Point of Care

The "Point of Care" section answers everyday clinical questions by providing practical information that aims to be useful at the point of patient care. The responses reflect the opinions of the contributors and do not purport to set forth standards of care or clinical practice guidelines. Readers are encouraged to do more reading on the topics covered. This month's questions were answered by members of the Canadian Academy of Periodontology.

CANADIAN ACADEMY OF PERIODONTOLOGY

QUESTION 1

What is the gold standard for treatment of periodontal disease?

Background to the Issue

Plaque-induced periodontal diseases are mixed infections associated with relatively specific groups of indigenous oral bacteria. Host responses to these periodontal pathogens are highly variable and determine the person's susceptibility to periodontal disease. Although our understanding of the causes and pathogenesis of periodontal infections is increasing, diagnosis and classification are still based on clinical assessment. Factors such as the presence of inflammation, probing depth, attachment loss, the patient's medical and dental history, and other signs such as pain, ulceration and amount of plaque and calculus help the dentist to make the correct diagnosis.[1]

Plaque-induced periodontal diseases have traditionally been divided into 2 general categories on the basis of absence (gingivitis) or presence (periodontitis) of attachment loss. Gingivitis is inflammation of the gingiva without the loss of connective tissue attachment, whereas periodontitis involves gingival inflammation plus pathological detachment of collagen fibres from the cementum and apical migration of the junctional epithelium (i.e., attachment loss).

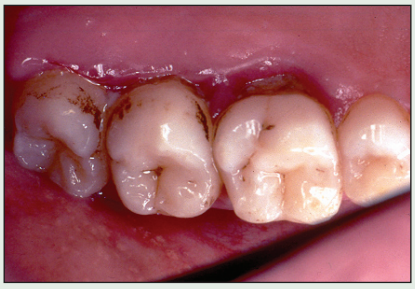

Figure 1a: Palatal view of molars in quadrant 2 before root planing.

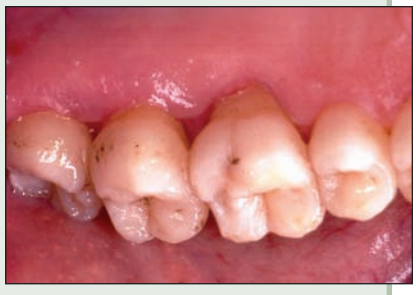

Figure 1b: Palatal view of molars in quadrant 2 approximately 6 weeks after root planing.

Management of the Issue

The ultimate goal of periodontal treatment is the preservation of the dentition for as long as possible, while maintaining patient health, function and comfort. A broad range of periodontal therapies are available, but no single approach can be used to treat every case. However, the use of an orderly and logical progression of treatment, from active periodontal therapy (nonsurgical and surgical modalities) to maintenance, is indispensable.

Initial periodontal therapy is intended to reduce or eliminate the major etiologic factors, namely plaque and calculus, as well as other contributing factors such as iatrogenic elements (e.g., amalgam overhangs), malpositioning of the teeth and potentially destructive habits (e.g., bruxism). Nonsurgical scaling and root planing is the most important component of initial therapy, since this procedure can result in reduction or elimination of bleeding on probing, edema, erythema and mobility (**Figs. 1** and **2**). Studies conducted in both research and private practice settings have shown the effectiveness of scaling and root planing for molar and nonmolar teeth and at shallow or moderately deep sites.[2] The primary caveat with nonsurgical therapy is that it may not be effective for some patients and/or at specific sites. This limitation becomes evident on re-evaluation, at which time appropriate alternative therapy — possibly surgery — can be contemplated.

There seems to be good evidence that subgingival scaling and root planing has limited effectiveness in the treatment of probing depths greater than 5–7 mm.[3–5] In such cases, especially in the presence of pocket activity such as bleeding on probing, suppuration, marginal edema and erythema, surgical flap procedures should be considered. The surgical approach to periodontal therapy is intended mainly to provide

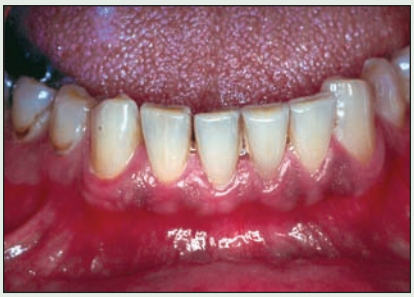

Figure 2a: Labial view of mandibular anterior teeth before root planing.

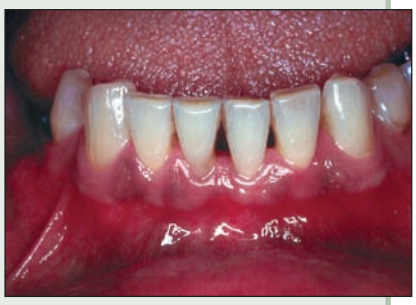

Figure 2b: Labial view of mandibular anterior teeth approximately 6 weeks after root planing.

greater visual access to the root surface and the periodontal lesion for mechanical debridement. However, promotion and facilitation of healing, re-establishment of physiological gingival and osseous contour, and creation of an environment amenable to routine periodontal maintenance have also been suggested as possible goals of surgical procedures. Following any type of flap surgery, frequent prophylaxis can help to prevent inflammation and limit increases in probing depths.

Upon completion of active treatment, follow-up periodontal maintenance visits are important to the long-term management of patients. Because every patient is different, there is no standard maintenance regimen; however, repeated reinforcement of oral hygiene procedures, in combination with debridement, leads to a more favourable response over time.[6] In determining optimum maintenance frequency, the degree of inflammation, the amount of plaque and calculus accumulation, and changes in probing depth should be taken into account, but maintenance intervals of 3 to 4 months are commonly needed.

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QUESTION 2

In a patient with gingival recession, what is the best procedure to obtain root coverage?

Background

Gingival recession is defined as migration of the marginal tissue apical to the cementoenamel junction, a process that exposes the root surface to the oral environment.[1] It can be the result of inflammation, trauma from tooth-brushing or orthodontic movement. Regardless of the cause, recession can present problems for some patients. In patients with a high lip line, for example, esthetics may be compromised. Dentinal hypersensitivity can occur, which may significantly affect a patient's quality of life by limiting the types of foods that can be eaten. If the tissue covering the root is primarily mucosa, which is thin, normal hygiene procedures may be uncomfortable, which in turn leads to increased accumulation of plaque and increased risk of further recession. For patients in whom plaque control is compromised, such as elderly patients or those taking xerostomic medications, gingival recession can increase the risk for root caries.

The current gold standard in root coverage procedures is connective tissue grafting (CTG). Other procedures that may be successful include the creation of coronally positioned flaps and use of a barrier membrane (also known as guided tissue regeneration [GTR]). Free gingival grafts, while effective at increasing the amount of keratinized tissue, do not provide predictable root coverage.

Management of the Issue

As with any health care intervention, there are risks and benefits to each option. CTG provides excellent root coverage but has one major drawback: the procedure necessitates the use of 2 surgical sites, the donor site (often the palate) and the recipient site (**Fig. 1**). It is therefore assumed that patients will be more uncomfortable after the surgery than if there were just one surgical site. With GTR using bioresorbable membranes, there is only one surgical site, although GTR with nonresorbable membranes requires a second procedure to remove the membrane and thus offers no advantage over CTG. Presumably, having one site produces less postoperative morbidity. There is, however, a dearth of evidence on any type of quality-of-life outcomes of GTR.[2] The most significant drawback to the use of GTR is cost, since it is much more expensive than CTG.

A recent meta-analysis by Roccuzzo and others[3] examined evidence from 20 clinical trials. CTG was the most successful in terms of gain in attachment level and percent root coverage. The

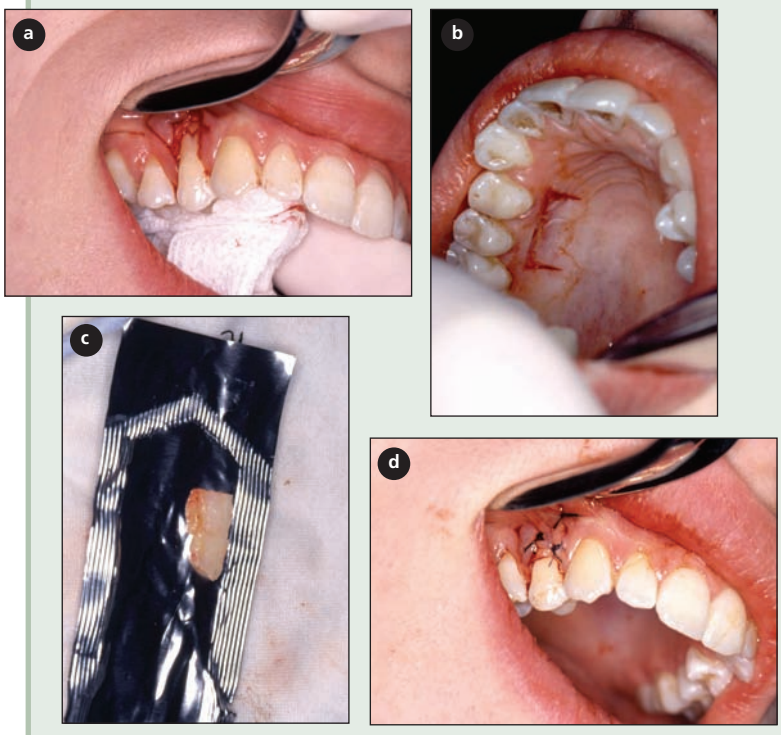

Figure 1: (a) Connective tissue grafting of tooth 14 begins with preparation of the recipient site. A split-thickness flap is extended into the moveable mucosa, such that the flap can be positioned coronally. **(b)** The split-thickness flap allows harvesting of subepithelial connective tissue from the palate, anterior to the greater palatine vessels. **(c)** The subepithelial connective tissue graft, harvested from the palate. **(d)** The graft is sutured in place over the recession defect. The flap is then positioned over the graft and sutured. The success of this procedure can be attributed to the dual blood supply of the recipient bed and the flap.

Factors affecting the success of root coverage

Local factors

- height of interdental bone and gingiva
- type of recession (**Figs. 2–5**)

Patient factors

- smoking
- plaque control

Type of procedure

- connective tissue graft greater than guided tissue regeneration and coronally positioned flap

Experience of clinician

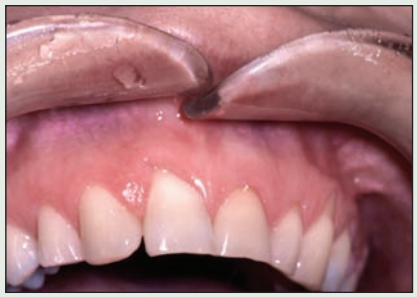

Figure 2: Class I recession, as defined by Miller.[4] The recession is shallow, and there is no interproximal bone loss. Complete (100%) root coverage can be expected.

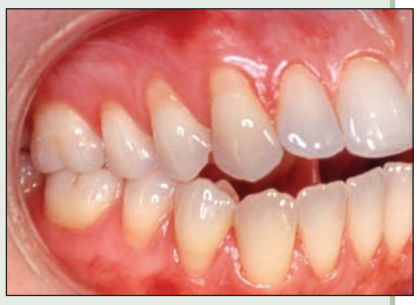

Figure 3: Class II recession. The area of recession is wide, and there is no interproximal bone loss. Complete (100%) root coverage can be expected.

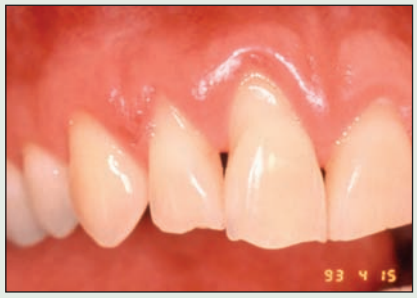

Figure 4: Class III recession. This category of recession is the same as Class I or II recession but with loss of interproximal bone. Root coverage will probably reach only to the level of bone.

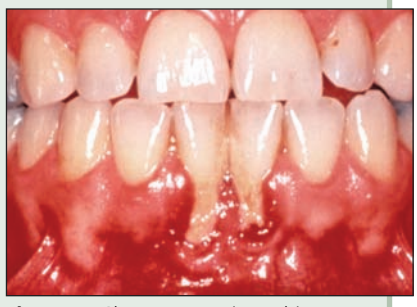

Figure 5: Class IV recession. This category of recession is the same as Class I or II gingival recession with loss of interproximal bone height such that the interproximal soft tissue is apical to the cementoenamel junction and at or apical to the facial gingival margin. Little to no root coverage can be expected.

authors found no difference between coronally positioned flaps and GTR or between resorbable and nonresorbable GTR membranes.

Technique is not the only factor in the success of root coverage. The height of the adjacent interproximal tissues is a key factor in predicting the amount of root that will be covered. Coverage should be expected to reach only to a line parallel to the interproximal bone height. People who smoke are poor candidates for root coverage procedures, because of reduced vascularity and interference with other healing factors. As with all clinical procedures, the skill and experience of the clinician also play an important (albeit rarely measured) role.

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QUESTION 3

What is the role of systemic antibiotic therapy in periodontal treatment?

Background

Periodontal disease attacks the supporting tissues of the teeth, causing bleeding on probing, pocketing, loss of bone and eventual loss of the dentition. Accumulation of a bacterial plaque biofilm (a structured community of bacterial cells enclosed in a self-produced glycocalyx matrix) is a prerequisite for this disease process. Regular removal of the biofilm through good home care by the patient and regular maintenance by a dental professional is the primary means of preventing periodontal disease.[1,2]

In spite of these measures, periodontal breakdown may continue (**Fig. 1**). Pockets continue to deepen, bleeding persists, support decreases and teeth are lost. At this point, it is necessary to reassess the case and determine the cause or causes for the ongoing periodontal breakdown. Refractory periodontitis may be diagnosed if conventional treatment has failed altogether, whereas recurrent periodontitis is diagnosed if conventional therapy was initially successful but the patient then failed to follow through with professional care at the suggested intervals or neglected his or her oral hygiene. The patient's systemic health may also be of concern. Diabetes, HIV/AIDS, cyclic neutropenia and other immunological conditions may leave the patient vulnerable to periodontal breakdown.[3] A genetic predisposition may be present. Smoking and tobacco use may hinder conventional treatment.[4]

With these factors in mind, systemic antibiotic therapy can be a useful adjunct to conventional treatment. Before antibiotics are prescribed, the patient must be re-examined and specific goals set. The area of concern should then be reassessed at appropriate intervals to ensure that the prescribed antibiotic has achieved these objectives.[5] Many studies have looked at the effectiveness of various antibiotic therapies, specifically penicillin, amoxicillin, clindamycin, metronidazole and tetracyclines. Almost all of these studies have examined conventional treatment (mechanical debridement) with and without the addition of an antibiotic.

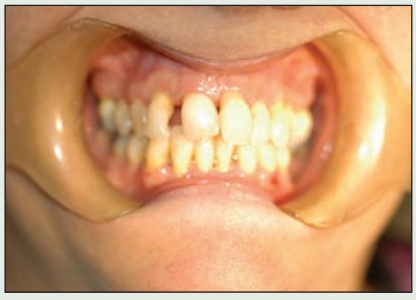

Figure 1: Anterior bite collapse due to ongoing periodontal disease.

Benefits and Risks of Systemic Antibiotics

Tetracyclines (in particular, minocycline and doxycycline), clindamycin and ethromycin are broad-spectrum bacteriostatic agents. In addition to their antibacterial effects, tetracyclines are capable of inhibiting collagenase, thus preventing tissue breakdown. Another benefit of tetracyclines is their ability to bind to tooth surfaces, which allows them to be released over time. Their use in recurrent and refractory periodontal disease has been well researched.[6] The primary drawback to clindamycin is its link to severe gastrointestinal disturbances, including colonization by *Clostridium difficile*, a potentially fatal condition.[5] For this reason, caution is needed in prescribing this drug, especially for use by elderly patients. Erythromycin may interact with other commonly used medications, such as benzodiazepines, ranitidine, oral anticoagulants, digoxin and methylprednisone.[7]

Metronidazole improves results when used in conjunction with scaling and root planing. However, it has no such improvement effect when combined with periodontal surgery. Use of metronidazole is contraindicated for patients taking warfarin.[8] Patients must also be advised to avoid alcohol, as they may suffer severe gastrointestinal upset, similar to the effects of disulfiram (Antabuse). Studies have shown that Augmentin (amoxicillin plus clavulanic acid) may improve clinical results when used in combination with scaling and root planing and, in rapidly progressive cases, surgery.

My 21 years of treating periodontal disease has led me to use antibiotic therapy only if conventional therapy has failed to control the disease. One potential exception occurs with patients who have diabetes, especially those with moderate to poor control of blood sugar. Diabetic control depends on eliminating chronic infections in these

patients. The routine addition of antibiotics has enhanced the results achieved by scaling and root planing or periodontal surgery.

People who smoke may also benefit from the addition of antibiotic therapy to conventional treatment, as they have higher levels of periodontal pathogens in shallower pockets. It can be difficult to monitor periodontal disease in these patients at maintenance visits because the microvascular effects of tobacco products lead to a lack of bleeding on probing, even when periodontal disease is active. Serial radiographs and serial recording of pocket depth are necessary for monitoring these patients.

In conclusion, antibiotic therapy can be a useful adjunct in the treatment of periodontal disease. However, it cannot replace scaling and root planing, with or without periodontal surgery, and good oral hygiene on the part of the patient. Appropriate maintenance of all patients with periodontal disease is critical to ensure that the results achieved with active treatment are retained. Antibiotic therapy, when applied in cases of refractory or recurrent disease or in immunocompromised patients, can help achieve good long-term results.

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QUESTION 4

What effect does smoking a water pipe have on the periodontium?

Current research, treatment and policy efforts related to smoking focus on cigarettes, whereas many people in developing regions smoke tobacco using water pipes. Water pipe smoking, also known as narghile, has been practised extensively for about 400 years, and its use is increasing globally, particularly among youth in the Eastern Mediterranean regions, where perceptions regarding health effects and traditional values may facilitate use of water pipes by women and children. Although cigarette smoking is known to increase the level of free radicals in periodontal tissues, which in turn may be responsible for the destruction associated with periodontal diseases, research about the epidemiology and health effects of water pipe smoking is limited, and no studies addressing treatment efforts have been published. Nonetheless, it is known that water pipe smoke contains harmful constituents, and preliminary evidence has linked this practice to a variety of life-threatening conditions.[1,2]

Adverse Effects of Water Pipe Smoking

Most western health care professionals are unfamiliar with the practice and health consequences of water pipe smoking (**Fig. 1**). The trend for increasing use of water pipes therefore represents a new challenge for health care providers. Studies that have examined narghile practitioners and the aerosol of narghile smoke have reported concentrations of carbon monoxide, nicotine, "tar" and heavy metals as high as or higher than those for cigarette smokers and cigarette smoke. The few scientific data about the adverse health consequences of water pipe smoking point to dangers similar to those associated with cigarette smoking. Additional dangers not encountered with cigarette smoking are infectious diseases that may result from sharing pipes and adverse effects caused by the frequent addition of alcohol or psychoactive drugs to the tobacco used in water pipes. Common misconceptions about water pipe smoking are that the nicotine content is lower than that of cigarettes, that the water filters out all toxins, that it is less harmful to the throat and respiratory tract than cigarette smoking, and that narghile tobacco contains fruit and is therefore healthy. Public health strategies for controlling the emerging epidemic of water pipe smoking include carrying out epidemiologic and toxicological research; implementing laws to limit acquisition and use; and providing health education, targeting adolescents in particular.[3]

A recent study[4] in Jeddah City, Saudi Arabia, explored whether water pipe smoking is associated with periodontal health in a manner similar to cigarette smoking. Participants were classified as water pipe smokers (33%), cigarette smokers (20%), smokers of both water pipe and cigarettes (mixed smokers; 19%) or nonsmokers (28%). Both cigarette consumption and water pipe smoking were associated with the presence of more than 10 sites with a probing depth of 5 mm or more. The relative risk for periodontal disease was 5.1 times greater among water pipe smokers and 3.8 times greater among cigarette smokers than among nonsmokers ($p < 0.01$). The relative risk associated with heavy smoking was about 8 times greater among water pipe smokers and 5 times greater among cigarette smokers than among nonsmokers, which suggests an exposure–response effect. Tobacco smoking was associated with a reduction in periodontal bone height. However, the cigarette smokers, water

Figure 1: Traditional water pipe.

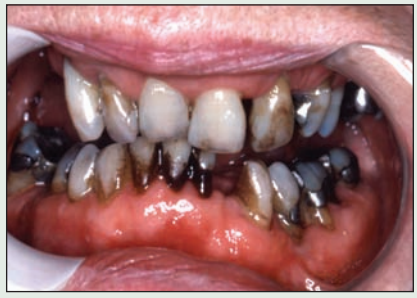

Figure 2: Smoker's mouth. In addition to nicotine staining, the thick fibrotic tissue and generalized recession are typical of people who smoke.

pipe smokers and nonsmokers all exhibited similar periodontal microflora (**Fig. 2**).

Management

Not only is smoking a major risk factor for periodontal disease, but the practice also adversely affects the response to therapy for this condition. Smoking patterns should be considered in treatment planning, since less favourable results (in terms of reduction in probing depth, gain in clinical attachment and reduction in gingival bleeding) have been seen in smokers than in nonsmokers following nonsurgical and surgical periodontal therapy.[5–10] In addition, susceptibility to recurrence of the periodontal infection and the need for retreatment are also greater among smokers.[11–13] Smoking counselling and assistance in smoking cessation should be incorporated as integral components of any dental practice.

Conclusions

The impact of water pipe smoking on the periodontium appears to be of about the same magnitude as that of cigarette smoking. However, more scientific documentation and careful analysis are required before the spread of water pipe use and its health effects can be understood and empirically guided treatment and public policy strategies implemented. Inquiries about water pipe smoking should be part of a complete periodontal examination.

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