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Prevention of Periodontal Diseases

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Prevention of periodontal diseases, including gingivitis and periodontitis, has been defined as a multistage process with primary, secondary, and tertiary components.¹ Primary prevention involves preventing inception of disease and includes the concept of health promotion and protection strategies. These health promotion strategies, aimed at enabling groups or individuals to control and improve their health, include providing oral hygiene education and protection strategies such as fluoridation. In developed nations, dentistry has been successful in these primary prevention areas. This success is illustrated by improvements in attitudes toward the importance of oral hygiene and the provision of fluoridated water supplies.^{2,3} Secondary disease prevention aims to limit the impact of disease by way of early diagnosis and treatment, thereby stopping disease progression in its earliest stages. The concept of tertiary disease

prevention is focused on the rehabilitation of the functional limitations that arise due to the disabilities encountered after advanced disease and includes such things as implants and prosthetic restoration of missing teeth.

Because the ultimate goal is to maintain the dentition over a lifetime in a state of health, comfort, and function in an esthetically pleasing presentation, this article focuses on the first two stages of periodontal disease prevention as they relate to gingivitis and periodontitis. Because these diseases are biofilm mediated and oral hygiene is important in all stages of prevention, certain concepts discussed here can and should be applied to all three phases of disease prevention. This article discusses risk assessment, mechanical plaque control, chemical plaque control, current clinical recommendations for optimal prevention, and future preventive strategies.

Risk assessment as an aid in prevention

The idea of applying risk assessment information to supportive [periodontal](#) care after thorough surgical or nonsurgical periodontal therapy is not new or untested. Axelsson and coworkers^{4,5,6} demonstrated that this approach essentially eliminates the recurrence of [caries](#) and [periodontal disease](#). Until recently, however, there has not been any validated and generally accepted tool for risk assessment. Through the 1960s, the concept of varying susceptibility to periodontitis was not widely accepted because early epidemiologic studies suggested that the disease was essentially pandemic in adults.^{7,8} Subsequent experimental work on animals demonstrated variability in disease expression,⁹ and more precise human population studies showed substantially lower prevalence rates for moderate to advanced periodontitis.¹⁰ These findings strongly suggested that differing susceptibilities for periodontal disease existed within a population. Much work has been done since that time to define local, environmental, and genetic factors that place individuals at a higher risk for developing periodontitis.^{11,12,13,14}

A risk factor is defined as some aspect of personal behavior or lifestyle, an environmental exposure, or an inherited characteristic that based on epidemiologic evidence is known to be hazardous to one's health and well-being.¹⁵ The presence of a risk factor increases the probability of developing the disease and represents a possible target for prevention or therapy. Although it is beyond the scope of this article to review the evidence that has accumulated over the last 2 decades regarding identification of risk factors and their [strength of association](#) with periodontitis, there are several factors that have repeatedly been documented. Examples of such risk factors include [cigarette smoking](#), uncontrolled [diabetes](#), irregular dental care, and [plaque](#) in the presence of attachment loss. This article focuses on recent research efforts to quantify the risk assessment because this represents a major step forward in the attempt to tailor preventive strategies for individuals.

As dentistry and periodontics begin to transition from a repair model of [oral health](#) care to a wellness model, the need to be able to predictably quantify levels of risk is crucial. In the last

World Workshop of Periodontics, the consensus report on prevention stated that the incorporation of risk assessment models for prevention of periodontal disease is an important goal. The concept of continuous multilevel risk assessment has developed and has been promoted as an important, if not [essential, factor](#) in proper patient assessment as part of treatment planning and reassessment during maintenance.¹⁶ One of the consistent therapeutic goals in the American Academy of Periodontology's Parameters of Care is to reduce or eliminate contributing risk factors for gingivitis and periodontitis.¹⁷ Moreover, the latest American Academy of Periodontology position paper on periodontal maintenance recommends that dental professionals should counsel people on the control of risk factors.¹⁸ The actual assessment and application of risk levels in prevention and management of periodontitis, however, is in its infancy. Although there are odds ratios and relative risk levels published for a variety of exposures as a crude means to assess the importance of each factor, there is currently little to no information on the interactions of these different risk factors. Clearly, not all of the relevant risk factors have been uncovered.

Recently, a few clinical risk assessment approaches or tools have been promoted in the literature. One tool is a relatively simple questionnaire¹⁹ that provides a vague but individual risk profile for the clinician and an educational tool for communication with the patient. A more sophisticated instrument employs a continuous multilevel risk assessment that incorporates the consideration of subject, tooth, and site risk evaluations.¹⁶ This approach generates functional diagrams that provide a more objective means of quantifying an individual's risk ([Fig. 1](#)).¹⁶ Depending on the area of the polygon, a patient may fall into low-, moderate-, or high-risk categories. The patient's individual periodontal risk assessment is low if all parameters fall within the low-risk categories or if only one category in the moderate-risk range ([Fig. 2](#)). A moderate periodontal risk assessment shows at least two parameters in the moderate category, with a maximum of one parameter in the high-risk area ([Fig. 3](#)). A patient with a high periodontal risk assessment has at least two parameters in the high-risk category ([Fig. 4](#)). Although this model still needs to be validated, it provides an objective quantification of risk into three categories. This assessment could provide justification for more or less aggressive preventive care.

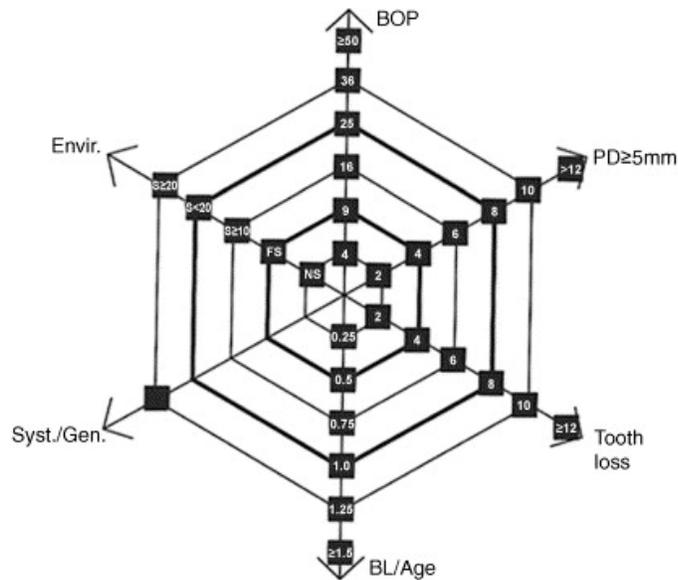


Fig. 1. Functional diagram to evaluate the patient's risk for recurrence of periodontitis. Each vector represents one risk factor or indicator with an area of relatively low risk, an area of moderate risk, and an area of high risk for disease progression. All factors have to be evaluated together; therefore, the area of relatively low risk is found within the center circle of the polygon, whereas the area of high risk is found outside the periphery of the second ring in bold. Between the two rings in bold, there is the area of moderate risk. BL, [bone loss](#); BOP, [bleeding on probing](#); Envir., environment; FS, frequent smoker; NS, nonsmoker; PD, pocket depth; S, smoker; Syst./Gen., systemic/general. (From Lang NP, Bragger U, Salvi G, et al. Supportive [periodontal](#) therapy (SPT). In: Lindhe J, Karring T, Lang NP, editors. Clinical periodontology and [implantology](#). 4th edition. Oxford (England): Blackwell Munksgaard; 2003. p. 788; with permission.)

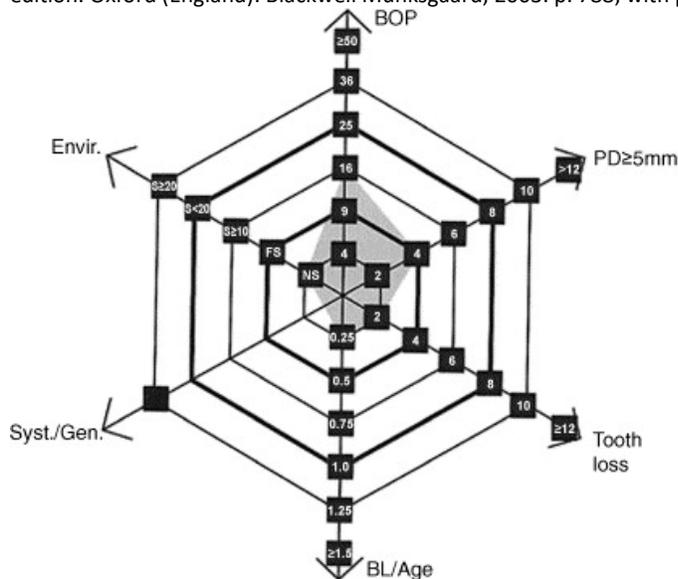


Fig. 2. Functional diagram of a low-risk maintenance patient. [Bleeding on probing](#) (BOP) is 15%, four residual pockets ≥ 5 mm are diagnosed, two teeth have been lost, the bone factor in relation to age is 0.25, no systemic factor is known, and the patient is a nonsmoker. BL, [bone loss](#); Envir., environment; FS, frequent smoker; NS, nonsmoker; PD, pocket depth; S, smoker; Syst./Gen., systemic/general. (From Lang NP, Bragger U, Salvi G, et al. Supportive [periodontal](#) therapy (SPT). In: Lindhe J, Karring T, Lang NP, editors. Clinical periodontology and [implantology](#). 4th edition. Oxford (England): Blackwell Munksgaard; 2003. p. 788; with permission.)

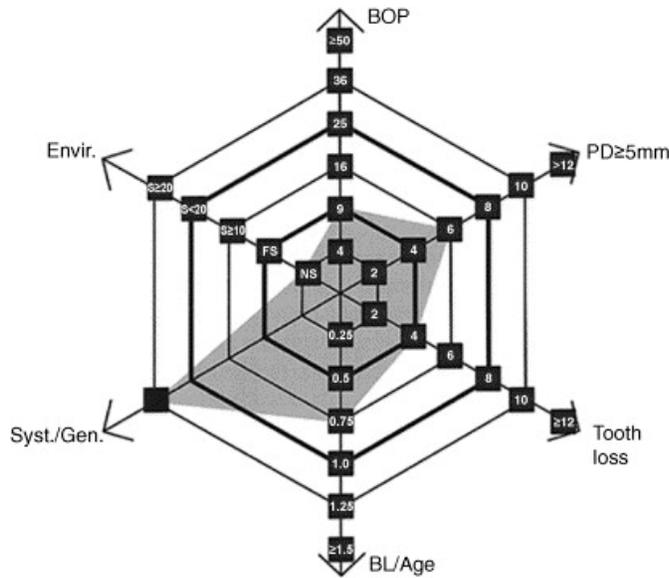


Fig. 3. Functional diagram of a medium-risk maintenance patient. [Bleeding on probing](#) (BOP) is 9%, six residual pockets ≥ 5 mm are diagnosed, four teeth have been lost, the bone factor in relation to age is 0.75, the patient has type I [diabetes mellitus](#), and the patient is a nonsmoker. BL, [bone loss](#); Envir., environment; FS, frequent smoker; NS, nonsmoker; PD, pocket depth; S, smoker; Syst./Gen., systemic/general. (From Lang NP, Bragger U, Salvi G, et al. Supportive [periodontal](#) therapy (SPT). In: Lindhe J, Karring T, Lang NP, editors. Clinical periodontology and [implantology](#). 4th edition. Oxford (England): Blackwell Munksgaard; 2003. p. 799; with permission.)

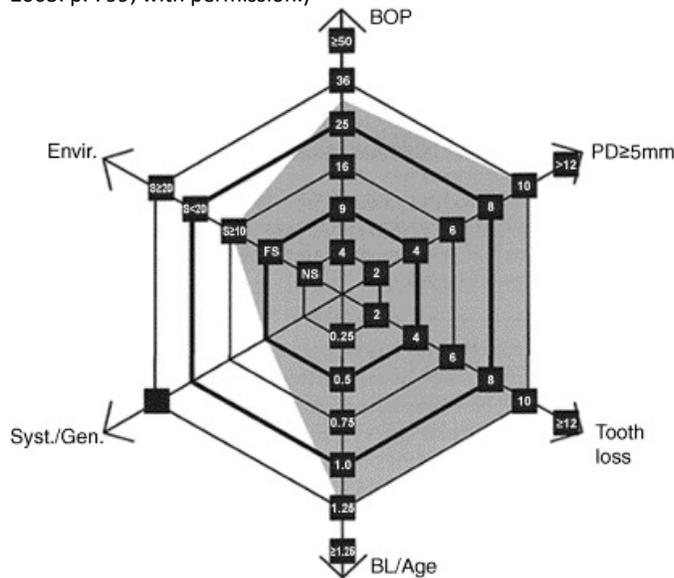


Fig. 4. Functional diagram of a high-risk maintenance patient. [Bleeding on probing](#) (BOP) is 32%, 10 residual pockets ≥ 5 mm are diagnosed, 10 teeth have been lost, the bone factor in relation to age is 1.25, no systemic factor is known, and the patient is an occasional smoker. BL, [bone loss](#); Envir., environment; FS, frequent smoker; NS, nonsmoker; PD, pocket depth; S, smoker; Syst./Gen., systemic/general. (From Lang NP, Bragger U, Salvi G, et al. Supportive [periodontal](#) therapy (SPT). In: Lindhe J, Karring T, Lang NP, editors. Clinical periodontology and [implantology](#). 4th edition. Oxford (England): Blackwell Munksgaard; 2003. p. 799; with permission.)

Using a different multifactorial model, Page and coworkers²² developed the “Periodontal Risk Calculator” (PRC). The PRC incorporates mathematic algorithms that are based on nine known

risk factors: age, smoking history, diabetes diagnosis, history of [periodontal surgery](#), pocket depth, [furcation involvements](#), restorations or [calculus](#) below the [gingival margin](#), [radiographic](#) bone height, and vertical bone lesions. This Web-based instrument can be accessed at <http://www.previser.com>, and to the authors' knowledge, provides the first validated tool to objectively quantify risk. Because risk assessments based on subjective expert opinion have been shown to vary too much to be useful in everyday [clinical decision making](#) [20], the development of the PRC may well represent a major step forward in the transition from the surgical/repair model of dentistry to the wellness or medical model of patient care. Data suggest that expert clinicians in the United States and Europe appear to base most of their risk assessment on severity of disease at presentation rather than on factors such as smoking, [diabetic](#) status, and poor oral hygiene.²⁰ Moreover, it has been observed that “risk scores generated for individual patients by subjective expert clinician opinion are highly variable and could result in the misapplication of treatment for some patients”.²¹ Overtreatment or undertreatment of periodontal diseases may be the result of subjectively forming such opinions.

Using a clinical patient data set from the VA Dental Longitudinal Study of [Oral Health and Disease](#), Page and coworkers^{22,23} retrospectively assigned risk scores to each of the 523 subjects on a 1 (lowest) to 5 (highest) risk scale using information from the baseline examination. They subsequently assessed radiographic [bone loss](#) and tooth loss over the next 15 years. The mean percentage of bone loss followed a consistent pattern from least bone loss in the low-risk category to greatest bone loss in the highest risk category (2<3<4<5). Several measures of tooth loss over time showed a similar relationship. Clearly, this approach to validation had weaknesses that included the retrospective nature of the study, the lack of enough low-risk subjects (periodontal risk assessment = 1) to allow for the full range of risk levels to be validated, and the lack of data on why teeth were lost. In addition, for teeth without periodontal disease at baseline, the PRC did not appear to discriminate between risk levels. This study, however, demonstrated the ability of the PRC to “predict” with accuracy and validity periodontal deterioration and even tooth loss for teeth with periodontal disease at baseline.

In addition to providing a quick and objective risk assessment, the PRC provides a [periodontal diagnosis](#), a quantitative disease score from 1 to 100, and treatment options based on the current American Academy of Periodontology standards of care. If this technology catches on, it may have a substantial impact on the dental profession. The PRC would provide a useful tool for individual patient assessment and may help sort out cost-effective treatments and even suggest rational approaches to preventive measures. Accurate and objective risk prediction should provide dentistry the ability to make serious strides in moving toward a wellness model of patient care. It has the potential to substantially improve the allocation of health care dollars by reducing the overtreatment of low-risk patients and applying more aggressive preventive strategies to high-risk patients.

Mechanical plaque control

Successful [primary or secondary prevention](#) is based on two major factors, the first being proper, thorough treatment during active therapy and the second being patient compliance with daily [plaque](#) removal and regular professional [supportive care](#). Because other articles in this issue of the *Dental Clinics of North America* deal with active surgical and nonsurgical treatments to eliminate disease, the current authors confine themselves to addressing current concepts in primary prevention of gingivitis and periodontitis and the personal and professional supportive methods to avoid recurrence after active therapy is finished. The authors' premise is that proper and thorough active treatment means leaving the patient with a maintainable [periodontium](#), which can be accomplished with nonsurgical therapy in the disease control phase of treatment or after the surgical corrective phase.

There is overwhelming evidence for the direct cause-and-effect relationship between the formation and accumulation of supragingival plaque and the development of gingivitis. It is also generally accepted that the loss of attachment and [alveolar bone](#) that defines periodontitis is preceded by [gingival](#) inflammation and subgingival plaque maturation.

It has been estimated that in a periodontitis patient with a full complement of teeth (28 teeth/168 sites) only 6% or less of these sites exhibit tissue destruction at any given time.²⁴ Although only a small percentage of gingivitis sites may progress to periodontitis, it still cannot be discerned which inflamed sites are actively breaking down. Traditionally, the default approach has been to try to eliminate all plaque-induced inflammation, particularly in subjects who have shown a susceptibility to a destructive [periodontal](#) inflammation. The first line of defense against plaque-induced gingivitis has always been daily meticulous mechanical plaque removal supplemented by periodic professional mechanical [tooth cleaning](#).

Toothbrushing: manual versus powered

Toothbrushing has played the major role in daily plaque control in developed nations for over 60 years. According to the Lemelson-MIT Invention Index at the Massachusetts Institute of Technology, when Americans were asked to list the five inventions that they believed they could not live without, the toothbrush was more appreciated than the car, the personal computer, the cell phone, and the [microwave](#) oven.^{24a}

Many novel manual toothbrush designs and methods of brushing have been described in the literature, with no one design or method showing a clear superiority. There is no consensus on the optimal frequency of toothbrushing, but it is clear that the average person is not very efficient or thorough in daily plaque removal.²⁵ Hawkins et al.²⁶ and Beals et al.²⁷ showed that most subjects spend less than a minute brushing their teeth with a manual toothbrush. It therefore seems reasonable for clinicians to ask whether it is time to start recommending powered brushes with automatic timers to moderate- and high-risk patients.

When Van der Weijden and coworkers^{28,29} performed professional plaque removal on subjects for varying duration using a manual or an oscillating/rotating powered brush, they

demonstrated that manual brushing, even after 6 minutes, only removed 75% of the plaque that the powered brush removed in 1 minute. Although one can argue the applicability of these particular data to direct patient use, there are additional studies that suggest that the electric toothbrush may prove advantageous for some patients. Several long-term (6 months or more) randomized [controlled clinical trials](#) have shown clinical benefits for certain powered brush designs compared with a manual brush in different populations with different levels of oral hygiene instruction.[30,31,32,33,34,35](#) The differences between manual and powered brushing are more evident when oral hygiene instruction is provided and reinforced during these long-term trials.

In perhaps the most comprehensive 6-month comparative clinical trial between a manual and a powered brush (oscillating/rotating type), Haffajee and coworkers[36,37](#) showed significantly better reduction in gingival index and attachment levels for the powered brush group, with mandibular and lingual surfaces showing the most benefit. They further demonstrated in both groups that a high level of personal supragingival plaque control had an unexpected and substantial beneficial effect on the subgingival flora. The decreased prevalence of [periodontal pathogens](#) from subgingival and supragingival plaque should lower the risk of [periodontal disease](#) initiation and recurrence. They further speculated that the lack of a significant microbiologic difference between the manual and the powered brush groups might be because the areas of greatest differences in clinical parameters (lingual and mandibular) were not the same areas that were sampled microbiologically (mesial-buccal site of each tooth).

Recently, two systematic reviews were published that addressed the comparative efficacy and safety of manual versus powered toothbrushing. Sicilia and coworkers[38](#) concluded that the counter-rotational and the oscillating/rotating brushes can be more beneficial than a manual toothbrush in reducing the levels of gingival bleeding. A Cochrane Review noted that no powered toothbrush designs except for the oscillating/rotating action were consistently superior to manual brushing. It was found that the oscillating/rotating brush achieves a modest reduction in plaque and gingivitis compared with manual toothbrushing and that it is safe to use.[39](#) The long-term implications of these findings are unknown.

There are limited data regarding the compliance of powered toothbrush use in periodontitis patients. Stalnacke and coworkers[40](#) found a 62% rate of daily compliance by maintenance patients at least 3 years after obtaining a powered brush. Only 3% had stopped using the powered brush altogether. In a study that specifically assessed low-compliance periodontal maintenance patients, another group showed an improved level of plaque control over 12 to 36 months in subjects using a powered brush.[41](#) Additional long-term studies would be useful.

More recently, McCracken et al.[42](#) recruited a small population of untreated periodontitis patients into a short-term powered toothbrush compliance study. Using a microelectronic device within each brush to record duration and pressure, the investigators gained further insight into powered toothbrush home use. Subjects were informed that the device would measure toothbrush performance only. No details were given as to what this meant in terms of

their compliance. McCracken et al.⁴² found that over 2 months of use, only about one third of these patients were truly compliant in using the brush for 2 minutes twice a day, nearly one third of the subjects overused the brush, and approximately one third substantially underused the brush. These data suggest that most but not all periodontitis patients using a powered brush are compliant. Patient education and motivation, as expected, play the greatest role in personal mechanical plaque control.

The plaque removal efficacy of the powered brush appears to come primarily from more efficient plaque removal on buccal and lingual approximal surfaces, with the problem of interproximal plaque removal still unresolved.⁴³ Clearly, although powered brushes are more efficient at plaque removal, maximum benefit can only be achieved by proper instruction and patient motivation.

Interdental cleaning

Based on current knowledge, it seems reasonable to conclude that some form of regular interdental cleaning is necessary to maintain periodontal health because no toothbrush effectively disrupts true interproximal plaque, particularly in the posterior dentition. In addition, there is a clear interdental site predilection for gingivitis, periodontitis, and [smooth-surface caries](#). Interdental areas truly are the key high-risk surfaces.⁴⁴

There are many different types of interdental cleaning aids, but the most widely recommended is [dental floss](#) or tape. Floss is most useful in the nonperiodontitis patient who has full [interdental papillae](#) and no exposed concave root surfaces. When used properly, it can penetrate the interproximal sulcus approximately 2 to 3 mm. Axelsson et al.⁴⁵ recommended that flat, fluoridated dental tape be used before brushing with a fluoridated toothpaste in children and young adults. It is unfortunate that traditional manual flossing is not an easy skill for patients to learn; it has been reported that only 20% of subjects report effective flossing behavior.⁴⁶ Even in those who have been adequately trained and who can demonstrate skill retention after 1 year, there is still a substantial drop-off in daily plaque removal with time.⁴⁷ Flossing forks, superfloss and, more recently, powered flossers have been introduced to make it easier for patients to clean interproximally. The studies that have been done on these interdental devices do not have enough statistical power to show anything but equivalence.

Interproximal brushes are better alternatives for periodontitis maintenance patients who have lost interdental papillae height and who are in a secondary preventive stage.⁴⁵ Interdental brushes come in a range of sizes. It seems reasonable to assume that the largest size that fits the space being deplaqueed would be the most efficacious; however, this has not been proved.⁴⁸ Considering the range of sizes needed for any given patient, it may be best to choose the size that can be used in all high-risk sites. Advantages of this type of brush include the ability to deliver topical [antimicrobials](#) or [fluorides](#) to interproximal sites while mechanically removing plaque and to provide positive stimuli to the [fibroblasts](#) in the col area. This latter benefit is speculative and is an extrapolation from recent studies in a canine model.^{49,50,51}

Fluoridated triangular toothpicks have also proved to be an excellent way to deplaque the interproximal areas up to 2 to 3 mm subgingivally and have been recommended as part of a needs-related oral hygiene regimen for patients with treated periodontitis as part of a [secondary prevention](#) program.⁴⁵

Single-tufted brushes and rubber-tipped stimulators tend to be site specific. They are most often recommended for [furcations](#), tipped teeth, the distals of terminal molars, or tooth surfaces directly adjacent to [edentulous](#) sites.

Chemical plaque control

Mouthrinses

Chemical preventive agents have been incorporated into [oral disease](#) management for centuries; however, it is only recently that these [adjunctive therapies](#) have been scientifically studied. Recently, Addy⁵² wrote a brief, informative, and rather humorous review on antiseptic use in [periodontal](#) therapy. These agents should be viewed as adjuncts and not replacements for effective mechanical [plaque](#) control. They are preventive agents, not [therapeutic agents](#). Given that many patients cannot maintain adequate levels of plaque control using mechanical oral hygiene methods alone, chemical plaque control has become a big business. Despite some over-reaching advertisements about the efficacy of some of the currently available antiseptics for plaque control, the dental profession can be thankful for the widespread public education that is occurring on the dangers of interproximal plaque. This section emphasizes developments in the area of chemical plaque control since the last World Workshop in Periodontics.⁵³

The biofilm inhibitory concentrations for a given [antimicrobial](#) are generally orders of magnitude higher than the standard minimal inhibitory concentrations determined for planktonic (free-floating) organisms. A recent [in vitro](#) study compared the antimicrobial activity of three mouthrinses with planktonic bacteria and their corresponding monospecies biofilms.⁵⁴ As anticipated, the bacteria contained in biofilms were shown to have a decreased susceptibility to [antimicrobial agents](#) versus those in planktonic form. The antimicrobial mouthrinses included in the study were an essential-oil (EO)-containing mouthrinse (Listerine Antiseptic; Pfizer, Morris Plains, New Jersey), an amine fluoride/stannous fluoride-containing mouthrinse (Meridol), a [triclosan](#) and polyvinylmethyl ether/maleic acid copolymer-containing mouthrinse (Plax), and a negative control (phosphate-buffered saline). All three mouthrinses produced statistically significant (99.99%) reductions in planktonic strains compared with the control. Effects on the biofilm forms of the organisms, however, were more variable. Exposure to Listerine Antiseptic produced statistically significant reductions compared with the control, whereas Plax (Colgate Palmolive [UK] Ltd., Guildford, United Kingdom) and Meridol (Wybert GmbH, Lorrach, Germany) produced much smaller reductions that were not statistically significant. These in vitro results provide a clear demonstration of the resistance to antimicrobial agents conferred by biofilms. The results also provide additional support for employment of tests using biofilms to more accurately assess the relative activities of

antiplaque agents in vitro (although when tested [in vivo](#), there are even more obstacles to overcome).

The in vivo use of an EO-containing antiseptic mouthrinse has a long clinical history (Listerine). The active ingredient of this mouthrinse is a fixed combination of EOs (0.064% thymol, 0.092% eucalyptol, 0.060% methyl salicylate, and 0.042% menthol). Many short-term (4–6 weeks) and long-term (3–6 months) clinical studies have demonstrated the effectiveness of EO rinses in plaque and gingivitis reduction. Although the “gold standard” for antiplaque and antigingivitis mouthrinsing is still chlorhexidine,^{55,56} the use of an EO rinse as an adjunct to mechanical oral hygiene can be beneficial, particularly in the secondary preventive phase because it does not induce [calculus](#) formation, taste alteration, or extrinsic tooth stain like chlorhexidine.^{57,58,59}

In a 6-month supervised-use trial specifically designed to compare the antiplaque and antigingivitis efficacy of EO and chlorhexidine mouthrinses, Overholser et al.⁶⁰ showed significant reduction in plaque formation and in gingivitis compared with a negative control. Although both mouthrinses had comparable antigingivitis effectiveness, the chlorhexidine mouthrinse was significantly more effective than the EO-containing mouthrinse in reducing plaque. In addition, the chlorhexidine group had more extrinsic tooth stain and calculus than the EO or the control group.⁶⁰ Charles and coworkers⁶¹ recently presented similar results in a 6-month randomized controlled clinical study on the antiplaque and the antigingivitis effects of a chlorhexidine or EO rinse compared with control. Although the patients were randomly assigned to the groups, it is difficult to discern how the observers remained masked to the treatments, given the obvious presence of stain and calculus. The investigators also stated that there was a statistically significant difference between the chlorhexidine group and the EO group at 3 months, but toward the end of the study (6 months), there was no difference. The chlorhexidine rinse group had significantly higher calculus formation and extrinsic tooth stain compared with the EO rinse group or the control group.

Because the occurrence of extrinsic tooth stain and calculus deposits may limit patient compliance, especially for long-term use, chlorhexidine mouthrinses have greater utility when short-term plaque control is critical. This situation includes the postoperative phase following [periodontal surgery](#), when mechanical oral hygiene is difficult. The use of an EO rinse is better suited as a long-term plaque-control adjunct for moderate- to high-risk patients for [primary and secondary prevention](#). For patients with [physical disabilities](#) that limit their mechanical oral hygiene skills, a chlorhexidine spray may prove useful.⁵²

The literature presents few studies that compare the efficacy of toothbrushing with interproximal plaque control using [dental flossing](#) or interproximal dental brushing^{62,63,64} or antimicrobial mouthrinses.^{57,58} Sharma et al.⁵⁸ and Bauroth et al.⁵⁷ compared the efficacy of plaque removal using an EO-containing mouthrinse or dental flossing in addition to brushing. They included three groups in a parallel-arm, single-masked, randomized [controlled clinical trial](#). The first group received the EO mouthrinse twice a day as an adjunct to the patient's regular twice daily brushing with a [fluoride dentifrice](#), the second group was advised to floss

once daily in conjunction with brushing, and the third group received a control mouthrinse to supplement brushing. The results showed significant improvement for the EO rinse and the dental floss group compared with baseline measurements. Reduction in the interproximal modified [gingival](#) index scores was “at least as good as” that provided by flossing. Although these studies were well controlled and well designed, the measurement of interproximal plaque seems problematic because the flossing group showed significantly less reduction compared with other studies that investigated the additional interproximal plaque reduction benefit comparing brushing alone to brushing and flossing.^{64,65,66,67,68} In other words, the positive control (brushing and flossing) was not as positive as one might have expected; the reason for this is not clear in the study. Possible factors could be the length of this study compared with others. Patient compliance may have waned by the first 3-month visit, leading to poor flossing technique or a decrease in patient motivation. It should also be noted that methods for measuring true interproximal plaque are limited.

A follow-up study compared a combination of mechanical and chemical hygiene regimens. The experimental group was instructed to brush twice a day (B), floss daily (F), and rinse with the antiseptic twice daily (EO). This BFE0 group represented optimal mechanical and chemical plaque control. A second group used a control mouthrinse (C) in conjunction with brushing and flossing (BFC group). A final group was assigned to brush and to use the placebo mouthrinse as a negative control (BC). A significant reduction in mean modified gingival index and plaque index was seen for the experimental group (BFE0) compared with the other two groups. The investigators suggested that the combined mechanical disruption of the interproximal plaque followed by the EO rinse provides a synergistic effect in plaque and gingivitis reduction due to better biofilm penetration. There was little comment on the low incremental reduction for interproximal mean plaque index and modified gingival index from the BC and BFC groups or on the sharp drop from 3 to 6 months in the BFC group interproximal scores.

As promising as these data are, it would be even more helpful to carry out similar studies on moderate- to high-risk patients because it has been suggested that antiseptic rinsing is not cost-effective for the general population. Recommending widespread antiseptic rinsing for the general population is essentially vast overtreatment to reduce disease only in the subpopulation of susceptible patients.⁵²

Toothpaste

Although the use of chlorhexidine as a mouthrinse has demonstrated significant plaque reduction, the incorporation of this agent into a dentifrice has proved difficult due to the interaction between chlorhexidine and calcium ions or anionic detergents such as [sodium lauryl sulfate](#).^{69,70}

The use of antigingivitis dentifrices has been recommended for primary prevention and as a maintenance measure as part of [secondary prevention](#) in treated and maintained periodontal patients. The addition of triclosan (a broad-spectrum phenol-derived antibacterial agent) to

different dentifrice formulations has been studied in various short- and long-term trials. Volpe et al.⁷¹ reviewed the use of various combinations of triclosan in reducing plaque and gingivitis in susceptible individuals.

One of the available combinations of triclosan is composed of 0.3% triclosan, 2.0% copolymer, and 0.243% sodium fluoride (Colgate Total; Colgate Palmolive Co., New York, New York). This combination was shown to alter the quality and composition of subgingival plaque.^{72,73}

A significant 5-year study by Cullinan et al.⁷⁴ supported and expanded the previous findings. This double-blind controlled clinical trial examined the effect of the same triclosan formulation as the previous studies on the progression of [periodontal disease](#) in patients with attachment loss ≥ 2 mm. It was concluded that the use of such a dentifrice significantly slowed the progression of periodontal disease in those individuals with existing disease. The clinical effect of a triclosan-containing dentifrice indicates that after a [periodontal pocket](#) has developed in a susceptible individual, the use of such dentifrice slows the progression of further pocket development. The effectiveness of triclosan dentifrice as a primary preventive measure against periodontitis has not been demonstrated; however, it has been used to prevent gingivitis, which is the necessary precursor to periodontitis.

One of the side effects of a triclosan dentifrice is tooth staining. A similar problem has been noted with more recent stable formulations of a stannous fluoride-containing dentifrice with proven antiplaque effects.^{75,76}

Irrigation

The effect of supragingival oral irrigation in periodontal therapy is to flush away loosely attached bacteria present in the gingival crevice, thereby diminishing the potential for developing gingivitis. There is contradictory evidence about the usefulness of supragingival irrigation in reducing plaque formation. Although a few studies have shown some benefits of oral irrigation (professionally applied and as a part of a home care program), the benefits have been unimpressive.⁷⁷ Recently, an improvement was demonstrated using this therapy at the clinical and subclinical level in a short-term daily home use study in periodontal patients. These results suggest that oral irrigation with water as part of a regular oral hygiene program can decrease the concentration of pro-inflammatory [cytokines](#) in gingival fluids.⁷⁸ Although these are promising findings, more work needs to be done before a strong recommendation can be made to add such a regimen to daily home care for these patients. One must weigh the benefit against the cost of the therapy. There is also the possibility of poor long-term compliance.

Clinical considerations for optimal prevention

For patients having resistance to periodontitis and who would thus be classified as low risk based on currently available risk prediction instruments, it is not clear how much mechanical and chemical preventive therapy represents overtreatment. Likewise, for [primary or secondary prevention](#) in moderate- to high-risk “susceptible” patients, there is very little understanding of

what may constitute the minimal effective therapy. The best available evidence compels us to continue to try to achieve “complete” [plaque](#) control by whatever means are best suited for the patient. For a toothbrushing population such as in the United States, this means there is more need for education and motivation in needs-related personal and professional [gingival](#) plaque removal (supra- and subgingivally), with an emphasis on interproximal plaque removal in high-risk sites. Axelsson and colleagues^{44,45} suggest that encouraging patients to perform interproximal plaque control on the posterior “key risk teeth” before toothbrushing may be adequate in achieving prevention because the behavior is linked with an already well-established brushing habit. In high-risk patients in a [secondary prevention](#) program, the authors believe it is wise to consider a powered toothbrush for home use. When there are no allergies or other [contraindications](#), a personal chemical plaque control regimen with a triclosan-containing toothpaste and perhaps an EO-containing mouthrinse twice daily would provide a cost-effective, evidence-based approach to prevention. Other adjunctive home care approaches for the high-risk patient include a dilute sodium [hypochlorite](#) solution (1 teaspoon household bleach/250 mL tap water) for subgingival irrigation as recommended by Slots et al.⁷⁹ As an adjunct to the professional supportive [periodontal](#) therapy, the use of a 1:9 ratio of povidone-iodine to water in the ultrasonic unit has also proved useful, particularly for advanced [chronic or aggressive periodontitis](#) cases.⁷⁹ When implants are present, plastic-tipped ultrasonics are preferred and metal instruments must be avoided.

Continuous multilevel risk assessment and management is now the standard of care. Clinicians must counsel patients on their risk profile and help them take decisive steps to reduce risk at each level.¹⁶ Perhaps the one area that could have the greatest overall public health benefit immediately is smoking cessation counseling.

Every dentist should be well familiar with the five “A’s” of smoking cessation ([Box 1](#)) and the five first-line pharmacotherapies approved by the Food and Drug Administration for smoking cessation ([Table 1](#)). Clinicians must be willing to guide patients through this process. There is a new current [dental terminology](#) code (D1320) that is available for reimbursement, and each year more insurance plans are reimbursing for this service. Because the prevalence for [adolescent smoking](#) has risen dramatically since 1990, clinicians need to be proactive in discouraging adolescent and [preadolescent](#) patients from starting smoking. It is estimated that over 3000 additional teens and preteens become regular tobacco users each day.⁸⁰ It certainly makes sense for dental professionals to take a leading role in combating this chronic addiction because they see these patients more frequently than physicians.

Box 1

The five “A’s” for brief intervention

1. Ask about tobacco use. Identify and document tobacco use status for every patient at every visit.

2. Advise to quit. In a clear, strong, and personalized manner, urge every tobacco user to quit.
3. Assess willingness to make a quit attempt. Is the tobacco user willing to make a quit attempt at this time?
4. Assist in quit attempt. For the patient willing to make a quit attempt, use counseling and pharmacotherapy to help him or her quit.
5. Arrange follow-up. Schedule follow-up contact, preferably within the first week after the quit date.

Table 1. Suggestions for the clinical use of first-line pharmacotherapies approved for smoking cessation by the Food and Drug Administration

Pharmacotherapy	Precautions/contraindications	Side effects	Dosage	Duration	Availability	Cost/d ^a
Bupropion SR	History of seizure or history of eating disorder	Insomnia, dry mouth	Begin treatment 1–2 wk prequit 150 mg 1×/morning for 3 d then 150 mg 2×/d	7–12 wk (up to 6 mo)	Zyban (prescription only)	\$3.33
Nicotine gum	—	Mouth soreness, dyspepsia	1–24 cigs/d: 2 mg gum (≤24 pcs/d) 25+ cigs/d: 4 mg gum (≤24 pcs/d)	≤12 wk	Nicorette/Nicorette Mint (OTC only)	\$6.25 for 10 (2-mg) pieces, \$6.87 for 10 (4-mg) pieces

Nicotine inhaler	—	Local irritation of mouth and throat	6–16 cartridges/d	≤6 mo	Nicotrol Inhaler (prescription only)	\$10.94 for 10 cartridges
Nicotine nasal spray	—	Nasal irritation	8–40 doses/d	3–6 mo	Nicotrol NS (prescription only)	\$5.40 for 12 doses
Nicotine patch	—	Local skin reaction, insomnia	21 mg/24 h then 14 mg/24 h then 7 mg/24 h	4 wk then 2 wk then 2 wk	Nicoderm CQ (OTC only) and generic patches (prescription and OTC)	Brand name: \$4.00–\$4.50 ^b
			15 mg/16 h	8 wk	Nicotrol (OTC only)	

Abbreviations: cigs, cigarettes; OTC, over the counter; pcs, pieces.

Information contained within this table is not comprehensive. Please see package insert for additional information. aCosts are based on retail prices of medication purchased in April 2000 at a national chain pharmacy located in Madison, Wisconsin.

bGeneric brands of the patch recently became available and may be less expensive.

Finally, dental care providers must assess the patient's disease status each time he or she returns for supportive periodontal therapy. Clinicians must use the data available from the medical history review, risk assessment, and clinical examination, including [radiographs](#) when taken. As part of the supportive periodontal therapy rotations at Marquette University School of Dentistry, the students are given a laminated card that lists some specific criteria they should employ to assess the stability or instability of their maintenance patients. These criteria are listed in [Box 2](#). Application of objective criteria such as these in the general practitioner's office may provide a more rational approach to appropriate referrals to the periodontist. Based on a recent retrospective survey of changes in referral patterns to periodontal offices over the past 20 years, it seems that some guidelines are needed if the goal of helping the public to keep their teeth in a state of health, comfort, and function in an esthetically pleasing presentation⁸¹ is to be reached.

Box 2

Site- and patient-level criteria to consider in assessing the stability of periodontal maintenance patients

Inflammation as measured by bleeding on probing

Full mouth bleeding on probing >15% suggests instability

Sites that consistently show bleeding on probing over time may be unstable

Sites that consistently show no bleeding on probing are likely stable

Probing depth measurements

Sites with a probing depth increase of ≥ 2 mm from baseline or previous visit are considered unstable

Number of significant periodontal pocket depths (10 or more sites with ≥ 4 mm probing depth are considered unstable)

Probing depth ≥ 6 mm at any site is considered unstable

Progressive gingival recession from baseline or previous charting

Radiographic considerations

Loss of crestal bone height based on vertical bite wings is considered unstable

Consistent presence of crestal lamina dura suggests stability

Patient-level considerations

Poor hygiene in the presence of attachment loss plaque index >30% is considered unstable

Smoking more than one-half pack per day is considered unstable

Diabetes mellitus with HBA1c ≥ 9 is considered unstable

High-stress events, divorce, loss of a loved one, and unemployment are considered unstable

Future preventive strategies

[Vaccine development](#) for periodontitis is a possible protection strategy for [primary prevention](#). Different groups have been developing vaccines that target important surface structures of certain periodontopathogens. These surface structures include the fimbrial protein and hemagglutinating subunits necessary for adhesion and colonization of *Porphyromonas gingivalis* and the surface [cysteine proteinases](#) Arg- and Lys-gingipains that allow *P. gingivalis* to bind to and degrade host tissues, causing destruction by way of a [prostaglandin](#) E₂-dependent mechanism.^{82,83,84,85}

New drug delivery systems are also in development that should provide more cost-effective, slow-release [antimicrobials](#) and anti-inflammatory agents for primary prevention in high-risk subjects and [secondary prevention](#) in more moderate- to low-risk cases. The possibility of using these systems to deliver antimicrobial, anti-inflammatory, or immune-modulating agents is currently being tested.^{86,87}

Another somewhat futuristic approach to primary or secondary prevention involves introducing nonpathogenic bacterial competitor strains (bacterial replacement therapy) as a means to prevent the colonization and establishment of pathogenic strains of bacteria. To date, most work done in this area has been geared toward [dental caries](#) rather than [periodontal disease](#); however, progress has been made in construction of a nonpathogenic [Streptococcus mutans](#) strain that can colonize the [oral cavity](#) but has no ability to produce acid due to a [lactate dehydrogenase](#) deletion mutation.^{88,89,90} These are just a few of the biologic therapies making their way into clinics. Considering how far we have come in 50 years, it is truly an exciting time to be a dentist.

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