TIME TO SHIFT: FROM SCALING AND ROOT PLANING TO ROOT SURFACE DEBRIDEMENT

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ABSTRACT

Non-surgical periodontal treatment has traditionally been based on the notion that bacterial plaque (dental biofilm) penetrates and infects dental cementum. Removal of this infected cementum via scaling and root planing (SRP) was considered essential for re-establishing periodontal health. In the 1980s the concept of SRP was questioned because several in vitro studies showed that the biofilm was superficially located on the root surface and its disruption and removal could be relatively easily achieved by ultrasonic instrumentation of the root surface (known as root surface debridement (RSD). Subsequent in vivo studies corroborated the in vitro findings. There is now sufficient clinical evidence to substantiate the concept that the deliberate removal of cementum by SRP is no longer warranted or justified, and that the more gentle and conservative approach of RSD should be implemented in daily periodontal practice.

Periodontology has been extensively researched over the past 50 years. This includes the fields of periodontal pathology and periodontal therapy. The seminal work performed by Löe et al in the mid-1960s[1] confirmed the cause-and-effect relationship between bacterial biofilms (then referred to as dental plaque) and the host inflammatory response. Periodontal therapy has focused on the importance of removing 'accretions' and 'infected cementum' from the root surface, with particular emphasis on removing calculus and infected cementum as part of cause-related (whether non-surgical and/or surgical) periodontal therapy. This mode of treatment persists to some extent today, even though the biofilm has been established as the principal cause of periodontal diseases. This has led to a rift between the theoretical knowledge and scientific basis of the aetiology of periodontal diseases (caused by the bacterial biofilm) and the clinical methods sometimes employed in treating the disease (removal of calculus and 'infected' cementum).

It was not until the 1980s that the traditional therapeutic method of scaling and root planing (SRP) employed in daily clinical practice was questioned by a series of studies. The rationale behind these studies was to ascertain whether the deliberate removal of tooth (root) tissue during SRP, in order to achieve periodontal health, could be justified. The results of these studies, both in vitro and in vivo, showed that the biofilm, rather than calculus or 'infected' cementum, was responsible for disease and that it was superficially located on the root surfaces of periodontally involved teeth and could be easily removed (via root surface debridement (RSD) in order to achieve periodontal health; thus the deliberate removal of root substance via SRP appeared to be unjustified.

Treatment of periodontal diseases: a historical perspective

The first microscopic evidence of bacterial accretions around teeth was brought to light by Antony van Leeuwenhoek in 1683[2] some 200 years prior to the key study by Löe et al (1965)[1] that established the cause-and-effect relationship between dental plaque and the initiation of periodontal diseases. In this study, a group of dental students who had good periodontal health were

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asked to refrain from daily toothbrushing for three weeks. Periodontal indices were monitored before and after these three weeks, after which toothbrushing was reinitiated. The clinical data collated at the end of the study showed that dental plaque accumulation caused gingival inflammation and its removal led to resolution of clinical symptoms and restitution of periodontal health. Although this was the first scientific study that showed the interaction between dental biofilm and the host response, the need for removal of dental deposits around teeth had long been observed and acknowledged. Indeed, a primitive form of the modern-day toothbrush dates back some 3500BC; the toothbrush, as we know it today, originated in China about 1600. In his 1728 thesis *Le Chirurgien Dentiste*, Pierre Fauchard acknowledged the need for 'oral cleanliness' and proposed that teeth should be cleaned periodically by the dentist.[3] This mode of treatment was propagated well into the 19th and 20th centuries as the non-surgical[4] and surgical[5] treatment of pyorrhoea alveolaris. Application of these scraping treatment protocols was facilitated by the development of sharp periodontal instruments by GV Black (1915),[6] which formed the basis of root planing as a therapeutic technique.

SRP or RSD?

Traditional non-surgical periodontal therapy encompasses oral hygiene instructions and SRP performed as initial therapy and then routinely repeated, usually on a three-monthly basis. The frequency at which this is performed is generally determined by the patient's susceptibility to disease; in other words, the more susceptible the patient, the more often SRP is repeated. All clinicians agree that the patient's role in periodontal therapy is crucial; effective daily removal of biofilm performed by the patient not only leads to resolution of gingivitis,[1] but also profoundly impacts on the successful management of periodontitis.[7] Furthermore, effective daily removal of supragingival plaque by the patient prevents recolonisation of periodontal pockets. But to what extent is the repeated planing of the root surface justified, given that this is a destructive process that removes tooth structure? It is useful to define first what is meant by 'scaling' and 'root planing'.

Scaling

Scaling has been defined as instrumentation to remove all supragingival uncalcified and calcified accretions and all gross subgingival accretions.[8,9] This mechanical removal of calcified deposits (calculus) on teeth has been the gold standard for periodontal therapy for centuries. Indeed, there was a time when this was the mainstay of non-surgical periodontal treatment, with the emphasis placed on the removal of all calculus, rather than the dental biofilm.

Both supragingival and subgingival calculus form following accumulation of undisturbed biofilm. The exact role of subgingival calculus in the initiation and progression of periodontal disease remains debatable.[10] Clinicians are aware that there is considerable variation between patients in the amount of calculus formation, with some being very prone to extensive calculus formation, whereas others manifest very little. Often the volume of calculus formation does not appear to be commensurate with the extent of disease present; thus patients with aggressive periodontitis often display negligible calculus formation. There is also considerable variation between ethnic patient groups in the amount of calculus formation.[11-13] Other factors that affect the amount of calculus formation include age, gender, diet, location in oral cavity, oral hygiene, bacterial composition, host responses and access to professional treatment.[14]

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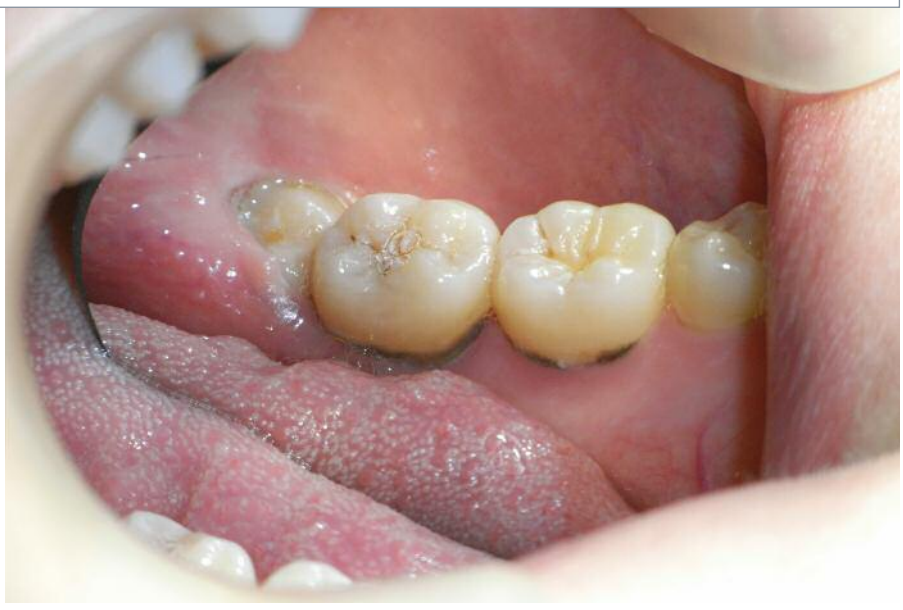

Figure 1: Periodontal healing in the presence of subgingival calculus

The pathological significance of calculus in the initiation and progression of periodontal disease has been implied based on a number of cross-sectional and longitudinal studies that have shown an association between the presence of calculus and periodontal disease;[15-18] however, a cause-and-effect relationship between calculus and periodontal disease has not been established. This is because of the difficulty in performing such a study given that calculus is invariably covered with a dental biofilm, which in itself initiates an inflammatory response in the host.[1]

Although calculus has a porous structure and has been shown to harbour several periodontal pathogens such as *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis* and *Treponema denticola*,[19] the essentially inert nature of calculus has been shown in a number of studies. For example, it has been shown that autoclaved calculus does not initiate an inflammatory response or cause abscess formation.[20] Furthermore, histological evidence is available to show that periodontal healing via a normal epithelial attachment is observed in the presence of calculus (Figure 1) once the biofilm has been removed from the surface of the calculus.[21,22] Clinical evidence of periodontal healing is evident in daily practice even though complete removal of calculus evades even the most experienced clinicians using a diverse range of instruments.[23-25]

As clinicians, we invariably leave behind extensive calculus deposits after scaling[26] and the amount of residual calculus seems to be proportional to the pocket depth and to tooth type, with molars manifesting the greatest degree of residual calculus.[27] However, despite our inability to remove all calculus, our patients still seem to improve following periodontal treatment – highlighting the importance of biofilm removal during therapy. The clinical benefit of scaling to remove calculus stems from the fact that calculus interferes with the patient's daily plaque control regimen; in other words, it presents an obstacle to self-performed biofilm control and also serves as an ideal substrate for bacterial colonisation. Subgingival calculus is no different in this respect from supragingival calculus. It should therefore be remembered that subgingival calculus forms as a result of the disease process rather than being the cause of it.[28]

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Root planing

Root planing has been defined as instrumentation to remove the microbial flora on the root surface or lying free in the pocket, all flecks of calculus and all contaminated cementum and dentine. The aim is to remove the softened cementum so that the root surface is made hard and smooth.[8,9] The rationale for root planing was based on the notion that once the root surface became exposed to the subgingival environment in periodontitis, it underwent both structural and pathological changes. Structural and topographical changes have been described, including the formation of resorption lacunae that could potentially lead to entry of bacteria and their products into cementum and radicular dentine.[29] Pathological changes in cementum were implied, based on the assumption that bacterial toxins or lipopolysaccharide (endotoxin) released by Gram-negative bacteria were adsorbed into the root surface. This led to the concept of cementum becoming 'infected' and therefore incompatible with attaching to healthy gingival/periodontal tissue.[30]

Endotoxin has several noxious properties: it is a potent inflammatory agent[31] and an inhibitor of cell proliferation, cell viability and gingival fibroblast reattachment to root surfaces.[30,32] It also inhibits bone growth[33] and induces bone resorption[34,35] and collagenase production by endotoxin-activated macrophages.[36] It was therefore inferred that removal of the bacterial endotoxin, located either supragingivally or subgingivally, was pivotal to the treatment of periodontitis.

Studies have shown that cementum from periodontally involved teeth had a significantly higher content of lipopolysaccharide when compared to non-periodontally involved teeth or control teeth (ie teeth extracted for non-periodontal reasons) when assayed using the Limulus amoebocyte lysate (LAL) test.[37] However, the extent to which endotoxin was adsorbed by cementum or whether it was only surface-move back became a contentious issue. Advocates of SRP claimed (and still do, to some extent) that the cementum became infected with endotoxin during the disease process, and that this layer of infected tooth structure had to be removed to achieve a biocompatible root surface to allow healing to take place, citing periodontal healing following SRP as evidence for this.

In the 1980s, several investigators[38,39] started to question the extent of penetration of endotoxin into cementum and therefore the actual clinical need to remove cementum as part of the therapeutic process, as performed during SRP. An in vitro study[38] used extracted teeth immersed in a solution of endotoxin for periods ranging between two to12 weeks, after which the teeth were subjected to radiographic and immunofluorescence techniques for localisation of the endotoxin. This showed that the endotoxin was located on the surface of the root and was not adsorbed, as had been surmised. A second in vitro study[40] demonstrated the ease with which endotoxin could be removed from the root surface of periodontally involved teeth which had been extracted. This study used the LAL test to assay the amount of endotoxin recovered from the root surfaces of teeth which had been extracted for periodontal reasons. Endotoxin quantification took place following rinsing and brushing the roots of these teeth (akin to root debridement) versus stripping the entire root surface (akin to root planing). The results of this study showed that over 99% of endotoxin was removed following rinsing and brushing only. These results were corroborated by findings from other in vitro [40,41] and in vivo studies.[42] These collective findings demonstrated unequivocally that bacterial endotoxin was superficially located on cementum and that it could be relatively easy to remove by simple measures not involving extensive removal of cementum.

Root surface debridement

The ease with which bacterial endotoxin can be removed from periodontally involved root surfaces[38-42] has profound clinical implications. Root planing can only be effectively performed with sharp hand instruments, in order to plane off the so-called infected cementum. However, if cementum does not become infected then removal of the root surface contaminants can be achieved with a much lighter form of instrumentation that does not remove tooth structure.

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A less invasive and gentler form of root surface instrumentation, which if used correctly does not result in tooth structure removal, can be provided by ultrasonic instrumentation. Smart et al (1990)[43] showed that by using ultrasonic instruments and adopting a 'conservative instrumentation regime of overlapping strokes and light pressure' for a limited period of time, periodontally involved root surfaces can be rendered free of bacterial endotoxin. Furthermore, this study showed that light ultrasonic instrumentation for less than 1s/mm^2 of root surface was all that was required to remove all bacterial contaminants. This amounts to about 17 seconds of instrumentation for an average pocket. This technique became known as root surface debridement, the aim of which is remove bacterial contaminants (principally the bacterial biofilm) without the intentional removal of tooth structure. Studies have provided evidence for the importance of the removal of subgingival plaque in the treatment of periodontitis,[44-46] and clinical studies in both animals and humans [42,47] have provided scientific evidence for RSD as a therapeutic modality for periodontal treatment. Ideally, RSD should be carried out using plain ultrasonic tips; use of diamond-coated ultrasonic tips should be avoided as this will lead to significant root surface loss.[48] The ultrasonic handpiece should be used on a low to medium power setting and the tip applied parallel to the tooth surface using multiple overlapping strokes.[49] Excessive instrument pressure, prolonged contact time or increased tip-to-tooth angle will all cause root damage.

The advantages of ultrasonic root debridement are multiple:

1. It is up to 10 times more conservative of root surface tissue.[50,51] Other studies differ in this regard,[52] possibly due to use of different types of instruments/instrument settings.
2. Micro-ultrasonic tips allow better access to the base of deep (>6mm) periodontal pockets[53,54] and within furcations.[55]
3. Being a non-invasive process, local anaesthesia is usually not required.
4. Greater cost effectiveness; manual instrumentation takes 20–50% longer to achieve the same clinical result.[56-58]
5. Greater comfort for the patient[59] and, possibly, the operator.
6. The possibility of full-mouth treatments, particularly if local anaesthesia is not employed.

Conclusion

Non-surgical periodontal therapy is effective because it removes the bacterial biofilm that is the primary aetiological factor initiating periodontal diseases; removal of other secondary factors, such as dental calculus, benefit the patient because calculus encourages plaque retention and interferes with effective biofilm removal. Once disease extends to involve the root surface, removal of the subgingival biofilm is crucial for the long-term success of periodontal therapy. Removal of the subgingival biofilm should be performed both by the patient, via effective supragingival cleaning and subgingival root brushing, and by the operator by RSD. Previous traditional methods such as SRP, which focused on removing the 'infected' cementum, inevitably involved excessive removal of root tissue and it is questionable whether such techniques, although effective, should have any place in modern periodontal therapy. Based on the available scientific evidence, it is time to shift from SRP to RSD.

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