MATTHIAS RZEZNIK

CLINICAL PERIODONTICS

A Modern and Preventive Approach

In collaboration with
Adrian Brun
Sébastien Jungo
and Frédéric Chamieh

QUINTESSENCE PUBLISHING

Berlin | Chicago | Tokyo
Barcelona | London | Milan | Mexico City | Paris | Prague | Seoul | Warsaw
Beijing | Istanbul | Sao Paulo | Zagreb

One book, one tree: In support of reforestation worldwide and to address the climate crisis, for every book sold Quintessence Publishing will plant a tree (https://onetreeplanted.org/).

Title of original issue:
PARODONTIE CLINIQUE
Une approche moderne et préventive

A CIP record for this book is available from the British Library.
ISBN: 978-1-78698-140-0

QUINTESSENCE PUBLISHING DEUTSCHLAND

Quintessenz Verlags-GmbH
Ifenpfad 2–4
12107 Berlin
Germany
www.quintessence-publishing.com

Quintessence Publishing Co Ltd
Grafton Road, New Malden
Surrey KT3 3AB
United Kingdom
www.quintessence-publishing.com

Editing: Quintessence Publishing Co Inc, Batavia, IL, USA
Layout and Production: Quintessenz Verlags-GmbH, Berlin, Germany

Printed and bound in Croatia

PREFACE

The idea of writing this book came to me during my years of teaching at the University of Paris-Descartes. At that time, I was proposing to my students a different vision of the management of patients with periodontal disease, and the students often asked me in which book they could find the principles of this more modern approach to periodontal management. Periodontal encyclopedias seemed too boring to them, as most of the books did not address the possibility of simplifying the protocols to make them accessible to the greatest number of people. The richness of my career in different universities (Reims, Paris, Corte), the encounters with talented and passionate practitioners, and the numerous exchanges that I had the chance to have with my colleagues in France and abroad gave me, I think, a certain open-mindedness, allowing me to get the best out of it, dare I say the "quintessence," which I tried to retransmit through this book.

I didn't want a book that was too classic. I am fortunate that the publisher gave me total freedom in writing, as much for the content as for the form, and I thank him for that. This book was conceived in the manner of a novel, with its "heroes" being our patients, whose "adventures" we will follow. Thus, throughout this book, in the course of the chapters, we will find our different characters undergoing treatment. All the steps of the periodontal treatments will be detailed, explained, and argued. The use of ultrasonic devices will be explained by means of videos (on models, on scaled teeth, and on patients) to allow the reader to better understand their use. From the management of simple gingivitis to the most complex periodontal treatments, multiple clinical videos are included in this book to help our readers visualize the concrete implementation of the therapies so that they can, I hope, put them into practice in the office tomorrow.

Dr Matthias Rzeznik

FOREWORD

Periodontal diseases are complex, multifactorial, and among the most prevalent chronic conditions affecting adults worldwide. Characterized by inflammation and destruction of the tissues supporting the teeth, periodontitis can lead to tooth loss, loss of masticatory function, and systemic complications that can have far-reaching consequences. The evidence base regarding the relationship between oral biofilm and the host inflammatory response to the etiology of periodontal disease continues to grow over the years. Periodontal disease initiation and propagation is through a dysbiosis of the commensal oral microbiota (dental plaque), which then interacts with the immune defenses of the host, leading to inflammation and disease. The traditional approach to periodontal therapy has focused on eliminating the bacterial biofilm, typically through mechanical and chemical means. While this approach has proven effective, it is often reactive and fails to address the underlying causes of periodontal disease. Therefore, a more modern and preventive approach to clinical periodontology is of utmost importance.

In recent years, there has been a growing recognition of the need for a more comprehensive approach to clinical periodontology. Simplified periodontal procedures that require less chairside time and surgical equipment, as well as reduced treatment costs and patient morbidity, have increased in popularity. An approach that recognizes the multifactorial nature of periodontal disease and aims to identify and address the various risk factors that contribute to its development and progression is of utmost importance. A proper diagnosis is key to the development of an individualized treatment plan, and emphasis on the importance of patient education, motivation, and self-care as integral components of periodontal therapy is essential to successful management of the disease.

The text you are about to read is a comprehensive guide that provides clinicians with the latest evidence-based knowledge and skills necessary to deliver simple, effective, and relatively painless periodontal care. This book is a gem. It covers a range of topics, including the etiology and pathogenesis of periodontal disease, risk assessment, diagnosis, treatment planning, and a detailed description of various clinical techniques. It also explores the role of various preventive measures in the management of periodontal disease, such as plaque control, patient motivation, reevaluation, and maintenance.

Written by young rising stars in periodontics who bring their expertise and insights, this comprehensive and informative text is a valuable resource for dental practitioners, hygienists, and students seeking to improve their understanding and management of periodontal diseases. *Clinical Periodontics: A Modern and Preventive Approach* aims to provide a comprehensive understanding of periodontal diseases and their management, emphasizing

the importance of prevention and early intervention to achieve optimal outcomes. It is organized in a user-friendly format, with extensive case examples, videos, clinical tips, and evidence-based recommendations throughout. It is designed to be a practical and accessible guide that can be used by clinicians at all levels of expertise. The focus is on a modern, novel, patient-centered approach, which involves thorough diagnosis and personalized treatment plans based on the patient's individual needs.

Dr Véronique Benhamou, BSc, DDS, Cert Perio
Periodontist
Associate Professor, Director, Division of Periodontology
Faculty of Dental Medicine and Oral Health Sciences, McGill University
Montreal, Quebec, Canada

FOREWORD

The periodontal interface constitutes a singular place where the epithelial envelope that protects us is broken during tooth eruption. A dynamic interface and not a passive barrier, this contact zone, before a place of confrontation, becomes a place of interactions between the host and the bacterial communities that reside in this oral ecosystem. These interactions result in a balance between the forces at work that guarantees the integrity of the tissues and their functionality. Microbiota are recognized today as an essential component in maintaining these local balances. It is not ignored by our immune system but tolerated, which represents a radical change of paradigm in the sense that tolerance to bacterial populations obliges us to redefine our biologic identity. This must now be presented as an open identity, articulating both exogenous and endogenous elements, and no longer as a closed entity ruthlessly eliminating all exogenous elements. At the level of the junctional epithelium, as with other interfaces, there is a tolerance process, which implies a precise and permanent microbial perception and a well-controlled basal immune response.

The great complexity of these interactions highlights the fact that in case of dysbiosis, the return to the initial equilibrium, or more likely to an alternative equilibrium between bacterial populations and the immune system, does not come naturally. It might seem a little vain to imagine that our inserts, curettes, or other scalpels will infallibly restore the subtle interplay of these interactions and bring about a return to equilibrium. This will require, beyond the local instrumentation, time. This time is often considered as a dimension to be reduced in our culture of urgency, but it is an ally in the return to homeostasis. Better biologic knowledge will allow the establishment over time of a sort of therapeutic gradient, allowing optimal use of the various therapeutic tools according to the very different healing dynamics from one patient to another.

Dysbiosis, along with bacterial pressure perceived as an aggression, involves genetics, health status, and the environment. If the reduction of the bacterial load is unavoidable, the treatment must integrate and be adapted to the individual characteristics of the patient. In other words, trying to stabilize a periodontal disease without taking care of the patient as a whole cannot be considered a satisfactory practice. This would constitute an "open door" to therapeutic failure or disease recurrence.

The management of a patient suffering from periodontal disease must be personalized and participatory, with the therapy offered based on scientific evidence. It is within the framework of long-term chronic pathologies, which

present high risks of recurrence, that therapeutic education of the patient aided by motivational interviewing takes on its full meaning. This approach aims at not only considering patients as the owners of their toothbrushes but as patients who benefit, thanks to their practitioner, from a certain expertise in their disease, allowing them to appropriate it and, to a certain extent, to manage it.

This participatory approach also resonates with the "healthy" person who only aspires to remain so. The identification of vulnerability factors upstream within the framework of primary prevention is only meaningful if it is accompanied by an awareness on the part of the patient, leading to possible behavioral, environmental, or general health modifications. The patient needs to understand the imbalance he or she has experienced or is at risk of experiencing and act accordingly to reduce the impact of this factor. It is interesting to observe that the evolutions in the understanding of the physiopathology of periodontal diseases are accompanied by evolutions in patient management, in particular through therapeutic education, the objective of which is to make patients more participative, not to mention more active, in the management of their health.

Dr Dominique Guez
Doctor of Dental Surgery, University of Paris-Descartes
Specialty Certificate (CES) in Periodontology (1987), University of Paris-Descartes
University Diploma (DU) of Periodontology, University of Paris-Descartes
University Doctorate, Laboratory of Bone Physiopathology, University of Paris-Descartes
Senior Lecturer, University of Paris-Descartes
Hospital practitioner, Public Assistance–Paris Hospitals (AP-HP), Bretonneau Hospital

AUTHOR

Dr Matthias Rzeznik
Doctor of Dental Surgery
Graduated from the Faculty of Reims in 2010
Former university hospital assistant in periodontics, University of Paris-Descartes, Bretonneau Hospital
Master 2 oral clinic specializing in periodontics (University of Paris V)
European inter-university diploma in implantology (University of Corsica)
Practice limited to periodontics and implantology (Lille)

President of the Paro Clap Association

CONTRIBUTORS

Dr Adrian Brun
Doctor of Dental Surgery
Specialty Certificate (CES) in Periodontics (Paris)
Master 2 applied oral physiopathology, periodontology, Paris-Descartes
Graduated from the Faculty of Paris-Descartes in 2013
University lecturer–Hospital practitioner (MCU-PH) in periodontology, Odontology Training and Research Unit, Montrouge
Orofacial Pathology, Imaging, and Biotherapies Research Unit
Head of periodontology, Oral Medicine Department, Hôpital Henri Mondor Public Assistance–Paris Hospitals (AP-HP)
Founding member of the Paro Clap Association

Dr Sébastien Jungo
Doctor of Dental Surgery
Graduated from the Faculty of Paris-Descartes in 2010
University hospital assistant in public health, University of Paris-Descartes, Bretonneau Hospital
Specialty Certificate (CES) in Periodontics (Paris)
Master 2 methodology and statistics in biomedical research
Inter-university diploma in liaison psychiatry and somatic care in mental health
Practice limited to periodontics (Montreuil)
Founding member of the Paro Clap Association

Dr Frédéric Chamieh
Doctor of Dental Surgery
Graduated from the Faculty of Paris-Descartes in 2011
Specialty Certificate (CES) in Periodontics (Paris)
Master 2 oral clinic specialized in periodontics (University of Paris VII)
European postgraduate in periodontology and implant dentistry, European Federation of Periodontology (EFP)
Clinical university diploma in advanced implant surgery, Paris VII
University diploma of expertise in dentistry, Paris VII
Practice limited to periodontics and implantology (Chartres)
Member of the Paro Clap Association

ACKNOWLEDGMENTS

To my family, my friends, my colleagues,
I am very lucky to be so well surrounded.

To my wife, Cathy, for the unconditional support she gives me every day.

To Gérard for your friendship and for sharing your passion for video with me.

To Sandrine, Marie-Pascale, and Véronique, you have inspired me a lot.

To Gilles for introducing me to ultrasound.

To my wonderful friends and associates Marc-Antoine and Sophie for your friendship and the pleasure I have every day to work with you. And to our global team members who give their best everyday: Sofiya, Pauline, Aurélie, Malika and Emmeline.

To the members of the Hoche firm:
Thank you, David, for your friendship and support, your humility and generosity. I am very lucky to work with you.
Pascal, thank you for your welcome to the firm and for sharing your knowledge with me.
Pierre and Zeina, you are great colleagues, passionate and motivated; it is a pleasure to work with you.
And a thank you to the rest of the team: Lucie, Caroline, Silvia, Florica, Leila, and Domecilia.

To the periodontics department of the University of Paris-Descartes, thank you for welcoming me and supporting me in my various projects.

To the Strass Group: You are a source of inspiration and sharing.

To my editors: I would like to thank Karim and Léon as well as the entire Quintessence team for trusting me with this project.

Adrian, Sébastien, and Frédéric, thank you for the honor of collaborating on this book.

Adrian, your keen eye for detail, your in-depth knowledge of periodontal pathophysiology, and your more academic approach to the subject have helped to bring a superior dimension to the writing of this book. I also thank you for your friendship and your listening skills; I admire the energy you put into your daily teaching.
Sébastien, thank you for being by my side for all these years; you are a great friend. Your knowledge of psychology was very useful in the writing of this book and is also useful to me every day in my practice. You taught me how to communicate better with my patients.

Frédéric, our friendship was born from our professional exchanges; you impress me a lot by your knowledge and your practice in surgery. Thank you also for your listening and your open-mindedness.

Véronique and Dominique, thank you for doing me the honor of writing the forewords to this book.

Dr Matthias Rzeznik

Videos in this book!

This book contains free augmented reality videos accessible from your smartphone or tablet. Easy, reliable, instant, and offline video viewing allows you to enrich your viewing experience thanks to the multimedia content selected by the author.

Download

Download the “Parodontie clinique” application from the App Store (iOs) or Google Play (Android).

Locate

Look for clinical photographs with the augmented reality pictogram.

Hover over

Hover over these photos with the app open on your smartphone or tablet to play the videos.

TABLE OF CONTENTS

1 Etiology and Epidemiology of Periodontal Disease

Why talk about periodontal disease today?

The World Health Organization (WHO) has described periodontal disease as a very common cause of tooth loss.

In 2007, studies by Bourgeois et al gave frightening statistics, with 95% incidence of interdental bleeding on probing in young people aged 20 to 25 and almost 50% incidence of signs of clinical attachment loss in patients older than 50 years.[1]

If we analyze these raw data, we realize that since more than half of the biofilm is located in the interdental area,[2] and there is a lack of effective brushing in this area in the general population, the use of an interdental brush in these spaces triggers bleeding, a sign of the already existing subclinical inflammation.

As these patients are rarely detected as "sick" because of the absence of macroscopically visible clinical signs, it is easier to understand that, 30 years later, without having modified or adjusted their brushing technique, they will be prone to gingivitis or, for those whose immunity is less favorable, periodontitis in the years to come.

The WHO has described three levels of prevention:

- Primary prevention
- Secondary prevention
- Tertiary prevention

These famous primary prevention patients, called "healthy carriers" because they have no obvious clinical signs, will develop gingivitis if their brushing habits are not corrected, thus leading them to secondary prevention. Their health condition is reversible with a possible return to a healthy state. However, if oral hygiene is not controlled in a more fragile periodontium, it can lead to periodontitis in almost 50% of patients.

Periodontitis is chronic because a cure cannot be envisaged, leading the patient to tertiary prevention (stabilizing and limiting recurrence).

Therefore, in our therapy, it is essential to manage patients as early as possible in order to avoid letting them go down the slope of periodontal disease.

We will see throughout this manuscript that the different levels of prevention will be managed with the same tools and similar protocols but to different degrees. Mastering primary prevention will therefore be the key to understanding all the treatments we will implement.

Bacterial Etiology

Periodontal diseases are inflammatory pathologies whose etiology is bacterial and, if not treated in time, will lead to the loss of teeth.

The gingival sulcus is bounded on one side by the tooth and on the other side by the junctional epithelial attachment of the gingiva, which forms a tissue barrier (Fig 1-1). Bacteria that are initially supragingival will accumulate and organize themselves into a biofilm (corresponding to a structural organization of bacteria), which during its maturation will develop in a subgingival direction. This will lead to a rupture of the junctional epithelial attachment ("loss of attachment") and finally to the destruction of the periodontium. We then observe the formation of a periodontal pocket, within which biofilm accumulates.[3]

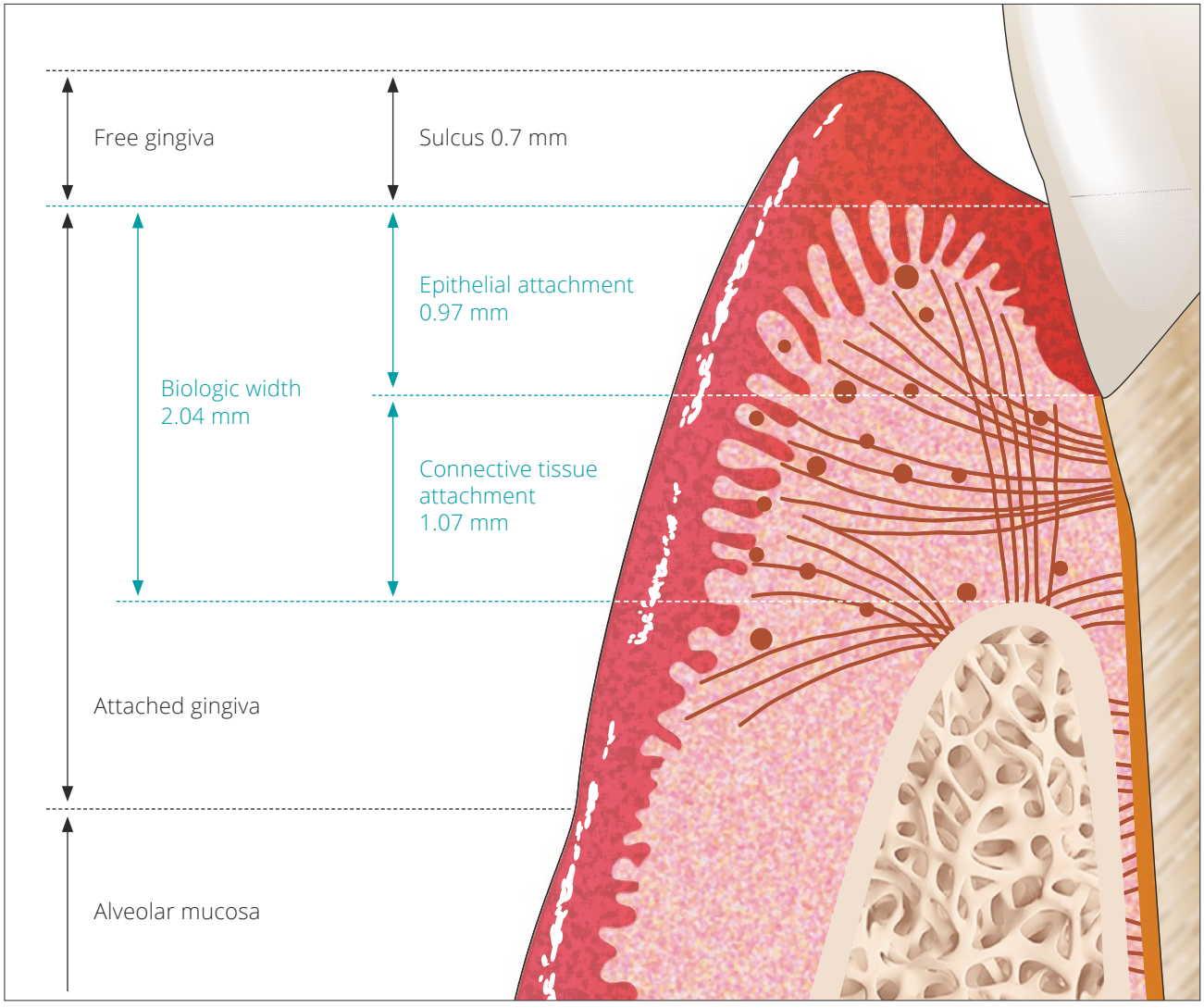


Fig 1-1 Diagram of the dentogingival attachment system.

Reaction of the periodontium to the accumulation of biofilm

As soon as the plaque accumulates, an inflammatory infiltrate can be observed in the underlying connective tissue, accompanied by an evolution of the microcirculation. The latter will dilate due to the increase in vascular diameter. There will also be a transformation of the endothelium of the capillaries, which will acquire a secretory phenotype and release cytokines into the environment, participating in the immune response.[4] A number of changes will then occur, including the arrival of a large number of neutrophils (extravasation), which will migrate through the junctional epithelium. Other cells, such as lymphocytes and macrophages, will also accumulate.[5] The body will then organize itself to protect the surrounding tissues. With time and without treatment, a migration of the epithelium in the apical direction will be observed, associated with a loss of attachment and alveolysis, which signals periodontitis.

The transition from periodontal health to periodontal disease is associated with a shift from a symbiotic to a dysbiotic microbial community that is itself faced with a paradigm. On the one hand, these bacteria need protection from the immune system to survive, but on the other hand, they need the immuno-inflammatory response to destroy the surrounding tissue. Indeed, neutrophils are the most commonly recruited leukocytes at the gingival sulcus and are necessary for periodontal tissue homeostasis.[6] However, a too rapid, too large, or poorly regulated response can cause collateral tissue damage due to the release of toxic substances or enzymes[7] and the activation of bone resorption. Neutrophils are thus partly responsible for the destruction of periodontal tissue.[8] The chronic recruitment of a number of neutrophils to the periodontal pocket is probably due to their inability to control the microbiota contributing to dysbiosis.[6]

Organization of the immune system and periodontal response

The immune system consists of two interconnected components, namely the innate system and the adaptive or acquired system. Innate immunity, which is of particular interest to us, is a nonspecific response of the organism to a pathogenic aggression. The immune system is organized into three levels[3]:

1. A cellular barrier, consisting of phagocytes (including neutrophils, macrophages, and dendritic cells) and natural killer lymphocytes
2. A chemical barrier, via antimicrobial agents—more than 1,700 antimicrobial peptides have been identified to date, and 45 antimicrobial peptides have been identified within saliva, sometimes within gingival fluid[9]—and proinflammatory cytokines
3. A physical barrier, ie, the oral mucosa

Because of the significant vascularization of the periodontium, neutrophils, in case of bacterial aggression, will quickly be mobilized at the involved site. This is followed by rolling, activation, adhesion, and then transendothelial migration of these cells, which will then go through the connective tissue and the junctional epithelium, to finish their course by forming a cellular barrier, called a "wall" of neutrophils, in contact with the dental plaque.

This response occurs following the stimulation of resident cells by bacterial virulence factors such as lipopolysaccharides (LPS), which will lead to the release of pro-inflammatory cytokines (eg, interleukin [IL]-1β, IL-8), resulting in the expression of adhesion molecules by vascular endothelial cells and the recruitment of a greater number of granulocytes (chemotactic gradient).[10] Adaptive immunity will subsequently come into play but will not allow a return to equilibrium without treatment.

The oral microbiota

The mouth is an incubator for bacteria in which they develop perfectly. The salivary environment ensures a permanent humidity conducive to the development of microorganisms. The pH of the mouth naturally varies between 6.75 and 7.25, and the temperature is between 35°C and 36°C. These parameters may vary according to external factors (eg, food) but remain stable overall. The oral microbiota is specific to the individual, and today there are more than 700 bacterial species. The vast majority of the microbiota is commensal, ie, resident, and nonpathogenic.[11]

The periodontal microbiota

The etiopathogenesis of periodontal diseases is due in large part to microorganisms. As previously mentioned, the transition between a microbiota associated with health and a microbiota associated with periodontal disease is called dysbiosis. Periodontal diseases are therefore not diseases caused by a single bacterium but by dental plaque, which is a mainly commensal bacterial community of several hundred species surrounded by a matrix, perfectly organized in biofilm. Plaque is much more than a simple aggregate of immobilized microorganisms and bacteria developed within a structured community.

A hypothesis evoked in the etiopathogenesis of periodontal diseases is that the oral microbiome is organized in bacterial complexes. Socransky et al were pioneers in this respect.[12] They have indeed shown that certain bacterial associations predominate according to the state of periodontal health (ie, health, gingivitis, or periodontitis). Deep periodontal pockets and sites associated with bleeding on probing were significantly correlated with the detection of *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia*.

Yellow, green, purple, orange, and red color coding defines the chronologic order of periodontal pocket colonization (Fig 1-2). The coaggregation capabilities of periodontal bacteria are critical in the establishment of dental plaque. Classically, "periodontopathogenic" bacteria meet four conditions:

1. They are transmissible to humans.
2. They colonize or invade host cells.
3. They escape the host's defense mechanisms.
4. They lead to destruction of the infected tissues.

The keystone pathogens

The notion of "keystone pathogens" has been evoked in the pathogenesis of periodontitis.[13] If periodontitis is a disease involving an association of bacteria in a biofilm, certain bacteria would allow, in a more significant way, the passage from the state of health to the state of periodontitis, compared to the others. These keystone pathogens would have the capacity to hijack the mechanisms of innate immunity (IL-8, complement system, and toll-like receptors [TLRs]) and adaptive immunity (T helper 17 [Th17] pathway).

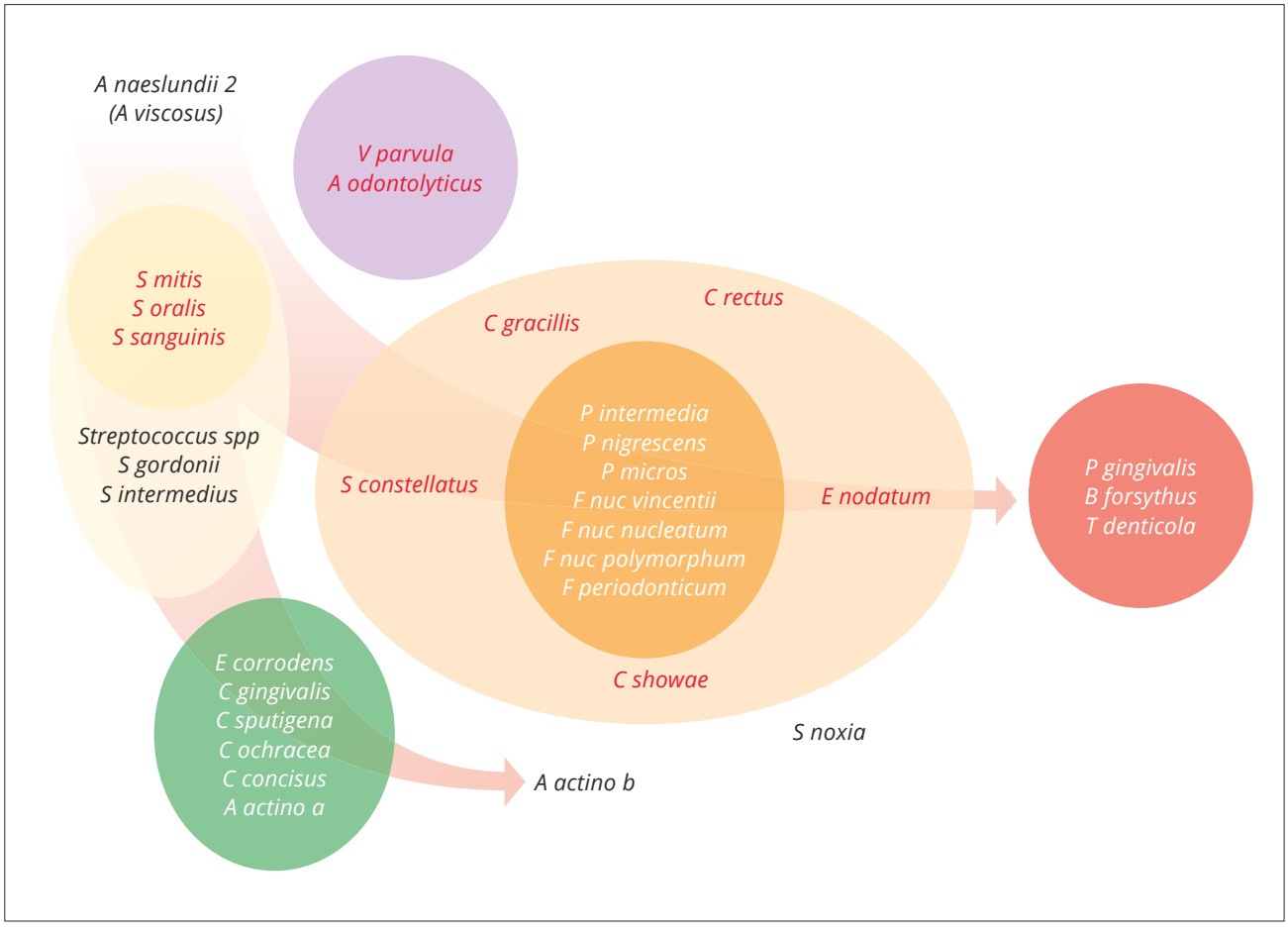


Fig 1-2 Socransky's bacterial complexes.

P gingivalis, mainly, would thus promote the transition from a symbiotic state to a dysbiotic state. However, this has only been demonstrated in a few animal studies.[14]

Porphyromonas gingivalis

P gingivalis is a gram-negative, strict anaerobic (aerotolerant), nonmotile, and asaccharolytic coccobacillus. Its diameter varies between 0.5 and 1 µm, and its length varies from 1 to 2 µm. Several serotypes have been described according to the antigenic properties of the capsule polysaccharides (K antigen) and of the LPS (O antigen). The main virulence factors of *P gingivalis* are fimbriae, hemagglutinins, LPS, and proteases (gingipains), which give it significant harmful potential. The expression of these virulence factors is dependent on oxygen conditions, pH, temperature, and heme availability. The relative ease of culture of *P gingivalis* and its good cellular, genetic, and biochemical characterization make it the most studied and best-known bacterium.

Polymicrobial infection

P gingivalis is able to aggregate with other bacteria of the oral microbiota, such as *Actinomyces viscosus*, *Streptococcus gordonii*, and *Streptococcus mitis*. This aggregation will potentiate the pathogenic effect of *P gingivalis*. Lamont et al[15] developed the idea of oral polymicrobial infection as a vector for the amplification of bacterial action. Indeed, the distinct microenvironments of the oral barriers harbor unique microbial communities regulated by sophisticated signaling systems and by host and environmental factors. The collective function of microbial communities is an important factor in homeostasis or dysbiosis and ultimately in health or disease. Despite different etiologies, periodontitis and caries are each induced by a direct linkage loop between the microbiota and host factors (inflammation and dietary sugars, respectively) that promotes the emergence and persistence of dysbiosis, highlighting emerging mechanisms governing oral polymicrobial synergy.

The etiopathogenesis of periodontal diseases therefore involves complex mechanisms involving bacteria (quantitatively and qualitatively), the host response (which can be variable over time), and the external environment.

Microorganisms

Clinical research clearly shows the prominent role of bacterial plaque dysbiosis in the etiopathology of periodontal disease.[20] Nevertheless, the numerous bacterial communities are not the only microorganisms detected in the mouth. Mycoplasmas, viruses, and parasites are found in the commensal and/or pathogenic flora.

Identified in 1849 by Gros, the parasites found in the oral cavity are mainly unicellular eukaryotic protozoa that feed by phagocytosis. By definition, a parasite is pathogenic because it is a microorganism that develops at the expense of its host.

Lyons et al were the first to attribute a pathogenic role to *Entamoeba gingivalis* and *Trichomonas tenax* in the incidence of periodontal disease.[21] Of the patients with periodontal disease, 62.5% had *E gingivalis* infection.

However, clinical research does not attribute a major role to them in the initiation, progression, or recurrence of periodontal disease. These parasites are widely found in healthy patients[22,23] whose plaque control is considered good. In addition, coinfections with herpes simplex virus (HSV), cytomegalovirus, and Epstein-Barr virus have also been described in the literature.[24] This can be explained by the fact that periodontal destruction is essentially inflammatory.

Parasite eradication as the primary treatment goal for periodontal disease does not appear to be necessary or sufficient for oral health.

The opportunistic nature of parasitic coinfections is most likely due to the increase in nutrients related to bacterial colonization and the inflammatory response.

Studies on amoebae, although interesting, have not been able to show a clearly demonstrated causal link with periodontal disease.

Since the elimination of these microorganisms also requires the disorganization of the microbiota, the recovery of periodontal health by their elimination is not proof of their pathogenicity.

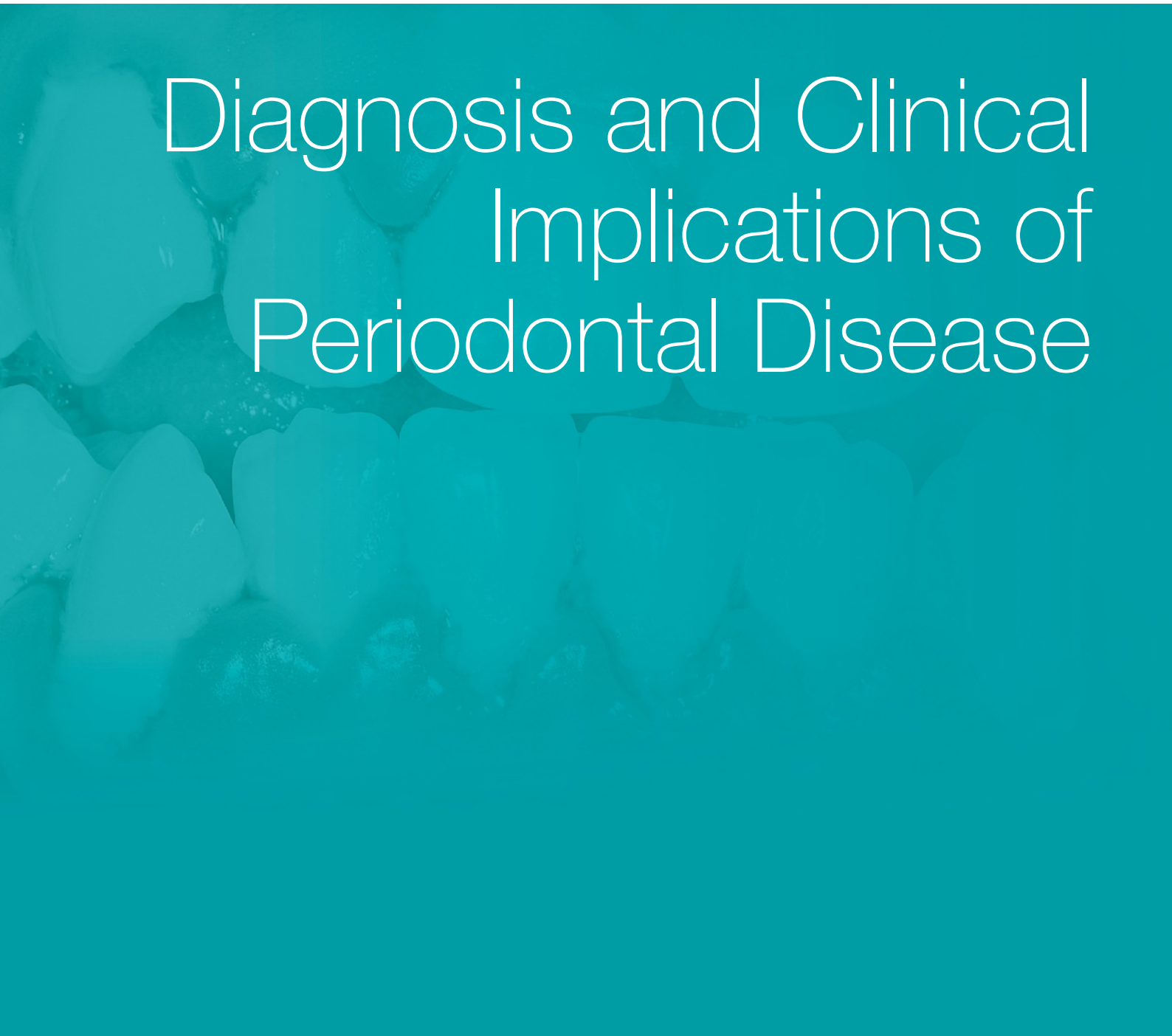
Conclusion

Let's go back to what is primary. Medicine today is essentially curative because of the fee-for-service system and also because of the lack of training in prevention that is really put in place during the first years of a dentist's studies. It is preferable to learn how to prevent the initiation of inflammatory problems before trying to treat proven pathologies. Indeed, to be able to treat these diseases, it is necessary to detect the first clinical signs early, and even to implement preventive measures beforehand. In order to avoid or limit the appearance of inflammation, whether in "at risk" populations or the entire population, it is necessary to detect it early and to implement measures that are applicable.

References

1. Bourgeois D, Bouchard P, Mattout C. Epidemiology of periodontal status in dentate adults in France, 2002-2003. J Periodontal Res 2007;42:219–227.
2. Carrouel F, Llodra JC, Viennot S, Santamaria J, Bravo M, Bourgeois D. Access to interdental brushing in periodontal healthy young adults: A cross-sectional study. PloS One 2016:11(5):e0155467.
3. Bouchard P. Periodontology and Implant Dentistry, vol 1 [in French]. Cachan, France: Lavoisier, 2014.
4. Zoellner H, Chapple CC, Hunter N. Microvasculature in gingivitis and chronic periodontitis: Disruption of vascular networks with protracted inflammation. Microsc Res Tech 2002;56(1):15–31.
5. Bosshardt DD, Lang NP. The junctional epithelium: From health to disease. J Dent Res 2005;84:9–20.
6. Darveau RP. Periodontitis: A polymicrobial disruption of host homeostasis. Nat Rev Microbiol 2010;8:481–490.
7. Chapple ILC, Matthews JB. The role of reactive oxygen and antioxidant species in periodontal tissue destruction. Periodontol 2000 2007;43:160–232.
8. Lee W, Aitken S, Sodek J, McCulloch CA. Evidence of a direct relationship between neutrophil collagenase activity and periodontal tissue destruction in vivo: Role of active enzyme in human periodontitis. J Periodontal Res 1995;30(1):23–33.
9. Gorr SU. Antimicrobial peptides in periodontal innate defense. Front Oral Biol 2012;15:84–98.
10. Page RC, Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. Lab Investig 1976;34:235–249.
11. Wade WG. The oral microbiome in health and disease. Pharmacol Res 2013;69:137–143.
12. Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL Jr. Microbial complexes in subgingival plaque. J Clin Periodontol 1998;25:134–144.
13. Darveau RP, Hajishengallis G, Curtis MA. *Porphyromonas gingivalis* as a potential community activist for disease. J Dent Res 2012;91:816–820.
14. Baker JP, Dixon M, Roopenian DC. Genetic control of susceptibility to *Porphyromonas gingivalis*-induced alveolar bone loss in mice. Infect Immun 2000;68:5864–5868.
15. Lamont RJ, Koo H, Hajishengallis G. The oral microbiota: Dynamic communities and host interactions. Nat Rev Microbiol 2018;16:745–759.
16. Ramseier CA, Anerud A, Dulac M, et al. Natural history of periodontitis: Disease progression and tooth loss over 4 years. J Clin Periodontol 2017;44:1182–1191.
17. Offenbacher S, Barros SP, Beck JD. Rethinking periodontal inflammation. J Periodontol 2008;79(8 Suppl):1577–1584.
18. Solbiati J, Frias-Lopez J. Metatranscriptome of the oral microbiome in health and disease. J Dent Res 2018;97: 492–500.
19. Palmer RJ Jr. Composition and development of oral bacterial communities. Periodontol 2000 2014;64:20–39.
20. Socransky SS, Haffajee AD. Periodontal microbial ecology. Periodontol 2000 2005;38:135–187.
21. Lyons T, Sholten T, Palmer JC. Oral amoebiasis: A new approach for the general practitioner in the diagnosis and treatment of periodontal disease. Oral Health 1980;70: 39–41, 108,110.
22. Wantland WW, Wantland EM. Incidence, ecology, and reproduction of oral protozoa. J Dent Res 1960;39:863.
23. Dao AH, Robinson DP, Wong SW. Frequency of *Entamoeba gingivalis* in human gingival scrapings. Am J Clin Pathol 1983;80:380–383.
24. Slots J, Slots H. Periodontal herpesvirus morbidity and treatment. Periodontol 2000 2019;79:210–220.

2



Diagnosis and Clinical Implications of Periodontal Disease

Classification of Periodontal Diseases

First of all, we can ask ourselves why classify periodontal diseases? We have to keep in mind that a classification is never fully accurate, but that it is the best way we have to talk about living beings. The purpose of a classification is to allow communication and interaction between several specialties. It is therefore normal that different classifications coexist depending on the context and the times.

Thus, in epidemiology the reference classification of periodontal disease is the CDC-AAP classification,[1] which must meet the requirements of sensitivity and specificity during an investigation. In clinical practice, the reference periodontal disease classification from 1999 until 2017 was the Armitage classification.[2] This classification has the advantage of being extremely clinical and of designating two patient entities: those that were called "chronic" and those that were called "aggressive," according to whether the pathophysiology of periodontal disease was mainly under the domination of external factors (bacterial load, risk factors, or comorbidities) or internal factors (host response).

A frequent criticism of this classification has been its subjectivity. Indeed, a lack of reproducibility was observed between the different clinicians on the classification between aggressive and chronic periodontitis when the age of the patient was more advanced, as well as on the severity of the disease, because of the weakness of the decision criteria.

To classify periodontitis as aggressive, three criteria were necessary:

- Healthy patient
- Known family history of periodontitis
- Rapid progression of pathology

It is the lack of reproducibility of such a classification that is problematic.

The new 2017 Chicago classification[3] has, in this sense, the merit of simplifying patient categorization, and although the impact on periodontal therapeutics is limited, it simplifies clinical research and comparative studies. The removal of the notion of aggressiveness seems to be an interesting advance and allows us to emphasize the notion that, whatever the "entity" of the disease, we are at the stage of tertiary prevention, ie, the therapeutic objective is not to cure but to stabilize the patient's chronic pathology in a state compatible with periodontal health. On the other hand, the disappearance of periodontitis "entities" in favor of stages and grades may be intellectually limiting but facilitates the categorization of the patient.

Chicago Classification of Periodontal Disease

The diagnosis of periodontitis is characterized according to its severity and complexity in four stages (Table 2-1) and according to its risk of progression in three grades (Table 2-2).

The main diagnostic criterion of periodontitis is the loss of interdental attachment. This is evaluated by means of a probe measuring the distance between the cementoenamel junction and the depth of the periodontal pocket. It is associated with radiographic bone loss to define the degree of severity of the disease. The number of teeth missing for periodontal reasons is also to be taken into account, if known.

The grade is based on the evaluation of the risk of disease progression, taking into account the host response. It includes secondary criteria related to general health (diabetes) or to harmful behavioral habits (eg, smoking),

Table 2-1 Summary of the stages of the Chicago classification[a]

		Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL[b]	1–2 mm	3–4 mm	≥ 5 mm	≥ 5 mm
	Radiographic bone loss[c]	Coronal third (< 15%)	Coronal third (15%–33%)	Middle or apical third (≥ 50%)	Middle or apical third (≥ 50%)
	Tooth loss due to periodontitis[d]	0	0	≤ 4	≥ 5
Complexity	Probing depth[e]	≤ 4 mm	≤ 5 mm	≥ 6 mm	≥ 6 mm
	Radiographic bone loss[c]	Mostly horizontal	Mostly horizontal	Vertical bone loss ≥ 3 mm	Vertical bone loss ≥ 3 mm
	Furcation involvement[f]	None or Class I	None or Class I	Class II or III	Class II or III
	Ridge defect[f]	None or mild	None or mild	Moderate	Severe
	Complex rehabilitation needs[f]	No	No	No	Yes

[a] Parameters defining the stage of periodontitis according to its severity and its complexity. (Modified from Papapanou et al.[3])

[b] Clinical attachment loss (CAL) is the pathognomonic sign of periodontitis. It is the main criterion to define its severity.

[c] Radiographic bone loss is an objective sign of periodontal destruction. It is assessed with periapical radiographs, which allow an accurate measurement, although radiographic bone loss often lags behind the reality of clinical demineralization. Vertical bone loss is a sign of severity.

[d] The number of teeth lost for periodontal reasons is often difficult to assess.

[e] If the loss of attachment evaluates the history of the progression of the periodontal destruction, the depth of probing evaluates its reality at the present time.

[f] Lesions reaching the interradicular areas of multirooted teeth are difficult to manage and thus complicate the treatment. The same is true for volume defects of the residual bone ridge after tooth loss or difficulties in prosthetic rehabilitation.

Table 2-2 Summary of the grades of the Chicago classification[a]

		Rate of progression	Grade A: Slow	Grade B: Moderate	Grade C: Rapid
Primary criteria	Direct evidence of progression	Loss of attachment or radiographic bone loss in the last 5 years[b]	None	< 2 mm	≥ 2 mm
	Indirect evidence of progression	Ratio of percent bone loss to age[c]	< 0.25	0.25–1	> 1
		Relationship of plaque quantity / periodontal destruction[d]	Heavy / mild	Normal	Mild / heavy
Modifying criteria	Risk factors[e]	Tobacco use (cigarettes/day)	None	< 10	≥ 10
		Diabetes diagnosis	No	Yes, HbA1c < 7.0%	Yes, HbA1c ≥ 7.0%

[a] Parameters defining the grade of periodontitis according to primary and modifying criteria. (Modified from Papapanou et al.[3])

[b] Evaluation of attachment loss or radiographic bone loss over the last 5 years is rarely possible but is a direct sign of disease progression.

[c] The ratio of percent bone loss to age is an indirect criterion of progression that is assessed on the most affected tooth.

[d] The ratio of plaque quantity to periodontal destruction is an indirect criterion for assessing host response and associated disease progression.

[e] Heavy smoking and uncontrolled diabetes are factors influencing the risk of progression.

which also play the role of a descriptor, allowing anticipation of the response to the planned treatment.

Uncontrolled diabetes will negatively influence the host's response, favoring the evolution of the disease. Tobacco consumption will have the same role, while directly attacking the periodontium (heat) and allowing the development of an ecosystem more favorable to certain bacteria, contributing to dysbiosis. Secondhand smoke probably also plays a role in these respects. Our job is to refer the patient to his or her physician to consider screening for diabetes (which is diagnosed very late in a portion of the population) or to monitor the patient's diabetes if it has already been diagnosed, but with randomized follow-up, recognizing that the control of diabetes necessarily varies over time. Our role is also to assist patients in a reduction or even cessation of smoking by explaining the implication of smoking in their disease and directing them toward organizations specializing in smoking cessation. Addressing these two elements will promote periodontal healing.

These classifications respond to a worldwide epidemiologic need to allow health professionals to agree on a common language but do not provide clinical guidance on a daily basis, as this is not their main purpose. The diagnostic elements proposed in this chapter are adapted to the therapies to be implemented and are mainly based on inflammation.

Clinical Diagnostic Elements: Early Intervention

We are certainly dealing with pathologies whose etiology is bacterial, but the destruction of the supporting tissues of the tooth is mainly carried out via the mechanisms of inflammation. These will be the first clinical signs to be observed.

Inflammation will result in a change in the shape, color, and texture of the gingiva. Its location can be papillary (interdental), at the buccal or palatal/lingual margin, or vestibular.

It is important at this stage to take into account the location of this inflammation, because at the papillary level (Fig 2-1), it is always related to ineffective interdental brushing, and therefore the use of the toothbrush alone will not be able to solve this problem. Only well-calibrated interdental brushes will have an impact. We will review this in more detail in the chapter on prophylaxis.

A marginal location (Fig 2-2) is associated with ineffective brushing at the sulcus. The roll brushing technique is often ineffective in cleaning the marginal area, although it may be useful for certain indications. The prescription of brushing equipment must then be made according to the technique taught

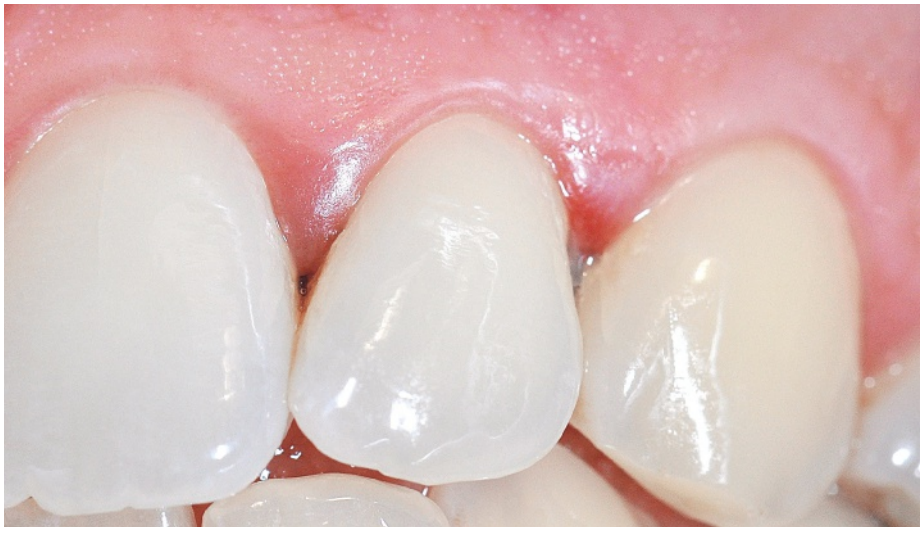


Fig 2-1 Papillary inflammation.

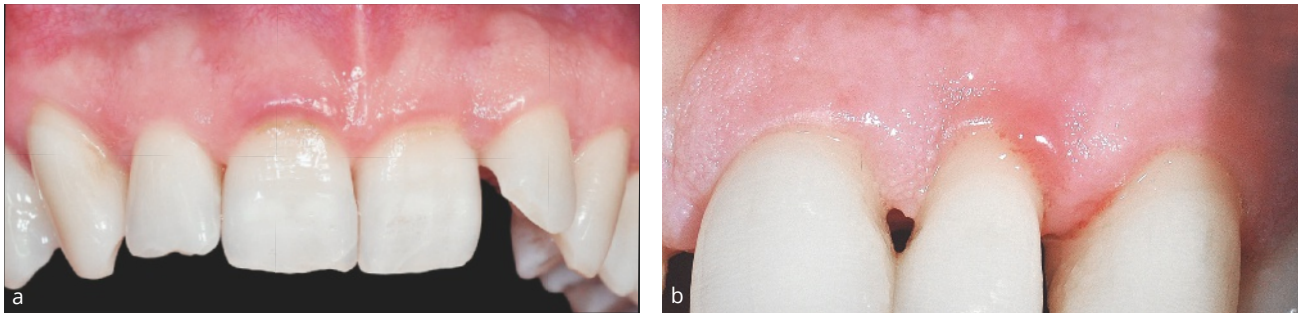


Fig 2-2 (a and b) Marginal inflammation.

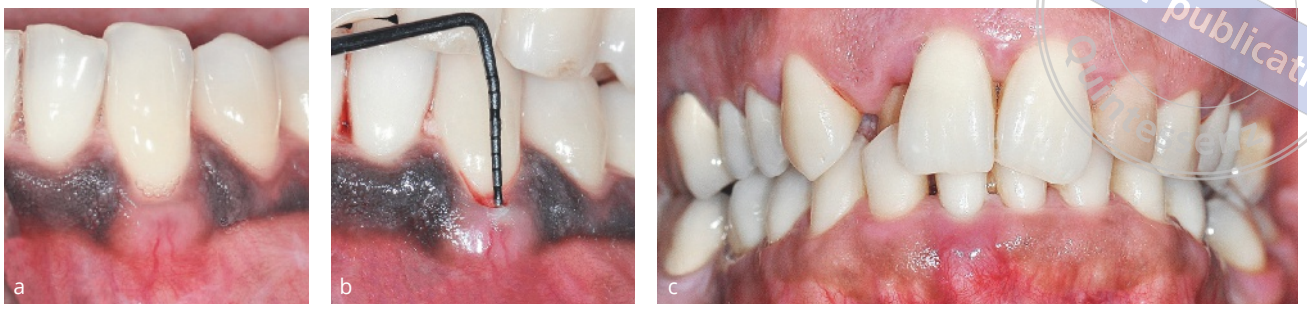


Fig 2-3 (a to c) Vestibular periodontal inflammation.

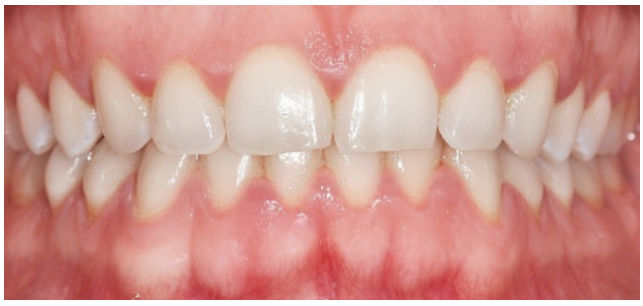


Fig 2-4 Superficial periodontal inflammation.

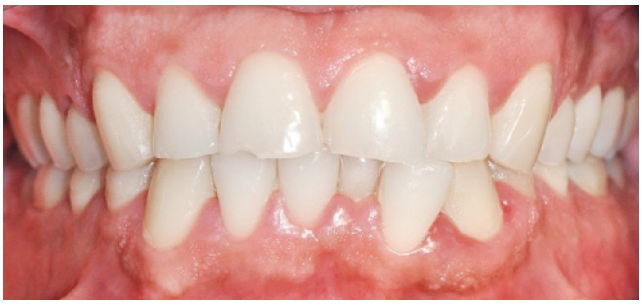


Fig 2-5 Moderate periodontal inflammation.

to optimize the control of plaque in this key area of the gingival margin. Finally, the vestibular location (Fig 2-3) will often be related to a deeper pocket or even a periodontal abscess, and brushing alone will not solve the problem.

Once the inflammation has been localized, the different degrees of severity must be identified, which are described as *superficial* (Fig 2-4) when there is a change in the color or texture of the gingiva, *moderate* (Fig 2-5) when there is objectively detectable edema with bleeding on stimulation, and finally *severe* (Fig 2-6) when the patient describes spontaneous bleeding even without stimulation by brushing.[4]

The severity of the inflammation will not always be related to the severity of the underlying destruction, so we will use other clinical criteria, such as loss of attachment associated with probing depth and, of course, radiographically quantifiable bone loss.

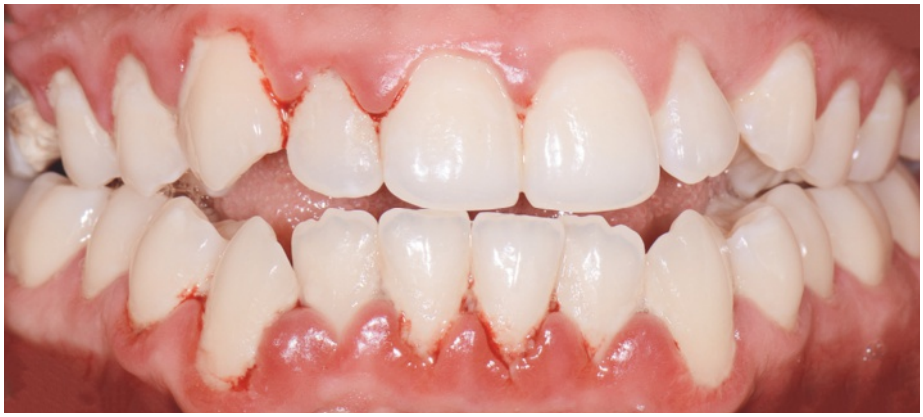


Fig 2-6 Severe periodontal inflammation.

Clinical Analysis

The best diagnostic tool available to us is our visual analysis, which will guide our clinical sense long before we take out a periodontal probe.

The eye as a diagnostic tool

The criteria for healthy gingiva have been well described (Fig 2-7; Löe, 1984):

- Pale pink gingiva
- Presence of orange peel stippling
- Firm to palpation
- Well-defined and even contour

However, visual access is almost impossible in interproximal areas, especially posteriorly, which can make it easy to miss well-established inflammatory pathology. The presence of orange peel stippling in a healthy area does not preclude a potentially inflammatory condition nearby (Fig 2-8). The inflammation present in the vestibular region may sometimes be very limited and discrete and yet much more severe in the lingual or palatal region (Fig 2-9).

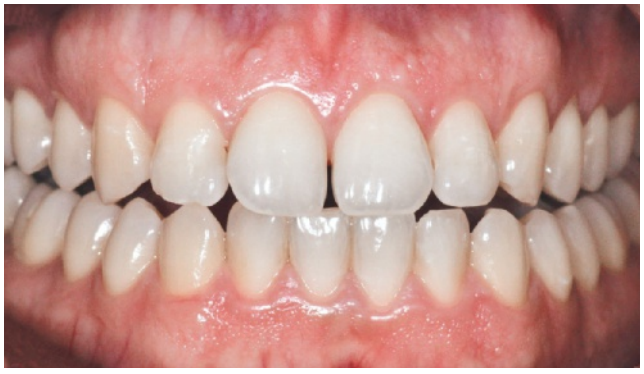


Fig 2-7 Healthy gingiva.

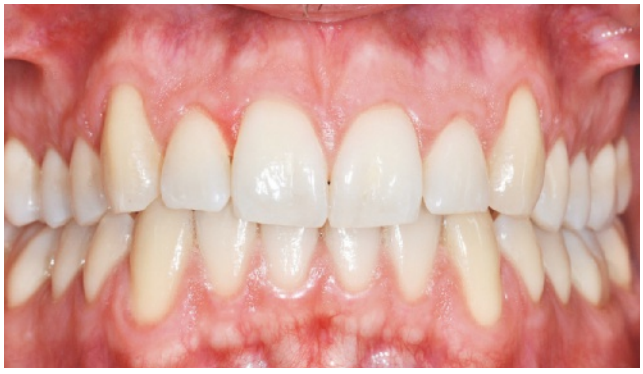


Fig 2-8 Localized inflammation.

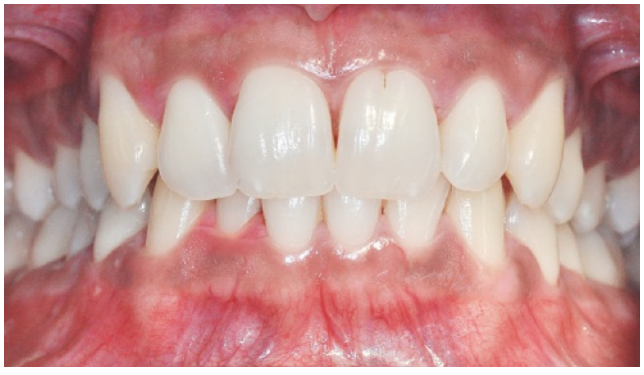


Fig 2-9a Vestibular view showing inflammation.

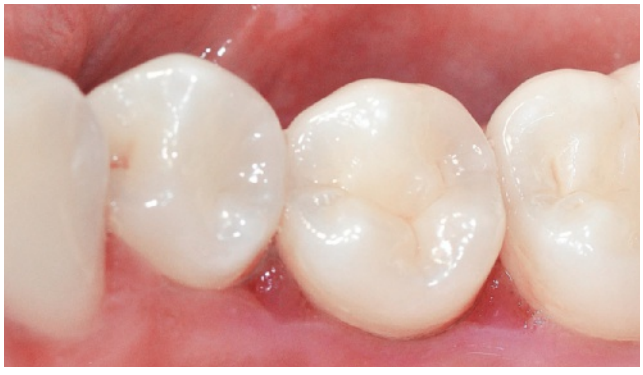


Fig 2-9b Lingual view of the same patient showing moderate inflammation.

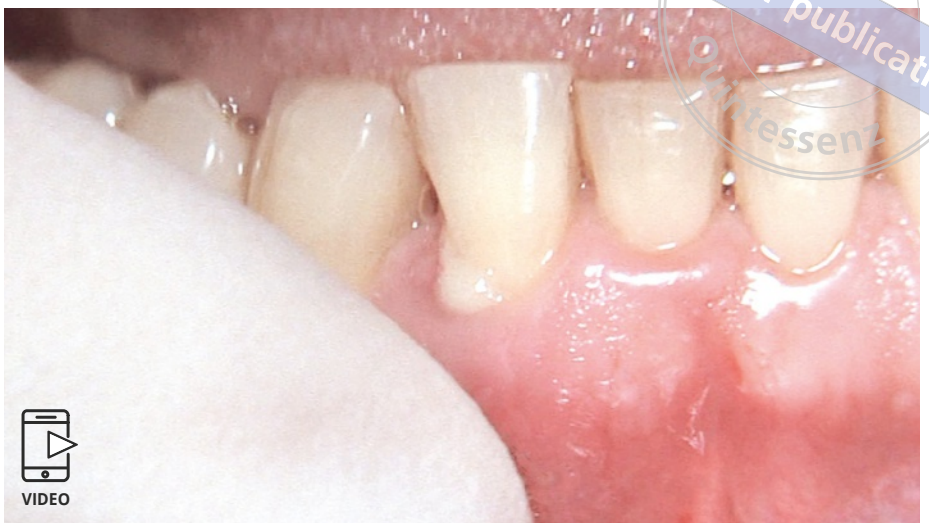
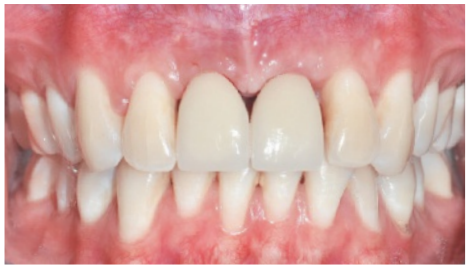


Fig 2-10 Suppuration following digital palpation of the vestibule.

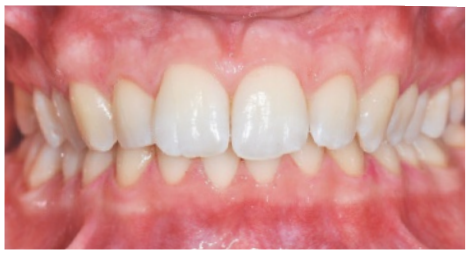
The gingiva must then be evaluated carefully, papilla by papilla, and massaged to reveal any suppuration (Fig 2-10).

Obviously, it is important to take advantage of this diagnostic session to look for the presence of dental pathologies as well as lesions of the oral mucosa in accordance with the principle of prevention.

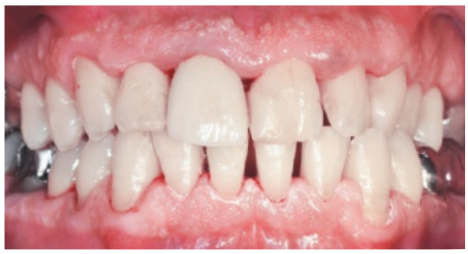
Fig 2-11 Periodontal biotypes.



a Thin periodontium.



b Intermediate periodontium.



c Thick periodontium.

At this stage, the first diagnostic tools are the eyes. We still have not used any instrumentation except a mirror to observe the areas not accessible by direct vision.

This rigorous visual inspection allows us to learn a great deal about our patients' brushing tools and techniques and to better guide them by explaining the causes and consequences of iatrogenic habits.

Assessment of the periodontal biotype can also be done by visual inspection[5] and, to simplify the diagnosis, the biotype can be categorized as follows (Fig 2-11):

- Thin
- Intermediate
- Thick

The thickness of the vestibular periodontal tissues can be assessed by palpation but is very difficult to quantify. This is why we limit our observation here to the quality of the gingiva. It is generally described that thick periodontal tissues will tend to mask the signs of inflammation more, and thin periodontal tissues tend to undergo more recession at the end of the treatment. This obviously depends on the bone level.

These parameters do not always allow us to say whether the patient has simple gingivitis or periodontitis, and it is the periodontal survey associated with the complementary radiographic examination that will allow us to make a diagnosis and especially to give a prognosis to the patient.

The periodontal probe: Clinical indications and limitations

Periodontal probing has always been given as the reference for clinical diagnosis to measure attachment loss and therefore to assess the periodontal health of patients. However, a complete assessment does not always appear necessary at first and especially involves a risk of bias.

In addition to causing pain, probing during the inflammatory period before treatment, may lack precision. Local plaque retention factors, such as the presence of subgingival calculus or fragile attachment during an inflammatory period, are all obstacles to accurate probing, which will then often be under- or overestimated.

Spot probing at certain sites is sufficient to make the diagnosis of periodontitis.

Differentiating between supra- and subgingival therapy is based on data from the scientific literature,[6] which shows that nonsurgical subgingival instrumentation of pockets smaller than 4 mm creates a loss of attachment, whereas the opposite is true if the pockets are greater than or equal to 4 mm. However, these conclusions must be correlated with the recommendations of the time. Indeed, the recommended treatment was to perform manual surfacing with sharpened curettes, creating a real trauma for the conjunctival attachment when handling them, but there are no studies evaluating the risk of loss of attachment in shallow pockets when using ultrasonic instruments that respect the periodontal tissues. However, it is important that the instruments are properly adjusted and handled (see chapter 7).

The clinical diagnosis to be correlated with the inflammation and therefore the bleeding[7] is as follows:

- Pockets less than or equal to 3 mm mean the patient is healthy.
- Pockets between 4 and 5 mm indicate moderate pathology.
- Pockets greater than or equal to 6 mm are associated with severe pathology.

However, we observe on a daily basis, in patients considered healthy and who have never presented periodontal pathology, that probing rarely goes beyond 1 mm, which is equivalent to the sulcus (0.8 mm on average).

How then can we explain that in the literature these 3 mm of probing depth are considered normal?

All of this work has been based on microbiologic analysis. For a pocket up to 3 mm, studies[8] reveal that oxygen is able to penetrate it, making the bacterial ecosystem compatible with periodontal health since the majority of bacteria are gram-positive aerobic bacteria (eg, *Streptococcus sanguinis, Streptococcus oralis, Streptococcus mitis*). For a pocket of 3 to 5 mm, we observe a mixed flora with an ecology combining gram-positive and gram-negative bacteria. A pocket larger than 6 mm is a real bacterial reservoir whose ecology may be favorable to the development of dysbiotic microbiota.[9] The risk of progression of the loss of attachment and/or recurrence of a treated pocket is then much greater.

As mentioned earlier, a complete diagnosis is not necessary, but spot probing of certain pockets is important to assess the loss of attachment. We will initially focus our diagnosis on the evaluation of the host inflammatory response.

The implications of this diagnosis in our daily practice allow us a great simplification of patient management because, whatever the degree of severity and damage associated with periodontal diseases, all will present inflammation whose etiology will be largely the same: the biofilm (ie, the main etiologic factor of periodontal diseases).

In this book, we will not detail the specific management of dermatologic pathologies (eg, fungal, viral) that can also induce inflammation (not induced by plaque).

We will consider two main types of periodontal disease:

- Gingivitis, which is damage to the superficial periodontium and is reversible. We will distinguish two major types of gingivitis: early and established.
- Periodontitis, which is a chronic pathology consisting of irreversible attacks on the periodontium that alter the supporting tissues of teeth and can lead to the loss of teeth in the absence of effective management.

> To say that a pathology is chronic is full of meaning from a medical point of view because it implies that one will not cure periodontitis, unlike gingivitis.

Diagnostic Tools

There are various tools today that are indicated as possible aids to diagnosis. However, there is no scientific evidence to date that shows the superiority of one test over another; the diagnosis will always be clinical and radiographic.

Phase contrast microscopes

These microscopes, much appreciated for their low cost and their educational aspect, allow the clinician to take a sample of biofilm from the patient's mouth and then show it to the patient to make him or her aware of the presence of the biofilm.

Since periodontal disease is insidious, with symptoms that are often considered benign by patients, direct visualization of the bacteria with an explanation from the clinician is a motivational technique.

However, from a diagnostic point of view, there is no association of these bacteria and/or parasites visualized under the microscope with a periodontal diagnosis. The use of a microscope will in no way modify the clinical management that we propose to the patient. The microscope remains a communication tool that, if mastered, can be effective but that cannot be used as a diagnostic aid or to monitor periodontal health (Fig 2-12).

Fig 2-12 Phase contrast optical microscope.

Bacterial samples

There are two main types of probes, namely culture and DNA probes. In both cases, we can identify only those bacteria that we know and/or know how to cultivate. Sequencing of the bacterial genome has made it possible to identify many subspecies of *Porphyromonas gingivalis,* showing that healthy patients can harbor *P gingivalis* in their mouths, but they do not all have the same subspecies, nor the same susceptibility to develop pathologies—hence the term *dysbiosis*.[10]

Again, these tests can be a tool for communication with our patients but will rarely guide our therapy. The decision to use antibiotics will be based primarily on a clinical diagnosis, and more rarely on a microbiologic diagnosis, since their prescription is probabilistic (the most commonly prescribed antibiotics cover a broad spectrum). Antibiotic therapy will not be modified based on this test but will be indicated by the recommendations, as we will see later.

Clinical findings indicate signs of periodontal disease, which in turn indicates a radiographic examination.

Radiography as a Complementary Tool for Diagnosis and Prognosis

Panoramic radiography is a good screening tool but is not sufficient to make an accurate diagnosis.

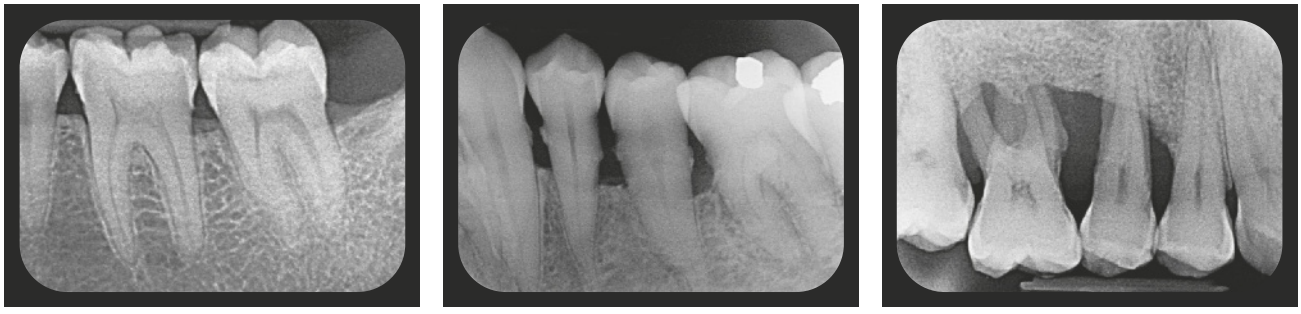
Periapical radiography remains the reference tool for quantifying bone loss. Several classifications have been proposed, and the one we have chosen for its simplicity and prognostic aid is that of Hugoson and Jordan.[11]

It does not consider the crown and measures bone loss at three equal portions of the roots:

- Less than one-third interproximal bone loss is considered superficial pathology.
- Bone loss between one- and two-thirds of the root is considered moderate pathology.
- Bone loss beyond two-thirds of the root is termed severe pathology (Fig 2-13).

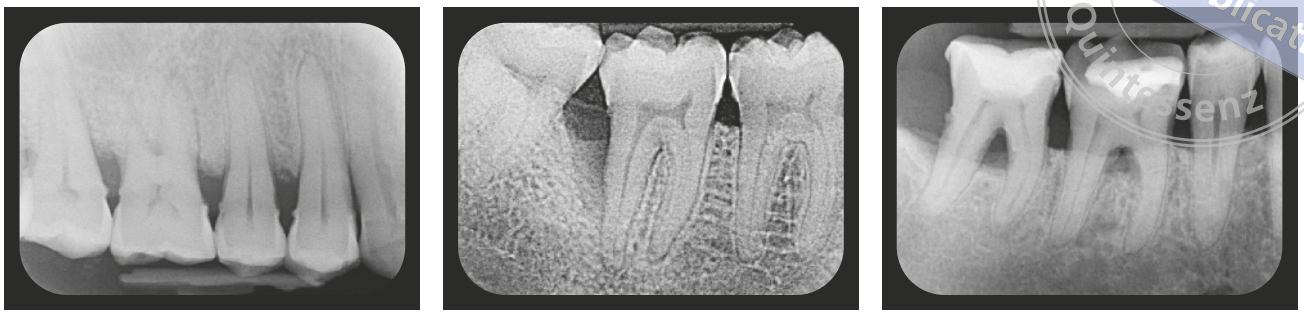
Thus, patients with superficial or moderate pathology (85% of patients with periodontitis) will have a low risk of losing their teeth if treated. For patients with severe pathology, the treatment can be more complex and require a multidisciplinary approach that can call upon one or more specialties (eg, periodontics, orthodontics, restorative dentistry).

Fig 2-13 Radiographic images of bone loss.



a Superficial. **b** Moderate. **c** Severe.

Fig 2-14 Radiographic images of bone loss.



a Horizontal. **b** Vertical. **c** Interradicular.

Bone loss is defined by several components (Fig 2-14):

- Horizontal: corresponds to the interproximal bone loss visible on a periapical radiograph
- Vertical: corresponds to angular defects
- Interradicular: corresponds to furcation damage; is not always visible radiologically and therefore will be assessed mainly clinically (Nabers probe)

Horizontal bone loss cannot be compensated for, which means that etiologic treatment will most often result in periodontal recession at sites affected by this type of bone loss. It is essential, at an early stage, to warn the patient of the possible consequences in terms of esthetics and comfort, which will be all the more significant if the bone loss is severe. However, purely horizontal bone loss with no other components is generally relatively simple to manage with etiologic therapy and without routinely resorting to a surgical approach.

Vertical bone loss can sometimes be repaired following etiologic therapy but will often be more complex to manage and may require surgical intervention.

Interradicular bone loss is the most complex in its management. The classification of the different types of furcation damage is clinical and will be of great prognostic help. Superimposition of images on radiographs limits precise interpretation, and it is impossible to differentiate Class II from Class III furcation involvement radiographically.

Bone loss can be considered localized if fewer than 30% of sites are affected and generalized if more than 30% are affected.

Bone loss can therefore be classified as simple (one component) or complex (several components) and as localized or generalized, and a degree of severity can be attributed to it.

The radiographic analysis will also look at the dental anatomy. Indeed, the longer the root of a tooth, the less the loss of attachment will have

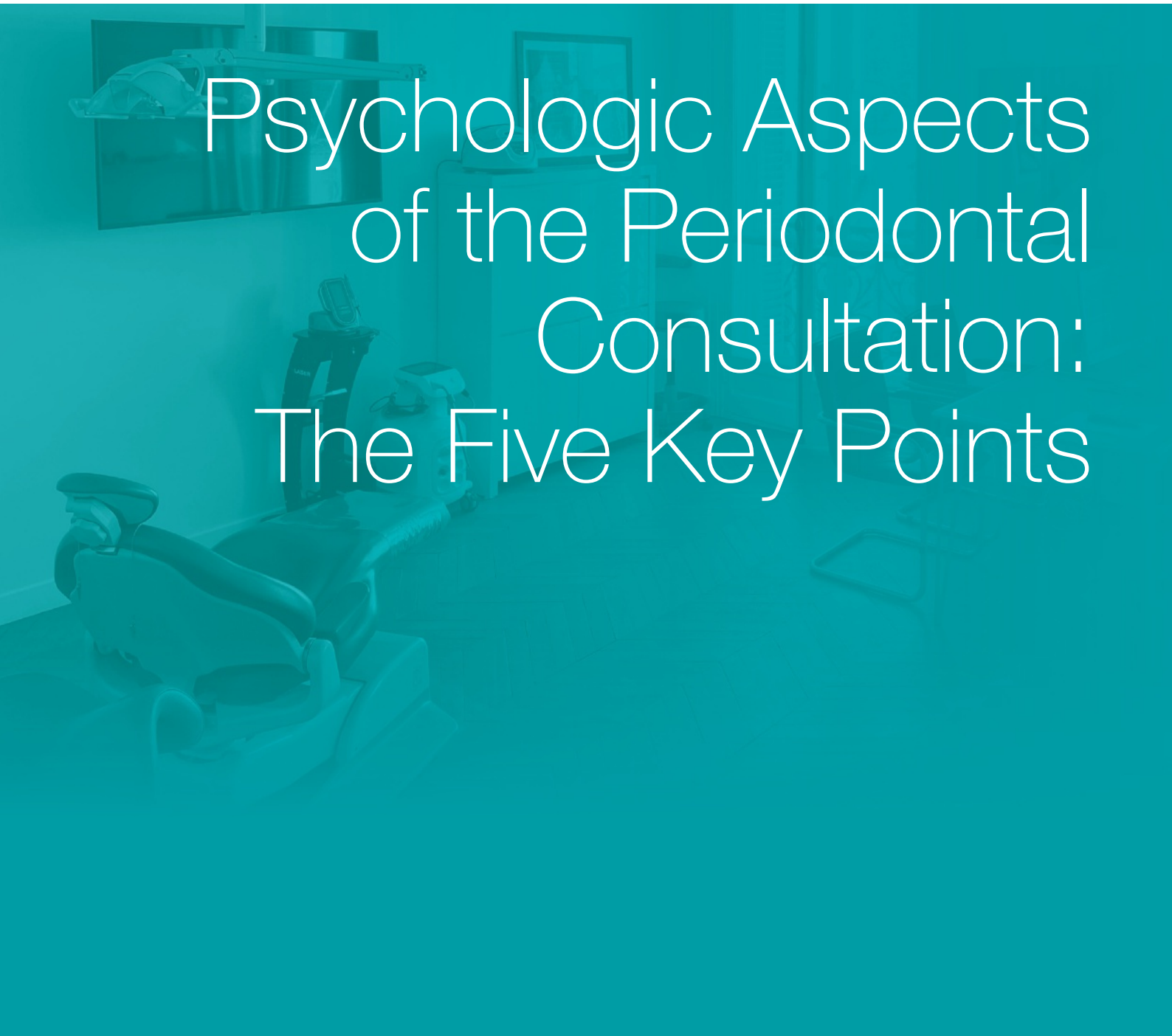
consequences in terms of survival rate and masticatory comfort. Similarly, a furcation with a more apical entrance will be affected later. However, once the lesion is established, it will be more difficult to treat.

But the Diagnosis, with a capital D, must relate a pathology to the patient's level of knowledge to lead to a true diagnosis of periodontal diseases, ie, a clinical and radiographic diagnosis plus an educational diagnosis.

The educational diagnosis corresponds to the patient's level of knowledge, their pathology, who they are, what they do, and how they conduct themselves.

References

1. Eke PI, Page RC, Wei L, Thornton-Evans G, Genco RJ. Update of the case definitions for population-based surveillance of periodontitis. J Periodontol 2012;83:1449–1454.
2. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4(1):1–6.
3. Papapanou PN, Sanz M, Buduneli N, et al. Periodontitis: Consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions. J Clin Periodontol 2018;45(Suppl 20):S162–S170.
4. Löe H. The gingival index, the plaque index and the retention index systems. J Periodontol 1962;38(6 Suppl):610–616.
5. Eghbali A, De Rouck T, De Bruyn H, Cosyn J. The gingival biotype assessed by experienced and inexperienced clinicians. J Clin Periodontol 2009;36:958–963.
6. Cobb CM. Clinical significance of non-surgical periodontal therapy: An evidence-based perspective of scaling and root planing. J Clin Periodontol 2002;29(Suppl 2):6–16.
7. Khan S, Cabanilla LL. Periodontal probing depth measurement: A review. Compend Contin Educ Dent 2009;30(1):12–21.
8. Meuric V, Gall-David SL, Boyer E, et al. Signature of microbial dysbiosis in periodontitis. Appl Environ Microbiol 2017;83(14):e00462–17.
9. Shi M, Wei Y, Hu W, Nie Y, Wu X, Lu R. The subgingival microbiome of periodontal pockets with different probing depths in chronic and aggressive periodontitis: A pilot study. Front Cell Infect Microbiol 2018;8:124.
10. Ambrosio N, Marín MJ, Laguna E, Herrera D, Sanz M, Figuero E. Detection and quantification of *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* in bacteremia induced by interdental brushing in periodontally healthy and periodontitis patients. Arch Oral Biol 2019;98:213–219.
11. Hugoson A, Jordan T. Frequency distribution of individuals aged 20-70 years according to severity of periodontal disease. Community Dent Oral Epidemiol 1982;10:187–192.



Psychologic Aspects of the Periodontal Consultation: The Five Key Points

The first essential points of a periodontal consultation are the general history and the recording of the reasons for the consultation in order to initiate patient management (see chapter 6).

It is therefore important to note all the pathologies and treatments of the patient, some of which have an impact on the periodontium, as well as certain harmful habits such as smoking. This history will be followed by a detailed clinical and radiographic examination (see chapter 2).

As periodontal diseases are chronic pathologies in the same way as diabetes and most cardiovascular pathologies, their treatment can only be envisaged with the help of a multifactorial approach based on the alliance that the practitioner builds with the patient.

This alliance consists of five essential points:

- Prescribe appropriate home hygiene tools for the patient
- Provide a simple and objective explanation of the patient's specific pathology
- Obtain patient confirmation of the treatment goals
- Work on ambivalences
- Avoid making the patient feel guilty

Prescribing Appropriate Home Hygiene Tools

Why is it important to prescribe the right home hygiene tools for each individual?

Let's imagine that a general practitioner diagnoses a patient with high blood pressure. It would never occur to the patient to go to the pharmacy to get an "antihypertensive." The doctor would never write on a prescription simply "antihypertensive." There would be many legitimate questions such as, "Which one? Are there no differences between this antihypertensive and that one?" And even if the practitioner were to specify the drug or even that one tablet should be taken per day, the prescription would still be incomplete. How do you know the dosage? Is it a 5- or a 10-mg tablet? And when should it be taken, in the morning or in the evening? Giving such a prescription would, of course, be a potential health risk for the patient and also pose a risk of inefficacy.

The doctor first prescribes a treatment in relation to the pathology, the general state of health, and sometimes even the abilities of the patient and then readjusts the treatment according to future clinical signs.

Let's go back to our periodontal patients: How many times have we told them to use toothbrushes that they can find in drugstores, pharmacies, and even supermarkets?

But should patients really be left alone with the choice of toothbrushs? Which type of brush? What kind of bristles? Which brand? What size?

Letting patients choose their own toothbrushes is equivalent to letting them self-medicate, which will certainly be ineffective and could even be harmful.

Providing a Simple and Objective Explanation

Why provide a simple and objective explanation to patients about their pathology? The patient comes to a consultation for their own problem; they do not care to know about the pathology of others. A personalized explanation, according to the patient's medical history, is therefore necessary. Studies have shown that keeping patients in the dark is detrimental to their involvement in their treatment.

Not all patients need to know all the ins and outs of how periodontal disease begins or the complex biologic processes of inflammation. They do, however, need to understand their essential role in the management of their periodontal disease, and in particular, their critical role in restoring a healthy balance. The explanation that we give patients must make them aware of the objectives of the treatment, of which there are three:

- Make the patient aware of the chronic (irreversible) nature of his or her pathology.
- Enable the patient to achieve effective plaque control on a daily basis.
- Make the patient a driving force and active participant in his or her treatment.

The story told to patients must allow them to become aware of these objectives. In the framework that we propose, we have intentionally simplified our language regarding the biologic mechanisms involved in periodontal diseases while maintaining the explanations that we consider necessary. Below is an example of how periodontal disease can be effectively explained to a patient.

"What you have is what is called periodontal disease*, which is a bacterial infection that has overwhelmed your gums' defenses. Normally, the gums are a 'waterproof' barrier, almost impermeable; bacteria accumulate but cannot pass underneath. More than half of the bacteria are located between the teeth, which means that brushing your teeth with only a toothbrush will only be, at best, half effective. The accumulation and stagnation of all these bacteria over the years irritated your gums, and one day they were no longer able to defend themselves. The bacteria overwhelmed the gums' defenses and went underneath. When they go underneath, they do two things: the first is to activate mechanisms of destruction of the bone around your teeth, and this is why we can see on your x-rays that you are losing bone; the second is to modify your gums, which have lost their seal*

and which we are not able to restore today. The treatments that we are going to put in place are designed to eliminate these bacteria that are stagnating above and below your gum line. But if we eliminate them and you are not able to prevent them from coming back and stagnating again, they will go back under your gums, which no longer have this defensive capacity, and all the work done will have to be done again and again. Moreover, since half of the bacteria are between your teeth, only interdental brushes will allow for effective cleaning, provided that they are adapted. Since the bone does not grow back, the gums will retract, and the spaces between the teeth will increase, which is normal. We will not be able to grow back the bone. However, if we don't lose more bone, we will keep your teeth and gums healthy.

We will do everything we can with you to preserve your teeth and your smile."

Ending on a positive note is extremely important. These explanations can be interspersed with more details, depending on the patient's reactions.

Confirmation of Treatment Goals by the Patient

Why is it extremely important at the end of the consultation to have the patient repeat the key points that have just been made?

Simply, to make sure that patients understand their health problems and, above all, to verify that they have understood what we expect of them moving forward. We sometimes find that the patient will focus on a completely different aspect than the one we wanted to emphasize, which is why the message must be as simple as possible, even if it means being somewhat inaccurate, if this allows the patient to retain the objectives of the treatment.

We are well aware that the gingiva is not an impermeable barrier, as it is a mucous membrane, but this simplification ensures the patient's understanding. Explanations that are too complex are often a source of misunderstanding in the therapeutic alliance and alter the relationship of trust between practitioner and patient.

Working on Ambivalences

The patient attaches great importance to being listened to. As proof of this, we all regularly hear, during the first contact with new patients, their complaints about the lack of listening by the colleague previously consulted.

Identifying the patient's grievances ensures that we can respond to them and, by gaining the patient's trust, point out important elements of which he or she was not aware.

To feel listened to, the patient must feel that he or she has been understood by the speaker. Repeating some of the patient's sentences to validate key points can be helpful, such as: "If I understand what you are telling me, you sometimes have bleeding when brushing, but it doesn't bother you so much. On the other hand, it is mainly cold sensitivity and loose teeth that bother you."

It is fairly obvious to us that the two are going to be related. Bleeding signals inflammation, probably related to the patient's periodontal disease, and existing recessions expose the dentinal tubules, causing these sensitivities.

Not wanting to hear the patients' complaints about these sensitivities or other aspects that they might bring up can cause them to reject the objectives toward which we would like to orient them. First, we must validate their words and perspective and explain, among other things, the solutions that we will implement to make them comfortable again.

Avoiding Making the Patient Feel Guilty

It is important to avoid saying, "It is because you brush your teeth badly that you have this problem." It is necessary to stay away from judgmental language. We need to get away from the notion of right and wrong to remain objective with patients. We all have patients who do not have effective hygiene and yet do not show signs of periodontal or caries disease, as everyone's immunity protects them differently. So, rather than being judgmental, try to offer help so patients can become more effective in brushing their teeth daily.

Reference

1. Deinzer R, Micheelis W, Granrath N, Hoffmann T. More to learn about: Periodontitis-related knowledge and its relationship with periodontal health behaviour. J Clin Periodontol 2009;36:756–764.