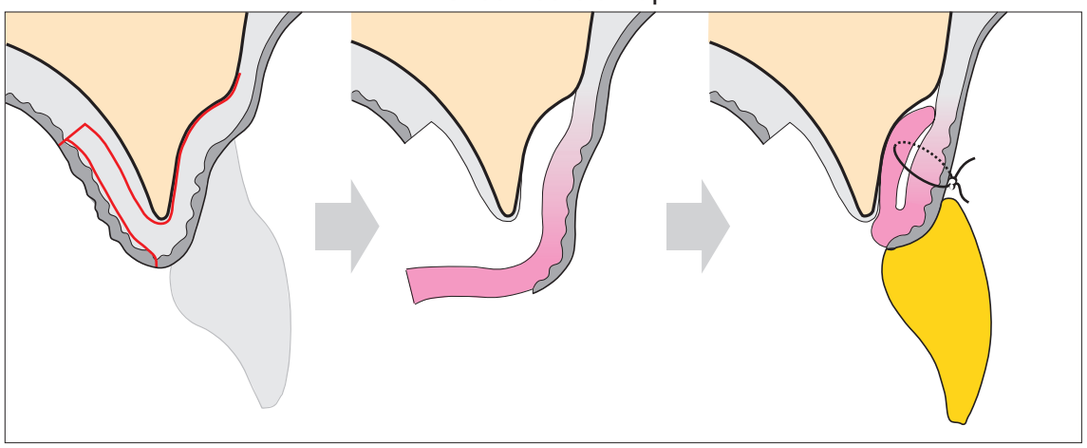
Techniques for Soft Tissue Augmentation

- *Rolled flaps* (Abrams 1980)
  Indication: Class I defect, minor severity, single tooth region
  Contraindication: Palatal mucosa too thin
  Advantages: Good esthetics; only one surgical site
  Disadvantage: Limited expanse

- *Subepithelial connective tissue graft* (Langer & Calagna 1982)
  Indication: Class I, II and III defects
  Contraindication: Thin palatal mucosa (donor site)
  Advantages: Good esthetics, minimal risk, no epithelial palatal defect, large tissue volume

- *Onlay transplant* (Seibert 1983a)
  Indication: Class II, but also Class I and III defects
  Advantage: Large graft volume
  Disadvantage: Deep palatal wound

Rolled Pedicle Flap

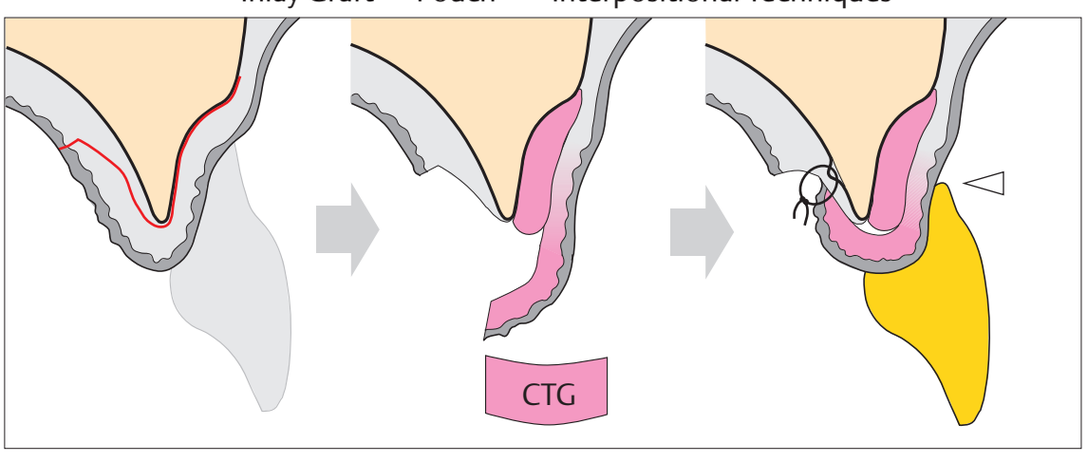


1252 Rolled Flap Technique
Left: De-epithelialization of the palatal mucosa and incision for the soft tissue flaps.

Middle: Mobilization and reflection of palatal and facial flaps (supraperiosteal soft tissue pocket).

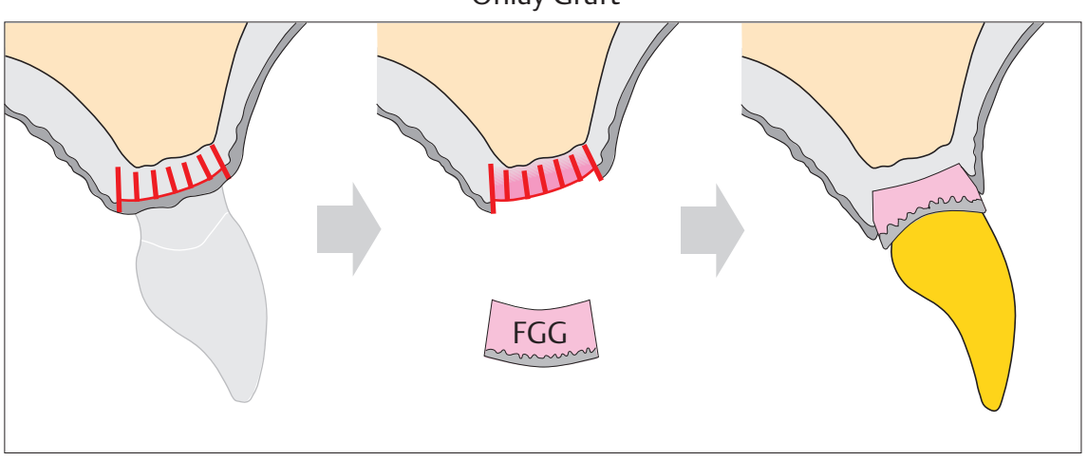
Right: Tucking the palatal flap into the facial "pocket." Suture fixation. Secondary healing at the palatal wound; periodontal dressing.
Caution: The adjacent papillae must *not* be lost!

Inlay Graft – "Pouch" – "Interpositional Techniques"



1253 Connective Tissue Transplantation—"Pouch" Technique ("Envelope Pouch")
Left: Creation of a large, thick, trapeze-shaped split-thickness flap from the facial aspect and distally onto the palate (lateral vertical incisions and palatal horizontal incision).
Middle: The connective tissue harvested from the *lateral* aspect of the palate is placed upon the periosteum and affixed with sutures.
Right: The split-thickness flap covers the tissue graft.

Onlay Graft



1254 Onlay Graft Technique
Left: The de-epithelialized recipient bed is incised several times in order to guarantee nutrition and revascularization of the graft. Partial failures are relatively frequent!

Middle: Recipient bed and FGG.

Right: FGG *in situ*. The supportive temporary must not apply pressure to the graft during the postoperative phase (*Caution*: swelling).

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Ridge Augmentation Using Partially Epithelialized Connective Tissue Graft

Surgical Procedure Step-by-Step

There exist various techniques to restore deep alveolar ridge defects following anterior tooth loss:

- Removable appliance that fills the ridge defect with acrylic
- Tooth replacement with a cast framework partial denture (inexpensive, easy) or a coping bridge construction (expensive)
- Soft tissue augmentation with subsequent tooth replacement via a fixed bridge
- Bone augmentation and dental implants

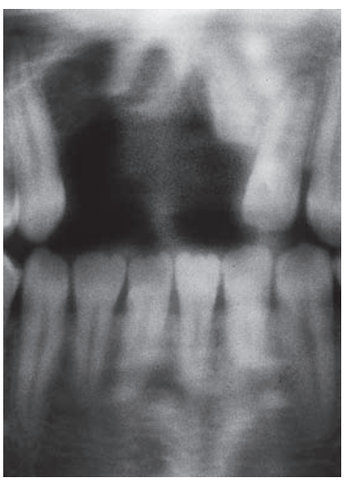
The choice of which method to use will depend upon the extent of the ridge defect, the planned definitive dental reconstruction, the prognosis, the patient's desires and financial possibilities, as well as patient compliance.

In the case depicted here, a 19-year-old patient presented with missing maxillary anterior teeth and a severe alveolar ridge defect following a moped accident. The treatment plan included soft tissue ridge augmentation and subsequent fixed bridgework; the expansive maxillary ridge defect was of Class III (Seibert 1983, p. 505).

1255 Preoperative Clinical View
Six months following the accident: The anterior teeth were lost; the ridge defect is moderate in the horizontal direction but severe vertically.
During the healing phase, the patient wore a simple, removable, clasp-retained temporary denture.
Planning: Ridge augmentation with soft tissue, combined inlay-onlay graft technique.

Right: Section from the panoramic radiograph. Note the expansive osseous defect!

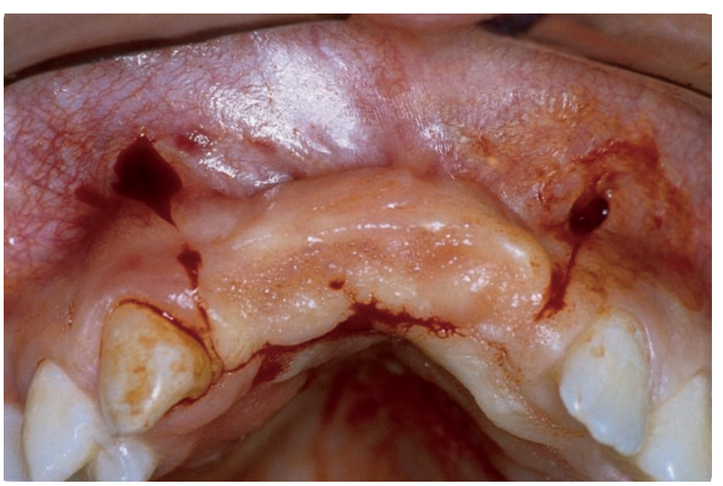


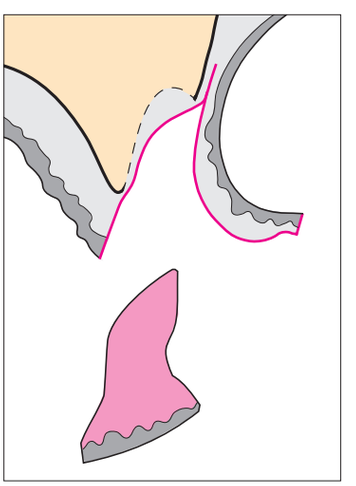


1256 Incision Lines
Horizontal incision 2–3 mm *palatal* to the alveolar crest.

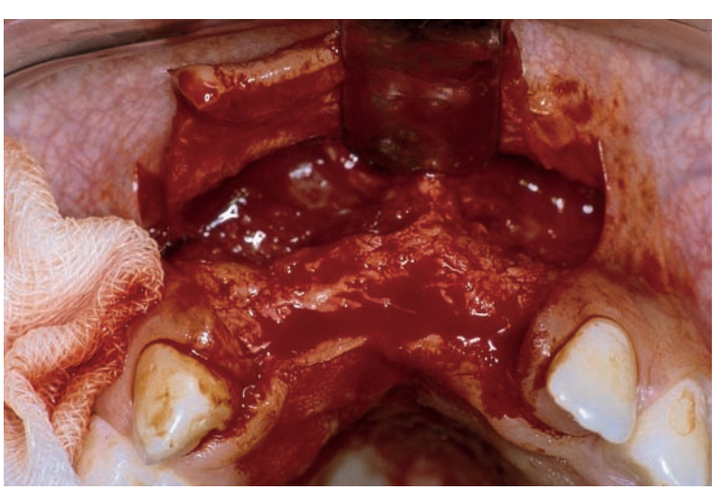
Vertical incisions into the mobile labial mucosa. The papillae adjacent to teeth 13 and 22 are protected and maintained.

Right: View in orofacial section after creating the split-thickness flap. The planned, large, wedge-shaped graft that will be taken later from the palate is smaller at its base than at the height of the planned new augmented ridge.



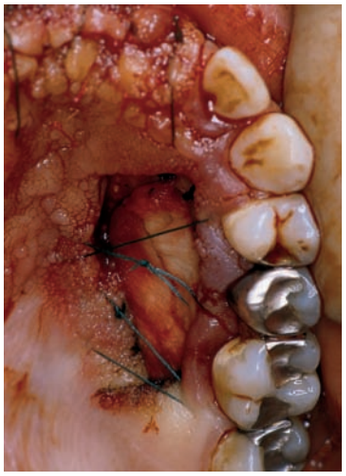


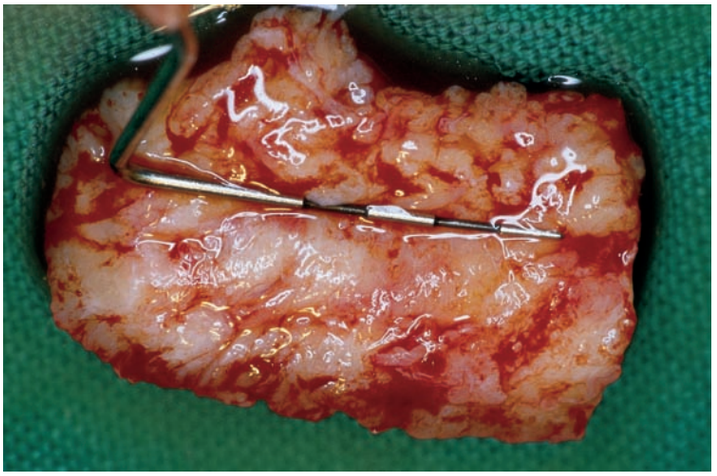
1257 Phase 1: Flap Reflection
Because of the old scar tissue and irregular osseous contour, it was relatively difficult to prepare the split-thickness flap. It is temporarily mobilized and left *in situ*, so that the surgeon can undertake phase 2 of the procedure, the removal of the graft from the palate.



Case courtesy of
S. Studer & A. Adler

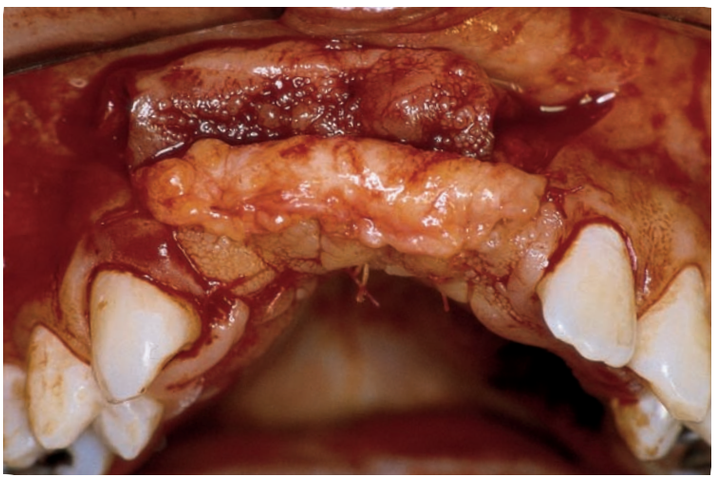
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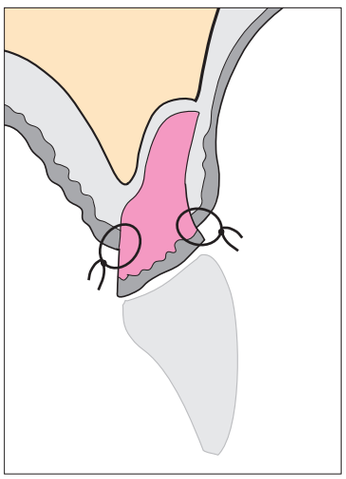


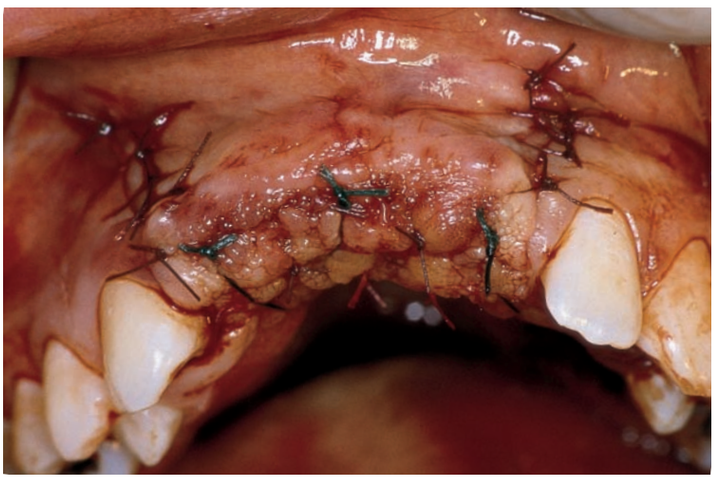
1258 Phase 2: Harvesting the Soft Tissue Graft
Large, *partially* epithelium-covered transplant from the region of 22–26 on the palate. Lipid tissue was retained in this case of ridge augmentation (without any supportive function!).

Left: Sutures to secure the donor site. An acrylic plate was also prepared to cover the wound.



1259 Phase 3: Graft *In Situ*
The horizontal and vertical aspects of the large defect extending over three teeth is completely filled by the graft. It is affixed palatally with *resorbable* sutures. Although it is not always possible, resorbable sutures should be used so that the “take” of the graft will not de disturbed during suture removal.





1260 Immediately after the Surgery
The split-thickness flap that was created initially is repositioned over the non-epithelialized portion of the graft, and its keratinized mucosa has been affixed to the margins using non-resorbable sutures. The vertical incisions are sutured and the patient is instructed concerning prevention of swelling.
Left: The facial flap covers the non-epithelialized portion of the palatal graft, while its epithelialized aspect is free.

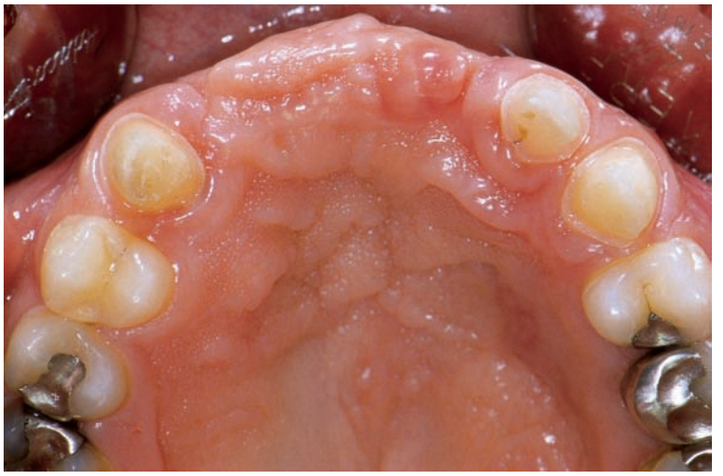


1261 Occlusal View, 8 Days Post-Surgically
Primary healing is almost complete. The graft was intentionally somewhat over-sized in order to compensate for the expected shrinkage (ca. 20 %).
For 14 days after the surgery, the patient rinsed with CHX solution, and subsequently used CHX spray at the surgical site.
It is imperative that the temporary denture exert no pressure on the surgical site.

Courtesy *S. Studer* & *A. Adler*

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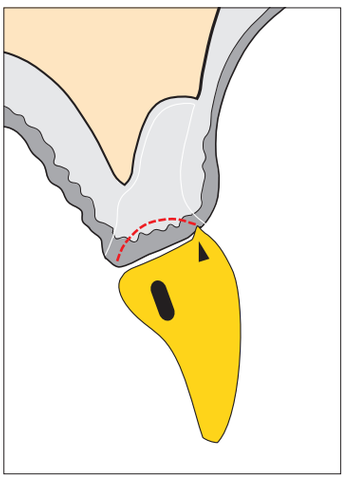
1262 Clinical, Occlusal View Two Months after the Surgery—Pre-Prepared Abutment Teeth
The edentulous ridge tissue has healed completely. The alveolar ridge exhibits almost physiologic shape and form both horizontally and vertically, so that the bridge pontics can be arranged in a normal dental arch form.
Teeth 13, 22 and 23 have been prepared to receive the initial *fixed temporary*.



1263 Clinical View Nine Months Later
The pontics (modified ridge lap) only lightly contacted the soft tissue during the maturation phase of the ridge augmentation procedure, and only on the facial aspect (phonetics, esthetics).

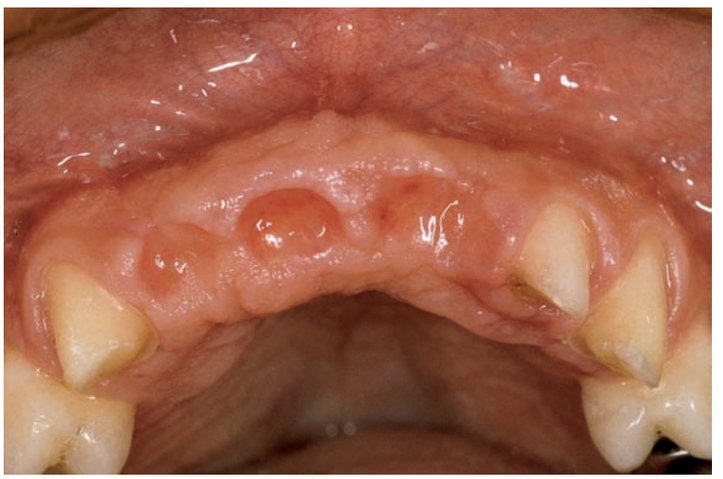
Right: The diagram shows the profile of the pontics. The dashed red line indicates the planned modification of the soft tissue to receive the ovate pontics on the minimally 3 mm thick alveolar ridge tissue.





1264 Surgical Modification of the Soft Tissue Ridge
During a minor surgical procedure, small depressions in the ridge are created for the three pontics; these re-epithelialize after only a few weeks (photograph). This provides a situation whereby the ovate pontics appear to naturally emerge from the ridge, and simultaneously new “papillae” are created.

Right: Instruments for creating soft tissue depressions: 5 mm diamond bur or electrosurgical loop.

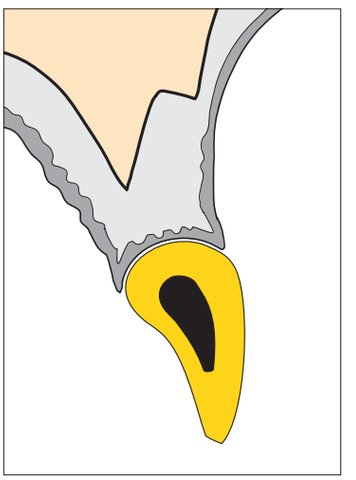




1265 Definitive Bridge Reconstruction—Two Years Following Surgery
The gingival margin has assumed a normal-appearing clinical course and the *emergence profile* of the pontics is ideal. The interdental spaces are for the most part filled with new papillae and in several sites with long interdental contact surfaces of the pontics (cf. Fig. 1262).

Right: The diagram reveals the easy-to-clean, ovoid shape of the pontics (PFM reconstruction).





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Ridge Defect—Build-up with Connective Tissue Graft—Summary

Ridge augmentation can be performed in various ways. The choice depends upon the planned reconstruction (prosthesis, bridge, dental implant). In addition, patient factors such as readiness for surgery, compliance, and esthetic expectations may play a determining role.

While for the osseo-integration of dental implants, bone *augmentation* is necessary, for fixed bridge constructions *soft tissue augmentation* is a satisfactory solution, particularly when working in the visible anterior region with ovate pontics ("emergence profile").

This type of soft tissue ridge augmentation, however, is limited in terms of expanse and volume (donor site!).

In the case of this 19-year-old patient, these prerequisites were certainly achieved. The very large defect, in both vertical and horizontal directions, demanded a very expansive soft tissue graft, and the healing was not completely predictable. In addition, the removal of such a large piece of tissue from the palate is not without its own problems, and is also difficult for the patient. Additional contouring and/or filling *surgical procedures* are often necessary.









(Fig. 1266 L/R, Fig. 1267 L/R)

Treatment Planning Considerations: Fixed Bridge or Dental Implant?

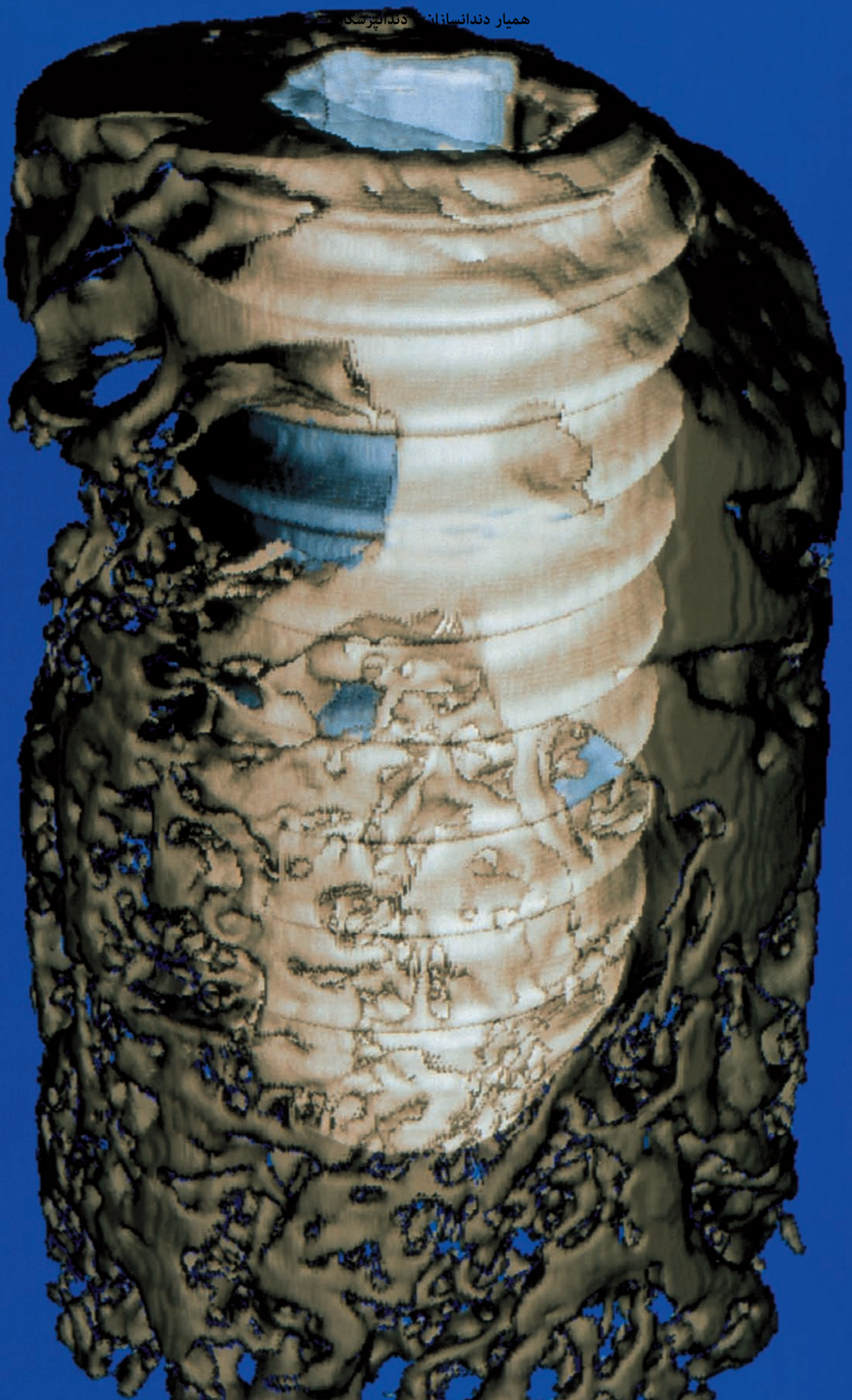
The dental stone models clearly show the expanse of the combined vertical (above, left) and horizontal (above, right) ridge defect: Class 3 (Seibert 1983). The clinical view at 2 months (below, left) and 9 months (below, right) reveal the risks involved in the case (expanse, magnitude). Almost in spite of the results of the comprehensive case analysis with a considerably less than predictable prognosis, the choice was made to provide this young man with a fixed bridge construction after expansive soft tissue ridge augmentation.

The alternative was a dental implant-borne construction, but in this case there were several *disadvantages*:

- *Male, age 19*: Jaw growth and tooth eruption not yet complete? Compliance?
- *Duration*: Healing after guided bone regeneration (GBR) and implant placement (maturation of the new bone); numerous temporaries?
- *Surgery*: For purely esthetic reasons (anterior segment) usually several surgical procedures are necessary (vestibuloplasty? connective tissue graft before GBR?)
- *Esthetics*: Possible problems with tooth position, "emergence profile," papilla build-up? Umbrella effect (p. 491).

Periodontology and dental implants: see next chapter.

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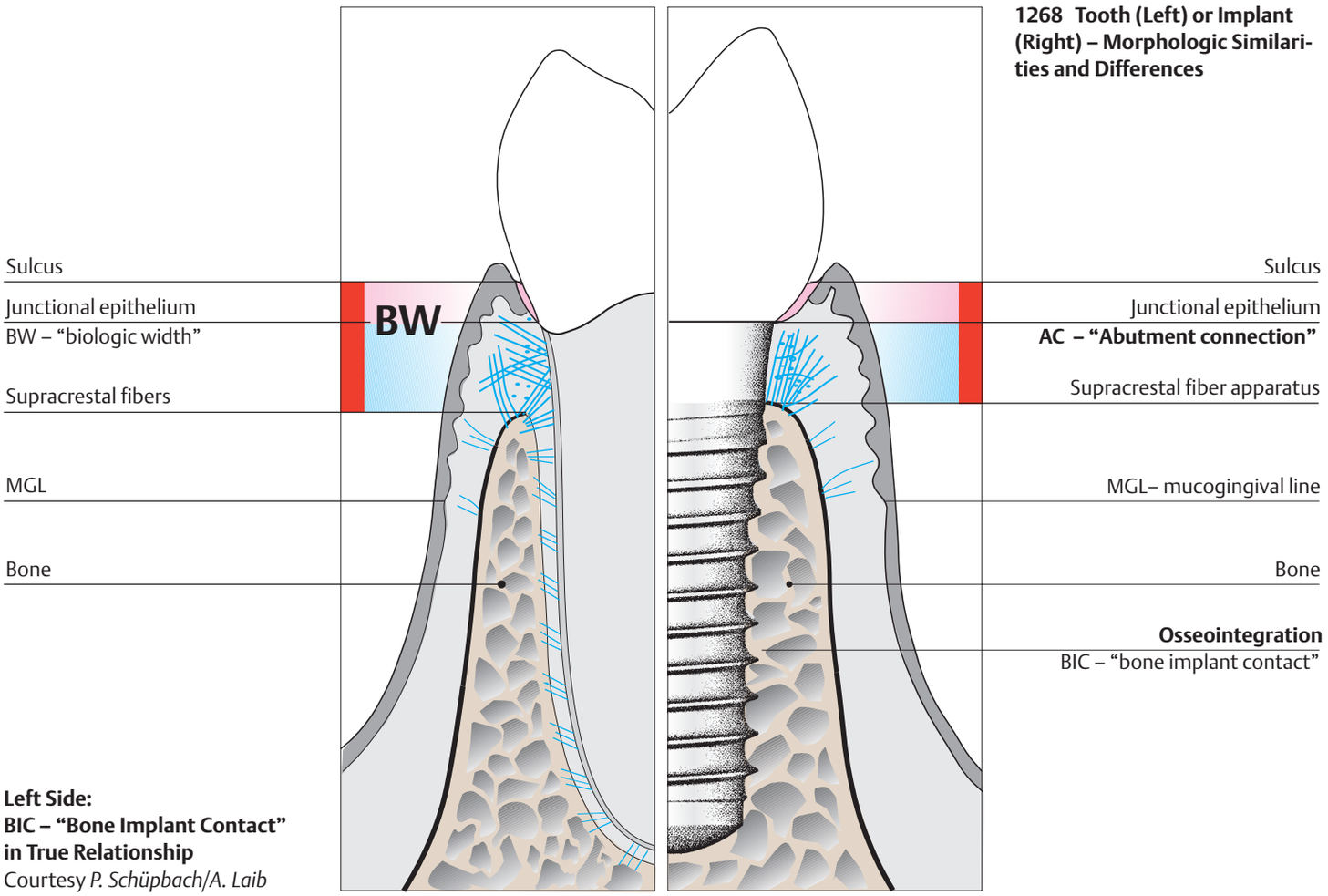
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Dental Implants—Implant Therapy

Natural Teeth or Dental Implants for the Treated Periodontitis Patient?

The increasing significance of dental implant therapy, also in periodontics, is no longer news (Nevins & Mellonig 1999, Lindhe et al. 2003). The comprehensive coverage of dental implant therapy appears in Volume 10 of this *Color Atlas of Dental Medicine series* (Spiekermann et al. 1994). This chapter will deal exclusively with some of the peculiarities of implant therapy in periodontitis patients who have been successfully treated.

In Figure 1268, a natural tooth and an osseointegrated, root-form titanium implant are depicted. Obvious differences are visible in the area of the bone (periodontal ligament vs. osseointegration) and with additional anatomic characteristics, but these are of minimal significance compared to possible differences between a periodontally compromised patient versus periodontal health. The question remains: Maintain a tooth or replace it with an implant? The decision will involve dental specialists who collaborate in diagnosis leading to proper treatment.



1268 Tooth (Left) or Implant (Right) – Morphologic Similarities and Differences

Left Side:
BIC – "Bone Implant Contact" in True Relationship
Courtesy *P. Schüpbach/A. Laib*

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Determinative Diagnostic Criteria

Tooth Maintenance or Dental Implant?

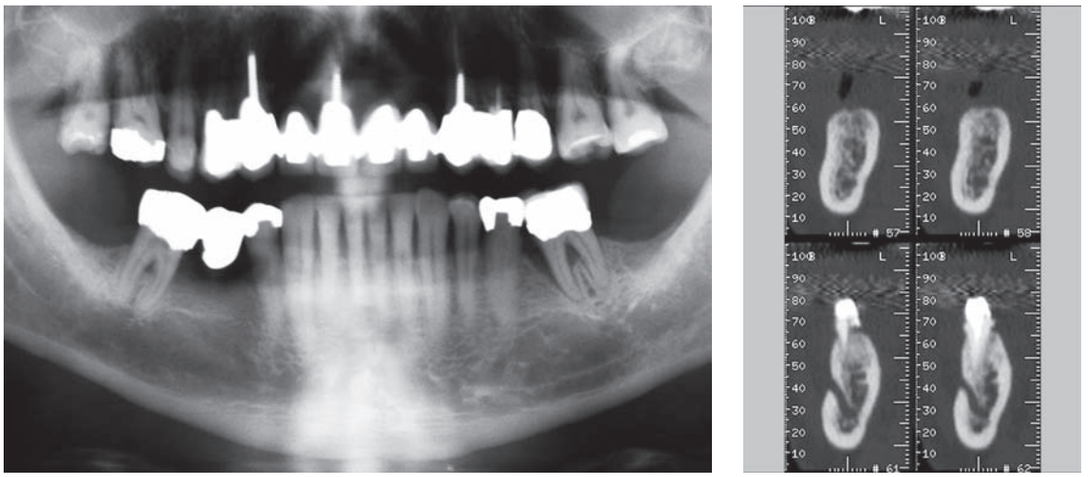
This question can only be answered on a case-by-case basis. In healthy patients, implant success rates of 99 % after 15 years have been reported (Lindquist et al. 1996). In patients suffering systemic disease, as well as those manifesting aggressive periodontitis, the success rate is much lower. Thus the predictability of success is also reduced, and there can be no "guaranteed" prognosis. Local risk factors (oral hygiene, compliance etc.), and above all general systemic risks (smoking, diabetes, osteoporosis, hematologic disorders) must be definitively characterized and diagnosed before making the decision about whether a tooth should be maintained "at all costs" (van Steenberghe 2003).

These decisions demand careful examination of the general medical history and all specific clinical data. It remains within the competency of the dentist to interpret the numerous aspects of each case in order to determine the appropriate and long-term functional treatment plan.

Maintenance of natural teeth
Not only in dental implantology, but also in regenerative periodontal therapy, enormous advances have been made. If periodontal therapy—alone—can lead to success, implant therapy is not indicated; this is especially true for teeth in the "risk areas" such as the maxillary posterior segment (maxillary sinus) or mandibular molars (mandibular canal/mental foramen).

If a prospective abutment tooth requires initial endodontic treatment, post build-up etc., it is important to consider and compare the length of treatment, the costs and the prognosis in comparison to dental implant therapy. Especially in such cases, the well-informed patient must participate in the decision.

Replacement via dental implants
In many partially edentulous patients, such decisions are not even necessary; the decision is simply between fixed bridgework including dental implants, or a removable partial denture.
Teeth that exhibit severe periodontal compromise and which are in danger of progressive disease are better extracted early on in order to preclude extensive loss of the osseous fundament that will be necessary for subsequent dental implant therapy.



1269 Panoramic Radiograph of an "At-Risk" Patient with Periodontally Involved Mandibular Molars Bilaterally
Questions: In this advanced case (risk factors: smoking, oral hygiene), should regenerative periodontal surgical therapy be undertaken or should dental implants be placed following extraction? *Right*: More complex diagnostic measures provide information about anatomic structures (jaw dimension, alveolar ridge width, mandibular canal etc.). CT (jaw cross section in a Dentascan).

Diagnosis before Dental Implant Therapy

Before making a definitive decision in favor of dental implants, the identical clinical examinations and findings must be collated as for all periodontitis patients (p. 165). Additional radiographic examinations (CT scans) provide information in complex or risk-rich cases concerning shape, structure, and thickness of the alveolar ridge, as well as concerning special structures such as the maxillary sinus, mandibular canal, persistent areas of ostitis, thickness of the compact bone layers and therefore indirectly concerning the bone *quality* (Lekholm & Zarb 1985).
Simultaneously, the prosthetically relevant parameters as they relate to the set-up plan and positions of the fixtures can be depicted in the radiographs (templates with marker points). This permits the observation of the implant sites before the surgical procedure.
In cases in which the alveolar process is significantly deformed, the implants may have to be positioned in unconventional areas of the alveolar ridge. Today, implants are not necessarily placed where bone remains, rather in locations where the prosthodontist needs them for her/his crown and/or bridge constructions ("backward planning"); additional surgical procedures may be necessary.

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Therapeutic Concepts—Therapeutic Results

Treatment Planning—Planning Problems

The prosthodontic concept of modern dental implant therapy is associated with the fact that implants must often be placed in areas where alveolar bone is minimal. Today there are numerous ridge-augmentation procedures that can be performed before placement of dental implants or, under certain conditions, simultaneously.
All of these surgical procedures can only be attempted with great care in *periodontitis-prone patients*. Above all the risk factor "heavy smoking," in combination with poor oral hygiene and compliance, as well as other risks—diabetes, IL-1 and other gene polymorphisms that lessen host response—must be considered when planning such surgical procedures.

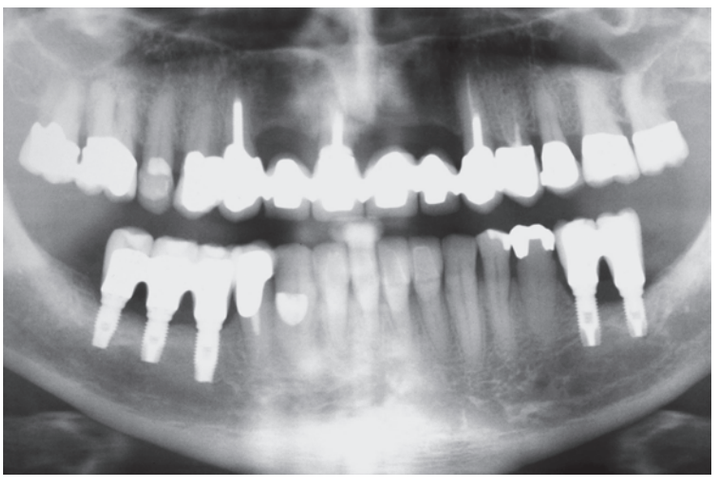
Patients today demand not only masticatory function, but also esthetically satisfying treatment, and this makes treatment planning all the more difficult. The dentist must sometimes reduce a patient's utopic thoughts by providing appropriate information and whenever possible using a written treatment plan, including costs and time considerations.

Augmentation Techniques

These surgical procedures are identical for the most part to those of periodontal and mucogingival surgery (p. 295, p. 397).
Soft tissue augmentation should usually be performed before hard tissue procedures. Using connective tissue grafts (CTG, p. 419), tissue augmentation and esthetics can be achieved. This can provide thickening of thin alveolar ridge mucosa so that the surgical site after flap reflection and GBR ("guided bone regeneration") can be closed tightly and without tension.
Even reconstruction of papillae can be accomplished using thick mucosa; the "emergence profile" of an ovate pontic (p. 502) can provide the appearance of a natural tooth.

Osseous Alveolar augmentation for stabilization of the implant bed can, depending on the anatomic situation, be performed *simultaneously* during the seating of the fixture, or as a *second* procedure.
Examples of these procedures include:
- Guided bone regeneration (GBR)
- Osseous block transplants
- Sinus lift procedures
- Distraction osteogenesis





1270 Panoramic Radiograph of the "At-Risk" Patient 3 Years after Implant Placement
Following bilateral extraction of the periodontally involved mandibular molars and a six-month waiting period for healing—without ridge augmentation—5 screw-type implants with micro-etched surfaces were placed.
Left: The oral hygiene compliance was significantly improved, permitting the patient to be placed on a 3–4 month recall schedule. Unfortunately, the patient did not stop smoking!

Long-Term Results—Oral Hygiene

The "chances for maintenance" of natural teeth and dental implants in a partially dentulous periodontitis patient are similar, but are clearly poorer than in a "periodontally healthy" patient. The chances of success depend upon the reaction of the patient (oral hygiene, compliance), her/his risk profile (smoking, clenching and bruxism), and above all the microbiologic colonization of the marginal soft tissues, and the "interface" between the tooth/implant and its surrounding soft tissue. A highly susceptible patient reacts to even a small colonization of shallow pockets with pathogenic microorganisms in a manner that is much less "controlled" than in a healthy patient. In the gingival sulcus and in pockets around dental implants, one finds virtually the same microbiologic flora as around natural teeth. Predominating are *Tannerella forsythensis*, *Peptostreptococcus micros* etc.

Fact: In a partially edentulous periodontitis patient with dental implants, the most important requirement for a favorable long-term prognosis is conscientious and careful plaque control and professional maintenance therapy (recall), as with natural teeth.

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Recall—Management of Implant Problems

Early failures of dental implants often result for purely biologic reasons. Overheating the bone during preparation of the implant bed may lead to necrosis and contamination of the wound bed, and to subsequent infection. Qualitatively or quantitatively insufficient bone, critical primary stability or premature loading can prevent successful osseointegration.
Late failures, on the other hand, in healthy patients are usually of a mechanical (overloading, parafunctions) or technical nature (fracture of implant components). It is different in treated periodontitis patients, where peri-implant inflammatory processes more often occur.

Peri-implant problems must be diagnosed as early as possible, depending upon the recall interval and the type of disease, in order to institute the simplest possible measures to halt the disease process (see CIST classes).
For this reason, at *every* recall appointment, the following biological parameters must be evaluated:
- Plaque accumulation on the implant structures
- BOP of the peri-implant tissues
- Suppuration, initial pocket formation
- Probing pocket depths (PPD)
- Peri-implant bone loss (radiograph)

1271 "Therapy Cascade"—CIST
C Cumulative
I Interceptive
S Supportive
T Therapy
The check-list (right) can be a big help to the dental hygienist; she is, after all, responsible for the recall organization and for early recognition of therapeutic problems. Treatment "package" **A** is initiated in all cases where the disease is *progressing*.
Note: **E** stands for *hopeless*, a virtual synonym for *implant loss*.

PPD mm	PI Plaque	BOP Bleeding	Bone Loss	CIST
<3	–/+	–/+	–	A
4–5	+	+	–	A + B
>5	+	+	<2 mm	A + B + C
	+	+	>2 mm	A + B + C ▶ D
Mobility / pain			Clinical and radiographic findings	E Explantation ?

Cumulative Management—Treatment Classes

A-E in the table (above) represent aspects of treatment that must be enhanced according to the "CIST" classes (Mombelli et al. 2003):

Procedure A—Mechanical Debridement

With inflammation (mucositis and probing depths to 3 mm): Mechanical cleaning of the implant using rubber cup and paste, calculus removal using plastic instruments.

Procedures A+B—Antiseptic Therapy

With suppuration, an early symptom of the destruction of peri-implant tissues, and 4–5 mm deep pockets: Treatment A plus pocket rinsing with CHX 0.2 %; the patient should also use CHX spray or at-home pocket irrigation.

Procedures A+B+C—Antibiotic Therapy

With probing depths above 5 mm and bone loss (radiography → peri-implantitis): Microbiologic tests may be indicated (Mombelli et al. 1987, Mombelli & Lang 1992, Luterbacher et al. 2000): Procedures A+B with additional antibiotic therapy against anaerobic microorganisms (systemic metronidazole, 1 g for 10 days, or topical application of "controlled released drugs," e. g., Atridox etc.).

Procedures A+B+C+D—Regenerative or Resective

With significant bone loss it may be necessary to intervene surgically (D) following antibiotic administration, in order to avoid osseo-disintegration of the implant. The surgical procedure may be resective, or one may attempt a regenerative-augmentive procedure in order to fill the osseous defect, following detoxification of the exposed implant surface (acids, mechanical removal of rough implant layers, "sandblasting" with biocompatible abrasive powders etc.); in extreme cases, re-osseointegration can be tried and may occur (Wetzel et al. 1999). Such more radical procedures may prevent loss of the dental implant.

Procedure E—Extraction, Explantation

Explantation. Pain and mobility indicate loss of osseointegration, a "failed implant." The implant should be quickly removed in order to give the "alveolus" the best possibility for regeneration. Thus, later, at the same site, a new implant can be placed.

Note: Oral hygiene and the absolutely necessary and regular implant follow-up professional care must always be performed using instruments that do not scratch the implant surface. With such routine care, implants can be maintained into old age.

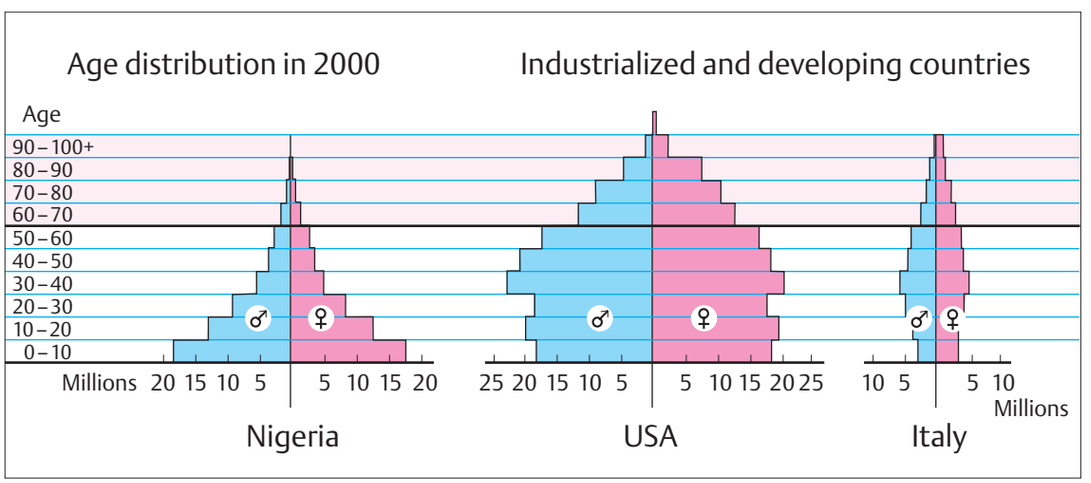
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Geriatric Periodontology?—The Periodontium in the Elderly

Different Circumstances—Modified Concepts of Treatment

The percentage of elderly individuals within the entire population continues to increase, but differences exist between the "Third World" and the industrialized nations (Fig. 1272).
Problem 1 for dentistry and dental medicine is the increasingly wide chasm between that which is *absolutely necessary* and that which is *possible*. This intriguing development brings with it socioeconomic considerations, technical health insurance matters and medical-ethical dilemmas, the magnitude of which has only been recognized in recent years.
The World Health Organization categorizes our aging population in four classes:

- Aging individuals 45–60 years old
- Older individuals 61–75 years old
- Old individuals 76–90 years old
- Very old individuals 91–100 years old



1272 Age Distribution in a Developing Country and in Industrialized Nations
In the "Third World" (example, Nigeria), one observes a high birth rate resulting in a broad and youthful population pyramid. The USA exhibits a "belly-like" age pyramid, which has resulted from a certain reduction in birth rate and a much higher life expectancy. The Italian curve exhibits the same shape as that of the USA, but is dramatically slimmer because of the significantly lower total population.

It is clear, of course, that the overall state of health of individual persons cannot be considered solely on the basis of age. Much more than age, each individual's physical, psychic and spectral conditions are most meaningful for individual quality of life. Diseases and the medications used to treat them may therefore prejudge dental treatment planning (ASA classification, medical history; p. 212).

Despite the increasing number of elderly persons, fewer complete dentures are fabricated today compared to earlier decades. This is likely due to enhanced knowledge of the etiology and pathogenesis of dental and periodontal structures in the second half of the twentieth century, which has increased enormously. This knowledge has led to comprehensive and intensive prophylaxis, which continues its effectiveness even into old age.

In addition, dental treatment techniques and preventive strategies have continued to develop and to be refined, so that a significantly extended maintenance of the natural teeth is virtually guaranteed.

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Different Circumstances—Different Prerequisites

The population's development toward ever more elderly individuals in industrialized nations obviously has a substantial effect in the dental specialty of periodontics.

In the chapter *Therapy* (p. 201), it was noted that for the treatment of gingivitis, periodontitis and gingival recession, scientifically validated therapeutic concepts now exist.

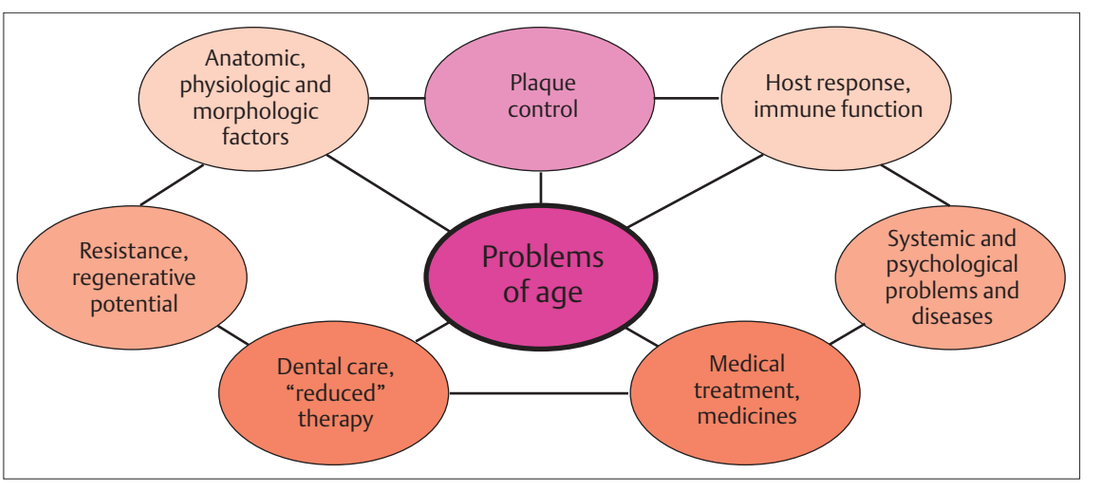
However, with the massive changes in the age distribution of the population, such concepts must be revisited:

Elderly individuals endure somatic and psychic changes, which may force the physician and the dentist to deviate from normal and usual treatment concepts. But this fact must never signify that our elderly and possibly also medically compromised patients should be "poorly" treated; rather, the significance is that these patients must simply be treated *differently* than young and medically healthy individuals.

It is important to note that the aging process does not necessarily reduce all physical and psychic functions and coordinations (Geering 1986):

Capabilities that remain or may even be enhanced:
- Long-term memory
- Experiences
- Ability to learn
- Judgment
- "Interpersonal skills"
- Speech capability
- Psychic and physical capabilities
- Reliability
- Self-satisfaction, compassion, kindness etc., but also the stubbornness of age

On the other hand, certain capacities and functions are reduced in the elderly:
- Immune response is reduced, and the danger of infection increases
- Systemic diseases and the ingestion of medicaments increases
- The regenerative capacity of tissues is reduced, also in the oral cavity
- Physical capabilities are reduced
- Oral hygiene is often neglected
- Oligosialia occurs
- Root surface caries increases



1273 Influences that can Lead to Periodontal Problems in Elderly Persons
Not all of the factors depicted here will be present in every elderly person. But one or two parameters—e. g., lack of compliance and/or reduced host response—may lead to significant problems with oral health. In addition, the individual factors may accumulate, and lead finally to the necessity for special treatment.

Modified from *Ratka-Krüger et al. 1998*

Structural/Biological Changes in the Periodontal Tissues of Elderly Individuals

In addition to the above-discussed factors that apply to the entire organism, there are also completely normal clinical and structural biological aging processes in all organs and tissues, which can also be recognized in the periodontium.
The chapter "Gingival Recession" (p. 155) described recession-like clinical appearances in elderly persons. Fig. 1274 depicts the clinical appearance of an old yet periodontally "healthy" man. The gingival recession/shrinkage—also interdentally—can be explained by external influences over many decades: Mild but chronic inflammation results in "shrinkage" of the gingiva and this is enhanced by improper oral hygiene and possible iatrogenic irritation.

Of significance for the periodontium are structural and biological aging processes of the gingiva (epithelium and connective tissue), the periodontal ligament and the alveolar bone. Healing, i. e., regenerative processes, appear to be less effective with increasing age (e. g., reduced quantity of precursor (stem) cells, p. 351).

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Epithelium

Changes in the *gingival epithelium* are directed for the most part by the subepithelial connective tissue. For the proliferation, and therewith the turnover of the epithelium, there are varying explanations: While some authors have described an increase in proliferative activity with age, others report stationary proliferation or even a decrease. Irregardless of these reports, there is consensus that the oral mucosa and also the gingiva becomes thinner, "softer," and drier (reduced saliva production) and there is a loss of gingival stippling. In elderly individuals, all mucosal surfaces are more susceptible to mechanical irritation in comparison to younger persons. Histologically, there is a reduction of keratinization of the gingiva, as well as atrophy in the region of the *Stratum spinosum*. All of these alterations are more common in females during menopause than in males of equal age, and may be explained by the reduction in ovarian function.

Research studies have not demonstrated any deviation from normal structural relationships in the *junctional epithelium* with age.

Connective tissue

Age-related connective tissue changes can be observed in the *gingiva* as well as in the *periodontal ligament*. The number of fibroblasts (and their mitotic activity) is reduced, as is collagen synthesis. The collagen within the periodontal ligament exhibits normal distribution, but the fiber bundles are thicker and more dense. Simultaneously, the organic matrix is reduced. Hyaline zones may form, and these (seldom) lead to cartilage-like or calcified regeneration. The number of Malassez epithelial rest cells becomes diminished.

The thickness of the cellular mixed-fiber cementum increases, especially in the apical third of the root surface and in furcations areas.

The periodontal ligament space becomes narrower, but this may also be the result of functional forces (afunction, hypofunction).

Vessels may exhibit atherosclerotic alterations, and vascularization generally is reduced.



1274 Anterior Clinical View of an "Old" 61-Year-Old Male Throughout his life he had performed toothbrushing with a horizontal scrubbing technique. Gingival recession (retraction) and wedge-shaped defects resulted. On tooth 31, the gingiva was complete lost. On the other hand, there were virtually no clinical symptoms of "pocketing", or periodontitis.

Bone

In elderly persons, osteoporotic changes in bone—resorption of compact bone and expansion of the marrow spaces—can also affect the jaw bones, but in this intra-oral localization, these lytic processes play a less significant role than was previously assumed. Osteoporosis is much more often observed in the long bones and vertebral column. Females, because of the reduction in estrogen production, are more often affected than males; women should be regularly tested following menopause (bone thickness/density measurements).

Healing

The question has often been raised whether with elderly patients periodontal procedures, especially surgical interventions, are at all indicated, or whether disturbances of wound healing are to be expected, leading to failure. This perceived danger is unsubstantiated. Even though in elderly patients fewer stem cells are present in all tissues, their potency remains unaltered. The single problem is the temporal sequence of biologic events leading to complete healing: This process can be significantly longer than in young individuals.

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Age-Related Changes—Influence Upon Treatment Planning

In this *Atlas*, we have repeatedly stated that certain prerequisites must be fulfilled before initiation of comprehensive periodontitis treatment:

- Time, understanding and resiliency of the patient
- Persistent and appropriate compliance with regard to oral hygiene
- Good general systemic health
- Reduction of risk factors

Even in elderly individuals, these prerequisites can surely also remain, if the patient is psychologically and physically healthy. Particularly, the elderly today often *demand* optimum treatment, similar to that offered to younger patients. These older patients seek no compromises in their treatment, simply because they are old! They often do not even want to compromise when it comes to oral esthetics.

On the other hand, some elderly patients will not fulfill the above-listed criteria for periodontal therapy, or fulfill them only partially. Their reduced capacities ("it is no longer worth it"!) must often lead to therapeutic compromises. In many cases, the mental capacity/understanding for systematic treatment is lacking. Only the "most necessary" or pain-alleviating treatment should be performed: The patient, in her/his age-related inflexibility, often knows better what to do—or what not to do—than the dental team itself.

Also severe systemic diseases such as diabetes, Alzheimer's, tumors, autoimmune disease, Parkinson's, hematologic disorders, and medication side effects must influence the oral/dental treatment plan significantly.

In general, manual dexterity decreases with age. Elderly patients with diseases such as those listed above may often not understand the importance of oral hygiene, or may be incapable of performing it. It is not always possible to replace the manual toothbrush with an electric device or by medicinal oral rinsing (CHX; cf. p. 235). The result is often greater plaque accumulation, and as a result, gingivitis and even periodontitis. Statistical studies have demonstrated that periodontal diseases develop more rapidly and more severely in the elderly as compared to young persons (Imfeld 1985). *Nevertheless*, periodontitis can no longer be categorized as a "disease of aging."

In addition to the previously discussed systemic health problems and oral hygiene difficulties of elderly patients, the dentition will also exhibit manifestations of age such as attrition, abrasion, gingival recession and tooth discoloration.

1275 80-Year-Old Male
This mentally alert patient suffered from mild Parkinson's disease. He had major difficulties with mechanical oral hygiene, but wanted to have "clean" teeth. Following professional tooth cleaning and periodontal therapy, an electric toothbrush was demonstrated, and a case-specific rinsing regimen with CHX (with the help of the patient's family), and the patient was scheduled for short-interval recall.

Right: CHX rinsing program.



CHX Rinsing Program—Two Possible Regimens

A

1 month long, daily 2 x CHX, 0.1–0.2 %
3 months *no* CHX
1 month CHX
3 months *no* CHX

B

Daily low dose CHX rinsing (0.06 %) as a permanent replacement for the patient's insufficient home care
(e. g., retirement home!)

Modified Treatment Planning

Old but still mentally and physically healthy patients usually do not require any changes in dental treatment planning. However, in patients who are mentally and/or physically handicapped, the treatment plan must be adapted to the actual situation. Teeth with a questionable prognosis should probably be extracted. A perhaps somewhat extreme approach would be to treat periodontally only those teeth that can be expected to be maintained until life's end.

Missing teeth in non-visible jaw segments beg the question of whether replacement is necessary. If replacement is unavoidable for functional reasons, a removable partial denture is often preferable to a fixed reconstruction.
It is better to incorporate a dental prosthesis at an age at which the patient can become accustomed to it, instead of persisting with years-long (decades-long) periodontal therapy, leading finally only to a complete denture that the patient can no longer successfully accept.

Teeth need not be maintained "at all costs" in elderly patients; rather it is the sense of oral well-being (health, function, phonetics, esthetics) and therewith the patient's own feeling of self worth.

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Classification of Periodontal Diseases

New Classification of Periodontal Diseases (1999)

As demonstrated in the chapter "Forms of Plaque-associated Diseases" (p. 79), new scientific and clinical findings, accumulating long-term experiences as well as the rapid exchange of this knowledge (internet) resulted in the necessity to freshly define the classifications and nomenclature of diseases and clinical "conditions."

Toward the end of 1999, a workshop in Oak Brook, Illinois, was convened with members of the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP), and the result of this meeting is presented on the following two pages as the original and unabridged illustration of the new classification (Types I-VIII), as published by Armitage (1999) in *Annals of Periodontology*.

Type	1999 Classification of periodontal disorders
I	**Gingival diseases**
II	**Chronic periodontitis**
III	**Aggressive periodontitis**
IV	**Periodontitis as a manifestation of systemic disease**
V	**Necrotizing periodontal diseases**
VI	**Periodontal abscess**
VII	**Periodontitis in combination with endodontic lesions**
VIII	**Developmental or inherited conditions**

1276 Periodontal Disease Types—Classification, 1999
The new classification of periodontal disease in eight primary groups/types will be depicted on the following two pages, including all subclassifications.

Classification 1999—For and Against

The "old classification" (AAP 1989) described five disease classes. This classification too heavily weighted the *patient age at disease onset*—e.g., "early onset periodontitis," EOP and "adult periodontitis," AP, as well as the course of the disease, e.g., rapidly-progressing (EOP/RPP). It was necessary to change this type of classification because "rapidly progressive periodontitis" (RPP) does not occur only in young patients, and also a chronic periodontitis (AP) in elderly patients with reduced immune function can unexpectedly and quickly evolve into an acute status.
But even the new 1999 classification will only last for a limited period of time: It is too all-inclusive and *combines* the disease entities that are *practice-relevant* and most common with those disease entities that are rather quite rare. The 1999 classification is similar in many ways to the extensive catalog provided by the WHO list of diseases, but does not consider the multifactorial character of periodontal diseases (risks!).
This problem has been described thematically in numerous subsequent publications (Van der Velden 2000, Burgermeister & Schlagenhauf 2002, Brunner et al. 2002, DGP 2002, Bengel 2003, Lang 2003).

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Classification 1999—Gingiva

1277 Classification—Original Version (AAP 1999)

This classification of periodontal diseases has been universally accepted world-wide, and is presented here in its original and unabridged English text version (abbreviated classification, p. 78).

Gingival Diseases

These occur in the periodontal tissues *without* simultaneous periodontal attachment or bone loss.

(Even with periodontitis, one of the primary symptoms is gingival disease.)

Plaque-Induced Gingival Diseases (A)

Above all, plaque-induced gingivitis (type I A) is a ubiquitous disease. It occurs more or less in all oral, periodontal diseases and is easy to treat. Deeper periodontal structures are not affected within this definition.

Non Plaque-Induced Gingival Lesions (B)

Also with these disease processes, an "additional" plaque-induced gingivitis may be present.
This is a group of diseases/lesions that are relatively seldom observed. Exceptions: Viral lesions that affect the periodontal tissues as well as the oral mucosa. Treatment may be difficult, with varying success results. In many cases, medical specialists must be called upon, especially in life-threatening forms (e. g., *Pemphigus vulgaris*; IB5a3).

Type I—Gingival Diseases

A. Dental plaque-induced gingival diseases

1. Gingivitis associated with dental plaque only
   a. without other local contributing factors
   b. with local contributing factors (see VIII A)
2. Gingival diseases modified by systemic factors
   a. associated with the 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
3. 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
4. Gingival diseases modified by malnutrition
   a. ascorbid acid-deficiency gingivitis
   b. other

B. Non-plaque-induced gingival lesions

1. Gingival diseases of specific bacterial origin
   a. *Neisseria gonorrhea*-associated lesions
   b. *Treponema pallidum*-associated lesions
   c. streptococcal species-associated lesions
   d. other
2. Gingival diseases of viral origin
   a. herpes virus infections
      1) primary herpetic gingivostomatitis
      2) recurrent oral herpes
      3) varicella-zoster infections
   b. other
3. Gingival diseases of fungal origin
   a. *Candida*-species infections
      1) generalized gingival candidosis
   b. linear gingival erythema
   c. histoplasmosis
   d. other
4. Gingival lesions of genetic origin
   a. hereditary gingival fibromatosis
   b. other
5. Gingival manifestations of systemic conditions
   a. mucocutaneous disorders
      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) toothpaste/dentifrices
         b) mouthrinses/mouthwashes
         c) chewing gum additives
         d) other
6. Traumatic lesions (factitious, iatrogenic, accidental)
   a. chemical injury
   b. physical injury
   c. thermal injury
7. Foreign body reactions
8. Not otherwise specified (NOS)

Type I—Gingival Diseases

- **Plaque-Induced Gingival Diseases**
  - gingivitis caused solely by plaque
  - gingivitis modified by systemic factors
  - Gingival diseases modified by medications
  - Gingival diseases modified by malnutrition

- **Non Plaque-Induced Gingival Lesions**
  - specific bacterial lesions
  - viral lesions
  - fungal lesions
  - genetically-elicited lesions
  - systemically-related lesions
  - traumatic lesions
  - foreign body reactions
  - others

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Classification 1999—Periodontium

Type II—Chronic Periodontitis, CP

Type III—Aggressive Periodontitis, AP

Type IV—Periodontitis with Systemic Diseases

– blood dyscrasia

– genetic factors

– non-specified

Type V—Necrotizing Gingivitis (NUG) and Periodontitis (NUP)

Type VI—Abscesses

Type VII—Periodontitis in Combination with Endodontic Lesions

Type VIII—Developmental and Inherited Deformities and Conditions

– localized dental factors, which enhance plaque retention

– mucogingival problems within the dental arch

– mucogingival problems within the edentulous alveolar ridge

– occlusal trauma

Type II **Chronic Periodontitis (CP)****
- **A.** Localized
- **B.** Generalized

Type III **Aggressive Periodontitis (AP)****
- **A.** Localized
- **B.** Generalized

Type IV **Periodontitis as a Manifestation of Systemic Diseases**
- **A.** Associated with hematologic disorders
  1. Acquired neutropenia
  2. Leukemia
  3. Other
- **B.** Associated with genetic disorders
  1. Familial and cyclic neutropenia
  2. Down syndrome
  3. Leukocyte adhesion deficiency syndromes
  4. Papillon-Lefèvre syndrome
  5. Chediak-Higashi syndrome
  6. Histiocytosis syndromes
  7. Glycogen storage disease
  8. Infantile genetic agranulocytosis
  9. Cohen syndrome
  10. Ehlers-Danlos syndrome (Types IV and VIII)
  11. Hypophosphatasia
  12. Other
- **C.** Not otherwise specified (NOS)

Type V **Necrotizing Periodontal Diseases**
- **A.** Necrotizing ulcerative gingivitis (**NUG**)
- **B.** Necrotizing ulcerative periodontitis (**NUP**)

Type VI **Abscesses of the Periodontium**
- **A.** Gingival abscess
- **B.** Periodontal abscess
- **C.** Pericoronal abscess

Type VII **Periodontitis Associated with Endodontic Lesions**
- **A.** Combined periodontal-endodontic lesions

Type VIII **Developmental or Acquired Deformities and Conditions**
- **A.** Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis
  1. Tooth anatomic factors
  2. Dental anatomic factors
  3. Root fractures
  4. Cervical root resorption and cemental tears
- **B.** Mucogingival deformities and conditions around teeth
  1. Gingival/soft tissue recession
     a. facial or lingual surfaces
     b. interproximal (papillary)
  2. Lack of keratinized gingiva
  3. Decreased vestibular depth
  4. Aberrant frenum/muscle position
  5. Gingival excess
     a. pseudopocket
     b. inconsistent gingival margin
     c. excessive gingival display
     d. gingival enlargement (see I A3 and I B4)
  6. Abnormal color
- **C.** Mucogingival deformities and conditions on edentulous ridges
  1. Vertical and/or horizontal ridge deficiency
  2. Lack of gingiva/keratinized tissue
  3. Gingival/soft tissue enlargement
  4. Aberrant frenum/muscle position
  5. Decreased vestibular depth
  6. Abnormal color
- **D.** Occlusal trauma
  1. Primary occlusal trauma
  2. Secondary occlusal trauma

1278 Periodontitis—Types II to VIII

Chronic (II) and Aggressive (III) Periodontitis

Chronic periodontitis (previously AP) is the most common form of the disease (> 80 % of all cases).

More aggressive forms (formerly EOP, PP, LJP, RPP; cf. Second Edition 1989) are rare.

In addition to the *pathobiologic* form (p. 96), the *pathomorphologic* expanse (p. 98) and the localization of the attachment loss must be considered:

** **Localized/Generalized**
In a case with less than 30 % involvement of all sites, the case is categorized as *localized*.
More serious and expansive involvement is categorized as *generalized*.

** **Degree of Severity**
Clinical attachment loss (CAL) is described as follows:

– "slight" up to 2 mm
– "moderate" 3–4 mm
– "severe" 5+ mm

**In addition to the globally accepted AAP-Classification (1999), one must also consider the degree of severity (CAL) alone and with the corresponding prospective therapeutic involvement, as described in the ADA/AAP classification of "*case patterns*" or "*case types*" (American Dental Association):

ADA—"Case Types"

• Grade	Severity Evaluation
I	Gingivitis – 3 degrees of severity
II	Early periodontitis
III	Moderate periodontitis
IV	Advanced periodontitis

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Changes—Comparing the 1989 and 1999 Classifications

Any classification of a complicated disease process can only be an attempt to describe the *overall* disease process in a short format, hopefully leading to better understanding of the complex nature over the long-term.

Therefore, it is not possible to assess/address alongside the name of the disease, also the etiologies, the initiation, the clinical condition and its severity and course, and ultimately the causes of the specific disease types in simple terms, or to distinguish the individual disease process from other diseases.

On the other hand, a classification may elicit and describe the relevance and the prevalence of the disease process, and therefore target the therapeutic energies of each dental practitioner!

The authors of this *Atlas* have attempted to use the new classification, while also noting the previous system. Above all, it was our goal to describe and portray the *most important periodontal disease forms* in our text and illustrations for the dentist and periodontist.

1279 Changes and Alterations within the 1999 AAP Classification
These are depicted in the table (right).

The term "apical" is retained for the more rare forms of periodontitis.

Note: Reasonable abbreviations for the new classifications are for the most part lacking!

Modified from *Meyle et al. 2002; DGP/QV*

1989/99 Changes in the classification		1989 5 classes	1999 8 types
⊕	• Enhancement of the classification "gingival disorders"	—	I
	• Enhancement of the classification "periodontal abscess"	—	VI
	• Enhancement of the classification "periodontitis in conjunction with endodontic lesions"	—	VII
	• Enhancement of the classification "developmental or genetic conditions"	—	VIII
△	• Differentiation within the term "periodontitis as a manifestation of systemic disease"	III	IV
►○◄	• Replacement of the term "adult periodontitis" with the term "chronic periodontitis"	I AP	II CP
	• Replacement of the term "early onset periodontitis" with the term "aggressive periodontitis"	II EOP PPP LJP RPP	III AP
	• Replacement of the term "necrotizing ulcerative periodontitis" with the term " necrotizing periodontal disease"	IV ANUG/P	V NUG/P
⊖	• Elimination of the terms "refractory and recurrent periodontitis" as individual categories	V RP	

Limitations—Examples of Criticism

The new classification (1999) has too many sub-classifications (hierarchy); for example, pregnancy gingivitis is found in IA2a3a/b! In addition, it would appear to be totally impractical that the most commonly occurring disease entities are not identified with simple abbreviations, except for CP, AP, NUG/NUP.
Possibly the most important chapter in this *Atlas*, "Oral manifestations of HIV-infection and AIDS " (p. 142) is not even *listed* in the new classification, even though this severe immunodeficiency not only manifests as necrotizing ulcerative gingivitis/periodontitis (NUG/NUP), but also as a specific "linear gingival erythema," and a long list of opportunistic secondary infections in the oral mucosa and periodontium (bacterial, viral and fungal infections, as well as tumors such as the Karposi sarcoma, p. 146).

"Classical gingival recession," which has become more and more common in industrialized nations in recent years (p. 155), is classified under VIIIB1a, "mucogingival deformations and conditions." Any "soft tissue recession" usually occurs only after some form of osseous dehiscence. Whether this "condition" should be classified as a disease or only as a morphologic variation of the healthy periodontium is really irrelevant for a dental practitioner or a periodontist (improper oral hygiene, functional dysfunction, p. 459).

Each dental practitioner must come to grips with the fact that the patient is not interested only in oral health but also, more and more, esthetic problems (gingival recession, "long teeth"), which can only be addressed through mucogingival/plastic surgery.

In the most recent classification, entire disease entities are not represented (infectious diseases—HIV/AIDS), or are inadequately represented (gingival recession), and some "conditions" such as pericoronal abscess (VI C) find themselves on the highest level.

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Recall—"Personalized Periodontology"

"Recall them all"

The authors of this Third Edition of the *Color Atlas of Periodontology* seek to acknowledge the many contributions of the numerous individuals who have served as critical contributors, as researchers and teachers during the more than 20 years during which this *Color Atlas* has evolved (see p. 448). We deeply appreciate the collegiality, friendship and patience with which our many colleagues have participated in this on-going effort.

At the same time, we beg forgiveness from the hundreds of other colleagues and friends who contributed to this decades-long publication effort, who, purely for space considerations, could not be included on our thumb tack wall!

In any and every case, however, we offer our thanks and professional consideration to all of our colleagues.

Names of the Periodontal Contributors as Displayed on Page 448

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31	32	33	34	35	36	37	38	39	40
41	42	**R**	**E**	**C**	**A**	**L**	**L**	49	50
51	52	53	54	55	56	57	58	59	60
61	62	63	64	65	66	67	68	69	70
71	72	73	74	75	76	77	78	79	80
81	82	83	84	85	86	87	88	89	90
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Acknowledgments for Figures

The individuals listed below provided us with illustrative materials for this publication.
All other illustrations and figures derived from the archives of the authors, their departments or their private practices. *Photographs of objects* were made exclusively by Herbert F. Wolf.

All of the *histologic illustrations*—with exception of special illustrations—were provided to us by Dr. Alice Kallenberger (emeritus).

All of the *schematic and graphic illustrations* were prepared and provided, according to detailed illustrations by H.F. Wolf, by the scientific illustration company B. Struchen & Partner (Zurich), with additional assistance in this Third Edition by J. Hormann, Graphic Design (Stuttgart).

Because many of the figures contain more than one photograph, each acknowledgement contains an exact description of the donor's contribution (e. g., L, M or R for left, middle or right; A, B, C etc.).

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J. Meyer	71, 71L
H. J. Müller	290
L. Ritz	638–643
B. Widmer	247

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Magne P., Belser U. Bonded Porcelain Restorations, Chicago: Quintessence; 2002, p. 142.

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References

The bibliography of this Atlas is divided into two sections:

- In the first section, *textbooks* of periodontology are listed on the following pages and are recommended for deeper study of each aspect of periodontology.
- The second section includes individual publications, and can be accessed over the internet via

http://www.thieme.com/perio-lit

On the basis of the increasing flood of information, the authors of this *Atlas* have limited themselves to only the most important publications that have appeared in the past 10–15 years. Included are the most supportive publications, especially the *Proceedings* of major conferences and workshops, *Position papers* and *Annals of Periodontology* (AAP) as well as literature summaries from *Periodontology 2000* that present the "state of the art" in our profession.

An exception in this *Atlas* remains the *classical milestones* of knowledge, i. e., older publications that maintain their value and viability even in the face of today's knowledge. For example, the *History of Periodontology* is reported in the excellent publication by A.J. Held—*Periodontology: From its origins up to 1980.*

In addition, the authors of this *Atlas* recommend that readers access the various scientific databases such as *Medline*, and also the link over the Thieme-Verlag homepage. By selecting appropriate "key words," one will be able to access "abstracts" as well as original scientific articles.

The literature that we present here does not pretend to be complete! In addition to the cited *papers*, more extensive literature references are indicated, that are not cited in our text. Simple access to these papers can be achieved via internet web sites for the various national periodontal professional organizations (e. g., AAP, SSP, DGP).

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Periodontology—Related specialties

- Basics
- Types
- Findings, diagnosis
- Prevention
- Treatment—Phase 1 therapy
- Medicaments
- Treatment—Phase 2 therapy
- Furcation treatment—Perio/endo
- Treatment—Mucogingival surgery
- Maintenance therapy—Recall
- Function—Orthodontics—Splinting
- Perio-prosthetics and implant therapy
- Esthetic integration
- New technologies—Laser
- Varia

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WWW.HIGHDENT.IR
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Index

A

Abfractions 398
Abscess 29, 60, 107, 114, 521
Abscess opening 219
Access flap (see also Widman procedure, modified) 299, 309, 354
  adaptation 312, 316
  contraindications 309
  defect coverage 311
  furcation treatment 386
  indications 308
  interrupted sutures 312
  partial mobilization 310
  pocket reduction 318
  post-operative measures 316
  root cleaning/planing 296
  suture removal 316
Acetylsalicylic acid 49
Acid etch technique 474
Acquired Immunodeficiency Syndrome, see AIDS
Acrylic bridge, temporary 481
Acrylic dental instruments 262
Acrylic resin, gingival mask 457
Acrylic splint, removable 473
Acrylic temporary, prosthetic, fixed 481
Actinobacillus actinomycetemcomitans (Aa) 30, 33, 51, 254, 287
  culture 181
  reservoir, oral 255
  virulence factors 36
Actinomyces 24
  israelii 24
  viscosus 24
Adenocarcinoma 127
Adhesins 41, 46
Adhesive bridge 479
Adhesive bridgework 475
  mandible 479
Adhesive splints 475
Adhesive technique, splinting 475
Age distribution 515
Age-related gingival recession 155, 160
AIDS (Acquired Immunodeficiency Syndrome; see also HIV infection) 139
  Candidiasis, oral 142, 144
  epidemiology 140
  fungal infection, oral 142, 144
  indicator diseases 141
  infection
    bacterial, oral 142
    opportunistic, therapy 150
    viral, oral 142, 145
    with human herpes viruses 145
  neoplasm, oral 142
  oral manifestations 142
  therapy, anti-retroviral 148
Air-scaler 240
Allergy, gingival alterations 120
Alloderm 340
Allograft 332
Alloplastic graft 332
Alveolar bone 1, 7
Alveolar mucosa 7
Alveolar process 16
Alveolar ridge defect 503
  classification
    qualitative 503
    semiquantitative 503
  connective tissue graft
    envelope technique 505
    inlay/onlay technique 506
    onlay technique 505
  definitive reconstruction 508
  soft tissue augmentation 503, 505
    technique 505
  soft tissue graft, planning, problems 509
  therapy
    prosthetic 504
    surgical 505
Alveolar ridge segment, edentulous 502
Alveolar wall 16
Amalgam restoration
  marginal discrepancy, plaque-accumulation 27
  overhang 27, 244
  pulp necrosis 446
  recontouring 245
Amelogenins
  porcine 351
  with flap reflection 352
Aminoalcohols 235
Amoxicillin 214
Amoxicillin-clavulanic acid combination 289
Angular cheilitis 144
Ankylosis 206
Anti-epileptic drugs 379
Anti-infectives 287
Anti-inflammatory drugs, non-steroidal (NSAID) 49, 294
Antibiogram 181
Antibiotic therapy 287
  decision criteria 288
  local 291
  systemic 291
Antibiotics (AB) 257, 287
  bacteriocidal 213, 289
  bactieriostatic 289
  concentration, subgingival, application-dependent 292
  resistance, bacteria 290
  sensitivity, bacterial 290
Antibodies 186
Anticoagulants 212
Antigen presentation 46
Antigen-antibody reactions 186
Antigens, surface, bacterial 38
Antigens, surface markers 186
Antiseptics, pocket rinsing 283
Aphthae
  expansive, with HIV infection 147
  recurrent 131
Apparatus, orthodontic 463
  maxilla 465
Arachidonic acid cascade 49
ARC syndrome 141
Arestin 293
Articulation, temporary, prosthetic, fixed 480
ASA classification, health status 212
Atridox 293
Atrigel technology 345
Atrisorb 339, 345
  adapted 347
  free flow 346
Atrisorb membrane, functional life 345
Attachment, connective tissue 12
Attachment gain 280
  via Emdogain 354
Attachment loss (AL) 1, 3, 95, 98
  bleeding upon probing 192
  clinical 169
  epidemiology 75
  gingival recession 161
  irregular 63
  labial 168
  maintenance 204
  periodontitis, degree of severity 95, 98
  periodontitis ulcerosa 89
  positive IL-1 genotype 190
  uniform 63
  with/without recall 449
Attachment niveau, clinical 171
Attachment structures, connective tissue 12
Augmentin 289
Autogenous fiber cementum, cellular 14
Autograft 332
Autoimmune disease, gingival alterations 120
Autoradiography 183
Azithromycin 289

B

Bacteremia 213
  dangerous procedures 214
Bacteria 23, 55
  adhesion 24, 34
  antibiotic resistance 290
  association 24
    with periodontitis 33
  cell wall 31
  fusiform 86
  gram-negative 30
  gram-positive 30
  inflammation-inducing products 21
  microcolonies 24
  morphotypes 180
  pathogens, elimination 202
  periodontopathic 1
  planktonic 24, 28
  resistance determination 181
  virulence 21
  virulence factors 34
  virulence transfer 34
Bacteria, rods 30
Bacterial attack, host defense reaction 21
Bacterial complex 37
Bacterial culture 277, 290
  anaerobic 181
Bacterial flora, IL-1 positive genotype 191
Bacterial invasion into tissue; see infection
Bacterial tests, enzymatic 187
Bacterial virulence 34
Bacteriophages 34
  DNA transfer 35
Bacteroides forsythus (Bf) (former name for *Tannerella forsythensis, Tf)* 30, 33, 36, 51
BANA test 187
Barricaid 370
Barrier membranes; see membrane barriers
Basal lamina
  external 10
  internal 10
Bass toothbrushing technique 228
BC-CP distance 496
"Bernese spider web" 193
β-TCP (tricalcium phosphate) 334
Betadine 275, 281
Beta-tricalcium phosphate (TCP) 336
  combined operation 334
BIC (bone implant contact) 510
Biofilm 23, 40, 203
  elimination 202, 287
  microbial 199
  subgingival 28, 62, 254
    changes during periodontitis 63
    removal 282
  tooth root 28
  vector ultrasonic device 282
Bio-gide 339
Bioglass, alloplastic 333
Biologic width (BW) 490
  crown lengthening via ostectomy 493
Biologic width (BW) 12
  implant therapy 511
Biomend 340
Biomodification factors 352
Bio-Oss
  collagen 346, 348
  perio-system 349
Bite plane, occlusal 462
Bite-wing radiograph 176
Bite-wing radiograph (BW)
  horizontal 176
  vertical 176
Bleeding index, see BOP
Bleeding on probing 192
Blood cell diseases 120
Blood coagulant 206
Blood thinners 212
B-lymphocytes 41, 43
BMP (Bone morphogenetic proteins) 351
Bone
  allogenic 325, 332
  alveolar, dehiscence 155, 158
  autogenous 325, 327, 332
    harvesting 328
  exostosis, re-contouring 357
  freeze-dried 332
  mandibular 17
  maxillary 17
Bone availability, dental implant therapy 512
Bone destruction, periodontal 61
Bone filter 328
Bone height 171
Bone loss 61, 99
  age-related 517
  horizontal 99
  IL-1 genotype, positive 190
  interdental 496
  irregular 329
  periodontal, radiographic 177
  vertical 99
Bone mill 329
Bone morphogenetic proteins (BMP) 351
Bone "nipper," fine 356
Bone quality, implant therapy 512
Bone replacement materials 325, 327
  xenogenic 333
Bone sounding 490
Bone-implant contact (BIC) 510
Bony pocket (see also pocket, intra-alveolar) 99, 104
  β-TCP filling 336
BOP (bleeding on probing) 67, 69, 81, 192, 224, 249
  following gingivitis treatment 252
  periodontitis, chronic, severe 111
  test validity 192
Brain abscess 64
Bridge flaps, lateral pedicle, coronally repositioned 440
Bridge framework, try-in 479
Bridge pontic 246
  corrections/modifications 246
  hygiene access 246
  incorrect planning 246
Bridge prosthesis 467
Bridgework
  definitive 480
  temporary 480
    requirements 481
Brush, round-head 230
Brush sticks 231
Bruxism 462
Bur, round 362
Burs 356
  cooled 328
BW (biologic width) 12
  implant therapy 511

C

Calculus
  subgingival 26
  supragingival 26
Calculus removal
  professional 208
  subgingival 90
  supragingival 243
Campylobacter rectus (Cr) 30, 33
Candidiasis
  atrophic/erythematous 144
  oral, AIDS 142, 144
  pseudomembranous 144
Canine (cuspid) 2
  labial version 470
  retained, impacted 176
  scaling, subgingival 260
Cantilevered bridgework 486
Capillary loops, subepithelial 18
Capnocytophaga gingivalis 24
Carbide bur 328
Cardiac pacemaker 213
Caries, dental
  in HIV infection 147
  subgingival 378
  with regular recall 449
  without recall 449
Case presentation 222
Cast framework partial denture 488, 504
Causal therapy 210
CD-markers 41, 46
Cell biology, wound healing 206
Cell surface molecules, regulatory 41, 46
Cells
  antigen presenting 41
  cement-forming 14
Cementoblasts 14
Cementocytes 14
Cementoenamel junction (CEJ) 26, 171
Cementum 20
  acellular-afibrullar 14
  resorption lacunae 20
Cervical caries 456
Chaikin wedge excision 319
Charting, computer-enhanced 195
Charting, diagnostic 194
Chediak-Higashi syndrome 53
Chemokine 48
Chemotaxis 55
Chewing force 459
Chewing gum, "tooth-friendly" 237
Chinolone (see Quinolone) 289
Chisel 242, 356
Chlorhexidine (CHX) 235, 281
  full mouth therapy 281, 283
  pocket filling 281
Chlorhexidine digluconate products 235
Chlorhexidine rinsing 90
  after periodontal surgery 302
Chondrosarcoma 127
CHX; see chlorhexidine
Ciprofloxacin 289
Classification of periodontal diseases 519
Cleft formation, gingival 399
Clindamycin 289
CMT (chemically-modified tetracycline) 294
CMV-infection in AIDS 145
Coagulation, course 212
Cocci 30
Col 7, 9
Col, interpapillary 7, 9
Community periodontal index of treatment needs (CPITN) 67, 72
  studies 75
  treatment necessity 73
Compact bone 7
Complement cascade 42, 44
Complex, dentogingival 490
Compliance 222
  insufficient 455
Composite splinting 469, 473
  semi-permanent 474
Computer tomogram, implant therapy 512
Concrement, subgingival 26
  removal 243
Connective tissue
  age-related changes 517
  gingival, function 20
Connective tissue breakdown 60
Connective tissue graft 163, 419
  alveolar ridge augmentation, partial epithelialization 506
  complications 430
  flap repositioning 428
  harvesting 421, 427
    incisions 422
    "open door" method 424
    using the double scalpel 424
    wound closure 423
  histology 421
  incision template 427
  vascular arrangement at the donor site 421
Connective tissue graft
  covering recession 419
  envelope technique 420, 430, 505
  multiple recessions 442
  onlay technique 505
  with alveolar defect 505
Contact position, retruded 175
Contouring diamonds 247
Controlled release drugs (CRD) 257, 292
Coronary disease 64
Course of treatment 208
COX-1 49, 52
  gene 53
COX-2 49, 52
COX-2 inhibitor 294
CPITN (community periodontal index of treatment needs) 67, 72
  studies 75
  treatment necessity 73
CPITN codes 72
Crater, interdental 151
CRD (controlled release drugs) 257, 292
Crown, clinical, lengthening 492
Crown lengthening
  crown replacement 494
  ostectomy 493
    indications 493
  osteoplasty 494
Crown margin
  non-transparent 491
  position 490

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

temporary, prosthetic, fixed 480
Crown margin gap 27
Crown replacement, following crown lengthening 494
Crowns
metal-reinforced 500
transparent 491
Culture, bacterial 181
Cumarin 212
antidote 212
Curettage 256
closed 253
gingival 300
open 253
Curette, Gracey 243, 258
areas of use 260
nomenclature 268
scaling technique 263
sharpening 268
Curettes 242, 257
double-ended 273
nomenclature 268
Cyanoacrylate 370
Cyclooxygenase, see COX
Cyclooxygenase-2-inhibitor (see COX-2-inhibitor
Cyclosporine (Cs) 123
Cyclosporine-induced gingival overgrowth 123, 380
Cytokine 40, 47
anti-inflammatory 47, 66
chemotactic 47
effect 48
immunoregulatory 48
pro-inflammatory 47, 66
Cytokine cascade 47
Cytokine network 47
Cytotoxicity 42

D

Darkfield microscopy 180
Debridement 152, 249
mechanical, instrumental 259
subgingival 253
effects 256
supragingival 242
Debridement, subgingival 271
anesthesia 272
checking the surface 275
Defect
genetic 52
intra-alveolar 297
mucogingival, surgical correction 296
Defect treatment 202
Defective function 174
Deformity 521
Dehiscence 398
Demineralized freeze-dried bone allograft (DFDBA) 332
Dental charting 165
facultative 166
obligatory 166
Dental cuticle 10
Dental floss 231
Dental hygienist (DH) 454
Dental implants 511
Dentin, hypersensitive 458
Dentin caries 164, 456
Dentin-cementum transition 15
Dentist 454
Dentocheck 187
Dentogingival complex (DGC) 490
Detoxification
root surfaces 253
with implant problems 514
Dentsply 259
Disinfection agents 257
subgingivally useful 283
DFDBA (demineralized freeze-dried bone allograft) 332
DGC (dentogingival complex) 490
Diabetes, retinopathy 133
Diabetes mellitus (DM) 64, 215
gingivitis 132
periodontitis 132
periodontitis therapy 215
Diagnoses
charting 194
microbial
plaque harvesting technique 179
test methods 178
Diagnosis 196
Differentiation factors, tissue regeneration 351
Dihydropyridine 122
Dihydropyridine/cyclosporine combination, gingival overgrowth 124
Dihydropyridine-induced gingival overgrowth 122
Diphosphonates 294
Disc probe 170
Disharmony, functional 398
DMDx-test, autoradiography 183
DNA probe hybridization 183
DNA probe test 183
results 185
DNA probe test, marker organisms 183
DNA transfer by bacteriophages 35
Down Syndrome (Trisomy 21) 53
periodontitis 134
Doxycycline 50, 289
Dressing materials, soft 370
Dressings, wound 370

E

Eikenella corrodens (Ec) 30, 33
Eikosanoids 41, 49
Electric toothbrushes 236
Electrosurgery, devices 368
Electrotome 364
attachments 368
Electrotome surgical loop 368
Elevator 305
ELISA (enzyme-linked immunosorbent assay) 186
Elyzol 293
EMD (enamel matrix derivatives) 351
Emdogain 285, 351
flap surgery procedures 352
with filler particles 352
Emergency situations 217
Emergency treatment 217
Empress ceramic crown 491
EMS airflow handy-2 241, 282
Enamel loss 164
Enamel matrix derivatives (EMD) 351
Enamel matrix proteins 351
Enamel "pearl" 388
Enamel projection 26, 172, 382, 388
Endodontics 445
Endodontic-periodontal problems, acute 217
Endocarditis 64
infectious 211, 213
Endocarditis prophylaxis 213
indication 213
Endotoxin, bacterial 38
Envelope technique, connective tissue transplantation 420, 430, 505
Enzyme test 188
Enzyme-linked immunosorbent assay (ELISA) 186
Epithelium
function 20
gingival, age-related changes 517
oral 10
Epulis
fibromatosis 127
giant cell 127
granulomatous 125
gravid 93
Erosion 164
deep 164
Erythroplakia 130
Esthetic correction, mucogingival surgery, plastic 441
Esthetic width (EW) 491
Eubacterium 33
nodatum 30
Eukaryotes 30
EW (esthetic width) 491
Excavator 362
Exostosis 126
following free gingival graft 412
Extraction, dental implant 514
Extraction, immediate 217
radiograph, preparatory 220
Extrinsic fiber cementum, acellular 14

F

FDBA (freeze-dried bone allograft) 332
FDIS (full mouth disinfection) 210, 253, 285
Feldspat ceramic crown 491
Fenestration, osseous lamellae 155
Fiber apparatus
gingival 12
periodontal 12
Fibroblasts, periodontal ligament 14
Files
manual 244
power-driven 244
Filler material, xenogenic 332
Fistula 29, 105, 220
periodontal 107
Flap reflection 303
facial 366
horizontal incision 303
vertical incision 303
wedge excision 321
Flap repositioning, apical 359
results 364
Flap surgery 303
contraindications 303
evaluation 358
indications 303
instruments 305
needles 306
suture loops 307
suture material 306
suture placement 308
sutures 307
with osseous surgery, evaluation 358
Flaps
coronally repositioned 415, 440
lateral pedicle, coronally repositioned 440
oral 342
adaptation 336
surgical procedure, combined 335
with amelogenic proteins 351
palatal 366
pedicle
covering areas of recession 415
half-moon shaped, coronally repositioned 415
laterally repositioned 415
surgical technique, combined 366
Florida Probe 171
Florida Probe System 170, 195
Fluorescence angiography 18
wound healing after free gingival graft 411
Fluorescence microscopy 186
Fluoride dentifrice 458
Fluorides 458
FMT, see full mouth therapy
Foetor ex ore (halitosis) 87, 237
Follicular cyst 437
Foramen, apical 445
Forceps, surgical 402
Freeze-dried bone allograft (FDBA) 332
Fremitus 174
Frenectomy 400
Frenotomy 400
Full mouth disinfection (FDIS) 210, 253, 285
Full mouth therapy (FMT) 210, 253, 281
hygiene phase 281
instrumental 282
pharmacologic 283
rate of healing 285
results 284
radiographically 284
Full-thickness flaps 301, 303, 352, 415
Functional analysis, small/limited 175
Functional disturbance 459
Fungal infection, oral, in AIDS 142, 144
Furcation 381
buccal 173
too narrow 389
closed 382
distal 173
mesial 173
position 172
special probe 172
Furcation abscess 105
Furcation involvement 172, 221, 297, 383
abutment teeth, prosthetics 385
degree of severity 383
F1 383, 385
F2 383, 385
F3 383, 385, 387
fully dentulous patient 385
hemisection 387, 392
horizontal 172
inadequate patient compliance 385
odontoplasty 388
root amputation 387
root resection 394
scaling 386, 388
tooth replacement, planning 385
treatment planning 385
trisection 387, 396
vertical 172
Furcation roof 382
Furcation treatment 384
closed 386
decision-making aids 385
influencing factors 384
mandible 386, 390
maxilla 387, 389, 394
open 386
planning 384
regenerative 390
methods 386
resective 387
long-term results 384
surgical 299
Furcationplasty 389
Fusobacterium nucleatum (Fn) 24, 33

G

Ganglion, Gasserian 19
GBR (guided bone regeneration) 340
Gene polymorphisms 53, 189
Gene probes 183
Genetic diseases 52
Genetic risk 189
Genome 53
Genotype, IL-1-positive 189
bacterial flora 191
probing pocket depths 191
risk factor combinations 191
Gingiva 1, 7
aging process, structural biology 516
attached 7
consistency 8
free 7
marginal 8
healthy 56, 65, 79
probing pocket depth 169
interdental 8
keratinized 512
marginal 10
oral pathological alterations 119
pigmented 8
thin, fragile 398
Gingival disease
classification 520
non-plaque induced 520
plaque-induced 520
Gingival epithelium, age-induced changes 517
Gingival erythema, linear, AIDS 143
Gingival extension 401
Gingival fibrosis 126
Gingival hyperplasia/overgrowth 303, 371
cyclosporine-induced 123, 380
dihydropyridine-cyclosporine combination 124
dihydropyridine-induced 122
following connective tissue graft 432
gingivectomy 372
gingivoplasty 372
hereditary base 126
hydantoin-induced 379
medication-induced 94, 120, 379
phenytoin-induced 121
Gingival index 23, 67, 69
Gingival margin 171
Gingival mask 457
bridge pontics 504
Gingival overgrowth 120, 125
Gingival recession, see recession, gingival
Gingival recession index 67
Gingival shrinkage 105
Gingival swelling 105, 171
edematous 81, 84
Gingival thickening (overgrowth)
idiopathic 126
surgical 442
Gingival tumors 120, 125
benign 125
malignant 127
Gingival width 9
Gingivectomy 299, 301, 367
anesthesia 372
contraindications 367
crown lengthening 492
flap surgery, combined 366
hydantoin-induced overgrowth 379
hyperplastic gingivitis 371, 377
incisions 373
indication 367
instruments 368
maxilla
buccal 377
palatal 377
oral 335
subgingival caries 378
surgical principles 367
tissue adhesive 370
very high smile line 492
wedge excision, combined 322
wound 374
wound debridement 374
wound dressing 370, 375
Gingivectomy knife 368, 373, 402
Gingivitis 1, 3
climacterica 91
desquamative 128
diabetes mellitus 132
epidemiology 74
etiology 21
general diagnosis 196
generalized 470
histology 56
histopathology 8
hormonally modulated 91
host factors 22
hyperplastic 371, 377
intermenstrualis 91
menstrualis 91
mild 80
moderate 80, 83
necrotizing 521
AIDS 143
oral hygiene effect 238
oral hygiene-dependent 23
periodontitis development 62
plaque-induced 1, 79
treatment 249
prevention, secondary 252
probing depth 169
prophylaxis/prevention 199
severe 80, 84
simplex 79
single-tooth diagnosis 196
symptoms 81
ulcerative 85
bacteriology 87
etiology 85
histopathology 86
symptoms 87
Gingivitis index 71
Gingivoperiodontitis, ulcerative 89
acute 217
emergency treatment 369
HIV infection 151
pain sensation 19
recurrent 163
treatment 90
Gingivoplasty 90, 126, 299, 301, 364, 367
anesthesia 372
contraindication 367
electrosurgical loop 369
indication 367
instruments 368
maxilla
buccal 377
palatal 377
surgical principles 367
tissue adhesive 370
wound dressing 370
Gingivosis 128
Gold standard 182
Gore membrane, furcation involvement 390
GoreTex barrier 339
GoreTex periodontal material 339, 341
Gracey curette set 258
color-coded 263
limited set 258, 263
Gram staining 31
Granuloma, pyogenic 127
Granulation tissue, removal 362
Granulocytes, polymorphonuclear (PMN) 40, 42, 44, 62
defective 52
interactions 44
migration 55
receptors 53
ulcerative periodontitis 86
Grooves 247
cervical area 247
Growth factors 325
activity 351
tissue regeneration 351
Guidor membrane 340, 438

H

HAART 149
Halitosis 87, 237
Hand instruments 262
root planing 258
scaling 258

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Hand instruments
sharpening 269, 270
manual 269
power-driven 270
tooth cleaning 242
Hand mucotome 402
Healing
periodontal 205, 206
regenerative 323
Healing time in the elderly 517
Health questionnaire 167
Health status, ASA classification 212
Hemidesmosomes 10
Hemisection, furcation involvement 387, 392
Hemorrhage 212
gingival, as a motivation factor 224
upon probing; see BOP
Herpes 131
Herpes simplex viral infection 131
Herpes virus 131
human, oral infection with AIDS 145
Herpetic gingivostomatitis 131
Herpetic stomatitis 145
Hertwig epithelial sheath (HERS) 338, 382
HI Virus (HIV) 1, 139
replication cycle 148
tropism 148
vaccine 149
HIV infection (see also AIDS) 139
alternating therapy 149
CDCP classification 141
classification 141
course 141
epidemiology 140
oral manifestations 142
periodontal treatment 150
periodontitis 120
post-exposure prevention by the dental team 150
prophylaxis for the dental team 150
treatment, antiretroviral 148
viral load 141
HIV periodontitis 151
osseous sequester 153
probing pocket depths 151
prosthetic treatment 154
tissue debridement 152
treatment 151
tooth mobility 151
Hollow chisel forceps 356
Hormones, gingival alterations 120
Host influences 202
Host reaction 199, 215
examination 166
immune-inflammatory 40
in diabetes mellitus 215
modulating substances 287, 294
test 178, 188
Host response 41
Hybridization technique 183
Hydantoin gingival overgrowth 379
Hydroxyapatite (HA) 327
Hygiene capability
bridge pontics 246
creation of 244
Hygiene phase during “full mouth therapy” 281
Hyperkeratosis, palmar and plantar (HPP); see PLS 136

I

IAI Pado test 184
IL-1 gene polymorphism 189
consequences 190
IL-1 gene test 190
Immune deficiency, periodontitis 120
Immune system 41
components
cellular 41
humoral 41
enhancement 199
Immunity
non-specific 41, 45
specific 41, 43, 45
Immunofluorescence
direct 186
indirect 186
Immunoglobulin gene superfamily 46
Immunoglobulins (Ig) 41, 43
IgG2 level, lowered 53
Immunoregulation 42
Implant 511
alloplastic 334
autogenous 4
delayed implantation 513
dental 511
healing 512
enosseous 5
extraction 514
failures 514
immediate implants 513
micro-movements 512
osseodisintegration 514
osseointegration 514
problem management 514
re-osseointegration 514
Implant, immediate 513
Implant therapy 511
biologic width 511
decision-making criteria 512
diagnostic criteria 512
Implantation, delayed 513
IN inhibitor 148
Indices
BOP 67, 69, 81, 111, 192, 224, 249, 252
CPITN 67, 72
PBI 67, 69, 82, 109, 113, 117, 224
PDI 67, 71
PI 67, 111, 192, 199, 249, 450
PSR 67, 73
Infant death, elevated 64
Infection 21, 29
bacterial, oral, in AIDS 142
classic 32
opportunistic 32
in AIDS, treatment 150
periodontal, systemic disease 64
pulmonary 64
specific, gingival alteration 120, 131
viral, oral in AIDS 142, 145
Infectious disease 211
Inflammation
periodontal, effect of periodontal surgery 296
reduction of 204, 208
Inflammatory cells 41, 59
Inflammatory reaction 55
Initial therapy 1 201, 221, 223
Initial therapy 2 201, 221
INR (international normalized ratio; cf. Quick) 212
Insulin resistance 215
Integrase (IN), inhibitors 148
Interdental brushes 231, 233
Interdental hygiene 231
Interdental ligature 473
Interdental space, temporary, prosthetics, fixed 480
Interleukin (IL), see Cytokines
Intoxication, gingival alterations 120
Invasion, bacterial, direct 63
Iodine test 161
Irrigators 236
Irritation, exogenous 54
Irritations, iatrogenic
correction 246
removal 244
Isograft 332

J

Jahnke classification 162
Jet shield 240
Junctional epithelium (JE) 10, 65
long 206
removal 256

K

Kaposi sarcoma 142
histology 146
oral 146
Kirkland knife 373
Kramer sharpening station 269

L

Labial frena 400
Lacunae, bacteria-filled 203
Lamina
cribriform 7
dura 16
propria 10
Laminate 497
Laser 368
Leukocyte adhesion Defficiency (LAD), Type 1 53
Leukocyte recruitment 55
Leukocytes 41
Leukoplakia 130
Leukotriene 49
Lichen
erosive 129
planus 129
reticularis 129
Lincosamide 289
Linen strips 245
Lingual scaler 242
Lipopolysaccharide (LPS) 38, 40
Lipopolysaccharide-host interaction 38
Long-term temporary
crown lengthening 495
prosthetics 476
metal-reinforced, fixed 482
Lymphocytes 41

M

Macrolide 289
Macrophages 38, 42, 44, 55
activation 58
overactivation 60, 189
surface molecules 46
Maintenance therapy, periodontal (“recall”) 449
goals 449
lacking 455
Mandible
adhesive bridge 479
furcation involvement
hemisection 292
odontoplasty 388
regenerative therapy 390
scaling 388
molar, tipped 466
molar up-righting 466
Marginal brush 388
Matrix metalloproteinase (MMP) 20, 41, 50
inhibition 50
stimulation 50
Matrix proteins 325
Mattress suture
horizontal 308
vertical 308
Maxilla
furcation involvement
furcationplasty 389
root resection 394
trisection 396
gingivectomy 377
gingivoplasty 377
Maxillary anterior segment
space closure 464, 468
teeth, too long, narrow 497
Maxillary reconstruction 484
McCall’s festoon, gingival 156
Mechanoreceptors 19
Mediators, pro-inflammatory 40
periodontitis 66
Medical history 167
Medicaments 287
anti-retroviral 149
side effects 54
Membrane barrier technique, principles 436
Membrane barriers 325, 338
biological factors 338
natural, bioabsorbable 340, 345
non-resorbable 339, 341, 435
plus filler substances 345
requirements 340
resorbable 339, 345, 435
synthetic 339
Membrane, immediate, individual 340, 345
Metabolic disturbances 120
Metastasis 127
Metronidazole 90, 114, 289
MHC molecule groups 41, 46
Michigan splint 462, 473
Microbiology 23
Microorganisms
beneficial 202
oral 30
pathogenic potential 32
tests 178
Microtabs, smoking cessation 216
Miller classification of gingival recession 414
Mini-gingivoplasty 364
Mini-hook 402
Miniplast medicament carrier 379
Miniplast splint 379
Minocycline 289
Mixed fiber cementum, cellular 14
MMP, see matrix metallo-proteinase
Molar uprighting, mandible 466
Molars
loss 486
replacement, influencing factors 486
scaling, subgingival 261
Molecular biology, wound healing 206
Monocyte stimulation, genotype
IL-1 negative 189
IL-1 positive 189
Morphologic odontoplasty 248
Motivation of the patient 222
Mucogingival junction (MGJ) 7
Mucogingival problems 398
conservative procedure 399
Mucogingival surgery 296, 299
“plastic” 397
esthetic corrections 441
indications 397
Mucoperiosteal flaps 301, 303, 352, 415
Mucosal flaps 303
Mucosal thickening, surgical 442
Mucosal/gingival graft
autogenous, without epithelium 405
free (FGG) 401
advantages 412
contraindications 401
coronal repositioning 418
disadvantages 412
donor site 421
fixation 406, 417
halting recession 405
histology 421
indications 401
instruments 402
mobilization 407
recession coverage 416
results 408
shape determination 404
template 403, 407
thickness 404
thinning 404
transplant bed preparation 406, 416
two-stage procedure 418
wound healing
clinical 410
fluorescence angiography 411
of the donor site 410
wound treatment 408
Mucositis, peri-implant 514
Mucotome 402
power-driven 402
Multi-pharmacy 211
Multimalacia 211
Mycoplasms 30

N

Needle holder 305
Needle-suture combinations, atraumatic 306
Needles 306
Necrosis 29
Neoplasms, oral, in AIDS 146
Nerve fibers, nociceptive 19
Nerves
mandibular 19
maxillary 19
ophthalmic 19
trigeminal 19
New attachment 206
connective tissue 206
epithelial 206
Nicotine substitution 216
Nitromidazole 289
Normofunction 459
NSAID (non-steroidal anti-inflammatory drugs) 294
Nutrition, inadequate 120

O

Occlusal contact, premature
manual test 175
marking/recording 175
Occlusion, temporary, prosthetic, fixed 480
OCT (osseous coagulum trap) 329
Odds-ratio 51, 191
Odontoplasty 247
furcation therapy 388
Ofloxacin 289
One-shot prophylaxis, antibiotic 214
Oral hygiene 23, 208
by the patient 223
motivation 224
inadequate/poor 192
information 223
problems in the elderly 518
temporary, prosthetic, fixed 480
Oral hygiene aids 227
Oral hygiene procedures, bacteremia 214
Oral rinsing 236, 281
bacteremia 214
in the elderly 518
Orban knife 373
Ornidazol 289
Orthodontic “edge-wise” segment technique 465
Orthodontic treatment 463
recession, gingival 437
risks 463
Orthodontics 463
papilla loss 497
Osseoquest membrane 340
Osseous coagulum 325, 329
Osseous coagulum trap 329
Osseous craters, intra-alveolar, elimination 357
Osseous implantation, autogenous 330
incisions 330
wound closure 331
Osseous lamellae, fenestration 155
Osseous margin, alveolar, course 490
Osseous recontouring
indications 357
instruments 356
Osseous regeneration 324
Osseous remodeling 61
Osseous resection 356
instruments 356
Osseous sequester 153
Osseous surgery, flap procedure, evaluation 358
Osseous thickening (exostosis), idiopathic 126
Osseous transplantation 325, 327
Ostectomy 301
crown lengthening 493
Osteoblasts 61
Osteoclasts 61
Osteoplasty 126, 301, 329, 335, 362
harvesting bone 327
indications 357
Osteotomy, harvesting bone 327

P

P-15 351
Palate, hard, histology 404
Palatal mucosa, “sounding” 421
Palatal roof, histology 421
Palatal rugae, transfer during free gingival graft surgery 412
Panoramic radiograph 176
implant therapy 512
Papilla biopsy, ulcerative gingivitis 86
Papilla bleeding index (PBI) 67, 69, 224
gingivitis 82
periodontitis
aggressive 113
initial stage 117
chronic 109
Papilla knife 368, 373
Papilla loss 496
classification 496
crown construction 500
etiology 496
prosthetic 497
treatment, orthodontic 497
Papilla necrosis 87
Papilla preservation flap 303
closure 308
Papilla reconstruction 496
Papilla rotation 441
Papilla width 162
Papillae
facial, interdental 7
hyperplastic 496
Papilloma virus, human 145
Papillon-Lefèvre Syndrome (PLS), periodontitis, prepubertal 136
Parafunction 174, 462
Patient examination 212
PBI, see papilla bleeding index
PCR (polymerase chain reaction) 183
PD (pocket depth) probe 170
PDI (periodontal disease index) 67, 71
Peep probe 170
Pemphigoid 128
Pemphigus vulgaris 128
Penicillin 289
Penicillin allergy 214

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همیار دندانسازان و دندانپزشکان

Peptostreptoccoccus micros (Pm) 30, 33
Peri-implantitis 514
Periapical radiographic status 176
Perio-prosthetics 477
Perio-prosthetics, esthetics 489
  defining the problem 489
Perio-Set 247
Perio-System 339
PerioChip 292
PerioStar 270
Periostat 294
Periodontal abscess 107, 217
  emergency treatment 219
  opening 219
Periodontal disease 1
  classification 78, 519
  diagnosis 165
  etiology 21
  plaque-associated 77
  prognosis 165
Periodontal disease index (PDI) 67, 71
Periodontal dressings 336
Periodontal index 67, 71
Periodontal ligament 1, 7, 12, 206
Periodontal ligament alterations, histologic, through occlusal trauma 461
Periodontal ligament space 12, 18
  age-related changes 517
  hour-glass shape widening 461
  widened 472
Periodontal problems, age-related 516
Periodontal screening and recording (PSR) 67, 73
  treatment need 73
Periodontal status 194
  charting, computer-enhanced 195
Periodontal status I 109, 113, 194
Periodontal status II 111, 194
Periodontal surgery 295
  defect factors 297
  follow-up care 302
  goal 295
  healing, monitored 302
  patient factors 297
  patient selection 297
  preparation 302
  regenerative 299, 301
  surgical procedures 296, 299
  treatment result
    influences 298
    long-term 298
Periodontal/endodontic problems, combined 445, 447
Periodontitis 1, 3
  accompanying symptoms 105
  acute 60
  aggressive 3, 66, 95, 112, 521
    acute 114
    bacteriology 112
    generalized 97
    hereditary factors 97
    initial stage 116
    interdental space, plaque index 113
    localized 97
    papilla bleeding index 113
    probing pocket depth 115
    radiographic status 113, 115
  attachment loss 95, 98
    irregular 63
    regular 63
  bone loss 98
  bone loss, types 99
  chronic 3
    bleeding index 111
    interdental space, plaque index 109
    papilla bleeding index 109
    plaque index 111
    radiographic findings 109, 111
    severe 110
    treatment before orthodontic therapy 464
  classifications 95, 521
  clinical course 4
  cyclic disease course 63
  debridement, closed 271
  degree of severity 95, 98, 521
  diabetes mellitus 132
  epidemiology 75
  etiology 21
  furcation involvement 98, 102, 381
    horizontal measurement 102
    vertical measurement 103
  generalized 98, 521
    treatment, closed 279
  genetics 40, 53
  histology 59
  histopathology
  host reaction 294
  host sensitivity 40
  implant 4
  infection 32
  juvenile 176
  limited 62
  local/localized 521
    with recession 405
  localized 98
  marker bacteria 36, 51
  mediators 66
  molecular biology 40, 58
  necrotizing 95
    AIDS 143
  oral hygiene, effect 238
  overall diagnosis 196
  papilla loss 496
  pathobiology 96
  pathogenesis 39
  pathomorphology 98
  periodontal pockets, types 99
  pocket epithelium, alterations 104
  prepubertal 118
    Papillon-Lefèvre Syndrome (PLS) 136
  prevention/prophylaxis 198
  probing pocket depths 98, 106, 169
  profound, chronic 447
  progressive 61, 63
    etiology 202
  prophylaxis 199
  pulpal inflammation 445
  radical surgical procedures 4
  risk assessment 193
  risk factors 40, 51
    alterable 51, 54, 199
    genetic 52
    non-alterable 51
  root cleaning/planing 4
  single-tooth diagnosis 196
  stable lesion 63
  systemic disease 521
  test sensitivity 182
  test specificity 182
  therapy 3, 96 (see also treatment; see also therapy), 204
    diabetes mellitus 215
    failure 455
    in smokers 216
    regenerative 4
    success 252, 280, 318, 354, 358, 455
  tooth mobility 98, 107
  Trisomy 21 134
  ulcerative 85, 89
    bacteriology 87
    etiology 85
    generalized 89
    histopathology 86
    localized 89
    symptoms 87
    treatment 85
  untreated, recession, gingival 155, 160
  Widman procedure, modified 313
Periodontitis treatment
  auxiliary personnel 454
  failures 455
  negative consequences 456
  oral hygiene 238
  planning, elderly patients 518
  success 455
Periodontium 1, 7, 445
  blood supply 18
  in the elderly 515
    structural biological changes 516
  loading 459
  loading effects 459
  marginal, surgical correction 296
  nerve supply 19
  oral pathologic changes 119
  reduced 461
  turnover 20
Perlèche 144
PFM (porcelain-fused-to-metal) crown 500
PGE2 (prostaglandin E2) 49
Phagocytes 28, 41
Phagocytosis 42
Pharmaceuticals, topical 257
Phase-2 201, 295
Phase-3 201, 448
Phase-1 therapy 201, 221, 253
Phase contrast microscope 180
Phenotype, gingival 8, 490
  thick 8
  thin 8
Phenytoin 121
  pregnancy gingivitis 94
Phyenytoin-induced gingival overgrowth 121
"Pill" gingivitis 91
Plaque 21, 51, 65, 203
  bacterial, demonstration 225
  development 24
  expanse, subgingival 25
  gingivitis 82
  growth 24
  initial reaction 58
  "loose" 28
  subgingival 28
    bacteria 37
  supragingival 25
    bacterial 37
    non-specific 33
Plaque accumulation 23, 80
Plaque control 199
  before periodontal surgery 302
  chemical 235
    permanent 379
  on ovate bridge pontics 502
  supragingival 235
Plaque disclosing agents 225
  fluorescent 225
Plaque harvesting, subgingival 179
Plaque index (PI) 67, 199, 249
  periodontitis, chronic, severe 111
  recall effect 450
Plaque indices 192
Plaque removal
  effect of dentifrice 234
  interdental 251
  subgingival 90
  supragingival, professional 208
Plaque retention 26
Plaque retentive areas, elimination 247
Plaque-host interactions 22, 25
Plasma cells 43
Plasmids 34
Plastic probes 170
Platelet-rich plasma (PRP) 351
Plexus, vascular 18
  gingival in gingivitis 82
PLS 136
Pocket
  active 34
  acute suppurative 217
    emergency treatment 218
  bacterial reservoir 36
  deep, pulpitis 446
  gingival 59, 79, 104
  histologic 171
  inactive 34
  infra-alveolar 99, 104
  intra-alveolar
    defect anatomy 324
    filling material 327, 332
    therapy, regenerative 324
  periodontal 99
    treatment, resective 355
  supra-alveolar 99, 104
Pocket abscess 105
  emergency treatment 219
    medical 219
  radiographic picture 177
Pocket activity 105, 107
Pocket depth (see also probing depth)
  clinical charting 194
  genotype, IL-1 positive 191
Pocket diagnosis, microbial 180
  culture 181
  darkfield microscopy 180
  phase contrast microscopy 180
Pocket elimination 4, 204, 323
  resective 323, 355, 359
    results 364
Pocket epithelium 80
  periodontitis 104
  removal 254, 256
Pocket filling with chlorhexidine gel 281
Pocket flora
  removal 184
  subgingival 28
Pocket formation, gingival 25
Pocket fundus 169
  irregular course 203
  probing 272
Pocket implant, surgery, combined 336
Pocket instrumentation 256
Pocket marking forceps 368
Pocket measurement 106
Pocket probes 170
Pocket probing (see also probing depths) 168
Pocket reduction 280
  surgical 295
Pocket re-colonization 256
Pocket resection 323, 355, 359
Pocket rinsing 275
  antiseptics 283
Pocket therapy
  conservative 254
  dental implant problems 514
Pocket treatment, mechanical/instrumental 210
Pocket-television 285
Polishing diamonds 247
Polymerase chain reaction (PCR) 183
Polypeptide, synthetic 351
Pontic
  double 504
  ovoid/ovate 502
    plaque control 502
  ridge-form 502
    modified 502
  shape/form 502
  temporary, prosthetic, fixed 480
Porcelain full crown 500
Porphyromonas gingivalis (Pg) 24, 33, 51, 114, 254, 287
  culture 181
  reservoir, oral 255
  virulence factor 36
Posterior dental arch
  debridement, subgingival 274
  scaling, subgingival 261
  use of Gracey curettes 265
Powder-water spray devices 240
Precancerous lesion 130
Pregnancy gingivitis 91
  severe 92
  with phenytoin 94
Premature birth 64
Premolars, subgingival scaling 260
Prevention 198
  anti-infectious 208
  definition 198
  individual 257
  primary 221
  recall effect 450
  secondary 221
Prevotella intermedia (Pi) 24, 33
PR inhibitor 148
Primary prevention 198
Probe test, bacterial 183
Probes 170, 257, 320
  checking for scaling effectiveness 250
Probing pocket depth (PD) 169, 171, 271
  charting 194
  interpretation of probing levels 171
Prognosis 197
  local factors 197
  systemic factors 197
Prophy pastes
  dentin abrasion 242
  standardization 242
Prophylaxis, definition 198
Prophylaxis team 454
Prostaglandin E2 (PGE2) 49
Prostaglandins 49
Protease (PR), inhibitors 148
Proxoshape-Set 244
PRP (platelet-rich plasma) 351
Pseudopocket 79, 380
  gingival 371
PSR (periodontal screening and recording) 67, 73
  treatment need 73
PSR codes 72
Puberty gingivitis 91
Pulpal infection 445
  with deep pockets 446
Pulpal necrosis beneath amalgam restoration 446
Pus drainage 114

Q

Quick, values 212

R

Radiographic examination 176
Radiographic status, periodontal diagnosis 177
Radiographic technique, digital 177
Ramping 357
RCP method (rubber cup and paste) 251
Re-attachment 206
  connective tissue 206
Recall 449, 523
  dental implant problems 514
Recall appointment
  duration 450
  risk assessment 451
  treatment measures 452
Recall effect 450
Recall hour 452
  checklist 452
Recall interval 453
Recall practice 450
Receptor molecule 41, 46
Recession, coverage 405, 413
  connective tissue graft 419
    complications 432
    envelope technique 420, 430
    flap adaptation 428
    flap reflection 431
    flap repositioning 432
    Nelson technique 420, 425
    post-operative findings 428
    root surface pre-treatment 419, 426, 431
    surgical technique 420
    wound healing 432
  flaps, pedicle 415
  gingival graft, free 401, 405, 416
    fixation 417
    results 417
    transplant bed preparation 416
    two-stage procedure 418
  membrane, resorbable 437
  membrane adaptation 436
  membrane barrier technique, principles 436
  multiple tooth involvement 440
  surgical diagnostic aids 414
  surgical technique 413
  tissue regeneration, guided 435
Recession, gingival 1, 171, 297, 398, 456
  age-related 155, 160
  attachment loss (AL) 161
  "classical" 155
  classification 162
  consequences 164
  defect anatomy 414
  diagnosis 161
  etiology 155
  FGG treatment 405
  following orthodontic treatment 437
  following periodontitis therapy 155, 160
  generalized 159, 430, 442
  horizontal 162
  Jahnke classification 162
  localized 158
    deep 157
  localized periodontitis 405
  measurement 162
  Miller classification 163, 414
  prophylaxis/prevention 2
  reduction
    after guided tissue regeneration 444
    after mucogingival surgery 444
  symptoms 157
  therapy 2
  untreated periodontitis 155
  vertical 162
Reflection test 269
Regeneration 206, 323, 355
  connective tissue 206
  epithelial 206
  osseous 206
  periodontal 206
  pocket reduction 232
  prerequisites 323
Regeneration methods 323
  biological 354
  mechanical 354
Re-osseointegration, implants 514
Repair 206
Reservoir, microbial, oral 255
Residual pocket 206
  distal, active, wedge excision 320
  following modified Widman surgery 318
Resistance, bacterial, determination 181, 290
Restorations, old
  polishing 244
  reconstruction 244
  recontouring 244
  smoothing, approximal 245
Retention cyst 176
Reverse transcriptase (RT), inhibitors 148
Rhabdomyosarcoma 127
Ridge lap 502
  modified 502

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WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Rinsing 236
Risk clarification 165
Risk cluster 193
Risk estimation
  patient-related 451
  recall appointment 451
  tooth surface-related 451
  tooth-related 451
"Risk hexagon" 193
Risk profile, individual 193
RNA probe test 183
  marker bacteria 183
  results 185
Rodogyl 298
Rogues gallery 290
Roll flaps, pedicle, with alveolar ridge defects 505
Roll test 161
Rongeur 356
Root amputation with furcation involvement 387
Root cementum 1, 7, 12
Root cleaning/planing 4, 254, 256, 381
  bone regeneration 324
  closed 208, 271
    attachment gain 318
    pocket reduction 318
  direct vision 296, 311, 315
  problems 203
  surgical preparation 335
Root formation 7
Root fusion 172
Root planing 253, 256
  hand instruments 258
Root resection with furcation involvement 394
Root resorption 206
Root surface 203
  biomodification 352
  detoxification 253
Root treatment 446
RT inhibitor 148
Rubber cup, tooth polishing 243, 251

S

Saliva 54
Saliva gland involvement, HIV infection 147
Salivary calculus 26
Scaler 242
Scaling 250
  furcation treatment 386, 388
  hand instruments 258
  subgingival 238, 260
    pre-operative 300
  supragingival 239
Scalpel 402
Scratch test 269
Screening/triage 176
Scrotal tongue 135
Secondary prevention 198
Selective grinding (occlusal adjustment) 471
Selenomonas sputigena (Ss) 30, 33
Sensitivity, of a test 182
Separator 244
Septum, interdental, width 99, 101
Serum calculus 26
Sharpening oil 268
Sharpening stone 268
Sharpey fibers 12
Sharpness test 269
Signal molecules 206
Single-knot suture, interdental 308
Single-tooth diagnosis, periodontal 196
Single-tooth prognosis, periodontal 197
Single-tuft brush 233
Smile line 492
  classes 492
  crown 483, 501
  extremely high 492
  height 492
  maxillary anterior teeth, too long, narrow 497
  maxillary bridgework 485
  veneers 499
Smoking 54, 216, 298
Smoking cessation 216
"Soft chemo" prevention 235
Soft tissue augmentation for alveolar ridge defect 503, 505
Soft tissue curettage 253
Solo brush 233
Space closure, maxillary anterior segment 464, 468
Specificity, of a laboratory test 182
Spiramycin 289
Spirochetes 30, 33, 86, 180
Splinting
  adhesive technique 475
  permanent 472, 475
  semi-permanent 472, 474
    removable 474
  temporary 472
    removable 473
Split-thickness flaps 303
  half-moon shape 415
Square knot suture 307
  securing loop 307
Stabilization 471
  prosthetic 476
Stabilized power system 240
Stem cells 206
Stent probe 170
Stillman cleft 157, 399
Stimulators 233
Stroke 64
Stomatognathic system, function 459
Streptococcus 25
  constellatus 254, 287
  sanguis 24
Stress 54
Strip, minimally abrasive 251
Sulcus 65, 79
  gingival 10
  normal findings 99
Sulcus fluid, enzyme test 188
Superficial diagnosis 168
Supportive apparatus, osseous 16
Suppuration (pus) 29
Surgery, combined 334
  flap "plasty" procedure 334
  pocket implant 336
  wound healing 336
Surgery, mucogingival; see mucogingival surgery
Suture, continuous/encircling 308
Suture knots 307
  surgical 307
    doubled 307
Suture material 306
Sutures
  braided 306
  twisted 306
Syrette 283
Systemic disease 54
  in elderly persons 518
  initial phase 211
  periodontitis 521
  with chronic periodontitis 64

T

T4-lymphocytes, HIV replication cycle 148
Tannerella forsythensis (*Tf*; previously: *Bacteroides forsythus*) 30, 33, 36, 51
Tapping test 174
Teeth
  long 456
  too long, narrow 497
  too short 492
Teeth, anterior
  danger of fracture 456
  debridement, subgingival 273
  scaling, subgingival 260
Telescope ("coping") bridges 487
Temperature, subgingival 188
Temporaries, prosthetics
  fixed 478, 480
    oral hygiene 480
    problem zones 480
  removable 478
Temporary, immediate 220
  prosthetic, removable 478
Tertiary prevention 198
Test results
  predictive value
    negative 182
    positive 182
  validity 182
Tests
  host reaction 188
  immunologic 186
  microbial 178, 185, 288
  molecular biologic 183
  periodontal, evaluative 182
  sensitivity 182
  specificity 182
Tetracycline 289
  chemically modified (CMT) 50, 294
Therapy (see also treatment) 3, 201
  anti-infectious, non-surgical
    battling bacterial reservoir 255
    full mouth therapy 281
    instrumentarium 257
    limitations 278
    medicinal 287
    treatment goal 254
  anti-retroviral 148
  concepts 202
  conservative 253
  corrective 202, 208
  course 210
  goals 204
  phases 201
  problems 203
  regenerative 4, 299, 301
  resective 296, 299, 301
    attachment gain 318
    pocket reduction 318
Third molar extraction, distal pocket 284
Three-headed toothbrush 227, 230
Tissue adhesives 370
Tissue homeostasis 20
Tissue regeneration
  after surgery 296
  differentiation factors 351
  growth factors 351
  guided (GTR) 163, 324, 338
    covering areas of recession 435
    furcation involvement 390
    furcation treatment 386
    healing 338
    membrane barriers, see membrane barriers
    periodontal surgery 299, 301
    surgical procedure 342
  proteins 351
  with Emdogain 354
Titanium dental implants 511
T-lymphocytes 41, 43
  receptors 46
Tongue cleansing 237, 255
Tongue, dorsal brushing with chlorhexidine 283
Tongue scraper 237, 255
Tooth
  distal, lone-standing, wedge excision 319
  multirooted 382
  non-vital, metal-reinforced crown 500
Tooth cleansing
  problems 203
  professional 249
    hand instruments 242
    subgingival 238, 251
    supragingival 238, 240, 250
      instruments 240
Tooth crowding 26
  plaque-retentive niches 248
Tooth eruption, incomplete 492
Tooth extraction 480
  deciding factors 477
  maxilla 484
Tooth immobilization, temporary 471
Tooth loss 107
Tooth migration 107, 468
Tooth mobility (TM) 107, 174, 271
  elevated 460
    treatment, etiology-dependent 472
  etiology 470
  healing, influences 472
  periodontal
    initial 460
    secondary 460
  physiologic 460
  progressive, caused by abnormal occlusal forces 461
  test, manual 174
Tooth mobility, localization 174
Tooth neck
  exposed 458
  exposure from periodontal therapy 456
  grooves 247
  hypersensitive 164
Tooth polishing 243, 251
Tooth positional abnormalities 463
  etiology 463
  periodontitis-elicited 463
  treatment planning 463
Tooth replacement 484
Tooth root
  biofilm 28
"Tooth sticks" 231
Toothbrush trauma 161
Toothbrushes 223, 226
  ADA type 226
  electric 230
Toothbrushing, bacteremia 214
Toothbrushing technique 228
  solo technique 229
  sulcus cleansing technique 228
  traumatic 398
Toothpaste 226, 234
  potassium-containing 458
Toothpicks 231
Total ridge lap 502
Trabecular bone 16
Transcriptase, reverse, inhibitor (RT-inhibitor) 148
Transparency 491
Trauma, periodontal 217, 460
  occlusal 461
    periodontal adaptation 461
    periodontal alterations, histologic 461
    primary 461
    secondary 361
Treatment (see also therapy)
  mechanical, closed 202
  orthodontic 463
    recession, gingival 437
    risk 463
  surgical, open 202
Treatment need (TN) 454
Treatment planning 208
Trephine bur 328
Treponema denticula (Td) 30, 33, 51, 254
Tricalcium phosphate (TCP) 327, 333
Trifurcation, probing 173
Trigeminal ganglion (Gasseri) 19
Trisection in furcation involvement 387, 396
Trisomy 21 (Down Syndrome) 53
  periodontitis 134
Tubuli 458
  coverage 458
  obliteration 458
Tumor screening in smokers 216
Tunneling, furcation treatment 386

U

Ulceration
  expansive 145
  in HIV infection 147
Ultrasonic brushes 230
Ultrasonic devices 259
  attachments 282
Ultrasonic scaler 240, 250, 257
Umbrella effect 491, 500
Universal curettes 242, 251, 258, 268, 300
Universal knife 368

V

Vector ultrasonic device 282
Veneers 497
Venule plexus, post-capillary 18
Verrucae 145
Vertical bite-wing radiograph 176
Vicryl 339
Viral diseases 211
Viral load in HIV infection 141
Vitamin K 212

W

1-Wall bony pocket 100
2-Wall bony pocket 100
3-Wall bony pocket 100
Waterpik 236
Wedge-excision 299, 319
  according to Chaikin 319
  distal, lone-standing tooth 319
  flap closure/suture fixation 322
  flap reflection 321
  gingivectomy 322
  incisions 321
  modified incision 319
  wedge removal 321
Wedge-shaped dental defects 164
Whitening toothpaste 234
Wickham's striae 129
Widman flap, modified (see access flap)
Widman procedure, modified
  attachment gain 318
  incisions 310, 314
  post-operative measures 316
  principles 310
  Ramfjord technique 310
  residual pockets 318
Width
  esthetic (EW) 491
  biologic (BW) 12
  implant therapy 511
Width, biologic 490
  after crown lengthening via ostectomy 493
Williams probe 320
Wings 500
Wire ligature splint 473
Wound healing 205
  histology 206
  failures 206
  phases 205
  results 206

X

Xenograft 332
Xerostomia with HIV infection 147

Z

Zbinden scaler 242
Zerfing chisel 242
Zyban 216

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