2008
Volume 83
Number 6
Journal of
Dental Hygiene

Supplement
to ADHA Access

Journal of Dental Hygiene

THE AMERICAN DENTAL HYGIENISTS' ASSOCIATION

Rationale for Comprehensive Nonsurgical Periodontal Therapy: A Review of the Clinical Evidence and Practice Protocol

- Microbes, Inflammation, Scaling and Root Planing, and the Periodontal Condition
- Locally Delivered Antimicrobials: Clinical Evidence and Relevance
- Periodontal Treatment Protocol (PTP) for the General Dental Practice

This supplement is sponsored by an educational grant from OraPharma, Inc.

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From the Editor-in-Chief of the *Journal of Dental Hygiene*

Over the last 30 years, we have learned much about the etiology, progression, and treatment of periodontal diseases. For example, we know that the accumulation of dental biofilm can trigger resultant inflammatory and immune responses. Dental biofilm contains a vast diversity of microbial species, some of which have been identified as etiologic agents for systemic diseases.

Risk factors for periodontitis can be grouped into categories such as microbial, systemic, behavioral, and local. Controlling risk factors is important to the management of periodontal diseases and is something that should be an overall goal for every dental hygienist. One risk factor for disease that can be controlled in the majority of cases is dental biofilm. However, control of dental biofilm is dependent on many factors including the knowledge of the dental hygienist regarding evidence-based strategies for disease prevention and treatment.

We have an extensive amount of scientific evidence available to educate every oral health care professional about periodontal diseases. However, dental practice management experts report that many clinicians are not adequately diagnosing, documenting, or monitoring disease status or making treatment recommendations to patients based on evidence-based strategies. Many questions arise about the best treatment techniques, products, and recommendations for patients who have chronic periodontitis or are at risk for the disease. The patient is dependent on the dental hygienist to be at the forefront of prevention. It is vital for dental hygienists to have up-to-date, accurate information so they can educate and make appropriate recommendations for the individual patient.

This supplement of the *Journal of Dental Hygiene* includes articles that will educate every dental hygienist about the evidence base for treatment of chronic periodontitis. Dr. Charles Cobb is an international expert on dental biofilm and the effect of nonsurgical methods for removing biofilm and hard deposits (calculus) on the tooth and root surfaces. He provides a comprehensive, evidence-based review of what dental hygienists can expect from nonsurgical therapies. Drs. David Paquette and Maria Ryan, 2 world-renowned periodontists, and I present a comprehensive paper on the evidence base for the use of locally delivered antimicrobials. Since their inception 3 decades ago, oral health care professionals have been utilizing locally delivered antimicrobials/antibiotics to treat chronic periodontitis. Still, questions arise about their utility and ability to treat and control this disease. This paper presents the clinical evidence for use of locally delivered antimicrobials in patient care. Finally, Dr. Larry Sweeting, Ms. Karen Davis, and Dr. Charles Cobb put the evidence into an action plan for dental hygienists. Dr. Sweeting and Ms. Davis are dental clinicians as well as professional speakers and consultants. Their paper discusses the effectiveness of using a Periodontal Treatment Protocol to assist in the early diagnosis and treatment of periodontal diseases. It also discusses insurance coding, vital verbal skills to use with patients, and considerations for implementation of locally delivered antimicrobials into a general clinical practice.

I want to extend sincere appreciation to OraPharma, Inc. for their support of this supplement. OraPharma, Inc. has been diligent in their goal of conducting evidence-based scientific investigations in order to help all oral health care professionals better diagnose and treat periodontal diseases.

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This special issue of the *Journal of Dental Hygiene* was sponsored by an educational grant from OraPharma, Inc.

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Journal of Dental Hygiene

special supplement

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The *Journal of Dental Hygiene* is published quarterly, online-only, by the American Dental Hygienists' Association, 444 N. Michigan Avenue, Chicago, IL 60611.

Subscription rates for nonmembers are one year, $45; two years, $65; three years, $90; prepaid.

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Supplement

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Microbes, Inflammation, Scaling and Root Planing, and the Periodontal Condition

Charles M. Cobb, DDS, MS, PhD

Abstract

Biofilms are a complex community of microorganisms characterized by the excretion of an adhesive and protective extracellular matrix, microbe-to-microbe attachment, structural heterogeneity, genetic diversity, and complex community interactions. Bacteria growing in dental biofilms display an increased tolerance to antibiotics and antimicrobial agents, including those used in dentifrices and mouthrinses.

The microbial challenge associated with the inflammatory periodontal diseases induces an immediate inflammatory and immune response in the host. The nature and magnitude of the response has an impact on the severity and rate of progression of the periodontal disease. It is this host inflammatory-immune response that ultimately leads to the clinical signs and symptoms of gingivitis and chronic periodontitis. The traditional treatment modality of scaling and root planing (SRP) remains the "gold standard" for the non-surgical management of chronic periodontitis. Even clinically successful treatment has a high probability of pocket re-infection. Re-infection of periodontal pockets results from residual biofilms, increased tolerance of microbes within a dense, mature biofilm to antibiotics, reservoirs of bacteria in calculus, and reservoirs of bacteria within the dentinal tubules of infected root surfaces. Thus, for maximum effect, a combination of scaling and root planing and locally delivered antimicrobials should be considered if non-surgical therapy is the treatment of choice.

Keywords: periodontal disease, periodontal infection, chronic periodontitis, scaling and root planing, dental biofilm

Introduction

Typically, the term "periodontal disease" refers to gingivitis and periodontitis, both common inflammatory diseases that involve a variety of pathogenic bacterial species and an innate host response to those bacteria.[1] Gingivitis, the most familiar form of inflammatory periodontal disease, has a high prevalence rate, affecting 50%-90% of adults worldwide.[2,3] By definition, gingivitis is limited to an inflammation that involves only the gingival soft tissues, ie, gingival epithelium and subjacent fibrous connective tissues. In spite of its high prevalence rate and worldwide distribution, biofilm (plaque)- induced gingivitis is preventable and rather easily reversed by routine oral hygiene measures.

Inflammation that extends into the deeper tissues to involve bone, resulting in resorption of tooth supporting bone, is termed periodontitis. Concomitant with the loss of bone is the formation of a deepened space between the root of the tooth and the gingiva, a periodontal pocket. Periodontitis can present as a chronic and slowly progressing disease (most common form) or as an aggressive disease causing loss of bone over a relatively short period of time. Periodontitis of advanced severity can result in tooth mobility, occasional pain and discomfort (generally associated with abscess formation), impaired ability to masticate food, and eventual tooth loss.

Although more common to adults, epidemiologic data indicate that periodontitis can also be found in children and adolescents.[4,5] In the United States, chronic periodontitis is more prevalent in men than women, and in African Americans, Native Americans, and Mexican Americans than Caucasians.[2,6,7] Various epidemiology studies, when considered in aggregate, suggest a progressive decrease in the prevalence of periodontitis between the years 1988-2004.[7-11] The more recent of these studies indicate a prevalence rate for moderate to advanced periodontitis ranging from approximately 5% to 15% for individuals > 18 years of age.[9-11] Given the current US Department of Census projections, a 5% to 15% prevalence rate translates to 11 to 33 million US adults that may exhibit periodontitis of moderate to advanced severity.[12] If one includes slight severity, the prevalence rate for periodontitis increases to approximately 30% of the US adult population, or roughly 65 million individuals.[9-12] However, all epidemiology studies that have reported on the prevalence of chronic periodontitis have utilized partial-mouth examinations, which tend to underestimate prevalence, extent, and severity of disease.[13-15]

Microbes and Biofilm

A biofilm is a complex community of microorganisms characterized by the excretion of an adhesive and protective extracellular matrix, microbe-to-microbe attachment, structural heterogeneity,

genetic diversity, and complex community interactions. Dental plaque is a microbial biofilm (Figure 1). As with any biofilm, the constituent microbes are tightly adherent to each other and to an oral substrate by means of an extracellular matrix, ie, slime layer or glycocalix, into which they are embedded.[16,17] The microbial populations in biofilm have 2 strategies that enable them to successfully survive within their community. The first is a high rate of reproduction for continued survival, and the second is physiologic adaptation to the available environmental resources or life-supporting capacity of the environment.[18]

Biofilms inherently dictate profound changes in the behavior of individual microbes, their relationship to the host, and their response to environmental conditions.[19] Indeed, oral biofilms, as distinct entities, are the causative agents of biological processes such as dental caries, periodontal disease, and periimplantitis, rather than any single microbe evading the host defense and causing disease.[20] Biofilms exhibit characteristics that impact the clinical management of inflammatory periodontal disease. For example, both altered patterns of microbial gene expression and the composition and density of the extracellular matrix reduce the susceptibility of microbes to antimicrobial agents.[21-23] Bacteria growing in dental biofilms display an increased tolerance to antimicrobial agents, including those used in dentifrices and mouthrinses.[24-27] In addition, confocal microscopy of *in situ* established natural biofilms showed that chlorhexidine only affected the outer layers of cells in 24 and 48 hour plaque biofilms, suggesting either quenching of the agent at the biofilm surface or a lack of penetration.[28] Further, biofilms of oral bacteria are also more tolerant of antibiotics (eg, amoxycillin, doxycycline, minocycline, and metronidazole) than planktonic cells.[29-31] In this regard, biofilms of *Porphyromonas gingivalis* have been shown to tolerate 160 times the minimum inhibitory concentration (MIC) of metronidazole that was determined for planktonic cells.[32]

Over 700 species of aerobic and anaerobic bacteria have been identified in the human oral cavity.[33,34] The microbes grow as complex, mixed, interdependent colonies in biofilms, and may achieve considerable thickness, achieving a thickness of 1 mm within 96 hours, if left undisturbed.[16,17] Oral biofilms, like all microbial biofilms, exhibit a successional colonization with gram-positive aerobic *Streptococci* species (spp.) being the initial colonizers, followed in sequence by *Actinomyces* spp., *Corynebacterium* spp., *Veillonella* spp., and then in more mature biofilm, a variety of gram-negative anaerobic microbes such as *Treponema* spp., *Fusobacterium* spp., *Porphyromonas* spp., *Prevotella* spp., and *Tannerella* spp.[17,35,36]

As the biofilm is allowed to mature with concomitant increases in thickness, the percentage of Gram-negative anaerobic microbes increases. Specific complexes of such microbes commonly cohabit subgingival sites and are consistently associated with inflammatory periodontal diseases.[35] These putative microbial pathogens include *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola*.[35]

In the human host, the transition from gingivitis to periodontitis does not occur automatically, either in every patient or every site, but depends on 3 factors: 1) degree of host susceptibility, 2) presence and numbers of pathogenic bacteria, and 3) presence and numbers of protective bacteria.[36] Pathogenic bacteria exhibit virulence features that decrease the effectiveness of the host response by inducing tissue degradation and retarding attempts at healing.

Host defense mechanisms are impaired through a variety of mechanisms. As one example, consider that *Aggregatibacter* (formally *Actinobacillus*) *actinomycetemcomitans* produces a leukotoxin that alters the cell membranes of neutrophils and monocytes and thereby alters chemotactic and phagocytic responses.[36] Infection with Gram-negative anaerobes is accompanied by the release of epitheliotoxins, endotoxins, leukotoxins, collagenase, gellatinase, elastase, fibrinolysins, and other proteolytic enzymes.[37] These bacterial toxins and enzymes are tissue irritants and/or cytotoxic and viewed by the host immune system as foreign proteins (Figure 2). The aggregate cellular/tissue insult activates the host immune system locally and is generally visualized at a clinical level as inflammation with all the inherent gingival changes, eg, vasculitis, edema and swelling, change in tissue color from white-pink to red or red-purple, and spontaneous gingival bleeding or bleeding on provocation.[38]

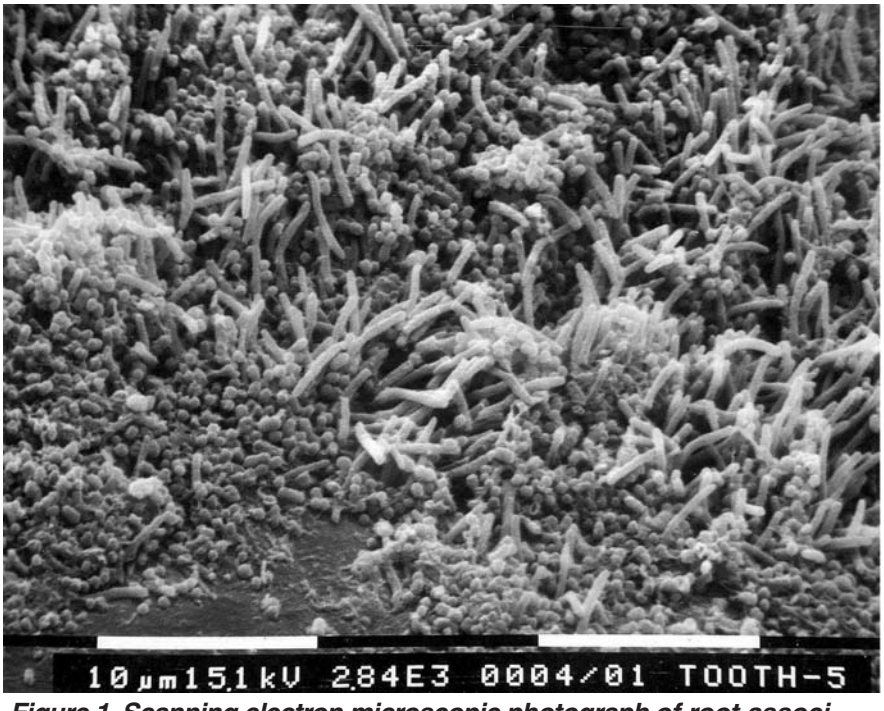

Figure 1. Scanning electron microscopic photograph of root associated dental biofilm (plaque). Bar = 10 micron at an original magnification of 2840x.

Role of the Host Immune Response

Bacteria are necessary but not sufficient by themselves to produce a destructive periodontal disease. Disease initiation and progression requires a susceptible host.[38] The microbial challenge induces an immediate inflammatory and immune response in the host. The nature and magnitude of the response have an impact on the severity and rate of progression of the periodontal disease.[39] Locally, bacteria and their metabolic byproducts stimulate a cellular immune response within the affected gingiva represented by a dense infiltration of neutrophils, macrophages, and lymphoid cells. These cells and host connective tissue cells within the developing inflammatory lesion are stimulated to synthesize and release proinflammatory cytokines, prostanoids, and proteolytic enzymes, eg, interleukin-1 (IL-1), interleukin-6 (IL-6), interleukin-8 (IL-8), tumor necrosis factor-alpha (TNF-α), prostaglandin E_2 (PGE_2), matrix metalloproteinases.[38] It is this host inflammatory-immune response that ultimately leads to the clinical signs of gingivitis and chronic periodontitis and their characteristic features of fibrous connective tissue degradation, resorption of tooth supporting alveolar bone, and periodontal pocket formation.

In contrast to the epidermis of skin, the epithelial lining of the soft tissue wall of a periodontal pocket lacks a stratum corneum and stratum granulosum. Consequently, the pocket epithelium is easily ulcerated and breached by invasive subgingival pathogenic bacteria.[40] In addition, endotoxins and other microbial antigens may gain access to the underlying connective tissues and gingival vasculature, leading to bacteremia and endotoxemia. There is considerable evidence that the locally produced proinflammatory cytokines and prostanoids gain access to the circulatory system and may, in turn, induce the production of liver-derived markers of a systemic inflammatory reaction, such as C-reactive protein, fibrinogen, serum amyloid-A, and haptoglobin.[41-45] Elevations in both the locally generated inflammatory mediators and systemic markers of inflammation have been associated with various systemic diseases such as atherosclerosis,[46] cardiovascular disease,[47] ischemic stroke,[48] pre-eclampsia,[49] and poor glycemic control 50 in diabetic patients.

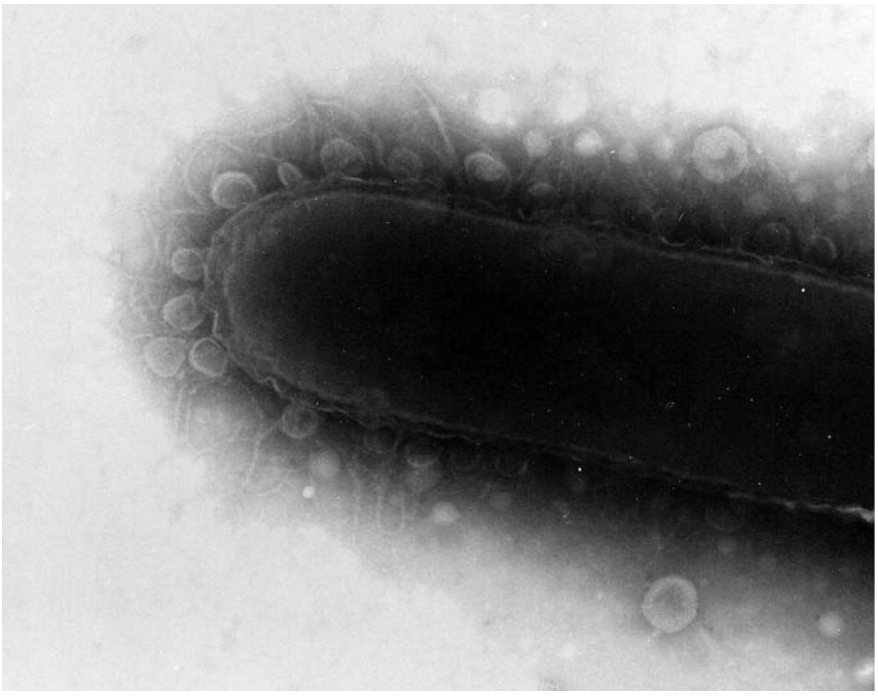

Figure 2. Transmission electron microscopic photograph of a negatively stained Phorphyromonas gingivalis featuring fimbriae and numerous surface blebs that likely contain endotoxin. Both fimbriae and endotoxin are potent antigens that solicit a host immune response. Original magnification of 35 000x.

Risk Factors Associated With Development of Chronic Periodontitis

In addition to the accepted associations of pathogenic microbes to the pathogenesis of inflammatory periodontal diseases, several genetic and environmental risk factors have been identified that affect the host response. It is well established that the prevalence and severity of chronic periodontitis increases with advancing age, poor oral hygiene, marginally or poorly controlled type I and II diabetes, and use of tobacco.[51,52] In addition, data from twin studies indicate that about 50% of the population variance in periodontitis can be attributed to genetic factors.[53,54] Several studies indicate that genetic polymorphisms (variations) in a cluster of at least 3 genes on chromosome 2q13, which control the production of proinflammatory cytokines, may affect the systemic inflammatory response in a significant percentage of people with chronic periodontitis.[55,56]

Scaling and Root Planing in the Control of Chronic Periodontitis

Periodontitis is a chronic and progressive inflammatory disease for which there is no known cure. It is now well-established that periodontitis is not associated with a single microorganism but rather the initiation and progression of periodontitis is the result of the host's immune response to a consortium of bacteria. For periodontopathic bacteria to initiate periodontitis, it is essential that they are able to colonize subgingival pockets and produce virulence factors that directly damage host tissue. Thus, a major goal of nonsurgical periodontal therapy is to suppress, to the extent possible, the subgingival pathogenic microbial flora and thereby signifi-

cantly reduce or eliminate the associated inflammatory lesion.

Dental calculus was the original etiologic agent associated with development of chronic periodontitis. In the 1960s and 1970s it was established that the rough, irregular surface of dental calculus was always covered with a nonmineralized microbial biofilm (Figure 3).[57-59] In addition to the surface biofilm, at least one recent study has identified the presence of several viable periodontal pathogens within the mass of dental calculus, ie, *Aggregatibacter actinomycetemcomitans*, *Treponema denticola* and *Porphyromonas gingivalis*.[60] Interestingly, the persistence of *Porphyromonas gingivalis* in the subgingival environment following periodontal therapy has been associated with progressive alveolar bone loss.[61] In support of this observation, Offenbacher et al[62] recently reported a significant association between serum immuneoglobulin G (IgG) titers against *Porphyromonas gingivalis* in patients that exhibit deep PDs (≥ 4 mm) and moderate (≥ 10% to < 50%) and severe (≥ 50%) bleeding on probing.

In spite of the fact that calculus can serve as a reservoir for pathogenic microbes, the role of subgingival calculus, as an etiologic agent in chronic periodontitis, was relegated to secondary status once microbial biofilm was declared the primary, extrinsic etiologic factor. Thus, the need for complete removal of subgingival calculus became a subject for debate.[63] However, the traditional treatment modality of scaling and root planing (SRP) remains the "gold standard" for the nonsurgical management of periodontitis.[64]

The periodontal literature is replete with studies showing that treatment of periodontitis by SRP results in reductions in probing depth (eg, a mean reduction of 1.29 mm for 4-6 mm pockets and a mean of 2.16 mm for pockets of ≥ 7 mm) and subgingival bacterial loads and gains in clinical attachment.[65-67] Probing depth (PD) reduction is generally greater at sites with deeper initial probing depths. The decrease in PD is the result of 2 phenomena: shrinkage of the pocket soft tissue wall manifested as recession of the gingival margin which results from a decrease in soft tissue inflammation and the inherent edema; and gain in clinical attachment.

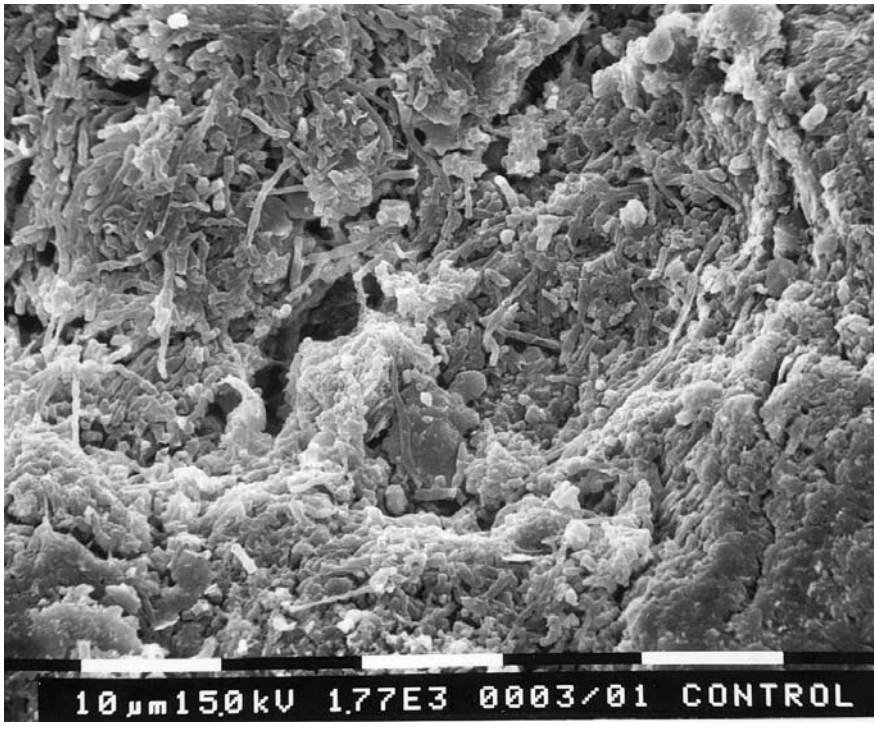

Figure 3. Scanning electron microscopic photograph of dental calculus characterized by a superficial layer of microbial biofilm. Bar = 10 micron at an original magnification of 1,770x.

The latter usually accounts for roughly one-half of the probing depth reduction.[65-67] In general, clinicians should evaluate post-SRP healing at 4 to 6 weeks following treatment. After 6 weeks, most of the healing has taken place but repair and collagen maturation may continue for an additional 9 months.[67,68]

Three relevant observations must be considered when deciding to use nonsurgical therapy as the primary modality for treatment of early to moderate chronic periodontitis. First, regarding SRP, clinicians must be careful when interpreting data from published clinical trials as they may not accurately reflect the private practice setting in terms of time, skill level, severity of disease, and diversity of patient population.[65] For example, university-conducted clinical trials often use highly skilled clinicians, select patients for level of disease, and report spending 10 minutes per tooth when performing SRP.[66,67] Ten minutes per tooth equates to about 70 minutes per quadrant. It is the experience of this author that in private practice a quadrant of SRP may be completed in approximately 60 minutes, regardless of the level of disease, and this allows approximately 10 minutes for setting of the patient and administration of anesthetic. Greenstein[67] has rightfully noted that decreased time devoted to SRP in more recent studies probably accounts for the diminished results reported when to the more classic clinical trials. Second, one must remember that microbes embedded in a mature, undisturbed subgingival biofilm may exhibit an increased tolerance to antimicrobial agents.[28-32] Third, even when chronic periodontitis is treated successfully, the reduction in subgingival pathogenic microbes is transitory. SRP of diseased root surfaces can open dentinal tubules, allowing invasion by periodontal pathogens into the exposed tubules, and possibly then serve as a reservoir for re-infection of the pocket.[69,70] Thus, the need for follow-up treatment, usually consisting of supra- and subgingival debridement at 3 to 4 month intervals, is necessary to maintain the initially gained beneficial effects.[71,72] Collectively considered, the distinct probability of less than ideal results from SRP and pocket re-infection by residual microbes is a forceful argument for the use of adjunctive treatment modalities in addition to SRP.

Clinical Implications

1. The prevalence rate for chronic periodontitis (slight, moderate, and advanced severity) is approximately 30% of the US adult population or roughly 65 million individuals.
2. Bacteria growing in undisturbed dental biofilms exhibit a significant increased tolerance to antimicrobial agents and antibiotics.
3. The transition from gingivitis to periodontitis does not occur automatically, either in every patient or every site, but depends on 3 factors: 1) degree of host susceptibility, 2) presence and numbers of pathogenic bacteria, and 3) presence and numbers of protective bacteria.
4. Even when chronic periodontitis is treated successfully, the reduction in subgingival pathogenic microbes is transitory. Thus, the need for follow-up treatment, usually consisting of supra- and subgingival debridement at 3 to 4 month intervals, is necessary to maintain the initially gained beneficial effects.
5. Due to limitations of SRP and reinfection of the periodontal pocket, adjunctive treatment modalities may increase the likelihood of improvement in the periodontal condition.

Disclosure

Dr. Cobb has served as a scientific advisor and consultant for OraPharma, Inc.

References

1. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol. 1999;4:1-6.
2. Albandar JM, Kingman A. Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988-1994. J Periodontol. 1999; 70:30-43.
3. Albandar JM, Rams TE. Global epidemiology of periodontal diseases: an overview. Periodontol 2000. 2002; 29:7-10.
4. Löe H, Brown LJ. Early-onset periodontitis in the United States of America. J Periodontol. 1991;62:608-616.
5. Jenkins WM, Papapanou PN. Epidemiology of periodontal disease in children and adolescents. Periodontol 2000. 2001;26:16-32.
6. Douglass CW, Fox CH. Cross-sectional studies in periodontal disease: current status and implications for dental practice. Adv Dent Res. 1993;7:25-31.
7. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. J Periodontol. 1999;70:13-29.
8. Brown LJ, Oliver RC, Löe H. Evaluating periodontal status of US employed adults. J Am Dent Assoc. 1990;121: 226-232.
9. Borrell LN, Burt BA, Taylor GW. Prevalence and trends in periodontitis in the USA: The NHANES, 1988 to 2000. J Dent Res. 2005;84:924-930.
10. Page RC, Eke PI. Case definition for use in population-based surveillance of periodontitis. J Periodontol. 2007; 78: 1387-1399.
11. Borrell LN, Crawford ND. Social disparities in periodontitis among United States adults 1999-2004. Community Dent Oral Epidemiol 2008;36:In Press. Published article online: 18-Oct-2007, www.Blackwell-Synergy.com
12. Cobb CM, Williams KB, Gerkovitch M. Is The Prevalence of Periodontitis in the United States in Decline? Periodontol 2000. 2008; In Press.
13. Kingman A, Morrison E, Löe H. Systematic errors in estimating prevalence and severity of periodontal disease. J Periodontol. 1988;59:707-713.
14. Hunt R, Fann S. Effect of examining half teeth in a partial periodontal recording of older adults. J Dent Res. 1991;70:1380-1385.
15. Eaton KA, Duffy S, Griffiths GS, Gilthorpe MS, Johnson NW. The influence of partial and full-mouth recordings on estimates of prevalence and extent of lifetime cumulative attachment loss: A study in a population of young male military recruits. J Periodontol. 2001;72:140-145.
16. Listgarten MA. Structure of the microbial flora associated with periodontal health and diseases in man. J Periodontol. 1976;47:1-18.
17. Cobb CM, Killoy WJ. Microbial colonization in human periodontal disease: an illustrated tutorial on selected ultrastructural and ecologic considerations. Scan Microsc. 1990;4:675-691.
18. Nishihara T, Koseki T. Microbial etiology of periodontitis. Periodontol 2000. 2004;36:14-26.
19. Marsh PD. Dental plaque as a microbial biofilm. Caries Res. 2004;38:204-221.
20. Caldwell DE, Atuku E, Wilkie DC, et al. Germ theory vs. community theory in understanding and controlling the proliferation of biofilms. Adv Dent Res. 1997;11:4-13.
21. Marsh PD. Dental plaque: Biological significance of a biofilm and community life-style. J Clin Periodontol. 2005;32(Suppl 6):7-15.
22. Gilbert P, Maira-Litran T, McBain AJ, Rickard AH, Whyte FW. The physiology and collective recalcitrance of microbial biofilm communities. Adv Microbial Physiol. 2002;46: 203–255.
23. Stewart PS, Costerton JW. Antibiotic resistance of bacteria in biofilms. Lancet. 2001;358:135-138.
24. Marsh PD, Bradshaw DJ. Microbiological effects of new agents in dentifrices for plaque control. Inter Dent J. 1993;43:399-406.
25. Kinniment SL, Wimpenny JWT, Adams D, Marsh PD. The effect of chlorhexidine on defined, mixed culture oral biofilms grown in a novel model system. J Appl Bacteriol. 1996;81:120-125.
26. Wilson M. Susceptibility of oral bacterial biofilms to antimicrobial agents. J Med Microbiol. 1996;44:79-87.
27. Pratten J, Wilson M. Antimicrobial susceptibility and composition of microcosm dental plaques supplemented with sucrose. Antimicrob Agents Chemother. 1999;43:1595-1599.
28. Zaura-Arite E, van Marle J, ten Cate JM. Confocal microscopy study of undisturbed and chlorhexidine-treated dental biofilm. J Dent Res. 2001;80:1436-1440.
29. Larsen T. Susceptibility of Porphyromonas gingivalis in biofilms to amoxicillin, doxycycline and metronidazole. Oral Microbiol Immunol. 2002;17:267-271.
30. Socransky SS, Haffajee AD. Dental biofilms: difficult therapeutic targets. Periodontol 2000. 2002;28:12–55.
31. Noiri Y, Okami Y, Narimatsu M, Takahashi Y, Kawahara T, Ebisu S. Effects of chlorhexidine, minocycline, and metron-

idazole on Porphyromonas gingivalis strain 381 in biofilms. J Periodontol. 2003;74:1647-1651.
32. Wright TL, Ellen RP, Lacroix JM, Sinnadurai S, Mittelman MW. Effects of metronidazole on Porphyromonas gingivalis biofilms. J Periodont Res. 1997;32:473-477.
33. Aas JA, Paster BJ, Stokes LN, Olsen I, Dewhirst FE. Defining the normal bacterial flora of the oral cavity. J Clin Microbiol. 2005;43:5721-5732.
34. Paster BJ, Olsen I, Aas JA, Dewhirst FE. The breadth of bacterial diversity in the human periodontal pocket and other oral sites. Periodontol 2000. 2006;42:80-87.
35. Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL, Jr. Microbial complexes in subgingival plaque. J Clin Periodontol. 1998;25:134-144.
36. Sbordone L, Bortolaia C. Oral microbial biofilms and plaque-related diseases: Microbial communities and their role in the shift from oral health to disease. Clin Oral Invest. 2003;7:181-188.
37. Zambon JJ. Periodontal diseases: Microbial factors. Ann Periodontol. 1996;1:879-925.
38. Offenbacher S. Periodontal diseases: pathogenesis. Ann Periodontol. 1996;1:821-878.
39. Page RC, Kornman KS. The pathogenesis of human periodontitis: an introduction. Periodontol 2000. 1997:14:9-11.
40. Hujoel PP, White BA, Garcia RI, Listgarten MA. The dentogingival epithelial surface area revisited. J Periodont Res. 2001;36:48-55.
41. Ebersole JL, Machen RL, Steffen MJ, Willmann DE. Systemic acute-phase reactants, C-reactive protein and haptoglobin in adult periodontitis. Clin Exper Immunol. 1997;107:347-352.
42. Noack B, Genco RJ, Trevisan M, Grossi S, Zambon JJ, De Nardin E. Periodontal infections contribute to elevated systemic C-reactive protein level. J Periodontol. 2001;72:1221-1227.
43. Amar S, Gokce N, Morgan S, Loukideli M, Van Dyke TE, Vita JA. Periodontal disease is associated with brachial artery endothelial dysfunction and systemic inflammation. Arterioscler Thromb Vasc Biol. 2003;23:1245-1249.
44. Slade GD, Ghezzi EM, Heiss G, Beck JD, Riche E, Offenbacher S. Relationship between periodontal disease and C-reactive protein among adults in the Atherosclerosis Risk in Communities study. Arch Intern Med. 2003;163:1172-1179.
45. Leivadaros E, van der Velden U, Bizzaro S, et al. A pilot study into measurements of markers of atherosclerosis in periodontitis. J Periodontol. 2005;76:121-128.
46. Tonetti MS, D'Aiuto F, Nibali L, et al. Treatment of periodontitis and endothelial function. N Eng J Med. 2007;356:911-920.
47. Kinane DF, Lowe GD. How periodontal disease may contribute to cardiovascular disease. Periodontol 2000. 2000;23:121-126.
48. Grau AJ, Becher H, Ziegler CM, et al. Periodontal disease as a risk factor for ischemic stroke. Stroke. 2004;35:496-501.
49. Siqueira FM, Cota LOM, Costa JE, Haddad JPA, Lana AMQ, Costa FO. Maternal periodontitis as a potential risk variable for preeclampsia: A case-control study. J Periodontol. 2008;79:207-215.
50. Hein C, Cobb CM, Iacopino A. Report of an independent panel of experts of the Scottsdale Project. The independent study initiative for collaboration in diabetes, cardiovascular disease and periodontal disease intervention. Grand Rounds in Oral-Sys Med. 2007;2(Suppl):2-27.
51. Abdellatif HM, Burt BA. An epidemiological investigation into the relative importance of age and oral hygiene status as determinants of periodontitis. J Dent Res. 1987;66: 13-18.
52. American Academy of Periodontology. Position paper: Epidemiology of periodontal disease. J Periodontol. 1996;67:935-945.
53. Michalowicz BS, Aeppli D, Virag JG, et al. Periodontal findings in adult twins. J Periodontol. 1991;62:293-299.
54. Michalowicz BS, Diehl SR, Gunsolley JC, et al. Evidence of a substantial genetic basis for risk of adult periodontitis. J Periodontol. 2000;71:1699-1707.
55. Kornman KS, Crane A, Wang HY, et al. The interleukin-1 genotype as a severity factor in adult periodontal disease. J Clin Periodontol. 1997;24:72-77.
56. D'Aiuto F, Parkar M, Brett PM, Ready D, Tonetti MS. Gene polymorphisms in proinflammatory cytokines are associated with systemic inflammation in patients with severe periodontal infections. Cytokine. 2004;28:29-34.
57. Baumhammers A, Conway JC, Saltzberg D, Matta RK. Scanning electron microscopy of supragingival calculus. J Periodontol. 1973;44:92-94.
58. Muhleman HR, Schroeder HE. Dynamics of supragingival calculus formation. Adv Oral Biol. 1964;1:175-203.
59. Mandel ID. Dental plaque: Nature, formation and effects. J Periodontol. 1966;37:357-367.
60. Calabrese N, Galgut P, Mordan N. Identification of Actinobacillus actinomycetemcomitans, Treponema denticola and Porphyromonas gingivalis within human dental calculus: A pilot investigation. J Inter Acad Periodontol. 2007;9:118-128.
61. Chaves ES, Jeffcoat MK, Ryerson CC, Snyder B. Persistent bacterial colonization of Porphyromonas gingivalis, Prevotella intermedia, and Actinobacillus actinomycetemcomitans in periodontitis and its association with alveolar bone loss after 6 months of therapy. J Clin Periodontol. 2000;27:897-903.
62. Offenbacher S. Barros SP. Singer RE. Moss K. Williams RC. Beck JD. Periodontal disease at the biofilm-gingival interface. J Periodontol. 2007;78(10):1911-1925.
63. Robertson PB. The residual calculus paradox. J Periodontol. 1990;61:65-66.
64. Ryan ME. Nonsurgical approaches for treatment of periodontal diseases. Dent Clin N Am. 2005;49:611-636.
65. Cobb CM. Non-surgical pocket therapy: Mechanical. Ann Periodontol. 1996;1:443-490.
66. Greenstein G. Periodontal response to mechanical nonsurgical therapy: a review. J Periodontol. 1992;63:118-130.
67. Greenstein G. Nonsurgical periodontal therapy in 2000: a literature review. J Am Dent Assoc. 2000;131:1580-1592.
68. Badersten A, Nilveus R, Egelberg J. Effect of nonsurgical periodontal therapy. I: moderately advanced periodontitis. J Clin Periodontol. 1981;8:57-72.
69. Adriaens PA, DeBoever JA, Loesche WJ. Bacterial invasion in root cementum and radicular dentin of periodontally diseased teeth in humans. J Periodontol. 1988;59:222-230.
70. Giuliana G, Ammatuna P, Pizzo G, Capone F, D'Angelo M. Occurrence of invading bacteria in radicular dentin of periodontally diseased teeth: microbiological findings. J Clin Periodontol. 1997;24:478-485.
71. Listgarten MA. A rationale for monitoring the periodontal microbiota after periodontal treatment. J Periodontol. 1988;59:439-444.
72. Listgarten MA, Levin S, Schifter CC, Sullivan P, Evian CI, Rosenberg ES, Laster L. Comparative longitudinal study of 2 methods of scheduling maintenance visits: 2 year data. J Clin Periodontol. 1986;13:692-700.

Locally Delivered Antimicrobials: Clinical Evidence and Relevance

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Abstract

Periodontitis is a common oral infection and inflammatory condition. Following treatment, residual or persistent periodontal inflammation is associated with disease progression and tooth loss. Cumulative evidence from clinical trials and meta-analyses support a complementary medical-mechanical model that combines locally delivered antimicrobials with scaling and root planing for the treatment of chronic periodontitis. Accordingly, greater pocket depth reductions and/or attachment level gains occur in patients treated with adjunctive locally administered antimicrobials (eg, tetracycline, chlorhexidine, doxycycline, and minocycline). These responses are clinically relevant because they are accompanied by a higher probability of patient maintenance or pocket resolution. Recent trials also indicate that locally administered antimicrobials may enhance the effects of periodontal surgical therapy and may reduce the signs of peri-implantitis. The consistency of these findings supports the use of locally administered antimicrobials for managing dental patients with chronic periodontitis.

Keywords: periodontitis, antibiotics, antimicrobials, local delivery, peri-implantitis, scaling and root planing

Introduction

Periodontal disease is a common, mixed oral infection affecting the supporting structures around the teeth. While 75% of the adult population has at least mild periodontal disease (gingivitis), 20%-30% exhibits the severe destructive form (chronic periodontitis).[1] Characteristically, the disease is silent until the advanced stage when patients may report symptoms like swelling (abscess), discomfort, shifting of the dentition, or tooth mobility. The clinical signs of periodontitis emanate from inflammatory and destructive changes in the gingiva, connective tissues, alveolar bone, periodontal ligament, and root cementum. These signs include the formation of periodontal pockets, loss of clinical attachment, and resorption of alveolar bone.[2]

Accordingly, periodontitis begins with a pathogenic shift in the bacterial flora around teeth. Gram-negative organisms, such as *Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola* and *Aggregatibacter* (formally *Actinobacillus*) actinomycetemcomitans, predominate in the subgingival space and organize as a biofilm.[3] Several of the gram-negative bacteria in the biofilm are particularly important because they have been identified as red-complex bacteria (*T. forsythia, P. gingivalis,* and *T. denticola*) and have been linked with important parameters of periodontal diagnosis, such as pocket depth and bleeding on probing.[3] This bacterial biofilm is in direct contact with host tissues along an ulcerated epithelial interface called a periodontal pocket. Locally, bacteria and their products (eg, lipopolysaccharide entotoxin) penetrate host periodontal tissues and stimulate host expression of inflammatory mediators like arachidonic acid metabolites (prostaglandin E2) and cytokines (interleukin-1).[4] These mediators in turn trigger local inflammatory and destructive changes in the tissues.

Longitudinal population studies indicate that these destructive changes (disease progression) are not continuous over time but appear restricted to "random bursts" of activity confined to short intervals (6 months or less).[5] Risk factors associated with progressive periodontitis include smoking, diabetes, obesity, poor plaque control, and certain genetic polymorphisms.[6-10] In addition, residual or persistent deep probing depths are associated with periodontitis progression.[11] Paulander and coworkers recently demonstrated that periodontitis subjects with moderate (4-5 mm) and deep (> 6 mm) probing depths were 2 to 3 times more likely to exhibit alveolar bone loss over 10 years.[12] Similarly for tooth loss, the odds ratio for moderate pockets was 2.9 (95% CI, 1.9-4.2), and the odds for deep pockets was 4.2 (95% CI, 2.4-7.3). These data imply pocket depth reduction (or resolution) is a clinically important treatment goal to ensure stability and maintenance in patients.

Complementary Medical-Mechanical Treatment Model with Adjunctive Antimicrobials

Strategies for treating periodontitis principally focus on addressing the etiologic bacteria or biofilm.[13,14] According to the mechanical model, the bacterial biofilm is disrupted and removed via scaling and root planing (SRP) procedures. These debridement procedures can be accomplished nonsurgically or surgically, and both approaches result in pocket depth (PD) reductions in patients.[15,16] In addition, a number of adjunctive chemotherapeutic approaches have been developed, tested and approved for use in patients with chronic periodontitis (Table 1). These "locally delivered antimicrobials" follow a complementary medical-mechanical treatment model since they are used in combination with SRP for enhanced efficacy. These formulations typically cou-

Table 1. Summary of FDA-approved locally administered antimicrobials and clinical evidence from pivotal trials.

Locally Administered Antimicrobial	Active Agent	Polymer	Pivotal Trial Reference	Number of Subjects	Experimental Treatment	Controls	Results
Periochip®	Chlorhexidine gluconnate (2.5 mg)	Cross-linked hydrolyzed gelatin	25	447	Periochip® plus SRP (adjunct)	Placebo chip plus SRP SRP alone	Periochip® plus SRP significantly reduced PD and increased CAL at 9 months compared to SRP alone.
Atridox®	Doxycycline (10% or 50 mg)	poly DL-lactide	29	411	Atridox® alone (monotherapy)	Placebo gel, SRP alone, no treatment	Treatment with Atridox® alone produced improvements in PD and CAL at 9 months that were equivlent to SRP alone.
Arestin®	Minocycline (1 mg)	Polyglycolide-co-dl-lactide	33	748	Arestin® plus SRP (adjunct)	Placebo microspheres plus SRP, SRP alone	Subjects treated with Arestin® plus SRP exhibited significantly greater PD reductions at 1, 3, 6, and 9 months versus SRP alone.

ple an antimicrobial or antibiotic with a drug polymer that extends drug release within the periodontal pocket (controlled-release delivery).[17]

A recent systematic review and meta-analysis conducted by Hanes and coworkers demonstrated that adjunctive locally administered antimicrobials improved PD over SRP alone in chronic periodontitis patients.[18] This group of investigators searched electronic databases and relevant dental journals and identified 32 clinical studies fitting selection criteria. The studies (28 randomized controlled clinical trials, 2 cohort, and 2 case-control studies) represented a variety of locally administered antimicrobials (eg, minocycline, doxycycline, tetracycline, metronidazole, and chlorhexidine formulations). The resulting meta-analysis indicated an overall significant reduction in PD with adjunctive local antimicrobials versus SRP alone. These findings strongly support the use of locally administered antimicrobials in combination with SRP in patients with chronic periodontitis, especially those at risk for disease progression.

The first local delivery system approved for use by the US Food and Drug Administration (FDA) was called Actisite® (ALZA Corporation, Palo Alto, Calif, USA) and was developed by Dr. Max Goodson in 1983.[19] This product consisted of a nonresorbable polymer fiber of ethyl vinyl acetate containing tetracycline hydrochloride (25% or 12.7 mg). Each fiber (23 cm) was placed subgingivally similar to retraction cord. Since that time, clinicians have been introduced to second generation locally delivered antimicrobials that are easier to utilize and produce greater clinically significant results. Following is a discussion about the 3 products currently available in the United States.

Chlorhexidine Gluconate Chip

The PerioChip® (Dexcel Technologies Limited, Jerusalem, Israel) is a biodegradable gelatin-based polymer system containing the active antimicrobial, chlorhexidine gluconate (2.5 mg). Each chlorhexidine (CHX)-gelatin wafer or chip is placed subgingivally with cotton pliers. While pharmacokinetic studies indicate that chlorhexidine is released from the system for 7-10 days in periodontal pockets, microbial studies have shown suppression of the pocket flora for up to 11 weeks following CHX chip treatment.[20,21] In the phase 3 clinical trials, CHX chip treatment plus SRP significantly reduced PD and maintained CAL at 9 months compared with SRP controls.[22] Importantly, SRP was limited in these trials to one hour of ultrasonic scaling. In addition, retreatment with CHX chip occurred at 3 and 6 months at sites with residual pockets (> 5 mm). Nevertheless, after 9 months of adjunctive CHX chip treatment, no sites exhibited bone loss, and 25% of the sites exhibited bone gain as measured with subtraction radiography.[23] In contrast, 15% of periodontal sites treated with SRP alone exhibited bone loss. Chlorhexidine gluconate chip has a documented safety profile, and unlike chlorhexidine mouthrinse, does not cause any visible staining of teeth.

Doxycycline Bioresorbable Gel

Atridox® (Atrix Laboratories, Fort Collins, Colo, USA) is a 10% formulation of doxycycline (50 mg) in a bioresorbable gel system (poly DL-lactide and N-methyl-2-pyrrolidone mixture). The system is supplied as 2 pre-filled syringes that are mixed chair-side and applied subgingivally to the base periodontal pockets using a syringe. The "flowable" polymer gel fills and conforms to pocket morphology, then solidifies to a wax-like consistency upon contact with gingival crevicular fluid. Doxycycline is released at effective concentrations over 7 days, and significant reductions (60%) in anaerobic pathogens are sustained for up to 6 months posttreatment.[24,25] In subjects with chronic periodontitis, the application of doxycycline gel (at baseline and 4 months later) reduced PD (1.3 mm) and improved CAL (0.8 mm) comparable to SRP alone at 9 months following treatment.[26]While current and former smokers within the trials did not respond as well to SRP alone, smoking status did not diminish the clinical improvements observed with doxycycline gel.[27] While these studies demonstrated equivalency of doxycycline gel (monotherapy) with SRP and supported regulatory approval, this system like other locally delivered antimi-

crobials is conventionally used as an adjunct to SRP in clinical practice.

One phase 4 or postmarketing trial investigated the use of doxycycline gel as an adjunct to SRP and demonstrated incremental benefits when the system was used in combination with SRP.[28] Accordingly, one arm of the adjunctive use trial involved initiating treatment with ultrasonic scaling plus doxycycline gel at baseline, and then isolated SRP at 3 months for those sites with residual pocketing (PD > 5 mm). The second arm of the study involved SRP alone at baseline, and then isolated ultrasonic scaling and doxycycline gel at those sites with residual pocketing. While both treatment strategies were equally effective at improving probing depths and clinical attachment levels over 6 months, responses were greater on average for the adjunctive doxycycline gel treatment at 3 months compared to SRP alone.

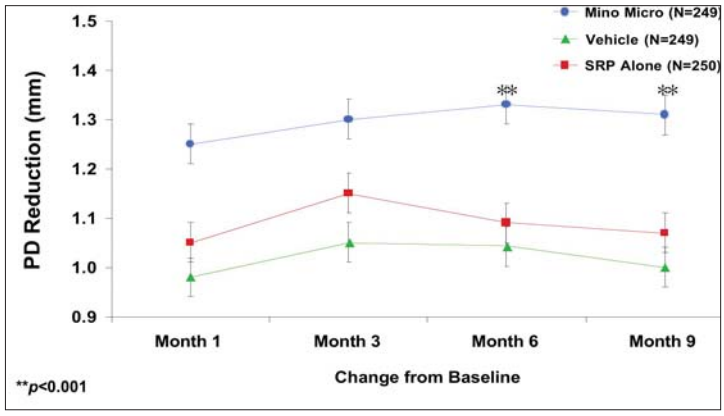

Figure 2. Mean probing-depth reductions over nine months for periodontitis subjects treated with adjunctive minocycline microspheres, adjunctive vehicle, or SRP alone. Adapted from Williams et al.[30]

Minocycline Microspheres

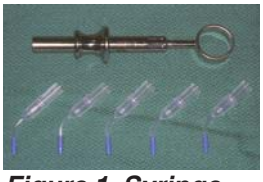

Figure 1. Syringe handle and premeasured cartridges for dispensing minocycline microspheres.

Arestin® (OraPharma, Inc., Warminster, Pa, USA) is an approved local delivery system featuring 1mg of minocycline hydrochloride microencapsulated in resorbable polymer microspheres (polyglycolide-co-dl-lactide). The delivery system (cartridge and syringe) is designed for quick and easy administration of one unit dose of Arestin subgingivally in periodontal pockets measuring ≥ 5 mm with bleeding on probing (BOP) (Figure 1). With this system, minocycline hydrochloride is maintained within pockets for 21 days at concentrations effective against periodontal pathogens. The agent may also block collagenases that are implicated in host tissue breakdown.[29]

The pivotal clinical trials of minocycline microspheres involved approximately 750 subjects with generalized moderate to advanced chronic periodontitis recruited at 18 centers.[30] Periodontitis subjects meeting inclusion criteria at baseline were randomized to 1 of 3 treatments: 1) scaling and root planing (SRP) alone (positive control); 2) SRP plus polymer vehicle (placebo control); or 3) SRP plus minocycline microspheres. Full mouth probing exams were performed at baseline (prior to treatment) and at 1, 3, 6, and 9 months. Figure 2 graphs mean probing depth reductions observed in the 9-month trial for all subjects (intent-to-treat population) in the primary analysis. Analyses of covariance adjusting for centers indicated significant-inter-group differences in probing depth reductions at all time points ($p < 0.001$). In particular, subjects treated with adjunctive minocycline microspheres exhibited significantly greater probing depth reductions as compared to control subjects treated with SRP alone. When smokers (Figure 3) or those with advanced periodontitis (mean baseline PD > 6 mm) (Figure 4), were considered in secondary analyses, again ANCOVA indicated significant probing depth reductions with adjunctive minocy-

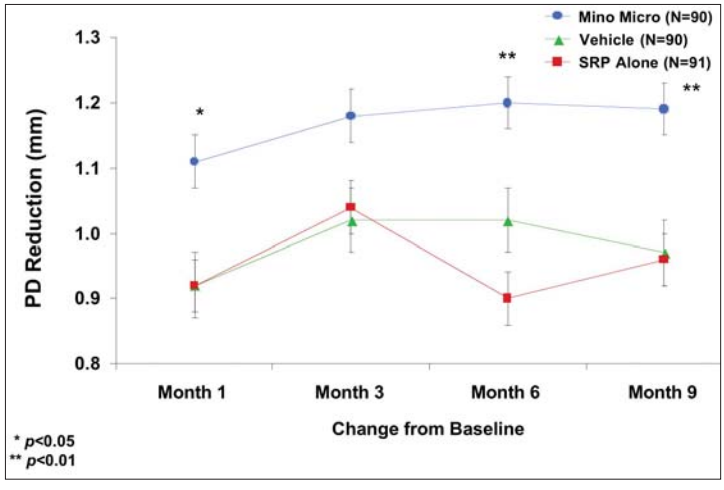

Figure 3. Mean probing-depth reductions over nine months for periodontitis subjects who smoke and were treated with minocycline, adjunctive vehicle, or SRP alone. Adapted from Paquette et al.[31]

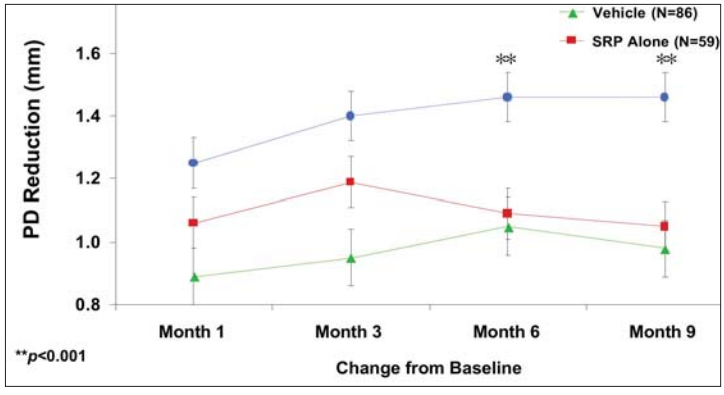

Figure 4. Mean probing-depth reductions over nine months for advanced periodontitis subjects (mean baseline probing depth ≥ 6 mm) treated with minocycline, adjunctive vehicle, or SRP alone. Adapted from Williams et al.[30]

cline microspheres over control treatments.[31] Indeed, inter-group differences in PD reduction were greater among advanced periodontitis subjects versus the overall population.

A priori, a shift in subject mean probing depth < 5 mm with treatment was considered a clinically relevant and "maintainable" response. When regression analyses were performed comparing response odds with adjunctive minocycline microspheres treatment versus SRP alone, the odds ratios for subjects who smoked or who had advanced periodontitis were 2.06 (95% CI 1.10, 3.85) and 2.86 (95% CI 1.45, 5.66), respectively.[32] These data indicate that patients with advanced periodontitis or smokers are 2 to 3 times more likely to respond, and that this increase in odds is clinically relevant. Site analyses on pocket resolution (posttreatment PD < 5 mm) were also designated as meaningful. Again, a significantly and consistently higher percent of pockets were "resolved" with adjunctive minocycline microspheres versus SRP alone for all subjects and smokers, respectively (Table 2).[33]

A large, phase 4 (postmarketing) trial involving 2805 patients and 895 dentists was conducted to evaluate the use of minocycline microspheres in private practices throughout the United States.[34] Accordingly, 1095 patients received 2 applications of minocycline microspheres (at baseline and 3 months) per protocol, and 1710 patients received only one minocycline microsphere application (at baseline). Mean 6-month pocket depth reductions were 1.82 and 1.94 mm for the patients receiving one and 2 minocycline microspheres treatments, respectively. Similar results were obtained in smokers, diabetic patients, and cardiovascular disease patients. After one minocycline microspheres treatment, 62% of sites had decreased to less than 5 mm, and after 2 treatments the corresponding proportion increased to 67%. This large private practice study demonstrated that minocycline microspheres plus SRP is effective in reducing pocket depth and that efficacy increased with retreatment (dose-response).

One recently published trial indicates that the effects of flap surgery may be enhanced with adjunctive minocycline microspheres treatment. Hellström and coworkers recruited 60 periodontitis patients and randomized them to either flap surgery plus minocycline microspheres therapy (baseline and weeks 2, 3, and 5) or surgery alone.[35] At week 25, the mean PD reduction from baseline was 2.51 mm in the surgery plus minocycline microspheres (test) group versus 2.18 mm in the control group. Smokers in the test group had a significantly greater probing depth reduction (2.30 mm) as compared to smokers in the control group (2.05 mm). In addition, the number of sites with probing depth reductions of 2 mm or more was significantly higher in the test group than in the control group. Hence, minocycline microspheres may be adjuncts to both nonsurgical and surgical therapies for patients with moderate to severe, chronic periodontitis.

These efficacy findings for minocycline microspheres have been extended to peri-implantitis, an inflammatory process around one or more osseointegrated implants in function, resulting in a loss of supporting bone and associated with a similar pathogenic flora. Renvert and coworkers conducted a clinical trial in which 32 subjects with peri-implantitis (one implant with PD > 4 mm, bleeding and/or exudate on probing and the presence of putative pathogens) randomly received debridement plus minocycline

Table 2. Percentage of periodontal pockets resolving with adjunctive minocycline microspheres versus SRP. Adapted from Paquette et al.[33]

Baseline PD	**5mm**		6mm		**7mm**		**>8mm**	
Treatment **All Subjects**	Mino Micro	SRP Alone	Mino Micro	SRP Alone	Mino Micro	SRP Alone	Mino Micro	SRP Alone
Month 1	76	69	47	39	22	20	10	8
	p<0.0001		p<0.001		p=0.31		p=0.24	
Month 3	78	71	52	48	28	23	19	14
	p<0.0001		p=0.01		p=0.01		p=0.02	
Month 9	75	66	54	49	34	27	22	16
	p<0.0001		p=0.0005		p=0.001		p=0.01	
Treatment **Smokers**	Mino Micro	SRP Alone	Mino Micro	SRP Alone	Mino Micro	SRP Alone	Mino Micro	SRP Alone
Month 1	73	66	40	34	17	15	6	3
	p<0.0001		p=0.003		p=0.53		p=0.09	
Month 3	74	66	44	41	22	15	16	5
	p<0.001		p=0.17		p=0.04		p=0.003	
Month 9	70	61	45	39	27	20	20	12
	p<0.0001		p=0.006		p=0.04		p=0.04	

microspheres or debridement plus chlorhexidine gel (0.2%) at baseline, 1 month, and 3 months.[36] While both treatments reduced putative pathogens, adjunctive minocycline microsphere treatment resulted in significant improvements in PD compared to chlorhexidine gel at 1 month, 3 months, and 6 months. Significant reductions in bleeding on probing were also noted for up to 12 months. This investigative group published the results from a second trial with 30 peri-implantitis subjects. Again, adjunctive minocycline microspheres improved PD and bleeding scores, whereas the adjunctive use of chlorhexidine gel had limited effects on bleeding scores.[37] Another investigative team, Salvi and coworkers, also noted consistent efficacy with minocycline microspheres for treating peri-implantitis.[38] Here, the investigators applied minocycline microspheres to implant sites exhibiting bone loss and PD > 5 mm following a 3-week debridement and hygiene interval. While 6 of 31 implants were either rescued or exited from the trial because of persistent peri-implantitis, all other implants (80.6%) showed significant reduction in both PD and BOP over 12 months with minocycline microspheres therapy. The investigators also examined peri-implant microflora using DNA-DNA checkerboard hybridization techniques and observed significant reductions in *A. actinomycetemcomitans* at 12 months and reductions in "red complex" bacteria (*T. forsythia, P. gingivalis,* and *T. denticola*) for 6 months.[39] Binary regression analysis showed that the clinical parameters and smoking history could not discriminate between successfully treated and rescued/exited implants at any observation time point. In addition, failures in treatment could not be associated with the presence of specific pathogens or by the total bacterial load at baseline. Collectively, these new data indicate improvements in the clinical signs of peri-implantitis over 12 months with adjunctive locally administered minocycline.

Goodson and coworkers conducted a clinical trial utilizing 124 subjects with moderate to advanced chronic periodontitis. Subjects were randomly assigned to either SRP alone or minocycline microspheres and SRP. All patients received full-mouth SRP at baseline, followed by treatment with minocycline microspheres if assigned to the SRP and minocycline microspheres group. The examiner was blinded to the patient's treatment. Clinical assessments were made and plaque samples were collected at baseline and at Day 30. The results demonstrated that adjunctive minocycline microspheres significantly reduced red-complex periodontal pathogens as compared to SRP alone by one month.[40]

Another investigation conducted by Oringer et al[41] investigated the effect of minocycline microspheres on gingival crevicular fluid (GCF) levels pyridinoline cross-linked carboxy-terminal telopeptide of type I collagen (ICTP) and interleukin 1-beta (IL-1). ICTP is a bone-specific degradation product and IL-1 is a potent bone-resorptive cytokine. Forty eight periodontitis patients were randomized to receive SRP followed by minocycline microspheres or vehicle. Eight healthy individuals served as a control group. Results found a potent short term reduction of ICTP and IL-1 in the SRP plus minocycline microspheres group.

Summary and Conclusions

Residual or persistent periodontal inflammation is associated with instability of dental tissues (periodontal disease progression and tooth loss). Cumulative data from clinical trials and meta-analyses support a complementary medical-mechanical model using locally delivered antimicrobials for treating chronic periodontitis. Overall, the clinical evidence accrued to date consistently shows that when locally administered antimicrobials are used adjunctively, significantly greater PD reductions and/or attachment level gains occur in patients. These responses are clinically relevant because they are accompanied by a greater likelihood for patient maintenance or pocket resolution. Recent trials also indicate that locally administered antimicrobials may enhance the effects of periodontal surgical therapy and may reduce the signs of peri-implantitis. The consistency of these findings supports the use of locally administered antimicrobials for managing dental patients with chronic periodontitis.

Clinical Implications

- Recent clinical trials indicate that locally administered antimicrobials may enhance the effects of periodontal surgical therapy and may reduce the signs of peri-implantitis.
- Patients with periodontitis exhibiting moderate (4-5mm) and deep (≥ 6 mm) probing depths were 2 to 3 times more likely to exhibit alveolar bone loss over 10 years.
- A systematic review and meta-analysis demonstrated that adjunctive locally administered antimicrobials improved PD over SRP alone in chronic periodontitis patients.
- Patients with advanced periodontitis or smokers are 2 to 3 times more likely to respond to SRP + minocycline microspheres than to SRP alone.
- Use of minocycline microspheres has been shown to be advantageous when used as an adjunctive therapy to both nonsurgical and surgical therapies in patients with moderate to severe, chronic periodontitis.
- Adjunctive use of minocycline microspheres has shown a reduction in red-complex periodontal pathogens as compared to SRP alone.

Disclosure

Dr. Paquette has served as a scientific consultant and investigator for OraPharma, Inc. Dr. Ryan and Ms. Wilder are scientific consultants for Orapharma, Inc.

References

1. Albandar J, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. J Periodontol 1999;70:13-29.
2. Flemmig TF. Periodontitis. Ann Periodontol 1999;4:32-38.
3. Socransky SS, Haffajee AD. Periodontal microbial ecology. Periodontol 2000 2005;38:135-187.
4. Offenbacher S. Periodontal diseases: pathogenesis. Ann Periodontol 1996;1:821-878.
5. Socransky SS, Haffajee AD, Goodson JM, Lindhe J. New concepts of destructive periodontal disease. J Clin Peri-

odontol 1984; 11:21-32.
6. American Academy of Periodontology Research, Science and Therapy Committee. Tobacco use and the periodontal patient. J Periodontol 1999;70: 1419-1427.
7. Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: a two-way relationship. Ann Periodontol 1998;3:51-61.
8. Saito T, Shimazaki Y, Sakamoto M. Obesity and periodontitis. N Eng J Med. 1998;482-483.
9. Ramfjord SP, Morrison EC, Burgett FG, Nissle RR, Shick RA, Zann GJ, Knowles JW. Oral hygiene and maintenance of periodontal support. J Periodontol 1982;53:26-30.
10. Kornman KS, Crane A, Wang HY, di Giovine FS, Newman MG, Pirk FW, Wilson TG Jr et al. The interleukin-1 genotype as a severity factor in adult periodontal disease. J Clin Periodontol 1997;24:72-77.
11. Halazonetis TD, Haffajee AD, Socransky SS. Relationship of clinical parameters to attachment loss in subsets of subjects with destructive periodontal diseases. J Clin Periodontol 1989;16:563-568.
12. Paulander J, Axelsson P, Lindhe J, Wennstrom J. Intra-oral pattern of tooth and periodontal bone loss between the age of 50 and 60 years. A longitudinal prospective study. Acta Odontol Scand 2004;62:214-222.
13. American Academy of Periodontology. Parameter on chronic periodontitis with slight to moderate loss of periodontal support. J Periodontol 2000;71:853-855.
14. American Academy of Periodontology. Parameter on chronic periodontitis with advanced loss of periodontal support. J Periodontol 2000;71:856-858.
15. Cobb CM. Non-surgical pocket therapy: Mechanical. Ann Periodontol 1996;1:450-490.
16. Palcanis KG. Surgical pocket therapy. Ann Periodontol 1996;1:589-606.
17. Drisko CH. Nonsurgical pocket therapy: Pharmacotherapeutics. Ann Periodontol 1996;1:491-566.
18. Hanes PJ, Purvis JP, Gunsolley JC. Local anti-infective therapy: pharmacological agents. A systematic review. Ann Periodontol 2003;8:79-98.
19. Goodson JM, Holborow D, Dunn RL, Hogan P, Dunham S. Monolithic tetracycline-containing fibers for controlled delivery to periodontal pockets. J Periodontol 1983; 54:575-579.
20. Soskolne WA, Chajek T, Flashner M, Landau I, Stabholtz A, Kolatch B, Lerner EI. An in vivo study of the chlorhexidine release profile of the PerioChip in the gingival crevicular fluid, plasma and urine. J Clin Periodontol 1998;25:1017-1021.
21. Stabholz A, Sela MN, Friedman M, Golomb G, Soskolne A. Clinical and microbiological effects of sustained release chlorhexidine in periodontal pockets. J Clin Periodontol 1986;13:783-788.
22. Jeffcoat MK, Bray KS, Ciancio SG, Dentino AR, Fine DH, Gordon JM et al. Adjunctive use of a subgingival controlled-release chlorhexidine chip reduces probing depth and improves attachment level compared with scaling and root planing alone. J Periodontol 1998;69:989-997.
23. Jeffcoat MK, Palcanis KG, Weatherford TW, Reese M, Geurs NC, Flashner M. Use of a biodegradable chlorhexidine chip in the treatment of adult periodontitis: clinical and radiographic findings. J Periodontol 2000;71:256-262.
24. Stoller NH, Johnson LR, Trapnell S, Harrold CQ, Garrett S. The pharmacokinetic profile of a biodegradable controlled-release delivery system containing doxycycline compared to systemically delivered doxycycline in gingival crevicular fluid, saliva, and serum. J Periodontol 1998;69:1085-91.
25. Walker CB, Godowski KC, Borden L, Lennon J, Nango S, Stone C et al. The effects of sustained release doxycycline on the anaerobic flora and antibiotic-resistant patterns in subgingival plaque and saliva. J Periodontol 2000;71:768-774.
26. Garrett S, Johnson L, Drisko CH, Adams DF, Bandt C, Beiswanger B et al. Two multi-center studies evaluating locally delivered doxycycline hyclate, placebo control, oral hygiene, and scaling and root planing in the treatment of periodontitis. J Periodontol 1999;70:490-503.
27. Ryder MI, Pons B, Adams D, Beiswanger B, Blanco V, Bogle G et al. Effects of smoking on local delivery of controlled-release doxycycline as compared to scaling and root planing. J Clin Periodontol 1999;26:683-691.
28. Wennstrom JL, Newman HN, MacNeill SR, Killoy WJ, Griffiths GS, Gillam DG et al. Utilisation of locally delivered doxycycline in non-surgical treatment of chronic periodontitis. A comparative multi-centre trial of 2 treatment approaches. J Clin Periodontol 2001;28:753-761.
29. Oringer RJ, Al-Shammari KF, Aldredge WA, Iacono VJ, Eber RM, Wang HL, Berwald B et al. Effects of locally delivered minocycline microspheres on markers of bone resorption. J Periodontol 2002;73:835-842.
30. Williams R, Paquette D, Offenbacher S, Adams D, Armitage G, Bray K et al, Treatment of periodontitis by local administration of minocycline microspheres: a controlled trial. J Periodontol 2001;72:1535-1544.
31. Paquette DW, Oringer R, Lessem J, Offenbacher S, Genco R, Persson GR, Williams R. Locally delivered minocycline microspheres for the treatment of periodontitis in smokers. J Clin Periodontol 2003;30:787-794.
32. Paquette DW. Pocket depth reduction as an outcome measure of inflammation and soft tissue changes in Periodontitis trials. J Int Acad Periodontol 2005;7/4 (Supplement):147-156.
33. Paquette DW, Williams RC, Hanlon A, Lessem J. Clinical relevance of adjunctive minocycline microspheres in patients with chronic periodontitis: secondary analysis of a phase 3 trial. J Periodontol 2004;75:531-536.
34. Lessem J, Hanlon A. A post-marketing study of 2805 patients treated for periodontal disease with Arestin. J Int Acad Periodontol 2004;6:150-153.
35. Hellström MK, McClain PK, Schallhorn RG, Bellis L, Hanlon AL, Ramberg P. Local minocycline as an adjunct to surgical therapy in moderate to severe, chronic periodontitis. J Clin Periodontol 2008;35:525-31.
36. Renvert S, Lessem J, Dahlén G, Lindahl C, Svensson M. Topical minocycline microspheres versus topical chlorhexidine gel as an adjunct to mechanical debridement of incipient peri-implant infections: a randomized clinical trial. J Clin Periodontol 2006;33:362-9.
37. Renvert S, Lessem J, Dahlén G, Renvert H, Lindahl C. Mechanical and repeated antimicrobial therapy using a local drug delivery system in the treatment of peri-implantitis: a randomized clinical trial. J Periodontol 2008;79:836-44.
38. Salvi GE, Persson GR, Heitz-Mayfield LJ, Frei M, Lang NP. Adjunctive local antibiotic therapy in the treatment of peri-implantitis II: clinical and radiographic outcomes. Clin Oral Implants Res 2007;18:281-5.
39. Persson GR, Salvi GE, Heitz-Mayfield LJ, Lang NP. Antimicrobial therapy using a local drug delivery system (Arestin) in the treatment of peri-implantitis. I: Microbiological outcomes. Clin Oral Implants Res 2006;17:386-93.
40. Goodson JM, Gunsolley JC, Grossi SG, Bland PS, Otomo-Corgel J, Doherty F, Comiskey J. Minocycline HCl microspheres reduce red-complex bacteria in periodontal disease therapy.J Periodontol. 2007;78:1568-79.
41. Oringer RJ, Al-Shammari KF, Aldredge WA, Iacono VJ, Eber RM, Wang HL et al. Effects of locally delivered minocycline microspheres on markers of bone resorption. J Periodontol. 2002; 73:835-842.

Periodontal Treatment Protocol (PTP) for the General Dental Practice

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Abstract

A sequence of interrelated steps is inherent to effective periodontal treatment: early and accurate diagnosis, comprehensive treatment, and continued periodontal maintenance and monitoring. A primary goal of periodontal therapy is to reduce the burden of pathogenic bacteria and thereby reduce the potential for progressive inflammation and recurrence of disease. Emerging evidence of possible perio-systemic links further reinforces the need for good periodontal health. In the private practice setting, the treatment of patients with periodontal disease is best accomplished within the structure of a uniform and consistent Periodontal Treatment Protocol (PTP). Such a protocol would reinforce accurate and timely diagnosis, treatment needs based on a specific diagnosis, and continual assessment and monitoring of outcomes. This is best achieved if everyone in the practice setting has a general understanding of the etiology of periodontal diseases, the benefits of treatment, and potential consequences of nontreatment. Communication skills and patient education are vital components of effective therapy since slight and even moderate stages of the disease often have few noticeable symptoms to the patient. Accurate documentation and reporting of procedures for dental insurance reimbursement, coupled with scheduling considerations, assist general practice settings in effectively managing the increasing volume of patients that can benefit from early diagnosis and treatment of periodontal diseases. This article presents the essential elements of a PTP including diagnosis, treatment planning, implementation of therapy, assessment and monitoring of therapy, insurance coding, introduction of the patient to periodontal therapy, and enhanced verbal skills. In addition, considerations for implementation of adjunctive local delivery antimicrobials is presented.

Key Words: periodontal diseases, periodontal diagnosis, treatment protocol, periodontal maintenance, periodontal assessment, patient education

Introduction

Hujoel et al[1] estimated a 31% decrease in the prevalence of periodontitis between the years 1955 and 2000. Further, these authors estimate an additional 8% decrease by the year 2020. In spite of the decreased use of smoking tobacco,[2] better understanding of the pathogenesis of periodontal diseases, and more refined and goal directed therapies, there remains evidence that dentistry is not consistently achieving a timely diagnosis and appropriate and timely treatment of existing periodontitis.[3,4] Although the evidence is limited, there is a strong suggestion that use of a periodontal probe for diagnosis and recording of periodontal status in treatment records in general dental practices has yet to achieve the level of a routine and consistent habit.[5-9] Indeed, McFall et al[8] determined that except for radiographs, most private practice patient records were so deficient in diagnostic information that periodontal status could not be established. It should be self-evident that treatment requires a definitive diagnosis, ie, a disease cannot be adequately treated unless first diagnosed. In this regard, it is interesting to note that at least one study has reported a disconnect between dentists' perception of treatment rendered and actual treatment as recorded in patient records.[10] As an example, prophylactic procedures outnumber periodontal procedures by a ratio of 20:1[11,12] and yet the prevalence of chronic periodontitis (slight, moderate, and severe) is estimated to range from a low of 7% (aged ≥ 18 years)[13] up to 35% (aged ≥ 30-90 years)[14] of the US adult population.

Cobb et al.[3] compared the pattern of referral of periodontitis patients in 1980 vs 2000 using patient record data from 3 geographically-diverse private periodontal practices. Results showed the following trends occurring over the 20-year time span: decreased use of tobacco; increase in the percentage of cases exhibiting advanced chronic periodontitis with a concomitant decrease in the percentage of mild-moderate disease cases; increase in the average number of missing teeth per patient; and increase in the average number of teeth scheduled for extraction per patient. A similar study by Docktor et al[4] based on patient records from 3 private periodontal practices located within a major metropolitan area reported the following: 74% of referred cases were considered advanced periodontitis, of which 30% were treatment planned for extraction of 2 or more teeth; periodontal treatment provided by the general dental office did not vary because of disease severity; and the average number of periodontal maintenance visits/patient/year in the general dental office was less than the standard of care according to severity of disease, eg, 68% of advanced periodontitis cases reported between 0 and 2 periodontal maintenance visits per year rather than the recommended every 3 months. Viewed in aggregate, the trends reported by Cobb et al[3] and Docktor et al[4] support the assertion that timely diagnosis and appropriate and timely treatment of chronic periodontitis have

not significantly improved over time. A major reason for the reported scarcity of timely diagnosis and appropriate treatment may be the lack of a well-established office protocol for the diagnosis, treatment, maintenance, and monitoring of periodontal disease, and involvement of the patient through education. Obviously, this requires dedication of energy, resources, effective communication skills, and a change in practice philosophy.

The Periodontal Treatment Protocol (PTP)

Diagnosis

Regardless of recent advances in our understanding of the etiology and pathogenesis of the periodontal diseases, the assessment of traditional clinical parameters remain the foundation for periodontal diagnosis.[15] Generally, such clinical parameters include probing depth (PD), bleeding on probing (BOP), clinical attachment level (CAL), degree of furcation involvement, extent of gingival recession, tooth mobility, and plaque score. Clinicians typically utilize the results from the periodontal exam, radiographs, and the patient's medical and dental histories to establish a diagnosis and evolve a goal/diagnosis-directed treatment plan. It has been clearly demonstrated that different interpretations of the same diagnostic information can have a dramatic impact on treatment decisions.[16] For this reason, a standardized approach to periodontal assessments and a working protocol as to treatment parameters would fill a logical need in the average general practice setting. However, due to extensive overlaps in most classification systems, any standardized approach is subject to variations in both clinical assessments (eg, variations in probing depth among clinicians) as well as the interpretation thereof.

All effective treatment protocols begin with a thorough and timely diagnosis. Six-point probing to measure PD and BOP is the standard of care. Based on the needs of the patient, current radiographs should be evaluated to determine the location and percentage of bone loss. The presence, location, and extent of furcation invasions should be noted, as well as the location of the gingival margin or CAL. Also, the patient's age is an important factor, especially in cases of rapidly progressing disease and determining overall long-term prognosis.

A modified version of the American Academy of Periodontology (AAP) proposed guidelines for a comprehensive periodontal examination is presented in Table 1.[17] However, with respect to a functional PTP for the general dental practice, only the following principal diagnostic criteria can be addressed: age, PD, CAL, BOP, tooth mobility, furcation involvement, and percentage of radiographic bone loss. It must be emphasized that these criteria represent the minimal parameters for determining a periodontal diagnosis. There are many other important risk and modifying factors that will impact development and progression of disease and all such factors must be taken into consideration when establishing a defin-

Table 1. Modified Version of the American Academy of Periodontology Suggested Guidelines for a Comprehensive Periodontal Examination.[18]

Assessment of medical history
Assessment of dental history
Assessment of periodontal risk factors 1. Age 2. Gender 3. Medications 4. Presence of plaque and calculus (quantity and distribution) 5. Smoking 6. Race/Ethnicity 7. Systemic disease (eg, diabetes) 8. Oral hygiene 9. Socioeconomic status and level of education
Assessment of extraoral and intraoral structures and tissues
Assessment of teeth 1. Mobility 2. Caries 3. Furcation involvement 4. Position in dental arch and within alveolus 5. Occlusal relationships 6. Evidence of trauma from occlusion
Assessment of periodontal soft tissues including peri-implant tissues 1. Color 2. Contour 3. Consistency (fibrotic or edematous) 4. Presence of purulence (suppuration) 5. Amount of keratinized and attached tissue gingiva 6. Probing depths 7. Bleeding on probing 8. Clinical attachment levels 9. Presence and severity of gingival recession
Radiographic evaluation of alveolar bone loss, bone density, furcations, root shape, and proximity, etc.

itive diagnosis and a diagnosis-driven treatment plan.[18]

Age is of relative value in that advanced amounts of periodontal destruction at an earlier age tend to indicate a more aggressive form of periodontitis. In contrast, chronic periodontitis may slowly progress towards severity over several years or decades. Young age combined with moderate to severe bone loss presents a tenuous long-term prognosis and requires more aggressive therapy compared to the older patient presenting with a chronic form of periodontitis.[19]

Probing depth (PD) is defined as the distance from the gingival margin to the base of the gingival crevice.[20] The periodontal pocket, represented by a probing depth > 3 mm, is the principle habitat for gram-negative, anaerobic pathogenic bacteria.[20] Deeper pockets tend to represent more extensive destruction of the underlying periodontium and, therefore, a potentially greater pathenogenic burden.

Clinical Attachment Level (CAL) is defined as the distance from the CEJ to the base of the probable crevice/pocket. In cases of gingival recession, the amount of recession is added to the PD to yield the total amount of CAL. Although more difficult to obtain, it is a better measure of the total extent of damage to the underlying periodontium.[20-22]

Mobility is best measured by the blunt end of 2 instruments alternating pressure in a facial-lingual direction and an apical direction to assess abnormal movement of the tooth. Simply assessed: Grade I mobility is slightly more than normal; Grade II is moderately more than normal; Grade III is severe mobility facial-lingually plus apical displacement.[23] Mobility patterns are suggestive of possible occlusal trauma, severe inflammation, and/or loss of supporting alveolar bone.

Furcations represent bone loss between the roots of multi-rooted teeth. A deeply invasive furcation lesion is the equivalent of a poor long-term prognosis for the involved tooth. Simply put, a Grade 1 furcation involvement is incipient bone loss only; a Grade 2 is partial loss of bone producing a cul-de-sac; a Grade 3 is total bone loss with through-and-through opening of the furcation; and a Grade 4 is similar to a Grade 3, but with gingival recession that visually exposes the furcation opening.[24]

Radiographic Evidence of Bone Loss is best determined with adequate and current radiographs,[17] most typically a full-mouth periapical survey, including vertical bite-wings, or a panographic radiograph supplemented with vertical bite-wings and selected periapical films. By definition, true periodontitis does not begin until bone loss occurs.[25] Radiographic evaluation of the distribution and severity of bone loss, bone density, root anatomy, and approximation to other teeth provides specific information that will help in determining a proper diagnosis, treatment plan, and prognosis.

Bleeding on Probing (BOP) is a simple assessment of the inflammatory status of the gingiva.[15,26] In patients with deeper pockets and/or loss of clinical attachment, the chances of disease progression are greater as the percentage of bleeding sites increase.[27] Conversely, lack of BOP is highly correlated with stability and a lack of inflammation.[28] This latter statement, however, does not apply to smokers as they tend to bleed less when compared to nonsmokers with equal amounts of disease.[29]

In addition to the usual clinical parameters, the clinician is well advised to consider other risk factors and their potential impact on the development and progression of plaque-induced periodontal diseases.[18] Risk factors that are sometimes overlooked in the diagnosis, treatment plan, and prognosis equation include, among others: diabetes, smoking, osteoporosis, compromised immune system, drug-induced gingival conditions, hormonal changes, and genetics. Patients at risk for periodontal disease are often allowed to "slip between the cracks" during a routine visit because they may be in the early stages of the disease. Risk factors increase a patient's chance of developing periodontitis. The presence of one or more of these risk factors may also indicate a benefit from specialty referral in some patients.

Case Types and Periodontal Diagnosis

As part of a PTP it is necessary to establish diagnostic guidelines that will provide a framework for organizing the treatment needs of the patient. Guidelines are not meant to replace clinical knowledge or skills, nor do they imply a one-size-fits-all treatment plan for periodontal disease. It is recognized that each dental practice setting is different. Consequently, guidelines are intended to be used in a manner that best meets the needs of the specific patient.

Generally speaking, plaque-induced periodontal diseases have historically been categorized into gingivitis versus periodontitis based upon whether attachment loss has occurred. The 1999 International Workshop for Classification of Periodontal Diseases[21] reclassified the plaque-induced periodontal diseases into 7 different classifications. In consideration of a working PTP that addresses only the common periodontal diseases, this paper will address health, gingivitis, chronic periodontitis (formerly adult periodontitis), and aggressive periodontitis (formerly early-onset periodontitis). The first 7 entries in Table 2 (see back cover) constitute a set of clinical criteria (PD, BOP, percent bone loss, tooth mobility, degree of furcation involvement, and CAL) that will facilitate differentiation of health from gingivitis and between the various levels of severity of chronic periodontitis. Further, Table 2 can aid the clinician in differentiating between chronic and aggressive periodontitis.

Some practice settings may prefer a system of "Periodontal Case Types" for purposes of diagnosis and record keeping. Table 3 presents the diagnostic clinical criteria as applied to Case Types for health, gingivitis, chronic periodontitis (slight, moderate, and severe), and aggressive periodontitis. These criteria and Case Types are generally appropriate but should be considered as guidelines only and not as a definitive diagnosis. As mentioned before, there are numerous modifying and risk factors to consider prior to evolving a diagnosis and a diagnosis-driven treatment plan.

Treatment Planning

Development of a logical and properly sequenced treatment plan is a derivative of the periodontal assessment and diagnosis. Periodontal therapy is diagnosis-driven and, to the extent possible, should address all modifying factors and risk factors that impact development and progression of plaque-induced periodontal disease. An overview of a typical periodontal treatment plan is presented in Table 4.[30]

Table 3. Clinical Criteria Assigned to Periodontal Case Types of Health, Gingivitis, Chronic Periodontitis (slight, moderate, and severe), and Aggressive Periodontitis.

Case Type		PD (mm)	BOP (Yes/No)	Bone Loss (%)	Mobility (Grade)	Furcations (Grade)	CAL (mm)	Visual Inflammation
0	(Health)	0-3	No	0	None	None	0	No
I	(Gingivitis)	0-4	Yes	0	None	None	0	Yes (localized or generalized)*
II	(Slight Chronic Periodontitis)†	4-5	Yes	10	I	1	1-2	Yes (localized or generalized)*
III	(Moderate Chronic Periodontitis)†	5-6	Yes	33	I and II	1 and 2	3-4	Yes (localized or generalized)*
IV	(Severe Chronic Periodontitis)†	≥ 6	Yes	> 33	I, II, or III	1, 2, 3, or 4	≥ 5	Yes (localized or generalized)*
V	(Aggressive Periodontitis)‡ (age is significant factor)	≥ 6	Yes	> 33	I, II, or III	1, 2, 3, or 4	≥ 5	Yes (localized or generalized)*

* Localized disease is defined as ≤ 30% of sites are involved; and generalized disease infers >30% of sites are involved.[21]

† Specialty referral may be indicated for additional treatment beyond initial therapy.

‡ Specialty referral should be considered.

Table 4. General Overview of the Major Steps in a Typical Periodontal Treatment Plan.[3]

Sequence of Major Phases
1. Address acute periodontal problems and/or pain
2. Review and update medical and dental histories
3. Assessment of systemic risk factors and refer for medical consultation as needed
4. Extraoral examination
5. Oral cancer evaluation
6. Assessment of periodontal risk and modifying factors
7. Periodontal examination to include dental implants
8. Dental examination to include occlusal relationships and dental implants
9. Radiographic examination
10. Establish a definitive diagnosis
11 Generate a diagnosis-driven periodontal treatment plan and sequence of treatment
12. Determine required adjunctive restorative, prosthetic, orthodontic, and/or endodontic treatments and sequence
13. Execute Phase I therapy (aka anti-infective or nonsurgical therapy) with consideration given to adjunctive use of chemotherapeutic agents
14. Re-evaluation (assessment) of Phase I therapy
15. If end-points are not achieved, consider selective retreatment, need for surgical therapy, specialty referral, or use of adjunctive diagnostic aides, eg, microbial, genetic, medical lab tests, etc.
16. Determine interval for periodontal maintenance and continued assessment of periodontal status

Implementation of Therapy

There are a wide variety of treatment options to be considered when confronted with gingivitis or chronic or aggressive periodontitis. However, thorough scaling and root planing (SRP) is still considered the gold standard in periodontal therapy. Beyond SRP, no one treatment modality is the answer in every case. However, the clinician must have specific endpoints or goals that therapy should achieve. If such endpoints are not achieved, then therapy must be re-evaluated and a decision made concerning retreatment or specialty referral for consideration of more advanced therapy options. Treatment options that should be considered include:[30]

- Patient education including plaque control and counseling in management of periodontal and systemic risk factors
- Scaling and root planing
- Consideration of adjunctive chemotherapeutic agents, eg, locally or systemically administered antibiotics and host response modification agents.
- Re-evaluation
- Consideration of referral to a specialist is an option that must be considered for both legal and ethical reasons.[31] There are a variety of reasons to consider referral to a periodontist, such as, SRP in the presence of extreme amounts of dental calculus or SRP with complications of systemic disease, gingival overgrowth and/or inflammatory hyperplasia, resective surgery, regenerative procedures for soft and hard tissues, periodontal plastic surgery, occlusal therapy, pre-prosthetic surgery, dental implants, management of perio-systemic complications, phobic patients requiring conscious sedation, etc.

Periodontal Maintenance Therapy and Continual Assessment

In general, data suggests that patients who have undergone definitive therapy for either localized or generalized periodontitis should be managed by periodontal maintenance (PM), performed at an interval of 3 months for an indefinite period of time following active therapy.[32] The 3-month interval is critical (and the standard of care for moderate and severe chronic periodontitis and aggressive periodontitis) as it has been repeatedly shown to be effective in reducing disease progression, preserving teeth, and controlling the subgingival bacterial burden.[33-35] Nonetheless, the PM schedule should be individualized and tailored to meet the needs of each patient. Factors such as home care, previous level of disease, tendency toward refraction, stability indicators, etc, should be used in making this assessment. More fragile patients may need intervals of 2 months or less, and conversely, patients intercepted in early disease states who demonstrate consistent stability may need less frequent intervals of 4-6 months. Regardless of the interval between appointments, the periodontal status of each patient should be re-evaluated at every maintenance appointment. Only through close monitoring can disease recurrence be detected and the maintenance interval adjusted accordingly. Continual assessment of the periodontium during maintenance affords the best opportunity for assuring long-term stability or providing interceptive care.[34,35]

Insurance Coding

The American Academy of Periodontology's Parameters of Care 2000[36] and the American Dental Association's Current Dental Terminology[37] are available to clinicians to guide decision-making related to providing therapeutic periodontal treatment and subsequent reporting of services for insurance reimbursement. In terms of nonsurgical periodontal therapy, familiarity with dental insurance codes provides a clear method to document treatment and select appropriate procedures to maximize insurance reimbursement for the patient.

Table 5 presents a modified description of the ADA insurance codes most commonly used in Phase I periodontal therapy (aka anti-infective therapy or nonsurgical therapy). The descriptions are intended to reveal distinctive differences between procedures, and guide the clinician in reimbursement procedures.

To simplify decisions made by patients, dental insurance should be referred to as "reimbursement," "benefit," or "assistance" by the clinician and other staff members rather than "coverage" since the word implies complete. Most patients with dental insurance will find it necessary to supplement whatever insurance benefit they receive toward lifetime periodontal care, as many plans have contract limitations for the percentage of reimbursement associated with various procedures and/or the length of time those benefits apply. For example, limitations of some insurance plans assign benefits for PM following SRP but only for 24 months following active therapy. As another example, exclusions found in other insurance plans assign benefits for SRP for generalized periodontal disease but not for localized infection. Many patients are reticent to proceed with treatment unless their insurance will "pay for it." Consequently, it is advantageous for practices to have clear explanations about the reality of dental insurance. Figure 2 presents a sample explanation of dental insurance that can

Understanding Dental Insurance

1. Dental insurance is a contractual agreement between the employer and insurance company. The percentage of reimbursement varies greatly dependent upon the premiums paid for a particular plan and limitations of the agreement.
2. Maximum payable benefits around $1000 - $1500 commonly found today with dental insurance plans are almost identical to the annual maximum benefit of dental insurance plans 40 years ago.
3. Dental insurance is a benefit designed to help defray the costs of quality dental care, but is not all-inclusive of what an individual may need or desire to obtain optimal dental health for a lifetime.

Figure 2. Facts about dental insurance to share with patients.

Table 5. Modified Description of ADA Insurance Codes Commonly Used for Phase I Periodontal Therapy (aka anti-infective therapy or nonsurgical therapy).

Code Number	Treatment Procedure	Description
D0180	Comprehensive Periodontal Evaluation	Indicated for new or established patients showing signs or symptoms of periodontal disease and for patients with risk factors such as smoking or diabetes. It includes evaluation of periodontal conditions, probing and charting, evaluation and recording of the patient's dental and medical history and general health assessment. It may include the evaluation and recording of dental caries, missing or unerupted teeth, restorations, occlusal relationships and oral cancer evaluation.
D1110	Adult Prophylaxis	Includes the removal of plaque, stain and calculus from tooth structures and is intended to control local irritation to gingival tissues, thereby preventing disease initiation.
D4355	Full Mouth Debridement to Enable Comprehensive Evaluation and Diagnosis	Initial removal of plaque and calculus that interfere with the ability to perform a comprehensive oral evaluation. This preliminary procedure is generally followed by a comprehensive periodontal evaluation for diagnosis and subsequent therapeutic periodontal procedures.
D4341	Scaling and Root Planing Generalized per Quadrant	Involves therapeutic treatment of 4 or more periodontally involved teeth per quadrant through definitive removal of subgingival plaque biofilm and root preparation in order to halt the disease from progressing, thereby creating an opportunity for healing. To be reported per quadrant inclusive of updated periodontal charting and radiographs for reimbursement.
D4342	Scaling and Root Planing Localized per Quadrant	Involves therapeutic treatment of 1 to 3 periodontally involved teeth per quadrant through definitive removal of subgingival plaque biofilm and root preparation in order to halt the disease from progressing, thereby creating an opportunity for healing. To be reported per quadrant with identification of specific teeth being treated inclusive of updated periodontal charting and radiographs for reimbursement.
D4381	Localized Delivery of Antimicrobial Agents via a Controlled Release Vehicle into Diseased Crevicular Tissue	Subgingival insertion of antimicrobial medications of a therapeutic concentration into periodontal pockets that are released over a sufficient length of time in order to suppress the pathogenic burden, and are intended to enhance the clinical results of scaling and root planing alone. To be reported per tooth, identifying multiple treatment sites per tooth, if indicated, inclusive of a narrative describing systemic considerations for reimbursement such as tobacco usage, diabetes, or heart disease.
D4999	Unspecified Periodontal Procedure, by Report	In the absence of a specific ADA code for complete periodontal re-assessment following definitive periodontal therapy, this procedure code is being utilized to determine healing response and future treatment recommendations.
D4910	Periodontal Maintenance	Follows the completion of active therapy to treat periodontal infection for the lifetime of the dentition or implant replacements and includes removal of plaque biofilm and calculus from supra and subgingival surfaces. It may also include site specific scaling and root planing for areas of localized disease recurrence. It is intended to keep periodontal diseases under control; therefore a patient may move from active therapy to periodontal maintenance and back to active therapy and/or referral during the lifetime of the dentition or implant replacements. It is not synonymous with prophylaxis, and is required at varying intervals to manage periodontal diseases and modify risk factors. To be reported by both general and periodontal practices on patients having undergone active therapy irrespective of where the procedure is performed. Current periodontal charting documenting the patient's on-going periodontal status should be submitted for reimbursement.

be shared with patients, assisting them in making independent decisions about treatment, regardless of the insurance reimbursement schedule.

Patient Education and Introduction to Periodontal Therapy

Effective implementation of the aforementioned concepts requires expertise in effective patient education and introduction of periodontal therapy so that patients are prepared to make wise health decisions. Being proficient in SRP procedures has little value to the patient who assumes they are visiting the dental hygienist for a "routine cleaning." This is particularly true if the patient already has a developing or existing periodontal infection and does not understand the need for therapeutic intervention. Chronic periodontal diseases often provide few noticeable symptoms, especially in earlier stages of development. Thus, the need for effective communication, education, and listening skills are of particular importance to today's dental patient.

The incidence of moderate and severe generalized chronic periodontitis in the US appears to affect only 5% to 15% of the adult population, whereas slight disease affects approximately 35% of the adult population.[13,14,38] Thus, most new patients and even many existing patients will ultimately be diagnosed with periodontal diseases. To be effective at enrolling patients into active therapy everyone in the practice setting must have a basic understanding of the etiology of periodontal diseases, treatment options, consequences of nontreatment, and direct benefits of therapy. Patients are more motivated to accept treatment recommendations when a clear diagnosis has been established, they are given the opportunity to see infection in their own mouths, their questions have been answered, and they understand the value of treating periodontal infection in relation to their overall health.

Many clinicians inform patients of their periodontal status while working in their mouths with sharp instruments, or give a summary of findings at the end of the visit. Most patients are visual learners. Consequently, patients need to see the condition of their own mouth. At the beginning of every appointment, during data collection and tissue assessment, the patient should be provided a mirror to visualize with the clinician the evidence of periodontal disease, caries, gingival recession, tooth mobility, furcation involvement, etc. (Figure 1). During periodontal probing, the patient should hear the pocket measurements as data is being collected and recorded. In a similar manner, during examination of the radiographs, the patient should be shown evidence of permanent bone loss, and contrast that to areas without bone loss. Involving the patient in the discovery process visually and audibly is a powerful tool to help patients take ownership in their own health.

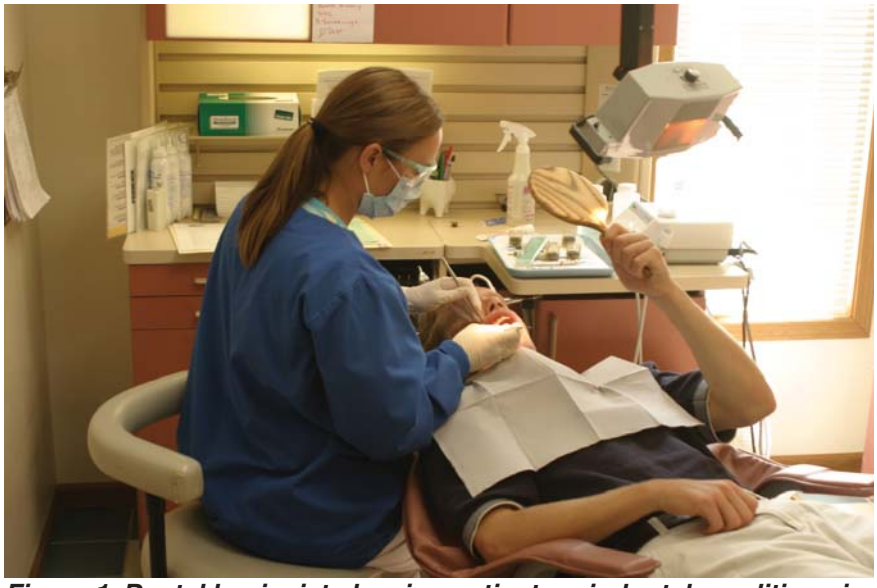

Figure 1. Dental hygienist showing patient periodontal conditions in patient's own mouth.

This is also an opportune time for the clinician to introduce adjunctive therapies to the patient such as the use of locally delivered antimicrobials and other agents. For example, the clinician can communicate that locally delivered antimicrobials have been on the US market for many years and have been shown to be a safe, effective treatment option. Important information to convey includes the ease of application; the high potency of the drug at levels that will kill bacteria; it does not affect the rest of the body; and there is no need for an additional appointment to remove the product since the agent biodegrades. Educating the patient to all of their treatment options is vital to clear and evidence-based communication.

Enhanced Communication Skills

Each clinician will develop his/her own style of case presentation for periodontal therapy and will individualize the message to different patients. However, there is significant advantage if the entire office staff has continuity in key words that are used when discussing periodontal therapy with patients. Equally important is the avoidance of minimizing messages such as "just a little bit of bleeding," or "a little bone loss," or "just a little bit of plaque." It is advisable to use language that does not trivialize conditions that are not yet severe. Terms such as "slight bleeding," "early bone loss," or "slight plaque" accurately describe findings without overstating them. Periodontal disease is a bacterial infection leading to a host immune response that is characterized by inflammation and degradation of periodontal tissues.[22] When informing patients of periodontal disease, using the word "infection" is more powerful than "gum inflammation" and can create a sense of urgency regarding treatment. The word "hemorrhage" indicates heavy bleeding and implies a condition outside healthy parameters. When the patient's gingival tissues hemorrhage easily upon provocation, "hemorrhage" rather than "bleeding gum tissue" should be verbalized to the patient. The words "scaling and root

Guide for Use of Locally Delivered Antimicrobials

Where to use locally delivered antimicrobials:

- Pockets ≥ 5 mm with bleeding on probing (BOP).
  - The locally delivered antimicrobial may be used at the time of scaling and root planing (SRP) or at the re-evaluation appointment 4-6 weeks following SRP. If used first at the re-evaluation appointment, the site must have additional SRP to remove biofilm and hard deposits that may have re-accumulated.
- Residual pockets of ≥ 5 mm with BOP or any site ≥ 6 mm following initial SRP.
  - Determined at re-evaluation appointment.
  - If ≥ 4 residual pockets in a given quadrant then consider either retreatment (SRP) with locally delivered antimicrobial or surgical intervention.
- Sites treatment planned for osseous grafting.
  - Locally delivered antimicrobial placed 3 weeks prior to surgical procedure.
- Periodontal abscess
- Probing depth at the distal-facial line-angle of 2nd molars related to 3rd molar extractions where surgical intervention will yield a compromised result.
- Ailing/failing dental implants (peri-implantitis) where surgical intervention is not indicated or will yield a compromised result.
- Grade II furcation involvements (shallow or deep) when surgical intervention is not planned.

Who might benefit from use of locally delivered antimicrobials:

- Periodontal maintenance patients with isolated probing depths of ≥ 5 mm that exhibit BOP or any pocket ≥ 6 mm (Figure 3).
- Patients wanting to avoid periodontal surgery.
- High risk surgery patients.
  - Poorly controlled (brittle) diabetic patients
  - Patients with a history of recent or recurrent coronary or cerebrovascular events.
  - Patients with a compromised immune system due to disease or medications.
  - Kidney dialysis patients.
  - Heavy smokers (>½ pack/day)
  - Patients with physical disability that impacts oral hygiene efficiency
  - Mentally handicapped patients
- Patient's with marginal oral hygiene that is not likely to improve and thereby represent a poor surgical risk.
- Please note that locally applied antimicrobials may need to be placed more than one time to achieve the desired result.

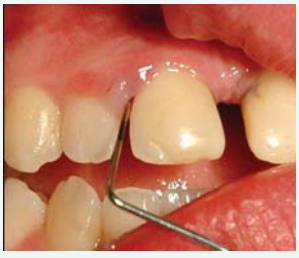

Figure 3. Pre-treatment clinical presentation showing PD of 6 mm

How to apply locally delivered antimicrobials:

- For optimal effect from locally delivered antimicrobials the following must be achieved:
  - Oral hygiene instructions and patient compliance regarding the necessary oral hygiene procedures, ie, tooth brushing, use of interdental hygiene aids such as dental floss and proxabrushes, and use of antimicrobial oral rinses.
  - Supragingival scaling and polishing.
  - Definitive subgingival SRP (generally under local anesthesia).
  - Place locally delivered antimicrobial according to manufacturer's directions. For example, in the case of minocycline microspheres, place one pre-measured dose per pocket. If the tooth has 2 pockets that need local delivery, 2 full doses should be administered.
  - The pocket should be as biofilm and deposit free as possible prior to insertion.
  - Insert the locally delivery product to the base of the pocket. In the case of minocycline microspheres, the tip should be placed as far into the pocket as possible before activating the syringe/handle (Figures 4 and 5).

Addendum:

- If probing depths are ≤ 4 mm, the clinician should consider a conventional adult prophylaxis coupled with oral hygiene recommendations and/or reinforcement.
  - If the patient exhibits multiple probing depths of 4 mm a periodontal maintenance interval of 3-4 months should be considered until it can be determined if the patient's periodontal status is stable and/or improving.

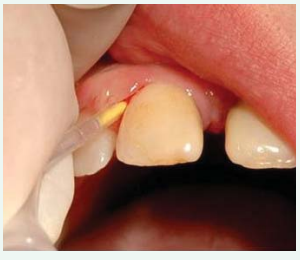

Figure 4. Initial Insertion of the pre-measured tip for administration of minocycline microspheres

Figure 5. Tip placement to base of pocket for administration of minocycline microspheres.

planing" may sound confusing to patients or imply discomfort. The words "periodontal therapy" are effective semantic choices when informing patients about necessary periodontal treatment. "We now know" are words that can introduce patients to new ideas or treatment options to explain why information may be different than what they have heard in the past, or expected to hear at their current visit. "Halting" or "arresting disease" can be used to describe a measurable goal for treating periodontal diseases that should be obtained through intervention. "Daily disease control" communicates to the patient that they share in the role in the effective removal of plaque bacteria beyond what it achieve through periodontal treatment.

Even though some states require written consent, effective communication between the clinician and the patient is the important consideration of informed consent,[39] not the completion of a form. Therefore, deliberate semantic choices should be shared by all members of the office staff to optimize patient understanding of their periodontal conditions.

Suggestions for Implementation of a Periodontal Treatment Protocol in the General Practice Setting

- General dentists and dental hygienists should schedule a meeting with referring periodontists and their dental hygienists to share philosophies of periodontal treatment and establish clarity for referrals.
- Schedule a team meeting workshop to bring all office staff up-to-date regarding periodontal assessments, diagnosis, case types, periodontal risk factors, individualized treatment of periodontal diseases, consequences of nontreatment (tooth loss and possible systemic involvement), and the value of periodontal maintenance.
- Establish continuity of the verbal skills and terminology the office staff will utilize to communicate effectively to patients about periodontal conditions.
- Include assessments and diagnosis of periodontal diseases in all new patient visits, routine prophylaxis appointments, and ongoing periodontal maintenance to insure no patient is overlooked regarding diagnosis of developing periodontal disease or recurring disease.
- Select appropriate ADA Insurance Procedure Codes for reporting periodontal procedures in order to maximize the patient's benefit.
- Share insurance information with patients to assist them in reducing their dependence on dental insurance benefits, thereby enabling them to make independent health decisions related to treatment of periodontal diseases.

Disclosure

Dr. Sweeting, Ms. Davis, and Dr. Cobb are scientific advisors for OraPharma, Inc.

References

1. Hujoel PP, Bergström J, del Aguila MA, DeRouen TA. A hidden periodontitis epidemic during the 20th century? Community Dent Oral Epidemiol 2003;31:1-6.
2. Mendez D, Warner KE. Adult cigarette smoking prevalence: Declining as expected (not as desired). Am J Pub Health 2004;94:251-252.
3. Cobb CM, Carrara A, El-Annan E, et al. Periodontal referral patterns, 1980 versus 2000: A preliminary study. J Periodontol 2003:74:1470-1474.
4. Dockter KM, Williams KB, Bray KS, Cobb CM. Relationship between pre-referral periodontal care and periodontal status at time of referral. J Periodontol 2006:77:1708-1716.
5. Bader JD, Rozier G, McFall WT, Jr., Sams DH, Graves RC, Slome BA, Ramsey DL. Evaluating and influencing periodontal diagnostic and treatment behaviors in general practice. J Am Dent Assoc 1990;121:720-724.
6. Cury PR, Martins MT, Bonecker M, De Araujo NS. Incidence of periodontal diagnosis in private dental practice. Am J Dent 2006;19:163-165.
7. Heins PJ, Fuller WW, Fries SE. Periodontal probe use in general practice in Florida. J Am Dent Assoc 1989;119:147-150.
8. McFall WT, Jr., Bader JD, Rozier G, Ramsey D. Presence of periodontal data in patient records of general practitioners. J Periodontol 1988;59:445-449.
9. Brown LJ, Johns BA, Wall TP. The economics of periodontal diseases. Periodontol 2000. 2002;29:223-234.
10. Helminen SE, Vehkalahti M, Murtomaa H. Dentists' perception of their treatment practices versus documented evidence. Int Dent J 2002;52:71-74.
11. Blair, C. The economic impact of the under diagnosis of periodontal disease in general practice. Triage 2005;1:21-25.
12. American Dental Association, Survey Center. 1999 Survey of Dental Services Rendered. Chicago IL: American Dental Association; 1999.
13. Borrell LN, Burt BA, Taylor GW. Prevalence and trends in periodontitis in the USA: The NHANES, 1988 to 2000. J Dent Res 2005;84:924-930.
14. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. J Periodontol 1999;70:13-29.
15. Armitage GC. Periodontal diseases: Diagnosis. Ann Periodontol 1996;1:37-215.
16. Papapanou PN, Wennstrom JL, Sellen A, Hirooka H, Grondahl K, Johnsson T. Periodontal treatment needs assessed by the use of clinical and radiographic criteria. Community Dent Oral Epedimiol 1990;18:113-119.
17. American Academy of Periodontology. Parameter on comprehensive periodontal examination. J Periodontol 2000;71(Suppl.);847-848.
18. Krebs KA, Clem DS, III. American Academy of Periodontology. Guidelines for the management of patients with periodontal diseases. J Periodontol 2006;77:1607-1611.
19. Novak KF, Goodman SF, Takei HH. Determination of prognosis. In: Newman MG, Takei H, Klokkevold PR, Carranza FA, eds. Clinical Periodontology, 10th ed. Philadelphia: Saunders/Elsevier; 2006; pp. 614-625.

20. Carranza FA, Camargo PM. The periodontal pocket. In: Newman MG, Takei H, Klokkevold PR, Carranza FA, eds. Clinical Periodontology, 10th ed. Philadelphia: Saunders/Elsevier; 2006, pp. 434-451.
21. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4:1-6.
22. American Academy of Periodontology. Position paper: Diagnosis of periodontal diseases. J Periodontol 2003;74:1237-1247.
23. Carranza FA, Takei HH. Clinical diagnosis. In: Newman MG, Takei H, Klokkevold PR, Carranza FA, eds. Clinical Periodontology, 10th ed. Philadelphia: Saunders/ Elsevier; 2006, pp. 540-560.
24. Carranza FA, Takei HH. Bone loss and patterns of bone destruction. In: Newman MG, Takei H, Klokkevold PR, Carranza FA, eds. Clinical Periodontology, 10th ed. Philadelphia: Saunders/Elsevier; 2006, pp. 452-466.
25. Armitage GC. Clinical evaluation of periodontal diseases. Periodontol 2000 1995;7:39-53
26. Haffajee AD, Socransky SS, Lindhe J, Kent RL, Okamoto H, Yoneyama T. Clinical risk indicators for periodontal attachment loss. J Clinical Periodontol 1991;18:117-125.
27. Claffey N, Egelberg J. Clinical indicators of probing attachment loss following initial periodontal treatment in advanced periodontitis patients. J Clin Periodontol 1995;22: 690-696.
28. Lang NP, Adler R, Joss A, Nyman S. Absence of bleeding on probing – an indicator of periodontal stability. J Clin Periodontol 1990;17:714-721.
29. Muller HP, Stadermann S. Multivariate multilevel models for repeated measures in the study of smoking effects on the association between plaque and gingival bleeding. Clin Oral Invest 2006;10:311-316.
30. American Academy of Periodontology. Position paper. Guidelines for periodontal therapy. J Periodontol 2001;72:1624-1628.
31. American Dental Association. Principles of ethics and code of professional conduct. January 2005. Available at: http://www.ada.org/prof/prac/law/code/index.asp. Accessed August 28, 2008.
32. American Academy of Periodontology. Position paper. Periodontal maintenance. J Periodontol 2003;74:1395-1401
33. Greenwell H, Bissada NB, Wittwer JW. Periodontics in general practice: Perspectives on periodontal diagnosis. J Am Dent Assoc 1989:119:537-541.
34. Hirschfeld L, Wasserman B. A long-term survey of tooth loss in 600 treated periodontal patients. J Periodontol 1978;49:225-237.
35. Tonetti MS, Muller-Campanile V, Lang NP. Changes in the prevalence of residual pockets and tooth loss in treated periodontal patients during a supportive maintenance care program. J Clin Periodontol 1998;25:1008-1016.
36. American Academy of Periodontology. Parameters of care. J Periodontol 2000;71: 847-880.
37. American Dental Association. Current Dental Terminology. 2007-2008;3-27.
38. American Academy of Periodontology. Position paper. Epidemiology of periodontal diseases. J Periodontol 2005;76:1406-1419.
39. American Academy of Periodontology. American Academy of Pediatric Dentistry. Guideline for periodontal therapy. Pediatr Dent 2005-2006;27(7 Reference Manual):197-201.

Table 2. Periodontal Diagnostic Guidelines.

Case Indicator	Healthy	Gingivitis	Slight Periodontitis	Moderate Periodontitis	Advanced Periodontitis	Aggressive/Refractory
Pocket Depth[a]	≤ 3 mm	≤ 4 mm	4 - 5 mm	5 -6 mm	≥ 6mm	≥ 6mm
Bleeding Upon Probing	No	Yes[b]	Yes[b]	Yes[b]	Yes[b]	Yes[b]
Six-Point Probing	Yes	Yes	Yes	Yes	Yes	Yes
Bone Loss	None	None	≤ 10%	≤ 33%	≥ 33%	≥ 33%
Tooth Mobility[c]	None	None	None	≤ Grade II	≤ Grade III	≤ Grade III
Furcation[d]	None	None	≤ Grade I	≤ Grade II	≤ Grade III/IV	≤ Grade III/IV
Clinical Attachment Loss (CAL)[e]	None	None	1 - 2 mm CAL	3 - 4 mm CAL	≥ 5 mm CAL	≥ 5 mm CAL
Other	No inflammation	Only gingival tissues affected by the inflammatory process • No alveolar bone loss • Localized or generalized	Signs of inflammation may be present, including • Edema • Redness • Suppuration • Alveolar bone level is 3 - 4 mm from CEJ • Radiographic bone loss present • Localized or generalized	Signs of inflammation may be present, including • Edema • Redness • Suppuration • Alveolar bone level is 4 - 6 mm from CEJ • Radiographic bone loss present • Localized or generalized	Signs of inflammation may be present, including • Edema • Redness Suppuration • Alveolar bone level is ≥ 6 mm from CEJ • Radiographic bone loss present • Localized or generalized	Signs of inflammation may be present, including • Edema • Redness • Suppuration • Same clinical signs as advanced but includes adolescents or young adults • Localized or generalized • Rapid cycles of disease progression
Assessment	• Prophy • OHI	• Prophy • OHI	• Comp. Oral Eval D0150 • Comp. Perio Eval D0180 • Four bitewings D0274 • Eight bitewings D0277 • FMX D0210 • Panoramic Film D0330	• Comp. Oral Eval D0150 • Comp. Perio Eval D0180 • Four bitewings D0274 • Eight bitewings D0277 • FMX D0210 • Panoramic Film D0330 • Full Mouth Debride D4355 • Occlusal Analysis D9950	• Comp. Oral Eval D0150 • Comp. Perio Eval D0180 • Four bitewings D0274 • Eight bitewings D0277 • FMX D0210 • Panoramic Film D0330 • Full Mouth Debride D4355 • Occlusal Analysis D9950 • Specialty Referral	• Comp. Oral Eval D0150 • Comp. Perio Eval D0180 • Four bitewings D0274 • Eight bitewings D0277 • FMX D0210 • Panoramic Film D0330 • Full Mouth Debride D4355 • Occlusal Analysis D9950 • Specialty Referral
Active Therapy	• Prophy • OHI	• Prophy • OHI	• Quadrant SRP D4341 - UR, UL, LR, LL • Localized SRP D4342 - UR, UL, LR, LL • Locally Administered Antimicrobials D4381 • OHI D1330 • Specialty Referral • Other Treatments	• Quadrant SRP D4341 - UR, UL, LR, LL • Localized SRP D4342 - UR, UL, LR, LL • Locally Administered Antimicrobials D4381 • OHI D1330 • Specialty Referral • Other Treatments	• Quadrant SRP D4341 - UR, UL, LR, LL • Localized SRP D4342 - UR, UL, LR, LL • Locally Administered Antimicrobials D4381 • OHI D1330 • Specialty Referral • Other Treatments	• Specialty Referral
Ongoing Maintenance	<u>6 Months</u> • Prophy • OHI	<u>6 Months</u> • Prophy • OHI	• Perio Maintenance D4910 - 3/4/6 months • OHI D1330 • Locally Administered Antimicrobials D4381 • Localized SRP D4342 - UR, UL, LR, LL • Other Treatments	• Perio Maintenance D4910 - 3/4/6 months • OHI D1330 • Locally Administered Antimicrobials D4381 • Localized SRP D4342 - UR, UL, LR, LL • Other Treatments	• Perio Maintenance D4910 - 3/4/6 months • OHI D1330 • Locally Administered Antimicrobials D4381 • Localized SRP D4342 - UR, UL, LR, LL • Other Treatments	• Perio Maintenance D4910 - 3/4/6 months • OHI D1330 • Locally Administered Antimicrobials D4381 • Localized SRP D4342 - UR, UL, LR, LL • Host Modulation

[a] Excluding gingival overgrowth and recession

[b] Bleeding upon probing may not be present in individuals with periodontal disease who are smokers.

[c] **Tooth Mobility**: *Grade I:* Slightly more than normal; *Grade II:* Moderately more than normal; *Grade III:* Severe mobility faciolingually and mesiodistally, combined with vertical displacement. Adapted from Newman MG, Takei H, Klokkevold PR, Carranza FA. *Carranza's Clinical Periodontology* 10th ed. Philadelphia, PA: Elsevier; 2006.

[d] **Furcation Grades**: *Grade I:* Initial attachment loss with most of the bone still intact in the furcation. No radiographic changes seen; *Grade II:* The bone defect is definite horizontal bone loss that does not extend all the way through. Vertical bone loss may also be present. There is an opening into the furca with a bony wall at the deepest portion. *Grade III:* Bone is lost across the whole width of the furcation so no bone is attached to the furcation roof; *Grade IV:* Bone loss across the furcation, accompanied with gingival recession at the furcation, is clinically visible. Adapted from Newman MG, Takei H, Klokkevold PR, Carranza FA. *Carranza's Clinical Periodontology* 10th ed. Philadelphia, PA: Elsevier; 2006.

[e] Adapted from Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999; 4(l):1-6

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