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COMMENTARY

A re-evaluation of scaling and root planing

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Abstract

Background: Extensive reviews on the role of scaling and root planing (SRP) in the treatment of periodontitis have been previously published. This commentary will address the importance of subgingival calculus in the progression and treatment of periodontitis and addresses factors that make the execution of a "definitive" SRP a critical part of therapy.

Methods: A search for articles, using keywords relevant to the subject , (e.g., periodontitis, dental scaling, root planing, dental calculus, biofilm, inflammation) was conducted using PubMed, Ovid Medline, Cochrane Reviews and the ADA Center for Evidence Based Dentistry data bases. Additionally, references cited in relevant articles were also considered.

Results: Surfaces of subgingival calculus are covered with a biofilm of metabolically active bacteria. Periodontal inflammation is clearly related to the presence of calculus and biofilm. The primary goal of SRP is removal of subgingival calculus and biofilm deposits to create a biologically compatible root surface and reduce the inflammatory burden. Current evidence suggests that inflammation associated with periodontal infections affects both the immediate oral environment and the patient's systemic health.

Conclusion: SRP is still critical to the treatment of periodontitis. SRP involving deep probing depths (≥ 5 mm) and root surfaces with anatomical and surface irregularities, regardless of the type of instrumentation, requires time, exceptional skill and perseverance, and patient compliance with periodontal maintenance. Sites with persistent nonresponding probing depths and signs of inflammation following a definitive SRP, should be considered for surgical intervention.

KEYWORDS
biofilms, dental calculus, dental scaling, inflammation, periodontitis, root planing

1 | INTRODUCTION

The purpose of this commentary is to reevaluate the impact of root surface accretions and alterations on the pathogenicity of periodontitis, the factors that influence treatment outcomes, and to offer recommendations that will promote optimal long-term success in controlling periodontal disease. The belief that a core of clinical and scientific facts exists independently of interpretation by the clinician is a fallacy. It is common for clinicians to consider the same data and derive different conclusions. What follows is influenced by the authors' combined 100+ years of academic and clinical experience. We present the facts as we know and interpret them and apply them to the clinical practice of scaling and root planing (SRP) as the essential element of non-surgical periodontal therapy.

Periodontal diseases have achieved the status of a worldwide public health burden. Epidemiology studies report that 42% of dentate U. S. adults between the ages of 30 to 79 years have some level of periodontitis. Based on census data, this age range represents roughly 144 million people. Thus, approximately 60.5 million people suffer from periodontitis of which 7.8% (≈ 4.7 million) suffer from Stage III or IV, Grade B or C periodontitis. Undiagnosed and untreated, or insufficiently treated periodontitis can have negative effects on oral health, systemic health and quality of life.

Much of the localized destructive effect and the systemic impact of periodontitis is the result of inflammation. Current theory holds that periodontal inflammation results from the interaction of the host immune system and a dysbiotic subgingival biofilm. Dysbiosis likely results from the interaction of "keystone" microbes, such as *Porphyromonas gingivalis* and *Filifactor alocis*, on an ever-expanding list of subgingival potentially pathogenic microbes.[1]

The choice of a treatment modality is not necessarily the most critical determinant of effective periodontal therapy.[2] Achievement of long-term success requires a combination of detailed root surface debridement, appropriate periodontal maintenance therapy and patient compliance and devotion to oral hygiene.[3] In addition, Stage III or IV periodontitis cases generally will present soft tissue and osseous defects that can only be corrected by surgical intervention. Even in these cases, the non-surgical phase of treatment may be essential to success.[4]

Given the importance of inflammation in periodontal and various systemic diseases, every effort should be made to determine if the goals of a "definitive SRP" have been accomplished. It is the intent of this commentary to emphasize the importance of definitive SRP, its role in treatment of periodontitis, and to recognize factors that inhibit attainment of the goals of nonsurgical therapy, i.e., characteristics of subgingival biofilm and calculus and their interactions with the root surface.

2 | SRP AND SUBGINGIVAL BIOFILM

SRP remains the cornerstone of nonsurgical periodontal therapy. An often-cited goal of SRP is the removal of all subgingival calculus and biofilm. Numerous studies report a significant reduction in the subgingival bacterial burden and/or specific periodontal pathogenic microbes following SRP in periodontitis patients.[5] However, there remains the issue of microbial repopulation and re-infection following SRP, particularly in those instances of inadequate instrumentation.[6,7]

Scaling and root planing is remarkably effective at reducing clinical inflammation and pocket probing depth.[8] Yet, multiple studies report that following SRP a significant percentage of treated teeth will exhibit residual subgingival biofilm and calculus.[4,9] Good clinicians understand that SRP is a technically challenging procedure. A well-done SRP requires time, patience, persistence, experience, skill, and training.[10] Paradoxically, a common site for post-SRP residual dental calculus is the cemento-enamel junction – an easily accessible site.[11] To date, no specific instrument used for SRP has demonstrated consistent superiority for the removal of both biofilm and calculus, be it manual, ultrasonic/sonic instrumentation or lasers.

Multiple studies have evaluated bacterial recolonization of periodontal pockets following SRP.[5–7,12] It is beyond the ability of current treatment modalities to achieve eradication of all bacteria. Scaling efficacy is reduced with increasing pocket probing depth, root concavities, grooves and microgrooves, restoration contours, degree of furcation involvement,[5,6,13,14] and invasion of root surface irregularities and dentinal tubules.[15,16] In addition, incomplete SRP or treatment over a prolonged period may result in translocation of bacteria from untreated sites to re-infect previously treated sites.[17,18] Lastly, there exist the possibility of transmission of pathogenic bacteria between family members by direct contact, familial interactions, and through saliva.[19]

The time needed for recolonization to reach pretreatment levels of mean counts and proportions of the subgingival microflora depends on disease severity and thoroughness of debridement. Other than inadequate removal of calculus and biofilms, the repopulation of treated periodontal pockets by microbial pathogens may also come from several oral reservoirs including, epithelium and lamina propria of the pocket wall and epithelium of the buccal mucosa, dorsum of the tongue, tonsillar crypts, and saliva.[18]

Prevention of rebound to pretreatment levels of periodontal pathogens requires repeated removal of subgingival biofilms at patient appropriate intervals. This underlines the importance of regularly performed periodontal maintenance therapy, including subgingival debridement and/or SRP of all pockets of ≥ 4 mm.

3 | MICROBIAL INVASION OF ROOT STRUCTURE

As noted above, potential reservoirs of pathogenic bacteria involve various oral soft tissues and saliva. Subgingival calculus and its inherent microbial components, both surface biofilm and internal of the calculus mass, will be

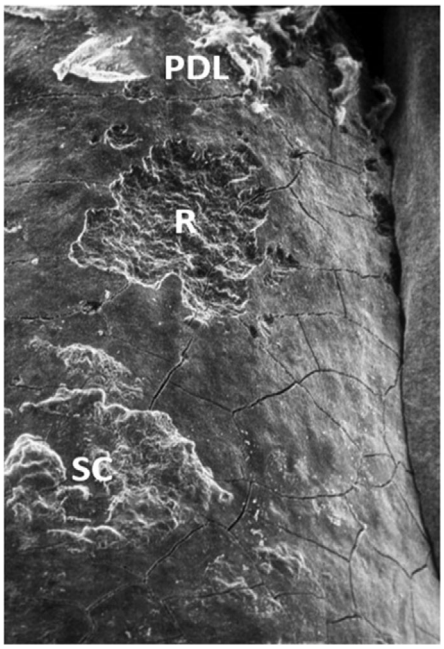

FIGURE 1 Surface view of a diseased root with a large cavitational defect. Remnants of periodontal ligament fibers (PDL); Resorption defect (R); subgingival calculus (SC). Original magnification of 160X

addressed as a separate subject. However, a seldom mentioned reservoir is that of bacteria that invade root cementum and dentin [15,16,20,21] or congregate in areas of root surface resorption, a.k.a., surface concavity or lacunar defect. (Figure 1) It has been suggested that such areas of root surface resorption are the result of persistent occlusal trauma, orthodontic movement, or inflammation from periodontal disease.[22,23] Regardless of the etiology of localized root resorption, teeth extracted because of severe periodontal disease and subsequently examined by scanning electron microscopy, frequently exhibit bacterial colonization within root surface cavitations (lacunae). (Figure 2) Scaling of a root surface where calculus is embedded in a resorptive defect is likely to result in incomplete removal of bacteria which, in turn, may facilitate recolonization of subgingival populations.

The presence of invasive bacteria in cementum and radicular dentin of periodontally diseased teeth has been demonstrated by both light and scanning electron microscopy and culture studies.[15,16,20,21] (Figure 3) Adriaens [24] reported that 83% of 69 caries free periodontally diseased teeth exhibited bacteria in radicular dentin. Most of the bacteria were located in the outer 300 μm of the dentinal tubules.[16] Microscopically, the morphotypes of the invasive bacteria resembled that commonly seen in early stages of developing subgingival biofilm, e.g., cocci and short and medium length rods.[20,21]

A more recent study by Giuliana et al.[20] cultured invasive bacteria from the roots of periodontally diseased teeth and reported that 14 of 26 (53.8%) of teeth yielded a positive culture. Using a commercial micro-method system for identification, the study detected the following invasive periodontal pathogens: *Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium nucleatum, Tannerella* forsythia (previously known as *Bacteroides forsythus*), *Micromonas micros* (previously known as P*eptostreptococcus micros*) and *Streptococcus intermedius*.

Collectively, these studies suggest that radicular dentin and cementum may act as a reservoir from which periodontal pathogenic bacteria can recolonize treated periodontal pockets and, thereby, contribute to reinfection. As emphasized previously, it is likely that incomplete or poorly executed SRP will facilitate a more rapid reinfection of the periodontal pocket. This in turn places more responsibility on the clinician and patient to adhere to a rigid and timely periodontal maintenance interval for continual monitoring and possibly retreatment of persistent or recurring disease.

4 | SUBGINGIVAL DENTAL CALCULUS

Dental calculus is considered to represent the calcified configuration of an undisturbed oral biofilm.[25] Historically, excepting the inherent surface biofilm, calculus was regarded a mineralized "dead" organic material, i.e., fossilized bacteria embedded in a mineralized extracellular matrix.[26] With the development of more sophisticated microscopy, it now appears that the a mineralized mass of a subgingival calculus is actually very porous, allowing for live microbes to exist within its structure.[27,28] Indeed, it has been demonstrated communities of viable bacteria in channels and lacunae of subgingival calculus, including filamentous microbes, spirochetes and short rods.[28]

Because of the ever-present surface biofilm, it is difficult to assign a cause-and-effect role to subgingival calculus in the initiation and progression of periodontal disease. Regardless, the presence of subgingival calculus has a strong association with inflammation in the soft tissue pocket wall,[29,30] further supporting the proposition that calculus, with its surface and internal populations of bacteria, is very capable of promoting inflammation. It has been estimated that 20%-25% of calculus is comprised of an organic matrix, non-mineralized channels and lacunae containing bacteria, and an extracellular polymeric matrix similar to that of other oral biofilms. As noted previously, the bacteria communities within the channels and lacunae are viable and comprised of a diverse collection of anaerobic Gram-positive and Gram-negative bacteria.

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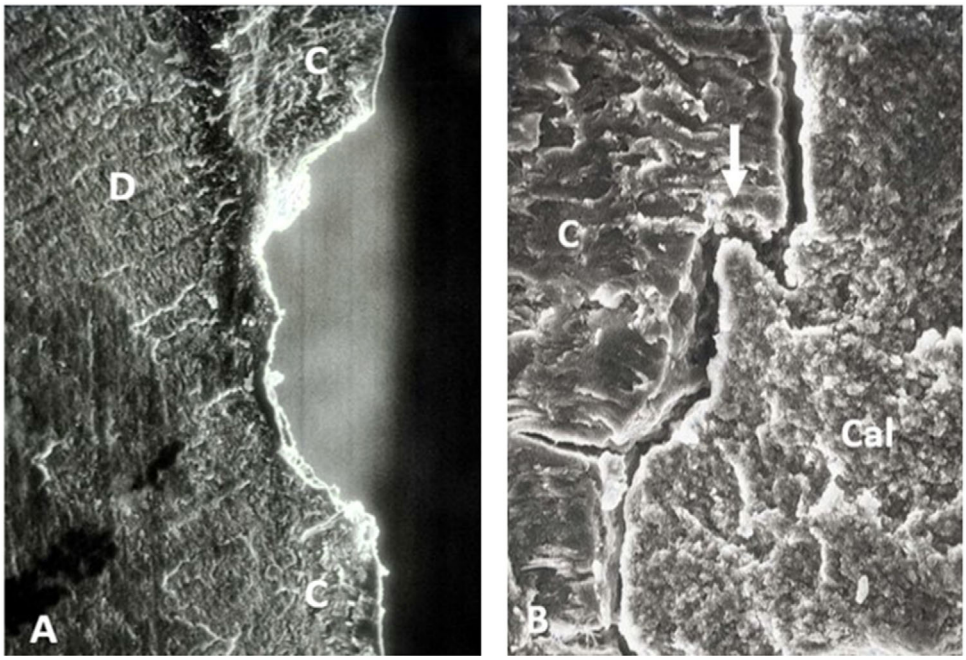

FIGURE 2 Cross-sectional views of root with resorption defects, without and with a calculus deposit. (**A**) The surface cavitation is approximately 50 μm in depth. Original magnification of 300×. (**B**) Calcified microbes are seen penetrating into a resorptive defect and engaging an undercut (arrow). Original Magnification of 600×. Cementum (C); dentin (D); and calculus (Cal)

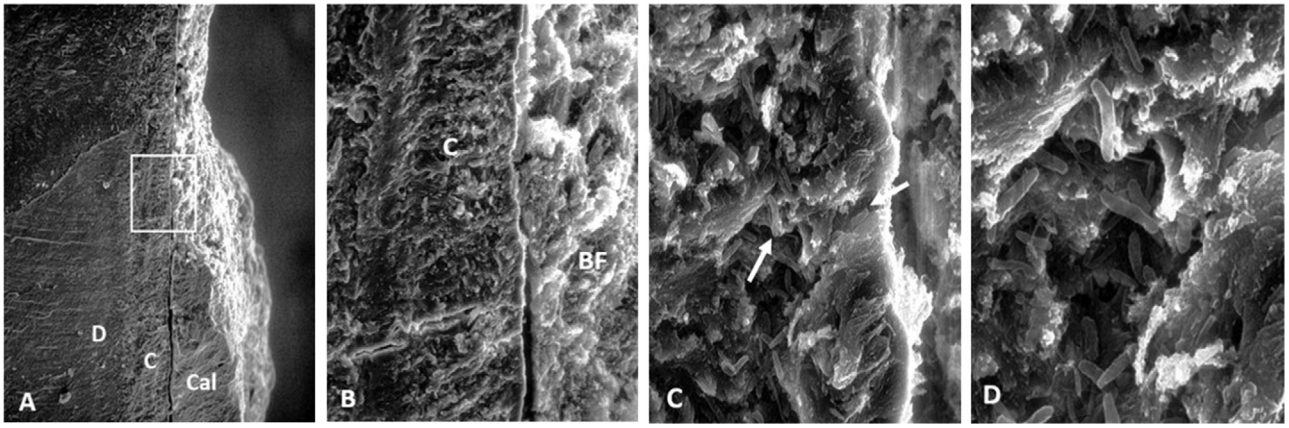

FIGURE 3 Progressive magnifications of a cross-sectional area showing calculus with attached biofilm and invasion of cementum and dentinal tubules by the biofilm microbes. (**A**) Area of Interest is outlined by box. Original magnification 160×. (**B**) Right 1/3 of photo shows a dense mat of biofilm attached to the cementum surface. Original magnification 300×. (**C**) Invasive rods in cemental microchannel (arrows). Original magnification 3,500×. (**D**) High magnification reveals numerous invasive short and medium length rods. Original magnification 7,000×. Dentin (D); cementum (C); biofilm (BF) and calculus (Cal)

Consequently, calculus may act as a reservoir for the continuous release of endotoxins and various microbial antigens.[26,31] Subgingival calculus, capable of initiating and/or promoting inflammation, prompted Mandel and Gaffar to label subgingival calculus a "slow releasing device".[31] Clearly, the incomplete removal of subgingival calculus leaves a residuum that serves as a reservoir of biologically active and noxious irritants that promote inflammation. Clinically, such a residuum is a contributing factor to reinfection of the periodontal pocket and recurrence of disease following inadequate treatment. In fact, Ramseier et al.[32] report in their analysis of the natural history of periodontitis over a 40-year period that smoking and calculus were associated with disease initiation and that calculus, biofilm and gingivitis were associated with loss of attachment and progression of disease.

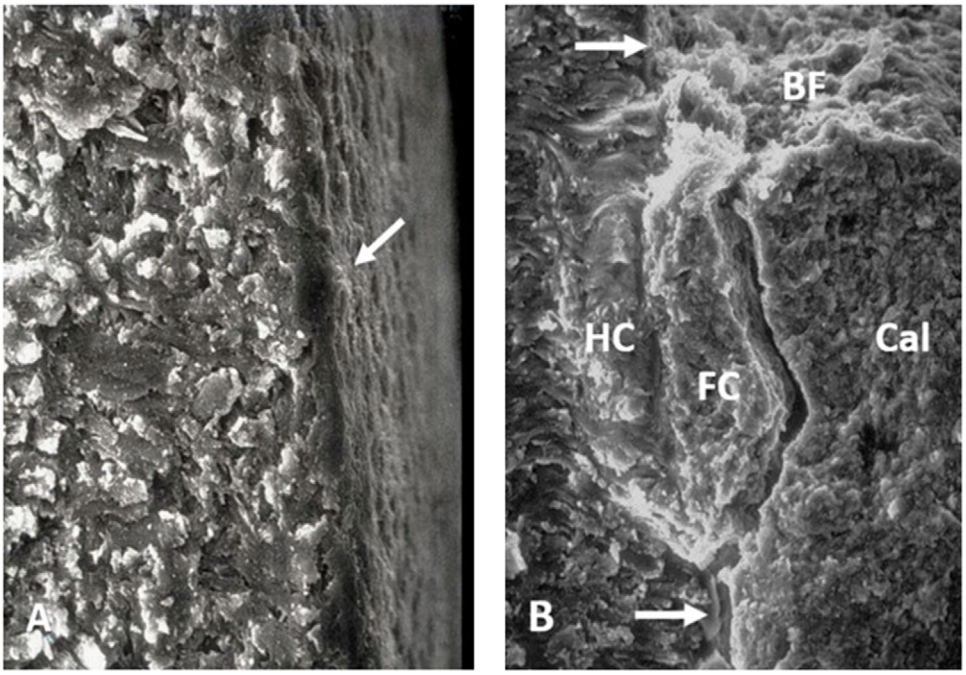

FIGURE 4 (**A**) Cross-sectional views of healthy cementum not exposed to the oral environment. Sharpey's fibers were removed by sodium hypochlorite application, revealing "mounds," that represent points of fiber insertion (arrow). Original magnification 700×. **B**) Diseased cementum with attached calculus and biofilm. Arrows indicate cementum surface. Note that part of the calculus deposit (Cal) has fractured (FC) with a portion tightly adherent to a zone of hypermineralized cementum (HC). Original magnification 350×

5 | CHARACTER OF THE DISEASED ROOT SURFACE

Root surfaces exposed to the toxic environment of a periodontal pocket undergo several changes. A root surface no longer protected by the periodontal ligament (PDL) or junctional epithelium represents a biological enticement for bacterial adhesion, and development of calculus. Compared to cementum that has not been exposed to the oral environment, the exposed and untreated root surface is uneven and generally hypermineralized.[33] (Figure 4)

The uneven surface results from elevated mounds that represent sites where PDL fibers were previously embedded (Figure 5) and resorption defects which are commonly observed.

In addition to root surface irregularities, several studies have identified adsorption of bacterial lipopolysaccharide (endotoxin) and penetration up to 10 μm in root surfaces of periodontally diseased teeth.[34] Cementum is sufficiently porous to allow penetration and diffusion of biologically active products derived from saliva, gingival crevicular fluid and biofilm, including bacteria. In this regard, Bosshardt and Selvig [35] noted that bacterial penetration of cementum is facilitated by the presence of surface cracks and microfractures.

Although bacterial endotoxin is adsorbed onto exposed root surfaces, it exhibits a weak surface binding. Hughes and Smales [34] suggested that the demonstration of root surface lipopolysaccharide (LPS) was likely more important as an indicator of residual bacteria and calculus than of cementum bound LPS per se. In spite of this view, Pitaru et al.[36] demonstrated, in vitro, that bacterial endotoxin disrupts migration, orientation and attachment of human gingival fibroblasts to type I collagen. Thus, one could argue the potential for residual endotoxin to create havoc during attempted healing following inadequate periodontal therapy.

In spite of the role attributed to endotoxin in periodontal disease pathogenesis, it must be noted that weak surface binding makes removal of endotoxin relatively easy during subgingival debridement. Several in vitro studies have demonstrated the ease of removing root surface bound endotoxin using manual and ultrasonic instrumentation or simply rinsing with water, all with equal effectiveness.[37] However, there is a caveat that must be confronted.

According to McCoy et al.,[38] in vivo SRP alone will markedly reduce but not eliminate adsorbed endotoxin. Moreover, the authors suggest that significant "retoxification" of treated surfaces may occur within a short period of time. If true, this would seem to compromise any long-term effect achieved by SRP and reinforces the recommendation of a short interval for periodontal maintenance (i.e., every 3 to 4 months).

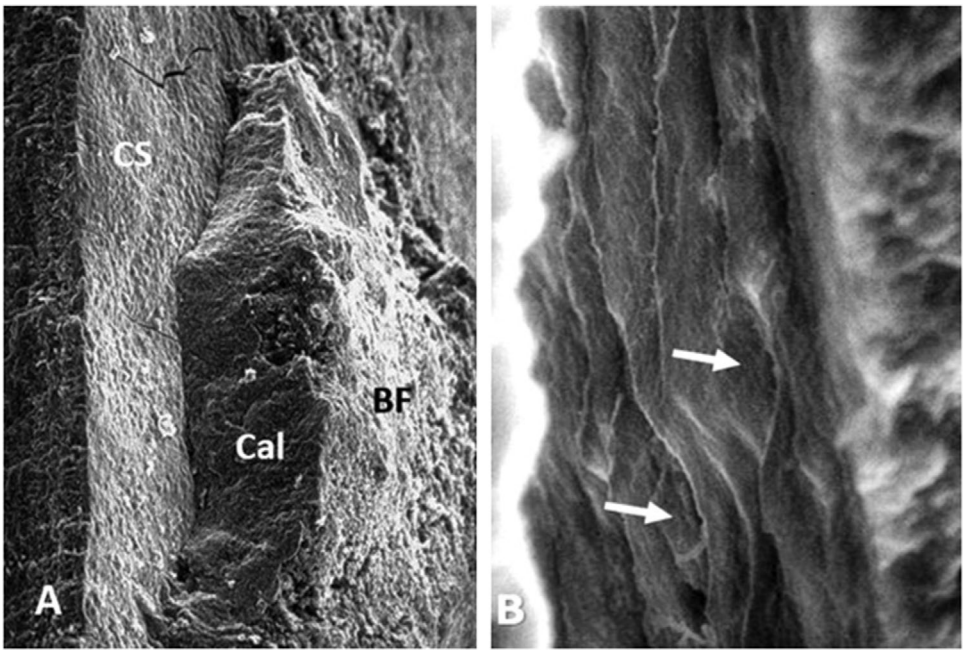

FIGURE 5 (**A**) Elevated mounds on the cemental surface (CS), representing insertion points of Sharpey's fibers are exposed after a section of calculus (Cal) with surface biofilm (BF) was removed. Original magnification 300×. (**B**) Calculus is lifted off a diseased root surface. Photo is focused on the underside of the calculus where the depressions (arrows) fit over the mounds on the cemental surface. Original magnification 1,500×

Lastly, root surfaces exposed as a result of periodontitis exhibit a surface coating that affords an attachment for microbiota and subsequent subgingival calculus. In the older literature such coatings were described as the dental cuticle and thought to be derived from epithelium. However, more recent evidence indicates the coating is derived from the adsorption onto the roots surface of components from gingival inflammatory exudate. Further, the coating is often mineralized and, like calculus, may be a reservoir for exogenous cytotoxic substances.[39] Perfunctory attempts at removing subgingival calculus and biofilm are unlikely to achieve removal of either the cuticle or the microorganisms. (Figure 6)

6 | CALCULUS ATTACHMENT TO THE ROOT SURFACE

As alluded to previously, calculus may not, in itself, induce inflammation in the approximated soft tissue pocket wall, but serves as an ideal substrate for subgingival microbial colonization and the concentration and release of bacterial toxins. Any discerning clinician has experienced that removal of subgingival calculus can on occasion be difficult, even when access is not constricted. The degree of difficulty encountered in calculus removal is related to location, hardness, and mode of attachment which, in turn, is mediated by cuticles, surface irregularities, bacterial penetration of cementum, undercuts in resorption lacunae, or penetration between separations of cementum.[40–42] It has been demonstrated, using transmission electron microscopy, that calculus can bind directly to the hydroxyapatite crystalline structure of cementum.[43] Fractographic analysis has shown such an attachment is stronger than the cohesive strength within the calculus itself—leading to incomplete removal of long-standing calculus deposits.[44] (Figure 4B) Direct binding of calculus to cemental hydroxyapatite may explain the findings of Harrell, et al.[45] that micro islands of calculus remain even after definitive SRP. Residual calculus deposits can promote further calculus formation by serving as a site for nucleation of calcium phosphate crystal growth and potential attachment sites for biofilm bacteria.

7 | RE-EVALUATION OF INITIAL THERAPY

The re-evaluation appointment after SRP is not an endpoint of treatment but rather is part of a decision matrix. Based the authors' clinical experience and a review of the periodontal literature, there is a high probability that all subgingival calculus and biofilm will not be removed during the initial SRP. It is prudent for the clinician to schedule a separate appointment 6 to 8 weeks after completion of therapy, allowing sufficient time to assess the presence of

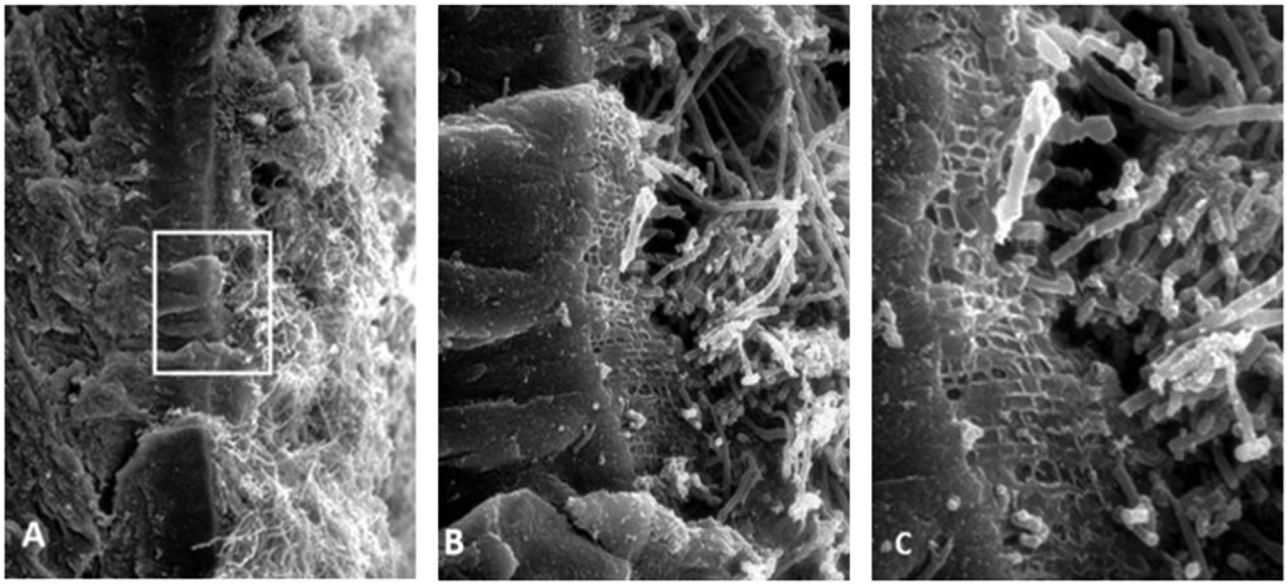

FIGURE 6 (**A**) Cross-section of cementum with subgingival biofilm attached to the surface. Boxed area indicates area of interest in **B**. Note the regimented and "lacy" morphology of the extracellular matrix with each space being the point of attachment of a single filament positioned at a 90^0 angle to the root surface. The fracturing process detached the organisms. Subsequent treatment with high powered water irrigation removed the blood and other contaminants related to the extraction process but did not disturb the bond of the bacteria to the cementum or to each other, indicating the cuticle has strong adhesive properties. Original magnification of 250× (**A**) and 1,500X×(**B**)

post-therapeutic health or disease. This assessment should use the same clinical parameters employed to diagnose the disease and to construct the treatment plan. Pocket depths and the presence of exudate or bleeding on probing (BOP) should be recorded. Exudate is a sign of acute pathology and should always receive further treatment until it is eradicated.

Although BOP is not an accurate indicator of progressive attachment loss, its absence is evidence of health.[46] Also, at the re-evaluation appointment, specific attention must be given furcation involvements as they represent sites that may experience rapid attachment loss.

Much like chronic systemic disease, the chronic nature of periodontal disease requires continuous monitoring and treatment. A "one size fits all" periodontal maintenance interval and/or protocol of treatment is inappropriate. Instead, the interval between maintenance appointments should be based on severity of disease and patient risk for recurrence of disease.

In this regard, Lu et al.[47] have reported that periodontitis patients that were treated and considered well-maintained still exhibit a subgingival dysbiotic microbial population than do healthy non-periodontitis patients. Thus, emphasizing the need for close monitoring and an individualized periodontal maintenance schedule.

8 | REFERRAL TO A SPECIALIST

It is widely known that practice management advisors encourage "soft tissue management" programs as an important income source for the general dental practice, often undermining timely referral to an appropriate specialist. SRP, with or without adjunctive therapies, may arrest periodontitis, but when it does not, the therapist is ethically obligated to inform the patient that more treatment is necessary and may require referral to a specialist. Indeed, the ADA *Principle of Ethics and Code of Professional Conduct* states: "Dentists shall be obligated to seek consultation, if possible, whenever the welfare of patients will be safeguarded or advanced by utilizing those who have special skills, knowledge, and experience."[48]

Ethical standards and the standard of care require that all clinicians keep current with both the clinical and scientific knowledge pertinent to the treatments rendered in their individual practices. This would include an understanding of the role of local factors in the initiation and perpetuation of periodontal disease and providing complete therapy to arrest the inflammatory process and achieve health. Simply stated, proper SRP mandates the removal of subgingival calculus and the biofilm. The scientific literature supports this requirement.

9 | CONCLUSIONS

Based on the extensive body of published evidence, the following conclusions can be made:

- A primary goal of SRP is the removal of calculus and biofilm deposits in order to create a biologically compatible root surface. Clinical studies have documented the beneficial effects of complete removal of subgingival calculus on the resolution of inflammation.
- Subgingival calculus surfaces are covered with a biofilm comprised of metabolically active bacteria. The bacteria of a subgingival biofilm can invade root surface irregularities and cavitational defects and root planing is required for eradication of such deposits.
- Endotoxin is adsorbed to the cementum and/or dentin surface but is easily removed without excessive instrumentation. However, the presences of calculus will impede removal of endotoxin.
- Periodontal destruction is strongly related to the presence of calculus.
- Because of increased or persistent probing depths, furcations, and anatomical and root surface irregularities, surgery may be required to remove residual calculus.
- As clinicians, our primary obligation is the best interest and welfare of the patient.
- Dentists are ethically obligated to seek consultation whenever the welfare of the patient will be safeguarded or advanced by utilizing those who have special skills, knowledge, and experience.

CONFLICTS OF INTEREST

Dr. Cobb and Dr. Sottosanti report no conflict of interest related to the content of this paper.

AUTHOR CONTRIBUTION STATEMENT

Both authors contributed equally to writing of the manuscript and the scanning electron microscope photographs were contributed by Dr. Sottosanti.

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