Principles and Practice of Periodontics

With an Atlas of Treatment

Edited by

Frank M. Wentz, D.D.S., M.S., Ph.D.

Assistant Dean and Professor of Periodontics
University of Nebraska
College of Dentistry
Lincoln, Nebraska

Consultant in Oral Pathology
Veterans Administration Hospital
Omaha, Nebraska

This text revolves around the biologic concept in periodontics as it applies to diagnosis, treatment planning, therapy and prognosis. The concept is further related to recent ultrastructural, molecular, biochemical and immunobiologic advances. The purpose of this text is to aid students and practitioners in planning therapy that is tailored to a specific individual. In meeting this goal, the contributors review the contributions of the Vienna School with regard to the biologic concept and revise the classification of periodontal disease. Practical applications of recent advances are then proposed for improving the process of treatment planning. The included treatment atlas is designed to serve as a review of specialized treatment techniques. The text in the atlas conveniently faces the illustration to which it refers.

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PRINCIPLES AND PRACTICE OF PERIODONTICS

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AMERICAN LECTURE SERIES®

A Monograph in
The BANNERSTONE DIVISION *of*
AMERICAN LECTURES IN DENTISTRY

Edited by
ALVIN F. GARDNER, D.D.S., M.S., Ph.D.
Bureau of Medicine
*Food and Drug Administration**
Washington, D.C.

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CONTRIBUTORS

Donald F. Adams, D.D.S., M.S. Associate Professor and Past Director of Undergraduate Periodontics, Loma Linda University, School of Dentistry, Loma Linda, California. Diplomate, American Board of Periodontology. Consultant in ADA Council on Dental Education, ADA Commission on Accreditation.

Charles R. Amen, D.D.S. Associate Clinical Professor of Dentistry, School of Dentistry, University of Colorado. Active Teaching Staff, Denver General Hospital. Courtesy Staff, Presbyterian Hospital. Consultant in Fitzsimons Army Hospital, Veterans Administration Hospital, Federal Correctional Institution.

Wesley C. Berry, Jr., D.D.S., M.S. Professor of Periodontics, Research Director, College of Dentistry, University of Nebraska. Member of State of Nebraska Nursing Home Council. Consultant in Veterans Administration Hospital, Grand Island, Nebraska, Nebraska State Nursing Home Association.

Richard E. Bradley, D.D.S., M.S. Dean, College of Dentistry, University of Nebraska. Professor of Periodontics, Consultant in Periodontics, Veterans Administration Hospitals in Lincoln, Omaha and Washington, D.C..

Mansoor H. Jabro, B.D.S., M.S.D., D.D.S. Associate Professor and Chairman, Department of Periodontics, Boyne School of Dental Science, Creighton University. Consultant in Periodontics, Veterans Administration Hospital, Omaha, Nebraska.

Kenneth L. Kalkwarf, D.D.S., M.S. Associate Professor, Department of Periodontics, College of Dentistry, University of Nebraska. Consultant in Periodontics, Veterans Administration Hospital, Lincoln, Nebraska.

Kenneth D. Keith, M.S., Ph.D. Coordinator of Research, Family Rehabilitation Program, Meyer Children's Rehabilitation Institute, University of Nebraska Medical Center. Assistant Professor, Department of Preventive Dentistry and Community Health, College of Dentistry, University of Nebraska.

Arthur J. Krol, D.D.S., B.S. F.A.C.D, F.I.C.D., Chief, Dental Service, Veterans Administration Hospital, San Francisco. Adjunct Professor, Department of Prosthodontics, University of California, San Francisco. Diplomate, American Board of Prosthodontics.

Connell L. Marsh, B.S., M.S., Ph.D., Professor of Oral Biology (Biochemistry and Nutrition), College of Dentistry, University of Nebraska.

Lanny L. McLey, D.D.S., M.S., Assistant Professor, Department of Periodontics, College of Dentistry, University of Nebraska.

Gerald J. Tussing, D.D.S., M.S.D., Professor and Chairman, Department of Periodontics, College of Dentistry, University of Nebraska. Professor of Periodontics and Medical and Educational Administration, University of Nebraska Medical Center. Consultant in Periodontics, Veterans Administration Hospital, Lincoln, Nebraska.

Frank M. Wentz, D.D.S., M.S., Ph.D., Assistant Dean and Professor of Periodontics, University of Nebraska, College of Dentistry. Consultant in Oral Pathology, Veteran's Administration Hospital, Omaha, Nebraska.

FOREWORD

The *American Lectures in Dentistry* series advances newer knowledge for progress of dental practice. Success in modern dental practice is dependent upon biologic as well as mechanical considerations. The interdependence of dentistry on oral biology is so great that dentists are turning to oral biologists and oral biologists to dentists in order to understand the local and systemic basis of oral disease. The oral biologic processes are currently becoming sound foundations for clinical dentistry resulting in a rather rapid extension of postgraduate instruction. Therefore, each of the books in this series unravels the oral mechanisms and provides the clinical management of many problems which have existed for decades.

The *American Lectures in Dentistry* series is charged with a striving ardor of dental wisdom, prepared *de rigueur* by the highly qualified oral Wissenschaftler. New insights and *Entscheidungsproblem* are discussed by distinguished dental colleagues. A tradition will be established of offering the dental practitioner comprehensive surveys of recent developments in the various fields of clinical dentistry while presenting self-contained independent presentations directed to the general practitioner and specialist. This series is charged with providing the most current concepts in developing the continuing education for the dental practitioner.

The *American Lectures in Dentistry* series is based upon the following principles: concern, conviction, competence, commitment, and courage. The series will show concern for numerous dental problems, have a conviction that dental problems can be solved, have competence that this series can contribute to their solution, have a commitment of time and energy in the search for answers, and have the courage to take the necessary action to present solutions to various dental problems.

Dentistry is both a science and an art which fulfills a social function. This series will, therefore, encompass clinical, oral biologic and social topics which are most applicable to the general practitioner of dentistry. It is our hope that the efforts of the contributors will assist the dental practitioner in fulfilling his responsibility to his patients through sound judgments, proper technical knowledge, and dispatch. The *American Lectures in Dentistry* will serve as extremely practical references to aid the dental practitioner to resolve some of the problems encountered in the practice of dentistry as well as to broaden the horizons of those progressive dentists who desire the postgraduate knowledge and continuing education presented in this series. Contributors will focus attention on those aspects of dental practice causing the general practitioner the greatest concern and difficulty. The contributors of this series will help the practicing dentist to meet the challenges of the various phases of dental practice. Their observations should be beneficial to dentists seeking to attain the best possible treatment for their patients. New oral diagnostic problems, techniques, instrumentation and therapeutic measures are emerging. It is hoped that a tradition will develop whereby the *American Lectures in Dentistry* serves the dental practitioner and dental specialist alike.

Continuing education is dependent upon communicating the newer concepts in dentistry to the dental practitioner. It is hoped that this series will provide the important link of communication and result in better patient care. The continued quest for newer knowledge is the responsibility of the dental practitioner. This series will attempt to put the written current concepts into actual dental practice and therefore combat obsolescence. The editor and publisher are interested in encouraging the correlation of oral biologic principles with the clinical problems encountered by the dental practitioner who will base all his therapy on sound biologic concepts. The undergraduate education of a dental practitioner includes, in part, the development of his knowledge and skills. The continuing education of a dental practitioner includes the further development of his knowledge and skills by means of advanced educational programs which have a profound influence on the services performed by the dental practitioner. This series is predicated on the concept that continuing dental education is no longer confined to a selected few, but rather is a requirement for every present and future member of the dental profession.

There are dramatic changes taking place in dental care which will influence the future of dental practice. The unprecedented growth of scientific knowledge now is being applied to dentistry which is due to create tremendous changes in the art and science of dentistry.

It is not humanly possible to assimilate all of the knowledge in dental school that will be needed for the practice of dentistry. In addition, new knowledge is increasing at a rapid rate. Therefore, the dental practitioner can only keep abreast of the times by showing an initiative for self-learning. It is our hope that the *American Lectures in Dentistry* will stimulate the inquiring mind and provide the dental practitioner with a foundation in basic and new knowledge, skills and attitudes of dentistry upon which he can prepare himself for dental practice in current and future years.

The modern-day dental practitioner is in need of every opportunity possible to extend his knowledge and clinical experience. It is the purpose of the *American Lectures in Dentistry* series to represent one kind of continuing education which will be readily available to him. The *American Lectures in Dentistry* series places emphasis upon the fact that dental knowledge is not a rigid, fully elaborated system of facts, but rather one that is dynamic and constantly changing as new facets of knowledge are developed into the mosaic pattern of the whole.

Dr. Frank M. Wentz, Assistant Dean and Professor of Periodontics, University of Nebraska, College of Dentistry, Lincoln, Nebraska, and Consultant in Oral Pathology, Veterans Administration Hospital, Omaha, Nebraska, presents the latest methods and clinical techniques for the most effective solutions to the clinical problems in periodontics confronting the dental practitioner. The editor of *Principles and Practice of Periodontics—with an Atlas of Treatment* is a charismatic dental educator, researcher, and administrator who has succeeded far beyond his modest aims for this textbook. He and his expert contributors have, in fact, produced a very definitive work in a pellucid style in the field of periodontics. The editor's treatment of this most vital topic is magisterial throughout all portions of this book.

The editor and contributors provide the general practitioner of dentistry with an excellent and unique *Atlas of Treatment of Periodontics* based on scientific principles for his daily clinical practice of periodontics. The authors also provide the modern day dental student with a textbook of clinical periodontics based upon numerous researches into the problems of clinical periodontics, together with the basic science data which is necessary for an understanding of the numerous advances in the clinical practice of modern periodontics.

The postgraduate and graduate dental students in periodontics will find that this excellent textbook represents a treatise that fills the gap between clinical periodontics and the basic science findings and permits the treatment of the periodontic patient wih better methods of therapy. The latter fortifies the postgraduate student in his knowledge that his practice of periodontics is backed by scientific data.

I am confident that the reader, whether he or she be a dental practitioner, dental student, postgraduate or graduate student, of this excellent treatise on clinical periodontics will conclude that the editor and expert contributors have collectively enlightened all dental practitioners on the modern up-to-date concepts of clinical periodontics. The present work reveals fresh insights based upon knowledge of the latest research in the field of periodontics. All of the selected topics receive highly critical attention and the most important areas of periodontics are clearly emphasized. The discussions of the mechanisms of periodontic diagnosis and therapy are highly critical, yet concise, with numerous important relationships based upon fundamental oral biologic principles. Dr. Wentz and contributors have come to grips with problems which have plagued the dental practitioner in his treatment of the periodontic patient. This textbook, therefore, provides both dental practitioner and dental student with a specialized assessment of the most important features and problems in modern-day periodontics.

Thanks are due to the editor and his expert contributors for sharing with the dental practitioner and dental student the knowledge and experience so carefully collected and so excellently presented in this textbook. May the Atlas of Treatment portion help all dentists in the daily treatment of their patients. The editor of this American Lecture Series expresses his gratitude and appreciation to Dr. Wentz and contributors, who assumed this vital task for dentistry despite already overburdened schedules.

Alvin F. Gardner
Editor
American Lectures in Dentistry

PREFACE

The practitioner, with a serious interest in periodontics, after ascertaining what is happening to his periodontal patient, then judges what is causing it to happen (etiology). Proper treatment designed to remove the etiologic factors and to correct the anatomic deformities caused by the disease will then follow. However, the treatment must be individualized for each patient. The specific treatment needs for each patient vary. To understand these needs, a "Biologic Conceptual Approach" must be had. The "Biologic Concept" is the basis of this book. Firm adherence to the concept will allow the practitioner to escape the pitfalls of the "Provider Effect", i.e. to fit the patient to the treatment because it is available, instead of individualized therapy.

Each contributor follows the format of the "Biologic Concept" and follows it through examination, diagnosis, treatment planning, treatment and maintenance.

The Atlas of Treatment section is concise and consistent with the "atlas" style and for convenience faces the illustration to which it refers. The atlas is intended to be used as a tool in reviewing examination and treatment procedures and is intended to supplement and not to replace more detailed texts.

Principles and Practice of Periodontics is intended to serve as a "chairside assistant" for the practitioner of periodontics.

CONTENTS

PRINCIPLES AND PRACTICE OF PERIODONTICS

Chapter 1

THE CONTRIBUTIONS OF THE "VIENNA SCHOOL" TO PERIODONTICS

FRANK M. WENTZ

"THE HERITAGE of the past is the seed that brings forth the harvest of the future."[1] The heritage of modern periodontics is rooted in the *Viennese School of Bernhard Gottlieb* and his co-workers *Orban, Weinmann, Kronfeld, Stein, Everett and Sicker.* Orban called Bernhard Gottlieb "the Father of the Biologic Concept in Periodontics" and dedicated to him the first edition of his book *Periodontics.*[2]

"The Biologic concept is still the basis of DIAGNOSIS, TREATMENT PLANNING, THERAPY AND PROGNOSIS." However, the concept has evolved to now include ultrastructural, molecular, biochemical and immunobiologic advances. The findings challenge us to update our methods of therapy. With this in mind, the purpose of this book is to plan therapy that is tailored for an individual patient. Therapy basically has not changed, but the timing of specific therapy has.

The first step is to rethink the classification of periodontal disease. Periodontosis as a clinical entity is not accepted by many clinicians. The effects of occlusal traumatism on the periodontium are no longer regarded as simple cause and effect or only as an aggravating factor in periodontitis. In addition, our group at Nebraska is beginning to question whether periodontitis is always a sequel of gingivitis or if some forms of gingivitis are truly destructive periodontal diseases.

The second step is to review the biologic concept of the Vienna School for the basic fundamentals upon which to build the new scientific advances in immunobiology, microbiology and biochemistry.

The Contributions of Bernhard Gottlieb and the Vienna School, circa 1920-1933

The basic views of the biologic concept which were developed in this time period are as follows:

1. *The Concepts of the Gingival Crevice* and the true relation of the epithelial *attachment* to the tooth
2. The Physiologic Mechanism of Continuous Eruption, i.e. *Continuous Passive Exposure and Continuous Active Eruption*
3. *The Concepts of Continuous Deposition of Cementum* as a physiologic process and of the tooth as a vital organ to the needs of which the immediate supporting tissues are subservient
4. *The Histopathologic Effects of Traumatic Occlusion.*
5. Finally, the emphasis upon correct evaluation of the normal physiologic mechanism in an understanding of Periodontal Diseases

The accumulated research of Gottlieb and Orban for the decade and a half was gathered into a text, *Zahnfleish Entzundung und Zahn Lockerung.*[3] The presentation was richly documented with *Histologic* evidence of *the biologic concept.* The very title indicates the first new classification for periodontal disease, a condition previously generalized as *Periodontoclasia* or Pyorrhea Alveolaris. *Gottlieb and Orban* in the introduction indicate the book is concerned with the problem of *Loose Teeth,* not the simple clinical designation of destructive periodontal disease as we know it. They intended to separate some forms of gingivitis from periodontal disease proper when they stated the following:

> This presentation is primarily a study of loose teeth with or without inflammation, of the gingiva or of gingival inflammation alone. If, in examining a case of chronic gingival inflammation, it appears that the mouth has been decidedly neglected from a hygienic standpoint, it might be well to consider the probability of this general lack of care as the causative factor of the existing inflammatory process. Instead of going through the involved procedure of differential diagnosis, it is simpler to render the mouth hygienically clean with a careful oral prophylaxis, in order to determine if this procedure

alone would not be sufficient to restore the mouth to normal. We are fully aware that this procedure is by no means adequate in all instances, but experience shows us that a review of incipient pathologic lesions may, in this way, be completely resolved or aborted. We therefore re-emphasize the importance of deferring any diagnosis until the mouth is hygienically clean and the routine habits of proper mouth hygiene are re-established. The responsibility for this rests equally with the dentist and the patient.

The authors concluded that, according to their experience, the clinical picture by and large remains the same despite its etiology. The variations in the end organ, i.e. the dentition, are similar in character regardless of the wide divergence in their etiology. They stated that it is therefore correct to conclude that the homogeneous disease entity should be recorded in accordance with the clinical symptoms even though the causative factors may be manifold (Table 1-I).

The development by Gottlieb of the cementopathia theory of periodontal disease occurred after his emigration to the United States. The basis of this theory is predicated upon a cementum tissue inferiority which, by its failure to constantly renew cemetoid layer for attachment of periodontal ligament fibers, allows the down growth of the epithelial attachment. This is followed by epithelial separation from tooth and pocket formation.

Orban's classification, in 1948, was primarily based upon identification of basic pathologic tissue changes. He stated, "A classification is not a permanent structure. It must be adaptable to change and discovery. Like a filing cabinet, its function is a logical and systematic separation and organization of knowledge about diseases."

The time has arrived to adjust the classification of periodontal disease to incorporate new concepts and new discoveries. The new concept relating to the varied host responses to the etiologic factors is identified as the immunological response. This evolutionary classification combines all that is valid in the old concepts with all that is valid in the new findings. The classification has progressed from simplistic cause and effect understanding to the realization that there is a complex interplay in the host-bacterial parasite relationship.

The following classification is a summary of the periodontal disease state.

TABLE 1-I

EVOLUTION OF THE CLASSIFICATION OF PERIODONTAL DISEASE

Periodontoclasia 1920	Premature Gingival Recession	Senile Gingival Recession		*Pocket Formation*
Gottlieb Orban 1933	Gingivitis (Due to Poor Hygiene)	*Parodontal Pyorrhea*	*Diffuse Atrophy* Alveolar Bone *Secondary Traumatism*	*Accelerated Eruption* (Gingival Atrophy)
Gottlieb 1940	*Gingivitis*	Marginal Cementopathia	Deep Cemento-pathia-pocket formation *Secondary Traumatism*	
Orban 1948	*Inflammatory* Gingivitis Periodontitis	*Dystrophic* Atrophy Hyperplasia Degenerative Gingivosis Periodontosis		*Traumatic*
Future	*Gingivitis* with or without immunodeficiency	*Periodontitis* with or without immunodeficiency	Traumatic Periodontitis (Periodontosis)	

PERIODONTAL DISEASE: A Classification based upon the clinical manifestations and the immunological response of the host. *(Modified from discussions with Doctor Tim McVaney)*

I. Normal

Negative to mild immune responses to bacterial environment.

No clinical manifestations of inflammation.

Epithelial attachment within normal physiological limits.

Slight infiltration of inflammatory cells in gingiva.

II. Inflammatory

A. Local

GINGIVITIS

Mild to moderate inflammatory and immune response, clinically apparent in gingiva as inflammation.

Friable epithelium, bleeding points.

Epithelial attachment within normal physiologic limits.

B. Systemic

Mild to moderate inflammatory and immune response, may not demonstrate classic signs of inflammation in gingiva.

Epithelial attachment within normal physiologic limits.

Gingiva demonstrates a *hyperplastic response* and hemorrhage.

III. Destructive Inflammatory (Humoral mediated inflammation)

A. Local

GINGIVITIS / GINGIVOSIS

Mild to moderate inflammatory response in gingiva, clinically apparent as inflammation, may be present as edematous hypertrophy.

Inflammation likely involves attached and marginal mucosa.

Epithelial attachment proliferating—may demonstrate early signs of rete ridge formation in sulcular epithelium.

Considerable *round cell* involvement in connective tissue.

B. Systemic

Hormonal level variations may potentiate the effects of inflammation.

Moderate to severe inflammation.

May demonstrate tissue necrosis (ANUG); atrophy; loss of basement membrane integrity, e.g. gingivosis.

The above classifications may be considered to be limited to the epithelial and connective tissues. Bone destruction is not prominent.

IV. Destructive Inflammatory (Cell mediated inflammation)

PERIODONTITIS

Mild to moderate inflammatory response in apical portions of the pocket.

Tissue may appear slightly abnormal, though the altered appearance may not be as striking as in inflammation characterized by readily bleeding tissue; in the sulcular area, tissue may appear more firm and fibrous, with a less-than-acceptable contour.

Epithelial attachment demonstrates apical migration.

Sulcular epithelium demonstrates rete ridge formation.

Alveolar process is involved with resorption associated with apical progression of the epithelial attachment.

Clinical signs ascribable to lymphocyte stimulation, e.g. fibroblast stimulation, osteoclast activating factor.

A. Modified Local

Magnified destruction of the alveolar process and proliferation of the epithelial attachment, associated with placement of the dental organ in an environment that induces more rapid destruction than repair mechanisms can cope with: We have exceeded the physiologic adaptability, e.g. occlusal trauma, iatrogenic (poor gingival margins, rubber bands).

B. Modified Systemic

PERIODONTOSIS

Increased rate of tissue destruction due to the following:

1. Antigenic overloads resulting in impaired immunal reactions.
2. Cellular overreaction resulting from excessive sensitization with increased levels in intercellular hormones, e.g. prostaglandins, cyclic AMP.
3. Hormonal imbalances which potentiate the effects of the inflammation, and immune reactions.
4. Metabolic deficiencies which can retard repair mechanisms, thus upsetting the balance between repair and

pathology, resulting in more severe destruction.

This chapter introduces the theme of the book. Logically, the classification prepared will be expanded and justified in Chapter 2, where the immunological host response to the etiologic factors will be discussed by Doctor Wesley Berry.

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Chapter 2

ETIOLOGY OF PERIODONTAL DISEASE

WESLEY C. BERRY, JR.

THE ROLE OF BACTERIA IN PERIODONTAL DISEASE

THE FACT THAT ORAL bacteria play the leading role in the etiology of inflammatory periodontal diseases has been well established by dental researchers. All evidence indicates that bacteria cause gingival tissue destruction and the oral clinical signs and symptoms of disease.

The oral cavity is an excellent incubator for many different species of microorganisms, a large number of which are considered to be *normal* residents of the mouth. However, the primary concern to the dentist and patient is the resident bacterial population associated with the gingival crevice area. This anatomic location is of significance because of the relationship of the gingival epithelial attachment to the surface of the tooth.

Host-Parasite Relationship

Socransky,[1] Gibbons[2] and co-workers have approximated total microscopic counts of microorganisms found in the healthy human gingival crevice. The microorganisms averaged more than 100 billion living and dead organisms in each crevice area. This is an astonishing number of organisms, particularly when one considers that a state of clinical health exists in the presence of so many potentially destructive bacteria. The predominant cultivable organisms were determined by dispersing the bacteria with ultrasound and plating them on nonselective media.[2] Table 2-I illustrates the types of the organisms found in the oral cavity.

If plaque is defined as the attachment and accumulation of bacteria on the surface of a tooth, then indeed a state of gingival health can exist in the presence of dental plaque. The inevitable question then arises concerning the factor(s) responsible for changing the host-parasite equilibrium observed in health to the host-parasite relationship resulting in gingival disease.

One of the classic investigations which lead to an awareness of the significance of the bacteria-gingival tissue relationship was reported by Loe, Theilade and Jensen.[3] Utilizing a sample of twelve dental students with healthy gingival tissue, they demonstrated all twelve subjects developed increasing amounts of plaque and associated clinical gingival inflammation. When all oral hygiene procedures were reinstituted, the gingival inflammation subsided. A later study confirmed that antibiotic therapy effectively reduced gingival exudate and leucocytic emigration.[4] The gingival exudate and leucocytic emigration were used as indicators of subclinical inflammation. These studies in humans and a similar study in dogs[5] have provided significant evidence that one of the major factors in changing the host-parasite relationship in the direction of disease is increasing the amount of and/or numbers of bacteria in dental plaque.

Other investigators have substantiated the significance of bacterial plaque in the initiation of periodontal disease by placing various mechanical irritants in the mouth.[6–8] These mechanical irritants, which included overhanging restorations, roughened tooth surfaces, cement and sutures, did not substantially increase the inflammatory response in the adjacent gingival tissues. A similar phenomenon is observed in germ-free animals, which form calculus in the absence of bacteria.[9–11] The germ-free calculus does not produce any evidence of disease such as inflammatory pocketing changes or alveolar bone destruction. However, the presence of certain bacteria in gnotobiotic animals *in the absence* of calculus can cause severe periodontal destruction.[12–14]

Clinical observations by nearly all of the dental