



## The surgical treatment of periodontal infections

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Identification of the specific pathogenic microorganisms responsible for periodontal destruction has led to newer treatment methods. The basis for this approach has been the application of well-established surgical principles of infection management: the identification of the responsible pathogens; mechanical débridement of the infected sites to decrease the bacterial load as well as products of the inflammatory immune response; and the introduction of systemic antibiotics known to be effective against the offending bacteria.

Experience has shown that eradication of the infection is possible and regeneration of attachment and bone regrowth in vertical defects is accomplished when several important guidelines are followed. The oral cavity is regarded as one ecosystem, with microorganisms readily transported to multiple sites by saliva, food, and the toothbrush. Treatment by debriding only one quadrant at a time allows reinfection of the site within the ecosystem from other untreated quadrants. The usual pattern of multiple oral hygiene visits before definitive full mouth debridement meant to “prepare” the mouth for surgery does not eliminate the infection that is harbored in biofilms in deep pockets. Eliminating the deep infection should take precedence over the repeated superficial scalings. Brushing, flossing and various mouth rinses do not reach pockets deeper than 4 to 5 mm despite the diligence of enhanced home care. The periodontal attachment continues to secrete extracrevicular fluid that inactivates and rapidly washes away any substances squirted into the pocket. It should be noted that topical antimicrobials (eg, chlorhexidine) as

well as topical antibiotics are cytotoxic [1]. They provoke the death of healthy cells and elicit their own inflammatory reactions.

The field of periodontal disease has undergone rapid expansion of basic information with respect to microbiology and the origin and characterization of endotoxins produced by gram-negative anaerobes, which are responsible for various kinds of periodontitis. The endotoxins (lipopolysaccharides) from the outer cell wall of the specific anaerobes are now known to be water soluble, and their localization on the cementum of the root is superficial and not deeply imbedded [2–5]. Smart et al [6] and others have reported that endotoxin can be washed away or brushed away without resorting to root planing, which destroys the cementum. Recently, the treatment concept has changed from aggressive root planing to debridement. It also has been shown that connective tissue regrowth in the periodontal crevice is positively influenced by the presence of adjacent cementum. In the absence of cementum, reattachment to dentin does not readily occur. Following periodontal connective tissue reattachment, bone fills the vertical defects, and the mobility of teeth decreases when the infection is resolved.

It is difficult to identify the origin of the practice of root planing. There is no scientific evidence to show that root planing is any more efficacious than debridement, without destroying cementum. The shibboleth of “scaling and root planing” with which we were taught to begin every treatment plan deserves to be seriously questioned. In addition to being destructive, the removal of cementum also leads to thermal hypersensitivity and more postoperative pain. It is likely that the original intent to remove scale, formed from salivary calculus, was extended down onto the exposed root surface and

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continued even in the absence of calculus deposits. Calculus deposits come from saliva and are most pronounced opposite the openings of Wharton's and Stensen's ducts. Calculus does not cause periodontitis but may constitute a mechanical irritant and be aesthetically objectionable. Calculus, when identified, is best removed using an ultrasonic cleaner or by discrete curetting, which clears away just the deposit and does not strip the cementum.

### Diagnosis of periodontitis

A major contribution was made by Loesche [7], who introduced the "specific plaque hypothesis." Other investigators have identified several periopathogens associated with patterns of bone loss, primarily by using the techniques of deep-pocket sampling and rapid anaerobic subculturing. Periodontitis was thus identified not as a single entity but as a series of infections based on microbiologic sources, host immunocompetency, and the interaction between these factors. Various syndromes of periodontal infections have been identified.

#### Rapidly progressive periodontitis

This is seen in children, young adults, and adults who are immunocompromised, especially by virtue of impaired cell-mediated immunity (eg, neutropenia, cancer chemotherapy, acquired immunodeficiency syndrome, etc.).

#### Juvenile periodontitis

Formerly known as periodontosis, this is a disease of late childhood and early adolescence caused by *Actinobacillus actinomycetemcomitans*, a gram-negative facultative organism that produces a potent leukotoxin. Early in childhood the organism is found in the tonsillar crypts and may cause repeated bouts of tonsillitis. Later on, in addition to dental pathology it may be found on damaged heart valves, causing infective endocarditis. This organism is susceptible to the tetracyclines and not consistently to the penicillin derivatives or clindamycin. Another important aspect of this syndrome is that there is usually a qualitative inherited defect in the behavior of the patient's neutrophilic leukocytes.

#### Acute necrotizing ulcerative gingivitis

This uncommon infection, once called "trench mouth," is most often noted in young smokers.

*Borrelia vincenti* and *Prevotella intermedia* are two major pathogens readily identified in this condition which is often associated with susceptible individuals who have been subjected to major psychologic stress, such as a divorce, familial death, or dismissal from college. Under these circumstances, direct invasion by the periopathogens has been demonstrated, leading to necrosis, loss of interdental papillae, lymphadenitis, gingival bleeding, and fetid odor. Treatment should be directed first to using a systemic antibiotic, such as penicillin or amoxicillin, to control the acute phase before launching into painful manipulations, such as curettage.

#### Chronic adult periodontitis

Several specific organisms have been associated with the localized areas of periodontal bone destruction (Table 1). These organisms include *Porphyromonas gingivalis*, *Bacteroides forsythus*, *Prevotella intermedia*, *Capnocytophaga species*, *Campylobacter rectus*, *Eikenella corrodens*, and *Fusobacterium nucleatum*. Other periopathogens will undoubtedly be identified as the field expands. Many of these organisms produce an endotoxin (ie, lipopolysaccharide) that provokes the release of host-tissue factors, contributing to further breakdown. Cytokines such as interleukin-1 $\beta$  and tumor necrosis factor- $\alpha$  from host lymphocytes and other mononuclear phagocytes are released. These and other substances can destroy collagen and bone, causing advancing destruction of the periodontium. Bacteriologic culturing

Table 1  
Species identified in refractory or recurrent periodontitis (n = 196)

Organism	Percentage of sites
<i>Bacteroides forsythus</i>	84
Spirochetes	83
Motile rods	76
<i>Fusobacterium species</i>	68
<i>Porphyromonas gingivalis</i>	63
<i>Campylobacter rectus</i>	47
<i>Capnocytophaga species</i>	38
<i>Prevotella intermedia</i>	23
<i>Peptostreptococcus micros</i>	18
<i>Actinobacillus actinomycetemcomitans</i>	16
<i>Candida species</i>	14
Enteric rods	9

Resistance to penicillin, tetracycline, and metronidazole was high.

Adapted from Listgarten MA, Lai C-H, Young V: Microbiota and antibiotic resistance (abstract). J Dent Res 1993;72:819. [8]

and the use of specific DNA probes (eg, University of Pennsylvania Microbiological Testing Laboratory, Philadelphia, PA) have served to identify putative organisms. Other tests have been proposed based on the enzymatic characteristics of the particular pathogens. Loesche et al [9] have reported on the use of the benzoyl-DL-arginine naphthylamide (BANA) test at chairside. BANA identifies three known pathogens: *P. gingivalis*, *B. forsythus*, and *Treponema denticola*. The ultimate aim is to develop a chairside test that will identify the predominant organisms and guide treatment and follow-up monitoring.

Periodontitis is readily transmissible between spouses and other cohabitants [10,11]. In the author's experience, 80% of patients requiring treatment have mates with similar organisms. To prevent "ping-ponging" the disease, both partners should be placed on antibiotics while the primary patient undergoes surgical débridement and proceeds into a maintenance phase.

Radiographs reflect the bone level (ie, bone loss) but do not indicate the current status of the infection. Bone loss does not necessarily translate into mobility or condemn a tooth to extraction. Satisfactory resolution of the infection leads to reduction in bleeding, pocket depth, and mobility, and new bone often forms and fills vertical defects [12–14].

### Surgical techniques

Current management of periodontitis has moved away from some of the prior practices (see Box 1). The gingivectomy is passé, as is the practice of bony recontouring. The author prefers early, one-stage, full-mouth débridement guided by the severity of attachment and bone loss. Pockets deeper than 4 or 5 mm merit use of a conservative access flap reflected just to the bone margin (Fig. 1).

This provides visibility for surgical curettage of the granulation tissue and avoids the postoperative edema and pain caused by excessive exposure of labial bone. Pockets that measure less than 4 or 5 mm are debrided and curetted at the same appointment without flap reflection.

With the patient under intravenous sedation and local anesthesia, the crevicular incisions are made in areas where pocket depths exceed 4 to 5 mm, preserving the interdental papillae and marginal gingivae. Bony crypts are debrided with a large Prichard curette followed by use of Gracey curettes to reach into and under bony defects. Copious saline irrigation is used during débridement instead of cytotoxic agents such as chlorhexidine and topical antibiotic

### Box 1. A summary of guiding surgical treatment of periodontal infections

1. Periodontitis is an infectious disease attributable to a relatively small number of gram-negative oral pathogenic bacteria that produce endotoxins.
2. Piecemeal mechanical treatment (by quadrant or sextant) via scaling and root planing above is incomplete and invites relapse.
3. The mouth is one ecosystem that must be treated as a comprehensive unit to eradicate the infection, preferably under intravenous sedation and local anesthesia.
4. Periodontitis may be considered a sexually transmitted disease.
5. Soft tissue débridement of periodontal pockets without destruction of root cementum (planing) is the goal, in conjunction with systemic antibiotics effective against the specific bacterial pathogens.
6. Topical irrigants, including antibiotic and antimicrobial agents, are cytotoxic and jeopardize healing and regeneration.
7. Regrowth of bone follows reattachment when the infection is resolved and the cementum is intact. It is a curable disease.
8. Reinfection is possible, and continued vigilance and follow-up care are important.
9. Individual immune status affects the incidence and long-term response to therapy. Examples of immunocompromised status include diabetes, HIV, and qualitative and quantitative white blood cell abnormalities.
10. Calculus has a minimal role in the initiation and progression of periodontitis.

The periodontal literature supports each of these principles and should lead to a modification of practice.

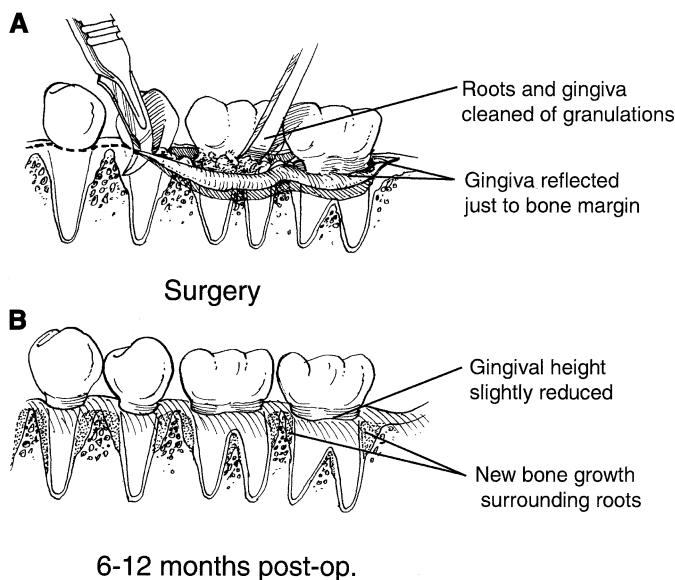


Fig. 1. (A) Crevicular incision reflected just to bony margin for débridement. (B) New bone regrowth in absence of infection.

solutions, which damage healthy cells [15,16]. It is important to remove the granulation tissue that adheres to the underside of the mucoperiosteal flap either by curettage or with a soft-tissue rongeur (Nipro Medical, Miami, FL) or nipper. With the flap reflected, deposits on the cemental surface can be discretely removed with a scaler or Cavitron.

Another important component of the recommended technique is to *avoid* root planing. The periodontal literature is finally recognizing that root planing is destructive and compromises reattachment. Cementum is necessary to induce and enhance fibroblastic proliferation and reattachment [4,5]. The apparent indication to remove diseased cementum is predicated on the presence of endotoxin [2,3]. Endotoxin, a product of gram-negative anaerobic organisms, is water soluble and not deeply imbedded in the cementum. It is limited to 40–50  $\mu\text{m}$  of the surface cementum and can be washed or even brushed away. Cementum has specific growth-enhancing factors that stimulate the fibroblasts necessary for repair and reattachment of the periodontal membrane. Therefore, planing the cementum is a waste of time and effort. Furthermore, root planing jeopardizes soft-tissue regeneration and bone support and often leads to thermal hypersensitivity.

The surgical objectives should be to clear the infection by mechanical débridement *and* to use antibiotics effectively. Minimal periodontitis can be read-

ily controlled by frequent periodic débridement probably because it disrupts the colonies that re-form in the pocket. When bone loss has advanced beyond 4 to 5 mm, however, it does not make good sense to rely only on conservative treatment and wait until the infection recurs and is finally classified as refractory. Treatment should be comprehensive and aim to debride with the least possible damage to cementum, bone, and mucoperiosteum. This should be accompanied by the administration of systemic antibiotics known to be effective against gram-negative anaerobes. Clindamycin, metronidazole, or amoxicillin/clavulanic acid have been used for 7 to 10 days with rewarding results. The tetracyclines usually are prescribed for 2 to 3 weeks to treat juvenile periodontitis [12–14,17,18].

Flaps are closed with interrupted sutures, avoiding apical repositioning (Fig. 1). Healing and bony regrowth are favored by adequate mucoperiosteal covering. The surgeon should not try to eliminate the pocket. The cervical defect fills initially with a blood clot, which then differentiates into connective tissue and subsequently into new bone. Periodontal packs are never used; they retard healing and are cumbersome, difficult to clean, and inappropriate when treating an anaerobic infection (Fig. 2).

Even in the advanced periodontitis cases, the operating time for this procedure is usually 1 to 2.5 hours. Very few patients undergo repeated sessions with the hygienist before the main surgical débridement. It is

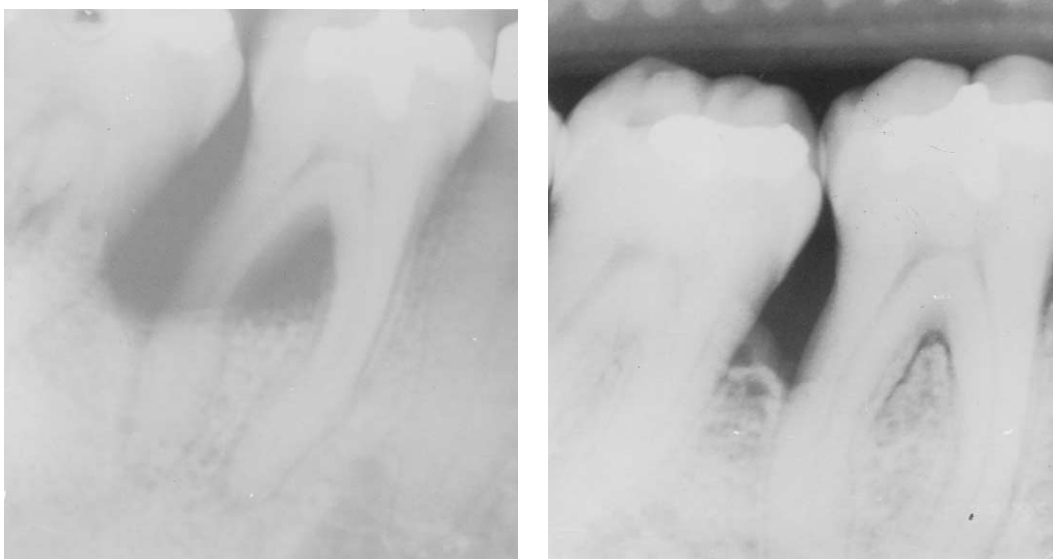


Fig. 2. (Left) Preoperative first molar bone loss. (Right) One year postoperative bone regeneration.

the primary intent to reduce the bacterial load in deep pockets that are beyond the reach of the patient's home care or the hygienist's scaling.

Postoperatively, the patient is given a prescription for a nonsteroidal antiinflammatory agent and instructed to continue the prescribed antibiotic for 7 to 10 days. Brushing is avoided in the sutured areas, and either saline irrigation or a chlorhexidine rinse is recommended for home hygiene. A new brush is provided on the 1-week return visit and a simple Bass brushing technique is demonstrated.

Patients are maintained on a soft diet and rarely report pain or swelling after the first day. The pockets are reprobated at 1 month to evaluate healing and pocket reduction. At this time, the patient can be referred back to his or her dentist for definitive restorative care. Individual persistent deep pockets may now be addressed if pocket elimination is requested by the restorative dentist. Successive 3-month follow-up visits are scheduled during the first year to evaluate the long-term results. Reprobing characteristically finds pockets significantly reduced, with no bleeding on probing or brushing. If this is not the case, additional antimicrobial therapy is pursued after culturing or taking a sample for DNA probe analysis [14].

Periodic (every 3 months) disruption of reforming colonies by deep scaling has been shown to provide adequate control if the case has been refractory to previous treatment. Topical application of fluoride gel

or use of a toothpaste with 1.1% sodium fluoride on a daily basis at home is effective in preventing reinfection from a supragingival source. The use of a chlorhexidine mouth rinse is also helpful in controlling supragingival organisms.

Concern has been expressed about the development of enterocolitis caused by *Clostridium difficile*, a gram-positive organism that produces exotoxin and damages the cells lining the intestinal tract. This complication is seen with a number of antibiotics, such as clindamycin, the cephalosporins, and amoxicillin. Patients are routinely alerted to this possibility when postoperative instructions are given. They are advised that approximately 10% of patients may develop diarrhea after taking the antibiotic for at least 4 to 5 days. If this occurs, they are instructed to discontinue the drug and call the office. The diarrhea usually abates within 24 to 48 hours. If it persists, oral metronidazole is recommended as the drug of first choice (250 mg four times per day).

The success rate of combined mechanical and antibiotic therapy has been cited by others [11–14, 17,18] to be superior to that of periodontal therapy alone. When treating a destructive infectious disease in the year 2002 it does not make sense to avoid using effective antibiotics. This technique also has distinct advantages in the salvage of failing implants. The principles of debridement and irrigation and the effective use of systemic antibiotics can enhance the

repair of endosseous implants that are losing bone support. No one knowingly root planes an implant. A soft-tissue débridement is indicated instead [12].

The recognition that specific cytokines, prostaglandins, growth factors, and interleukins, are active participants responding to the elaboration of bacterial lipopolysaccharide sheds new light on methods that may influence the clinical outcome. Golub and coworkers [19] have shown that the tetracyclines, in low doses, also inhibit matrix metalloproteinases such as collagenase and prevents damage to the attachment apparatus.

Perhaps most intriguing is the realization that immune-mediated injury is not only an integral part of the host response to periodontitis, but it has also been shown to be a significant contributor to myocarditis [20]. Cytokines released from activated T-lymphocytes have been identified as releasing tumor necrosis factor- $\alpha$  and other injurious molecules that also cause myocardial and vascular injury. Subsequent myocardial fibrosis correlates with the presence of T-lymphocytes and macrophages, which release fibrogenic cytokines such as fibroblast growth factor and tissue growth factor- $\beta$ . Our knowledge base is growing rapidly, and the future will bring fascinating new therapies for the management of periodontal infections.

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