1

The Treatment of Periodontal Disease

The Shift from "SRP" to "Periodontal Debridement"

CHAPTER OBJECTIVES

1) To provide a historical context regarding the development of instrumentation concepts in periodontal therapy.
2) To consider the historical focus on endotoxin, and how this led to the preeminence of root planing as a treatment strategy to remove calculus and cementum.
3) To consider the role of the plaque biofilm in driving periodontal inflammation, and the importance of the inflammatory host response in periodontal tissue breakdown.
4) To explain current understanding of periodontal pathogenesis, and how this has informed the development of modern periodontal treatment strategies.
5) To review the evidence that supports the paradigm shift away from root planing (a damaging form of periodontal instrumentation) to periodontal debridement, which achieves the aims of biofilm disruption and removal while at the same time preserving cementum.

Periodontal disease is not new. Archeologists have revealed evidence of alveolar bone loss indicating the presence of periodontitis in human remains dating from around 700,000 years ago (Dentino et al., 2013). Descriptions of what we now call periodontitis can be found in a number of ancient textbooks and manuscripts, such as *al-Tasrif*, the medical encyclopedia written by Albucasis (936–1013) in Moorish Spain, which also included depictions of instruments to remove calculus. This document was translated into Latin during the twelfth century and was one of the primary medical texts used in European universities until the seventeenth century.

Over the centuries, and especially during recent decades, our understanding of periodontal diseases has developed significantly, and as a result, so have the treatment strategies that are used to manage the condition. To help us decide which treatment methods are the most appropriate for modern day clinicians to utilize, it is important to briefly review some of the recent advances that have been made in periodontology, as these have greatly influenced the treatment protocols that have been used in clinical practice over recent years.

Early Concepts of Perodontal Pathogenesis

Calculus the Irritant

If we spend a moment to consider the likely oral health status of many of the people living in the Middle Ages, in the time of Albucasis,

Ultrasonic Periodontal Debridement: Theory and Technique, Second Edition. Marie D. George, Dani Botbyl, Timothy G. Donley, and Philip M. Preshaw.

Companion Website: www.wiley.com/go/george/ultrasonic

for example, we might imagine abundant calculus deposits, inflamed gingival tissues, gingival bleeding, and halitosis. It is understandable that early dentists focused on the role of calculus "accretions" as the cause of the problem, and developed methods for trying to remove the deposits. The etiological role of calculus in the pathogenesis of periodontal disease was unquestioned for many centuries. In the USA, the pioneering clinician Riggs (1810–1885) regarded calculus as the cause of periodontal disease and advocated treating the condition by the meticulous removal of calculus from pockets, curettage of the soft tissues (explained shortly), and oral hygiene instruction (Dentino et al., 2013).

The emergence of microbiology as a discipline, coupled with improvements in microscopy, led to studies of the bacterial composition of dental plaque. The term *pyorrhea alveolaris* was introduced in the late nineteenth century to denote conditions in which gingival pockets developed, which permitted bacteria to "infect and destroy" the periodontal tissues and the alveolar bone. During this era, the importance of local factors in the etiology of periodontal disease was unquestioned and calculus was viewed as being directly responsible for the tissue damage that was observed in patients with periodontitis. This concept led to the emergence of treatment strategies that focused on calculus removal as the endpoint of periodontal therapy.

The Role of Plaque

The etiological role of plaque in the development of gingival inflammation was confirmed in experimental gingivitis studies conducted in the 1960s: upon cessation of oral hygiene practices over periods of 3–4 weeks, plaque accumulation resulted in gingivitis (Figure 1.1), which was reversed following plaque removal and resumption of normal oral hygiene (Loe and Silness, 1963; Loe et al., 1965). These studies were revolutionary in that they moved the focus of attention away from calculus and more toward plaque as the predominant etiological factor of periodontal diseases.

But how did plaque cause periodontal disease? Over the years, various theories were established based on prevailing knowledge at the time. For example, the *nonspecific plaque hypothesis* assumed that periodontal disease resulted from the production and release of harmful substances from the entire plaque mass. Inherent to this theory were the suppositions that (i) there must be a threshold for these substances to cause disease, above which periodontal disease will develop and below which it will not, and (ii) the quantity of plaque is the main determinant of risk for disease.

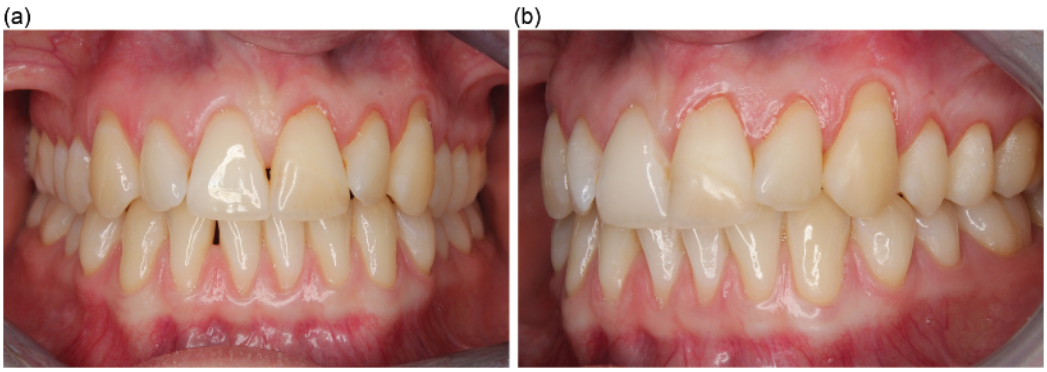

Figure 1.1 Experimental gingivitis in a 20-year-old male volunteer (anterior view – Figure 1.1a, buccal view – Figure 1.1b). Oral hygiene practices were suspended for three weeks in the upper left quadrant (with oral hygiene continuing as normal in the rest of the dentition). Note the gingival inflammation that has developed, affecting the gingival margins in the upper left quadrant (compare with the rest of the dentition).

In other words, this hypothesis suggested that the more plaque a person has, the more periodontal disease they will have. But clinicians will recognize that this does not always hold true; some patients have very poor oral hygiene and lots of plaque, and even though they may have gingivitis, they do not develop advanced periodontitis. Conversely, some patients with good oral hygiene and minimal plaque levels can develop advanced periodontitis, even at a relatively early age.

Further microbiological investigations led to the emergence of the *specific plaque hypothesis* (Loesche, 1976). This theory held that only certain types of plaque cause disease, because they contain specific bacteria that are particularly pathogenic; for example, they release irritants such as endotoxin (see following paragraphs), H_2S, lactic acid, and bacterial collagenase, which cause injury to the periodontal tissues. This concept was supported by evidence that the composition of subgingival plaque was different in healthy as opposed to diseased sites. It is noteworthy that both the nonspecific and the specific plaque hypotheses considered periodontal tissue breakdown to result from direct effects of harmful substances released from plaque bacteria.

Endotoxin

The term *endotoxin* was originally introduced to denote toxic substances within bacterial cells that were released upon death of the bacteria. Today, the term is typically used synonymously with the term *lipopolysaccharide* (LPS), which is a component of the cell wall of gram-negative bacteria. LPS consists of a polysaccharide chain linked covalently to a lipid moiety, and it is essential for maintaining the structural integrity of the bacterial cell wall. LPS induces strong immune and inflammatory responses in higher order species such as humans and other animals, which is why it is so important in the pathogenesis of a number of diseases, including periodontitis. LPS invokes strong immune–inflammatory responses precisely because it is present in gram-negative bacteria; higher order species have evolved to be able to detect and respond to LPS because it signals the presence of such bacteria.

Research in the 1960s and 1970s identified that endotoxin was present in the outer surfaces of cementum in teeth affected by periodontitis (Daly et al., 1980). It was hypothesized that this endotoxin would limit the effectiveness of periodontal therapy, because even if plaque and calculus were removed from the root surface, the endotoxin still present in the cementum would continue to irritate the tissues and thereby compromise healing following treatment. This presumption led to the preeminence of the treatment concept known as "root planing," often combined as a treatment strategy with scaling, and abbreviated to "SRP" ("*scaling and root planing*").

Scaling and Root Planing (SRP)

SRP became established as a periodontal treatment concept because of the prevailing belief that calculus, endotoxin, and necrotic cementum should be removed from the root surface. Necrotic cementum was considered to be that part of the cementum (the outer layer) that was impregnated with endotoxin from the overlying plaque mass. *Root planing* was therefore employed to remove this outer layer of cementum, by planing the root to remove the surface layer (*think about planing an over-sized door to make it fit the door frame better*).

What were the objectives of SRP?

SRP refers to two separate treatment techniques, scaling and root planing. Root planing was described as a treatment procedure in the early parts of the twentieth century (Hartzell, 1913; Stillman, 1917) and since then, there have been many different definitions of scaling and root planing in the periodontal literature. In the 1953 (first) edition of Glickman's *Clinical Periodontology* textbook, scaling is

described to remove calculus deposits and to "smooth the tooth surface" by removal of "softened, necrotic cementum" (Glickman, 1953). Another treatment strategy, *curettage*, was also described as the "management of the inner surface of the soft tissue wall" of the pocket, in which the epithelial lining of the pocket was forcibly removed (with a curette) to create a bleeding connective tissue surface against the root, which was believed at that time to result in better healing. This was achieved by applying pressure to the outside of the pocket (i.e., the free gingiva) with a finger while performing the upstroke with a curette located in the pocket, so that the pocket epithelium was stripped off. However, curettage is no longer undertaken, because of the pain and tissue damage it causes, and also because it was shown that outcomes following SRP with curettage were the same as those following SRP alone (Echeverria and Caffesse, 1983).

To add to the complexities that have existed around periodontal treatment terminologies, the term *curettage* has sometimes been used interchangeably with the term *root planing*, even though the two treatments had different objectives. Our interpretation of these various terms is presented in Box 1.1. For additional information, we also present MeSH terms that describe periodontal treatments (Box 1.2). As explained shortly, the preferred treatment strategy is periodontal debridement, defined as instrumentation performed to disrupt and remove the subgingival biofilm and to remove calculus, but without intentional removal of cementum.

Box 1.1 Periodontal treatment terminology as utilized in this book

Scaling Instrumentation performed to remove calculus deposits, both supragingival and subgingival, and without damaging the tooth surface

Root planing Instrumentation performed to remove subgingival calculus and necrotic cementum (and thereby endotoxin) from the root surface

Curettage Instrumentation performed to remove the soft tissue lining of the periodontal pocket (obsolete treatment strategy)

Periodontal debridement Instrumentation performed to disrupt and remove the subgingival biofilm and to remove calculus, but without intentional removal of cementum

Box 1.2 MeSH terms that describe periodontal treatments (*Source:* National Library of Medicine / Public Domain)

MeSH terms

MeSH terms are *Me*dical *S*ubject *H*eadings. They are a series of definitions created by the United States National Library of Medicine that are used for indexing journal articles and books. MeSH terms in relation to periodontal treatment include:

Dental scaling

Removal of dental plaque and dental calculus from the surface of a tooth, from the surface of a tooth apical to the gingival margin accumulated in periodontal pockets, or from the surface coronal to the gingival margin (year introduced: 1991, updated from 1972).

Root planing

A procedure for smoothing of the roughened root surface or cementum of a tooth after subgingival curettage or scaling, as part of periodontal therapy (year introduced: 1992).

(Continued)

Box 1.2 (Continued)

Subgingival curettage

Removal of degenerated and necrotic epithelium and underlying connective tissue of a periodontal pocket in an effort to convert a chronic ulcerated wound to an acute surgical wound, thereby insuring wound healing and attachment or epithelial adhesion, and shrinkage of the marginal gingiva (year introduced: 1965).

Periodontal debridement

Removal or disruption of dental deposits and plaque-retentive dental calculus from tooth surfaces and within the periodontal pocket space without deliberate removal of cementum as done in root planing and often in dental scaling. The goal is to conserve dental cementum to help maintain or reestablish a healthy periodontal environment and eliminate periodontitis by using light instrumentation strokes and nonsurgical techniques (e.g., ultrasonic, laser instruments) (year introduced: 2011).

Authors' comments

These MeSH definitions are generally well aligned with the terminology utilized in this book (Box 1.1). Importantly, the MeSH definition of *periodontal debridement* emphasizes that cementum is not deliberately removed and distinguishes the procedure from *root planing*. By simply referring to smoothing of roughened surfaces, the MeSH definition for *root planing* does not fully convey the damaging nature of the procedure. Finally, as noted in Box 1.1, *subgingival curettage* is an outdated treatment modality.

Notes: These terms were taken from the National Library of Medicine website: https://www.nlm.nih.gov/mesh/meshhome.html (accessed May 2023).

The rationalization for root planing

Given the historical belief that calculus was the primary etiology of periodontitis, it is understandable that treatment strategies focused on the complete removal of all calculus. As written in 1953, "every speck of it must be removed" (Glickman, 1953). However, we recognize now that although calculus is plaque retentive and compromises effective oral hygiene (as well as being unsightly), it is not the primary etiology of periodontitis (Akcali and Lang, 2018). Furthermore, complete removal of calculus is rarely achieved. A number of studies have addressed this issue. In a study of 690 root surfaces in 11 patients with periodontitis, the percentage of surfaces with residual calculus following instrumentation with a sonic scaler was 32%, with manual instruments was 27%, and with both types of instruments used together was 17% (Gellin et al., 1986). The researchers also found that deeper pockets were associated with more residual calculus following instrumentation. In another study, 476 surfaces on 101 extracted teeth were instrumented using both ultrasonic and hand instruments. Following the instrumentation, 19% of surfaces had residual calculus that could be detected clinically, and 57% of surfaces had residual calculus on examination under the microscope (Sherman et al., 1990). In a study of 21 patients requiring extractions, SRP was provided prior to the extractions and the percentage of subgingival surfaces that were free of calculus was determined with a stereomicroscope (Caffesse et al., 1986). In 4–6 mm pockets only 43% of surfaces, and in pockets >6 mm only 32% of surfaces, were free of calculus after SRP. The extent of residual calculus was directly correlated with probing depth, and was greatest at the cemento–enamel junction, and in association with grooves and furcations (Caffesse et al., 1986).

Taken collectively, the outcomes from these and other studies confirm that instrumentation is effective in significantly reducing the amount of calculus on root surfaces. However, complete calculus removal is not usually achieved, and deeper pockets are more likely to harbor residual calculus following treatment, with anywhere from approximately 3% to 80% of instrumented root surfaces showing some residual calculus after instrumentation (Claffey et al., 2004).

Furthermore, it is difficult for clinicians to reliably determine the removal of calculus during treatment (Figure 1.2). For example, in the study described earlier, there was a high false-negative response rate in that 77% of surfaces that were determined to have residual calculus present by microscope had been clinically scored as being free of calculus (Sherman et al., 1990). This underscores the difficulties of determining the thoroughness of subgingival instrumentation; calculus detection is technically challenging due to the complex anatomy of the pocket environment. While supragingival calculus can be identified by direct vision and drying of the tissues, subgingival calculus is much more difficult to identify unless the deposits are very large and can be detected by a periodontal probe or calculus explorer, or can be seen at the pocket opening. Radiographic detection of calculus is similarly unreliable; a sensitivity for radiographic detection of calculus of only 44% has been reported (i.e., only 44% of surfaces known to have calculus present clinically could be detected on radiographic examination) (Buchanan et al., 1987).

Besides calculus removal, the other objective for root planing is removal of cementum as the means by which to remove endotoxin (sometimes described as the removal of "contaminated cementum"). Early investigators reported that endotoxin was present in the cementum of teeth affected by periodontitis and that it was biologically active with an inhibitory effect on cellular function (Aleo et al., 1974). Further studies reported that fibroblasts did not attach to periodontally compromised roots until after the cementum was removed, or the endotoxin was removed chemically (Aleo et al., 1975; Assad et al., 1987). These studies implied that for periodontal treatment to be successful, there needed to be meticulous removal of cementum and endotoxin from the root surfaces. Further justification for this approach came from studies that showed that root planing resulted in significant reductions in the amount of endotoxin present, typically rendering the root surfaces nearly free of endotoxin (Jones and O'Leary, 1978). It was considered that removing the endotoxin creates a root surface that is more "biologically acceptable" or "compatible with wound healing," though this concept has rarely been defined in a clinical context (most studies that investigated the impact of endotoxin removal were laboratory-based studies that

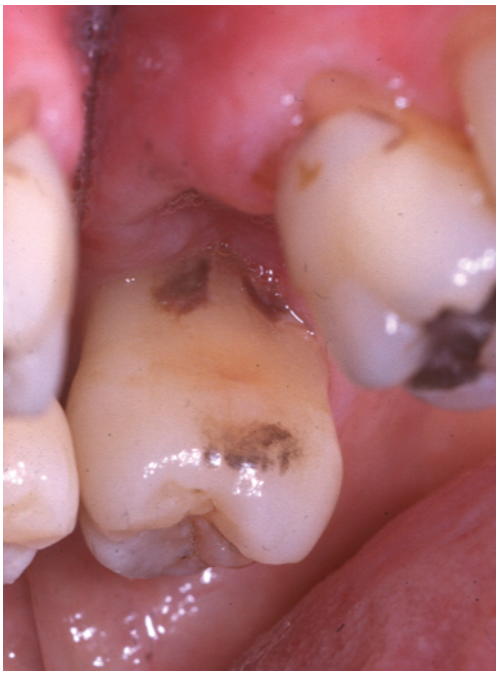

Figure 1.2 Residual calculus that had been missed during previous periodontal instrumentation. This patient had received previous periodontal instrumentation on several occasions and was in the supportive periodontal care phase of therapy. Tooth #14 (FDI 26) was extracted because of endodontic problems, and as the extraction site healed, the tissues at the mesial aspect of #15 (FDI 27) receded to reveal subgingival calculus that had not been removed by the prior episodes of instrumentation (dark brown areas in two locations close to the gingival margin). *Note: mirror view.*

investigated, for example, colonization of root surfaces by fibroblasts before and after cementum/endotoxin removal). The outcome of these studies was that root planing was considered a beneficial treatment strategy because it resulted in cementum and endotoxin removal, but it was never fully clear as to how much cementum should be removed or whether excessive cementum removal could have unwanted effects.

Unwanted outcomes of root planing

As already described, root planing has two main aims: (i) removal of subgingival calculus, and (ii) removal of the outer layer of cementum (and thereby endotoxin). In order to achieve this, the root surface is planed with sharp instruments to remove the outer layers of cementum together with any overlying calculus. The main disadvantage of this treatment is that tooth substance (i.e., cementum) is physically removed and the main consequence of this is dentin sensitivity. Other disadvantages of root planing are that it can be unpleasant for the patient as a result of the force applied and scraping sensations, and it can result in significant post-treatment pain, often leading patients to self-medicate to relieve the pain (Pihlstrom et al., 1999). Root planing usually requires the use of local anesthetics, can take a long time to perform (raising concerns about cost effectiveness), and can be tiring (for both the patient and the clinician).

How much cementum is removed during root planing depends on many factors, such as the sharpness of the instrument, the adaptation of the instrument to the root surface, the number of strokes used, and the lateral force applied. The issue of tooth substance removal when using manual or ultrasonic instruments is discussed in more detail in Chapter 2. What constitutes a clinically acceptable amount of tooth surface removal during periodontal instrumentation is difficult to define. However, it has been suggested that a defect depth of 0.5 mm (i.e., a total cumulative removal of 0.5 mm of cementum) over 10 years (e.g., in a patient undergoing long-term periodontal maintenance) is the maximum that is clinically acceptable (Flemmig et al., 1997, 1998a, 1998b). On this basis, and assuming that the same root surfaces are instrumented each year, cementum removal per year should not exceed 50 μm (i.e., 0.05 mm per year, in other words 1/20th of a millimeter per year). Assuming one maintenance visit per year, this suggests that a single episode of instrumentation should not remove more than 50 μm of cementum. If there were four episodes of instrumentation per year, then each episode of instrumentation should not remove more than 12.5 μm of cementum to remain within the 50 μm per year limit. However, it has been shown that root planing instruments can rapidly exceed these limits (see Chapter 2). While these concepts are important to consider, it must also be borne in mind that it is actually impossible to quantify the depth of cementum removal that may be occurring during any particular root planing episode.

It is certainly possible for root planing to result in removal of the full thickness of cementum from a root surface (Figure 1.3). Achieving this could have significant negative impacts in terms of root sensitivity but was once considered a desirable aim of treatment (the removal of all "diseased cementum" was a treatment endpoint). The complete removal of cementum as a treatment strategy was tested in an experimental periodontitis study in dogs in which a surgical approach to expose root surfaces was utilized (Nyman et al., 1986). In test quadrants, all the cementum was planed from the root surfaces, whereas in control quadrants the roots were polished with rubber cups and polishing paste. After two months, histological assessments indicated that healing was similar in all quadrants, whether or not the cementum had been removed. It was concluded that the removal of root cementum for the purpose of eliminating endotoxin does not seem to be necessary for achieving healing following therapy.

The same research design was then applied in a human study of 11 patients treated surgically using a split-mouth design. In two quadrants,

Figure 1.3 This lower premolar was an abutment for a cantilever bridge and was extracted as a result of progression of periodontitis, severe mobility, and sensitivity. The patient had received from a previous clinician many courses of root planing, which resulted in the removal of all the cementum and much of the dentin at the distal aspect of the root, with subsequent pulpal involvement. (*Source:* Courtesy of Dr. Ian Dunn).

teeth were root planed to remove all calculus and all cementum while in the other two quadrants, only calculus was removed (Nyman et al., 1988). The patients were monitored for 24 months. Outcomes of treatment (probing depth reductions and attachment gains) were similar following both treatment modalities, suggesting that there was no benefit of complete cementum removal. This led the authors to further question the prevailing dogma that "infected" root cementum should be removed by root planing (Nyman et al., 1988).

LPS is loosely adherent to the cementum

The historical justification for root planing was supported by studies which indicated that LPS was firmly attached to the cementum, and this was considered to negatively impact on healing (Aleo et al., 1974, 1975; Assad et al., 1987). Cementum that contained LPS was considered to be "infected," "necrotic," or "contaminated," and this led to the rationale for root planing, which aimed to remove the outer layer of the cementum in order to remove endotoxin (Jones and O'Leary, 1978; O'Leary, 1986).

However, subsequent studies reported contrasting findings that questioned the necessity for root planing in order to remove endotoxin. It was identified that LPS did not penetrate significantly into the cementum (it was located at the surface), and most of the LPS present was associated with tooth deposits and bacteria on the root surface (Hughes et al., 1988; Hughes and Smales, 1986, 1990; Ito et al., 1985). Furthermore, studies of extracted teeth indicated that LPS was superficially (i.e., loosely) bound, and could be removed by brushing, leading to the suggestion that root planing to remove cementum was not indicated. For example, in a study of teeth that had been extracted because of advanced periodontitis, it was identified that 39% of the LPS present in the cementum could be removed by gentle washing in water for 1 minute and a further 60% could be removed by brushing for 1 minute (Moore et al., 1986). In other words, 99% of the LPS was removed by comparatively gentle procedures. Further studies confirmed that as few as 15 strokes with a hand instrument resulted in significant reductions in the quantity of endotoxin within root surfaces, suggesting that extensive root planing was not warranted as a periodontal treatment strategy (Cheetham et al., 1988). The concept that LPS should be regarded as being *associated* with cementum, rather than being *bound* to cementum, was further supported by research on extracted teeth that were immersed in LPS for periods of 2–12 weeks. The researchers identified that LPS adhered to the surfaces only (whether teeth were healthy or had been affected by periodontitis) and did not penetrate into the cementum, and that the binding of LPS to the root surfaces was weak (Nakib et al., 1982).

To summarize, while it is clear that LPS is present in cementum of periodontally affected teeth (Maidwell-Smith et al., 1987; Wilson et al., 1986),

the great majority of the LPS is located at the cementum surface and is mainly associated with the subgingival biofilm, rather than being firmly bound to the cementum itself. LPS can be removed by gentle techniques such as washing with water or polishing the root surface, and therefore root planing in order to remove LPS cannot be justified given the damage that this procedure causes to the root surface.

Current Concepts of Periodontal Pathogenesis

Some of the most significant research that has influenced our understanding of periodontal pathogenesis was conducted in Sri Lanka. A population of 480 male laborers working in two tea plantations underwent periodontal assessments at regular intervals over a 15-year period (Loe et al., 1986). These individuals had no access to dental care and had not received any periodontal treatment at any point in their lifetime. The participants did not follow any conventional oral hygiene measures and, as a result, had abundant plaque and calculus deposits throughout the dentition. Yet, despite the fact that plaque and calculus were present in large quantities in all participants, they did not all have periodontitis. In fact, the population could be subdivided into three groups: (i) those with rapid progression of periodontitis (approximately 10% of the total population), (ii) those with moderate progression (~80%), and (iii) those with no progression of periodontal disease beyond gingivitis (~10%). The annual rate of disease progression varied between 0.1 mm and 1.0 mm in the rapid progression group, and between 0.05 mm and 0.5 mm in the moderate progression group. This study confirmed that not all people are equally susceptible to periodontitis (Figure 1.4), despite the fact that plaque bacteria are ubiquitously present.

Biofilm: the Driver of Periodontal Inflammation

Dental plaque is more accurately described as a biofilm. *Biofilms* are composed of microbial cells (e.g., bacteria) encased within a matrix of extracellular polymeric substances such as polysaccharides, proteins, and nucleic acids (Jakubovics et al., 2021). Biofilm structures vary according to the environmental conditions, but several features are common to most biofilms. For example, water channels are present which remove waste products and bring nutrients to the deeper layers of the biofilm. Surface structures such as fronds dissipate the energy of fluid flowing over the biofilm (and thereby protect against mechanical shearing forces). Microcolonies of bacteria may exist at

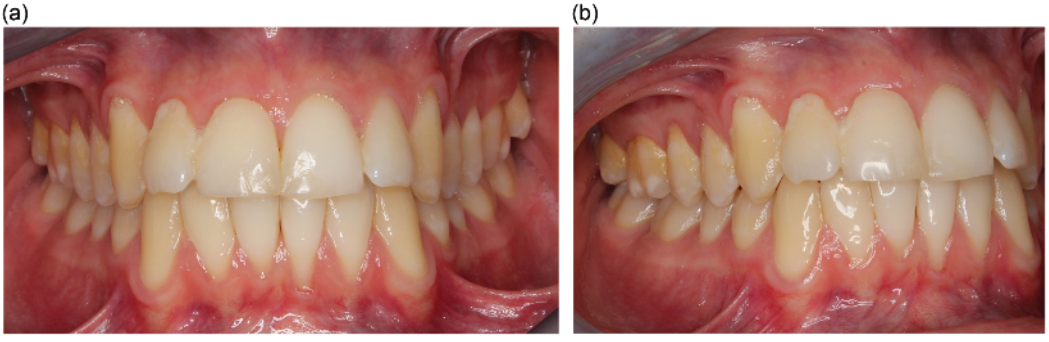

Figure 1.4 Experimental gingivitis in a 19-year-old male volunteer (anterior view – Figure 1.4a, buccal view – Figure 1.4b). Oral hygiene practices were suspended for three weeks in the upper right quadrant (with oral hygiene continuing as normal in the rest of the dentition). Note that although abundant plaque deposits are present and gingival inflammation has developed, the degree of gingival inflammation is relatively modest (compare and contrast with the patient shown in Figure 1.1, who developed more pronounced gingival inflammation in response to a lesser quantity of plaque, potentially indicating a greater disease susceptibility).

discreet areas of the biofilm according to the local environment, and steep chemical gradients (e.g., in oxygen levels or pH) can exist, which create distinct microenvironments within the biofilm.

The formation of the plaque biofilm can be divided into phases: (i) formation of pellicle on the tooth surface, (ii) early attachment of bacteria, and (iii) increased complexity and maturation. *Pellicle* (usually referred to as acquired pellicle) is an organic material that coats all hard and soft surfaces in the oral cavity. Proteins and glycoproteins from saliva continually adsorb onto the tooth surface, forming the acquired pellicle. These interact with oral microorganisms, functioning as adhesion sites for bacteria.

The initial attachment of bacteria to the pellicle commences within minutes, and the early colonizers (i.e., those bacteria which adhere to the pellicle first) are typically those which possess surface adhesins (molecules that allow them to attach to the pellicle). In health, gram-positive cocci and rods predominate. *Actinomyces* species (gram-positive rods) such as *Actinomyces naeslundii* are among the first species to colonize the tooth surface. They can coaggregate with other early colonizers such as *Streptococcus* to form early plaque biofilm. Other gram-positive species also highly prevalent in health include *Streptococcus sanguinis*, *S. oralis*, *S. intermedius*, *S. gordonii*, and *Peptostreptococcus micros*. Gram-negative species are also found in early biofilm, such as *Veillonella parvula, V. atypica*, *Capnocytophaga ochracea*, and *C. gingivalis*. In addition, the gram-negative filamentous rod *Fusobacterium nucleatum* is also found in health, further indicating that both gram-positive and gram-negative species are present at healthy sites (Curtis et al., 2020).

The early colonizers provide additional binding sites for adhesion by other bacteria which are not able to attach to pellicle. Furthermore, the metabolic activity of the early colonizers affects the local biofilm environment, for example, they utilize oxygen which results in lower oxygen tension that permits the survival of more anaerobic species. The coadhesion of multiple bacterial species leads to increasing complexity and maturation of the biofilm. As the clinical signs of gingivitis develop, there is increased abundance of species such as *V. parvula, Actinomyces* species, *F. nucleatum, S. mitis*, and *Prevotella* species. This shift in composition characterized by reduction of gram-positive species and enrichment of gram-negative species is also accompanied by an increase in the physical mass of the biofilm.

As periodontitis develops, there are further major shifts in the composition of the subgingival biofilm, characterized by the predominance of gram-negative bacterial species, including those classically described as the red-complex species (*P. gingivalis, Tannerella forsythia*, and *Treponema denticola*), which are much more commonly found in deep pockets in patients with advanced periodontitis compared to shallow sites in healthy patients (Socransky et al., 1998). Abundant species at periodontitis sites also include *Treponema* species, *Prevotella intermedia*, *T. denticola*, and *Fretibacterium* species. During periodontitis, major shifts in the composition of the subgingival biofilm characterized by enrichment of mainly gram-negative anaerobic species occur (Abusleme et al., 2021). Similar to gingivitis, the increase in complexity of the biofilm in periodontitis is also accompanied by a large increase in the overall bacterial biomass (Curtis et al., 2020; Sedghi et al., 2021).

The biofilm associated with disease is, therefore, a highly complex environment of multiple bacterial species cohabiting within the matrix that the bacteria produce. The matrix is a defining characteristic of bacterial biofilms, and typically contains proteins, carbohydrates, nucleic acids, peptidoglycans, and lipids (Jakubovics et al., 2021). The matrix plays a key role in adhesion of the biofilm to the underlying tooth surface, as well as in maintaining a homeostatic environment for the bacterial cells contained within it (Hobley et al., 2015). Communication between bacteria is enabled by the biofilm, a process known as quorum

sensing. For example, bacteria may secrete a signaling molecule that accumulates locally and triggers a response (e.g., the expression of specific genes) once a certain cell density has been reached; in other words, when the bacteria sense that the bacterial population has reached a critical mass, or quorum.

Importantly in the context of oral hygiene procedures and periodontal treatment, the biofilm is able to resist mechanical challenge to a certain degree, due to the physical properties of the matrix. The extracellular proteins, polysaccharides, and macromolecules in the matrix enable the biofilm to resist deformation and removal, which coupled with the complex anatomy of the tooth and root surfaces, ensures that biofilm accumulation occurs readily in anatomical niches such as the subgingival environment.

As knowledge of the complexity of the plaque biofilm has increased, our understanding of the role it plays in disease pathogenesis has also evolved. Thus, as discussed earlier, we have progressed from the *nonspecific plaque hypothesis* to the *specific plaque hypothesis*, and then to the *ecological plaque hypothesis* (Marsh, 1994; Marsh et al., 2011). This hypothesis holds that both the total amount of plaque biofilm and its specific microbial composition contribute to disease development and progression. In health, the biofilm composition is relatively stable, in a state of dynamic equilibrium (or microbial homeostasis), and in balance with a steady-state low level immune-inflammatory response in the gingival tissues (but without clinical signs of inflammation at this stage). Changes to this steady state may result from perturbations of the host response, such as brought about by an increase in the accumulation of biofilm, or changes in host factors (e.g., changes in hormone levels such as those occurring during pregnancy), or changes in environmental factors (e.g., caused by smoking). Accumulation of biofilm results in increased inflammation in the gingival tissues (leading to the clinical signs of gingivitis) and in turn, the inflammatory response alters the biofilm environment; for example, gingival crevicular fluid (GCF) flow increases, which may favor the growth of certain disease-associated species that utilize GCF constituents as a nutrient source (i.e., an ecological shift occurs). Tissue degradation, increased GCF flow and inflammation all can result in further shifts in the microbial population, favoring the growth of the predominantly anaerobic pathogenic species that have been associated with more advanced disease.

Building on these concepts further, the importance of *dysbiosis* of the biofilm has been recognized. Dysbiosis is characterized by an imbalance in types of bacteria present in the biofilm, changes in the distribution of the microbiota and their functional and metabolic activities, and disruption of microbial homeostasis. As the biofilm accumulates at the gingival margin, inflammatory responses are triggered that result in increased GCF flow which in turn encourages the growth of species that can metabolize GCF components. Increased thickness of the biofilm alters oxygen levels and encourages growth of species that can tolerate reduced oxygen tension. Tissue disruption and presence of blood cells in the subgingival region permits enrichment of the biofilm with species that utilize hemin (derived from hemoglobin) as a source of iron. As biofilm growth continues, proliferating species themselves contribute to further dysbiosis. For example, *P. gingivalis* has been shown to affect the composition of the biofilm by disabling and deregulating aspects of the host immune-inflammatory response, creating an environment that permits further enrichment by other pathogenic species (Hajishengallis et al., 2012).

It is increasingly clear that there is a direct link between the subgingival environmental conditions and the quantity and composition of the biofilm. In other words, the inflammatory response is both precipitated and perpetuated by, and influences the composition of, the biofilm. In health, the inflammatory response and subgingival microbiome can be regarded as being in balance (homeostasis), whereas in disease, there is dysbiosis and imbalance

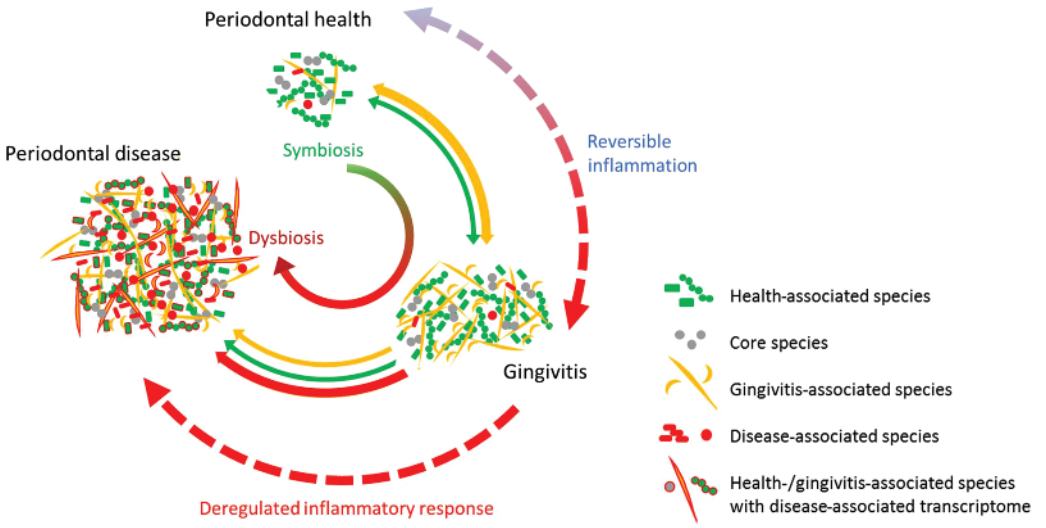

Figure 1.5 Bidirectional relationship between the subgingival microbiome and the immune-inflammatory response. In health, there is symbiosis, and the microbiota predominantly comprises health-associated species (green) with low abundance of disease-associated species (orange, red). In gingivitis, there is increased proliferation of gingivitis-associated species (orange), increased biofilm mass, and an increase in inflammation (which at this stage is reversible). In periodontitis, the microbiota becomes increasingly dysbiotic, with a reduction in health-associated species (green) and enrichment of disease-associated species (orange, red) together with increased biofilm biomass (green, orange, and red arrows). As a result, the immune-inflammatory response increases and extends further into the tissues, becoming increasingly dysregulated with tissue destruction occurring as periodontitis develops. (*Source:* Curtis MA et al., 2020 / John Wiley & Sons).

(Figure 1.5). Disruption and removal of biofilm by periodontal treatment and improved oral hygiene will not only control inflammation in the tissues but also play a role in the reversal of the dysbiosis (Curtis et al., 2020).

Periodontitis is an inflammatory disease

As described earlier, periodontitis was historically thought to result from tissue damage caused directly by noxious products released from bacteria in plaque. However, we now know that the majority of the tissue breakdown results from the host immune-inflammatory response to the biofilm (Van Dyke, 2008). Periodontitis is a complex inflammatory disease with multiple causative factors, including the presence of a dysbiotic and pathogenic biofilm which initiates and perpetuates the immune-inflammatory response, the nature of which is influenced by genetic, epigenetic, and environmental factors (such as smoking or diabetes), as well as behavioral factors (e.g., patient compliance) and site-specific characteristics (e.g., anatomical factors which influence biofilm growth). Periodontitis is characterized by an exaggerated, but poorly effective chronic (non-resolving) inflammation in the periodontal connective tissues, that results in tissue destruction and the development of clinical signs of disease (Chapple, 2009; Meyle and Chapple, 2015).

The recognition that the host immune-inflammatory response is responsible for the majority of tissue breakdown helps to explain why some people are more susceptible to periodontitis, why periodontitis can sometimes be observed to affect members of the same family (because aspects of immune-inflammatory responses can be genetically determined), and why some individuals appear to be relatively resistant to developing periodontitis despite the fact that they might have poor plaque control and gingivitis.

Even in clinically healthy tissues, evidence of (sub-clinical) inflammation can be detected at

the histological level, with a continuous (but low) flow of GCF, and migration of small numbers of neutrophils from the gingival capillaries through the tissues toward the sulcus in response to the presence of subgingival bacteria. As biofilm accumulation increases, the clinical signs of gingivitis develop in parallel with histological changes such as increased vascular permeability and vasodilatation, increased GCF flow, and increased infiltration of the tissues by leukocytes, particularly neutrophils and lymphocytes. Macroscopically, the tissues become red and swollen as a result of increased blood flow and permeability of the vessels, allowing fluid (and cells) to accumulate. As dysbiosis develops, the inflammation becomes more established, with accumulation of inflammatory cells in the connective tissues, increased release of matrix metalloproteinases (MMPs) and lysosomal contents from neutrophils, collagen destruction (resulting in collagen-depleted areas), and apical migration of the junctional epithelium to maintain an intact epithelial barrier. As the area of inflammation extends deeper into the underlying connective tissues, alveolar bone resorption commences, which retreats from the advancing inflammatory front. Thus, the clinical signs of periodontitis become evident as a result of breakdown of collagen fibers in the gingival connective tissue and periodontal ligament, apical migration of the junctional epithelium (resulting in increased probing depths), and alveolar bone resorption. A complex network of cytokines, destructive enzymes, and other inflammatory mediators plays a key role in the inflammatory process, and excessive, prolonged, and dysregulated inflammatory responses are responsible for the majority of the tissue damage that is observed (Kinane et al., 2011; Preshaw and Taylor, 2011).

Periodontal Debridement

Given that knowledge of periodontal microbiology and pathogenesis is continually evolving, it is important to interpret this information in the context of the clinical situation to inform the best treatment strategies for patients. Periodontal inflammation is initiated and perpetuated by the subgingival biofilm, and the inflammatory response causes the majority of tissue breakdown that leads to clinical signs of disease. Regularly disrupting and removing biofilm enables health-promoting bacterial species to become reestablished, and results in a reduction in inflammation (Meyle and Chapple, 2015). Resolution of inflammation and a return to a more health-promoting biofilm lead to symbiosis, with restoration of normal (non-inflamed) tissue function. Disease susceptibility influences the degree of biofilm removal necessary to achieve symbiosis; for some individuals (disease susceptible) even a small quantity of biofilm accumulation may trigger a destructive immune-inflammatory response and tissue damage (i.e., disease recurrence), whereas others appear to be disease resistant despite the presence of biofilm. Accordingly, periodontal treatment strategies should be tailored to the clinical situation, taking into account patient compliance, disease extent and severity, presence of risk factors, and response to treatment.

Treatment of periodontitis consists of active periodontal therapy (APT), which can be considered in a series of treatment steps (Sanz et al., 2020). A fundamental prerequisite to treatment is to inform the patient of the diagnosis, explain the etiology and role of risk factors in a personalized way, and discuss the treatment options and their risks and benefits. This discussion will pave the way for the development of a personalized care plan and the initiation of treatment. The first step of treatment includes improving patient oral hygiene, identifying and modifying risk factors (e.g., smoking cessation support), behavioral modifications (e.g., in relation to oral hygiene improvement or risk factor reduction) and removal of supragingival biofilm and calculus (Table 1.1). Step two is aimed at disrupting and removing subgingival biofilm and calculus, and response to treatment should be evaluated following this intervention. Step three is aimed at further treatment of those sites that have not responded fully (sites with pockets ≥4 mm with bleeding on probing (BOP) or presence of

Table 1.1 Treatment steps for the management of periodontitis.

Step 1	Behavior change and motivation	• Explain about the disease, risk factors, treatment options, and their risks and benefits (including no treatment) • Explain importance of patient-performed plaque control and support behavior change to improve oral hygiene • Risk factor reduction, including supporting behavior change (e.g., smoking cessation support) and removal of local plaque-retentive factors (e.g., calculus, overhangs) that impair oral hygiene • Supragingival dental biofilm and calculus removal (periodontal debridement)
Step 2	Subgingival periodontal debridement	• Continue to reinforce oral hygiene, risk factor reduction, and behavior change • Subgingival dental biofilm and calculus removal (periodontal debridement) • Evaluate treatment response (typically after approx. three months)
Step 3	Management of non-responding sites	• Continue to reinforce oral hygiene, risk factor reduction, and behavior change • Identify non-responding sites (sites with pockets ≥4 mm with bleeding on probing (BOP) or pockets ≥6 mm) • Repeat subgingival dental biofilm and calculus removal (periodontal debridement) at non-responding sites • Particularly for deeper non-responding sites (e.g., pockets ≥6 mm) consider alternative causes for the lack of response, and whether additional interventions (and/or referral) may be indicated, including surgical approaches (access flap surgery, resective surgery, or regenerative procedures) • Evaluate treatment response, and if endpoints of treatment are achieved (no pockets >4 mm with BOP and no pockets ≥6 mm) the patient can move into SPC (though it is recognized that these endpoints are not achieved in all patients)
Step 4	Supportive periodontal care (SPC)	• Ongoing provision of oral hygiene instruction, risk factor reduction, behavior change and support, according to the clinical situation • Regular targeted supragingival and subgingival dental biofilm and calculus removal (periodontal debridement) if disease recurrence is detected and to remove plaque-retentive factors • Recall frequency should be defined according to clinical need (typically will range from every 3–12 months)

pockets ≥6 mm) and may include repeated subgingival instrumentation to disrupt and remove biofilm, or surgical interventions. The response to the third step should be evaluated, and if the endpoints of treatment are achieved (no pockets >4 mm with BOP or no deep pockets ≥6 mm), the patient can be placed into supportive periodontal care (though it is recognized that these endpoints of therapy may not be achievable in all patients). Step four, supportive periodontal care (SPC) aims to maintain periodontal stability in treated periodontitis patients by ongoing provision of preventive and therapeutic interventions as defined in steps one and two, according to the clinical situation. SPC should be provided at regular intervals according to patient need, and at any SPC recall visit, re-instrumentation may be required if disease recurrence is observed. Reinforcement of the recommended oral hygiene procedures will need to be undertaken throughout all steps of treatment. At any stage of treatment, extractions may be considered for teeth that are considered to have a hopeless prognosis (Sanz et al., 2020).

This structured approach to periodontal therapy enables clinicians to have an endpoint in mind as they provide treatment for patients. For the majority of periodontitis patients, SPC will involve a life-long commitment to optimizing oral hygiene, compliance with periodontal recall visits, and potentially

involve episodes of re-instrumentation if disease recurrence occurs. It is important that patients are made aware of this as they proceed through their treatment. Plaque control (i.e., improved oral hygiene by the patient) and biofilm disruption and removal (i.e., instrumentation performed by the clinician) are the vehicles by which to reduce dysbiosis and periodontal inflammation, and thereby promote a return to symbiosis, with resolution of inflammation and stabilization of the periodontal tissues. Reduction of inflammation results in less bleeding and reduced pocket depths, which become easier to maintain and less likely to undergo disease progression compared to deeper pockets with persistent BOP. Reductions in pocket depths also result in environmental changes in the subgingival environment which favor health-promoting species in the biofilm.

Subgingival instrumentation (periodontal debridement) is a key component of all steps of periodontal therapy and may be undertaken with hand (manual) or powered (sonic/ultrasonic) instruments, either alone or in combination (Sanz et al., 2020). We consider that ultrasonic instruments are especially suitable for periodontal debridement because of their efficacy in disrupting and removing biofilm and calculus, yet with maximal conservation of tooth structure.

The objectives of modern periodontal debridement fit with our current understanding of periodontal disease processes, and include:

- disruption and removal of the subgingival biofilm
- removal of plaque-retentive factors such as calculus
- conservation of tooth structure
- creation of a biologically acceptable root surface
- resolution of inflammation.

These objectives are considered in more detail in Box 1.3. In broad terms, the overall aim of periodontal treatment is resolution of inflammation and a return to symbiosis, because uncontrolled and persisting inflammation in the tissues results in ongoing tissue damage (i.e., periodontitis progression). Improved oral hygiene by the patient and disruption and removal of biofilm by the clinician remain the cornerstones of periodontal therapy. Reduced dysbiosis, enrichment of health-promoting species in the biofilm, and reduced inflammation in the tissues result in pocket reductions (resolution of inflammation) and the shallower pockets are easier to maintain (by both patient and clinician).

The objectives of periodontal debridement listed in Box 1.3 would not be achieved by root planing, which has the objectives of removal of calculus and necrotic cementum from the root surface. The realization that LPS does not penetrate deeply into the cementum and is only loosely retained on root surfaces led to a paradigm shift in treatment concepts for periodontitis. This approach was pioneered by Kieser's research group who introduced the term *root surface debridement* (RSD) to indicate a light-touch, gentler form of instrumentation to promote biofilm removal, yet with preservation of cementum (Moore et al., 1986; Smart et al., 1990). This treatment approach seemed to fit well with the use of ultrasonic instruments which, as a result of their biophysical properties (discussed later in this book), could be utilized to facilitate biofilm disruption, but with the preservation of cementum.

In a study of periodontally compromised extracted teeth that were free of visible calculus, ultrasonic periodontal debridement applied with a force of approximately 50 g for a mean duration of 78 seconds (s) per root utilizing an overlapping "cross-hatching" instrumentation technique resulted in a reduction of LPS to levels that were similar to those found in healthy control teeth (Smart et al., 1990). Later work by the same group revealed that even in the presence of calculus, the use of ultrasonic instruments with light pressure and overlapping strokes resulted in mean reductions in LPS content per tooth from 1,900–29,200 ng prior to instrumentation to <22 ng post-instrumentation (for comparison, LPS levels at non-periodontally involved control

Box 1.3 Objectives of periodontal debridement

Disruption and removal of subgingival biofilm

The subgingival biofilm initiates and perpetuates the inflammatory response in the periodontal tissues, which in turn is responsible for the majority of the tissue breakdown that characterizes periodontitis. Treatment should aim to disrupt and remove the biofilm, with the aim of altering the pocket environment to reduce dysbiosis and promote a reduction in inflammation. This in turn will have clinical benefits as shallower pockets are easier to maintain by both the clinician and patient.

Removal of plaque-retentive factors such as calculus

Calculus does not cause periodontal disease. However, it is unsightly and biofilm-retentive. Therefore, visibly and tactilely detectable calculus deposits should be removed. However, it is recognized that complete calculus removal is seldom achieved, and this should not be the sole aim of treatment.

Conservation of tooth structure

Removal of cementum by periodontal instruments can result in sensitivity, and grooves or gouges in the root surface can be biofilm-retentive. Periodontal debridement techniques should therefore cause minimal damage to the root surface and should conserve tooth structure. This is particularly important given that supportive periodontal care (SPC) is a life-long requirement for most periodontitis patients, who may undergo multiple episodes of periodontal instrumentation over their lifetime.

Creation of a biologically acceptable root surface

This indicates a root surface that, following debridement, does not hinder resolution of inflammation or healing. It will be free of biofilm-retentive factors (such as visibly or tactilely detectable calculus), and will have undergone instrumentation procedures to disrupt, reduce, and remove biofilm.

Resolution of inflammation

The overall aim of periodontal debridement is resolution of inflammation, and that is achieved by the objectives outlined above, together with optimal plaque control by the patient.

teeth ranged from 15 to 28 ng) (Chiew et al., 1991). These reductions in LPS levels occurred despite the fact that not all the calculus was removed by the instrumentation procedure (planimetric assessment revealed that the mean percentage coverage of the roots by visible calculus was 74% before debridement and 34% after). The authors concluded that the therapeutic benefits of periodontal instrumentation are derived from the removal of biofilm rather than cementum or calculus.

Kieser proposed that periodontal therapy should be performed in a pragmatic, staged approach, adopting a periodontal debridement methodology (as opposed to root planing) that involves the use of periodontal instruments at light pressures with multiple overlapping strokes, the aim being to remove biofilm and to minimize removal of cementum. The success of treatment would be assessed not by surface characteristics following instrumentation (e.g., whether the root felt rough/smooth or soft/hard) but by the soft tissue response following therapy. The biological response should be the main measure of the success of therapy; in other words, whether inflammation has reduced, and healing has occurred. Kieser advocated that probing depths should be measured at three months following therapy, and for sites with persistent BOP, further instrumentation could be performed. By utilizing ultrasonic instruments, this treatment model would permit multiple episodes of instrumentation to disrupt and remove biofilm and reduce

Table 1.2 Key differences between Scaling and Root Planing (SRP) and Periodontal Debridement.

SRP	Periodontal Debridement
Previously, the nonsurgical treatment strategy was SRP (scaling and root planing). Why was this?	Modern understanding of the disease process indicates that periodontal debridement is the treatment of choice.Why is this?
Answer: Calculus was regarded as *the* etiological factor in periodontal disease, and therefore, every effort should be made to remove it.	*Answer*: Periodontitis results from a complex interplay between the dysbiotic subgingival biofilm and the host immune-inflammatory response. Calculus does not cause disease (though it is biofilm retentive).
And: Research seemed to suggest that endotoxin was embedded in the cementum, therefore, surface layers of cementum should be removed too.	*And*: It has been shown clearly that LPS is only loosely adherent to cementum; therefore, there is no need to intentionally remove cementum. Furthermore, cementum removal is damaging to the root surface and results in problems of pain and sensitivity (particularly if done repeatedly over the years).
The upshot of this: There was an emphasis on using sharp, bladed instruments to remove calculus and cementum and create a smooth, hard root surface.	*The upshot of this*: Ultrasonic instruments are indicated for root debridement, that is, to disrupt and remove biofilm and calculus, but without intentional removal of cementum. This is a gentler, less destructive form of treatment that achieves the same clinical outcomes, but without causing tissue damage. It is also more time efficient, and appropriate for use year-on-year in supportive periodontal care.

inflammation, but without causing extensive tissue damage or cementum removal. An overview of some key differences between SRP and periodontal debridement is shown in Table 1.2.

Following active periodontal treatment, patients with periodontitis enter supportive periodontal care, and may require repeated episodes of instrumentation if there is evidence of disease recurrence. Calculus should be removed as it is biofilm retentive, but calculus removal alone is no longer considered the endpoint of therapy. Root planing cannot be justified as a treatment concept because (i) it causes unnecessary damage to the root surface, (ii) its target outcome (i.e., calculus and cementum removal) cannot be verified, and (iii) it is not necessary to plane roots for the purposes of endotoxin removal. Instead, we consider that ultrasonic periodontal debridement is the treatment of choice for modern nonsurgical periodontal therapy.

References

Abusleme L, Hoare A, Hong B-Y, Diaz PI. Microbial signatures of health, gingivitis, and periodontitis. *Periodontol 2000* 2021; 86: 57–78.

Akcali A, Lang NP. Dental calculus: the calcified biofilm and its role in disease development. *Periodontol 2000* 2018; 76: 109–15.

Aleo JJ, De Renzis FA, Farber PA, Varboncoeur AP. The presence and biologic activity of cementum-bound endotoxin. *J Periodontol* 1974; 45: 672–75.

Aleo JJ, DeRenzis FA, Farber PA. An *in vitro* attachment of human gingival fibroblasts to root surfaces. *J Periodontol* 1975; 46: 639–45.

Assad DA, Dunlap RM, Weinberg S, Ahl D. Biologic preparation of diseased root surfaces. An in vitro study. *J Periodontol* 1987; 58: 30–33.

Buchanan S, Jenderseck R, Granet M, Kircos L, Chambers D, Robertson PB. Radiographic detection of dental calculus. *J Periodontol* 1987; 58: 747–51.

Caffesse RG, Sweeney PL, Smith BA. Scaling and root planing with and without periodontal flap surgery. *J Clin Periodontol* 1986; 13: 205–10.

Chapple ILC. Periodontal diagnosis and treatment – where does the future lie? *Periodontol 2000* 2009; 51: 9–24.

Cheetham WA, Wilson M, Kieser JB. Root surface debridement – an in vitro assessment. *J Clin Periodontol* 1988; 15: 288–92.

Chiew SYT, Wilson M, Davies EH, Kieser JB. Assessment of ultrasonic debridement of calculus-associated periodontally involved root surfaces by the limulus amoebocyte lysate assay: an in vitro study. *J Clin Periodontol* 1991; 18: 240–44.

Claffey N, Polyzois I, Ziaka P. An overview of nonsurgical and surgical therapy. *Periodontol 2000* 2004; 36: 35–44.

Curtis MA, Diaz PI, Van Dyke TE. The role of the microbiota in periodontal disease. *Periodontol 2000* 2020; 83: 14–25.

Daly CG, Seymour GJ, Kieser JB. Bacterial endotoxin: a role in chronic inflammatory periodontal disease? *J Oral Pathol* 1980; 9: 1–15.

Dentino A, Lee S, Mailhot J, Hefti AF. Principles of periodontology. *Periodontol 2000* 2013; 61: 16–53.

Echeverria JJ, Caffesse RG. Effects of gingival curettage when performed 1 month after root instrumentation. A biometric evaluation. *J Clin Periodontol* 1983; 10: 277–86.

Flemmig TF, Petersilka GJ, Mehl A, Hickel R, Klaiber B. The effect of working parameters on root substance removal using a piezoelectric ultrasonic scaler in vitro. *J Clin Periodontol* 1998a; 25: 158–63.

Flemmig TF, Petersilka GJ, Mehl A, Hickel R, Klaiber B. Working parameters of a magnetostrictive ultrasonic scaler influencing root substance removal in vitro. *J Periodontol* 1998b; 69: 547–53.

Flemmig TF, Petersilka GJ, Mehl A, Rudiger S, Hickel R, Klaiber B. Working parameters of a sonic scaler influencing root substance removal in vitro. *Clin Oral Investig* 1997; 1: 55–60.

Gellin RG, Miller MC, Javed T, Engler WO, Mishkin DJ. The effectiveness of the Titan-S sonic scaler versus curettes in the removal of subgingival calculus. A human surgical evaluation. *J Periodontol* 1986; 57: 672–80.

Glickman I (1953). The scaling and curettage technique for the eradication of the periodontal pocket. In: *Clinical Periodontology*, pp. 716–24. Philadelphia: WB Saunders.

Hajishengallis G, Darveau RP, Curtis MA. The keystone-pathogen hypothesis. *Nat Rev Microbiol* 2012; 10: 717–25.

Hartzell TB. The operative and post-operative treatment of pyorrhea. *Dental Cosmos* 1913; 55: 1094–101.

Hobley L, Harkins C, MacPhee CE, Stanley-Wall NR. Giving structure to the biofilm matrix: an overview of individual strategies and emerging common themes. *FEMS Microbiol Rev* 2015; 39: 649–69.

Hughes FJ, Auger DW, Smales FC. Investigation of the distribution of cementum-associated lipopolysac charides in periodontal disease by scanning electron microscope immunohistochemistry. *J Periodontal Res* 1988; 23: 100–06.

Hughes FJ, Smales FC. Immunohistochemical investigation of the presence and distribution of cementum-associated lipopolysaccharides in periodontal disease. *J Periodontal Res* 1986; 21: 660–67.

Hughes FJ, Smales FC. The distribution and quantitation of cementum-bound lipopolysaccharide on periodontally diseased root surfaces of human teeth. *Arch Oral Biol* 1990; 35: 295–99.

Ito K, Hindman RE, O'Leary TJ, Kafrawy AH. Determination of the presence of root-bound endotoxin using the local Shwartzman phenomenon (LSP). *J Periodontol* 1985; 56: 8–17.

Jakubovics NS, Goodman SD, Mashburn-Warren L, Stafford GP, Cieplik F. The dental plaque biofilm matrix. *Periodontol 2000* 2021; 86: 32–56.

Jones WA, O'Leary TJ. The effectiveness of in vivo root planing in removing bacterial

endotoxin from the roots of periodontally involved teeth. *J Periodontol* 1978; 49: 337–42.

Kinane DF, Preshaw PM, Loos BG. Host-response: understanding the cellular and molecular mechanisms of host-microbial interactions-consensus of the Seventh European Workshop on Periodontology. *J Clin Periodontol* 2011; 38(Suppl 11): 44–48.

Loe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan labourers 14 to 46 years of age. *J Clin Periodontol* 1986; 13: 431–40.

Loe H, Silness J. Periodontal disease in pregnancy I. Prevalence and severity. *Acta Odontol Scand* 1963; 21: 533–51.

Loe H, Theilade E, Jensen SB. Experimental gingivitis in man. *J Periodontol* 1965; 36: 177–87.

Loesche WJ. Chemotherapy of dental plaque infections. *Oral Sci Rev* 1976; 9: 65–107.

Maidwell-Smith M, Wilson M, Kieser JB. Lipopolysaccharide (endotoxin) from individual periodontally involved teeth. *J Clin Periodontol* 1987; 14: 453–56.

Marsh PD. Microbial ecology of dental plaque and its significance in health and disease. *Adv Dent Res* 1994; 8: 263–71.

Marsh PD, Moter A, Devine D. Dental plaque biofilms: communities, conflict and control. *Periodontal 2000* 2011; 55: 16–35.

Meyle J, Chapple I. Molecular aspects of the pathogenesis of periodontitis. *Periodontol 2000* 2015; 69: 7–17.

Moore J, Wilson M, Kieser JB. The distribution of bacterial lipopolysaccharide (endotoxin) in relation to periodontally involved root surfaces. *J Clin Periodontol* 1986; 13: 748–51.

Nakib NM, Bissada NF, Simmelink JW, Goldstine SN. Endotoxin penetration into root cementum of periodontally healthy and diseased human teeth. *J Periodontol* 1982; 53: 368–78.

Nyman S, Sarhed G, Ericsson I, Gottlow J, Karring T. Role of "diseased" root cementum in healing following treatment of periodontal disease. An experimental study in the dog. *J Periodontal Res* 1986; 21: 496–503.

Nyman S, Westfelt E, Sarhed G, Karring T. Role of "diseased" root cementum in healing following treatment of periodontal disease. A clinical study. *J Clin Periodontol* 1988; 15: 464–68.

O'Leary TJ. The impact of research on scaling and root planing. *J Periodontol* 1986; 57: 69–75.

Pihlstrom BL, Hargreaves KM., Bouwsma OJ, Myers WR, Goodale MB, Doyle MJ. Pain after periodontal scaling and root planing. *J Am Dent Assoc* 1999; 130: 801–07.

Preshaw PM, Taylor JJ. How has research into cytokine interactions and their role in driving immune responses impacted our understanding of periodontitis? *J Clin Periodontol* 2011; 38(Suppl 11): 60–84.

Sanz M, Herrera D, Kebschull M et al. On behalf of the EFP workshop participants and methodological consultants. Treatment of stage I–III periodontitis-the EFP S3 level clinical practice guideline. *J Clin Periodontol* 2020; 47: 4–60.

Sedghi L, DiMassa V, Harrington A, Lynch SV, Kapila YL. The oral microbiome: role of key organisms and complex networks in oral health and disease. *Periodontol 2000* 2021; 87: 107–31.

Sherman PR, Hutchens LH, Jewson LG, Moriarty JM, Greco GW, McFall WT. The effectiveness of subgingival scaling and root planing. I. Clinical detection of residual calculus. *J Periodontol* 1990; 61: 3–8.

Smart GJ, Wilson M, Davies EH, Kieser JB. The assessment of ultrasonic root surface debridement by determination of residual endotoxin levels. *J Clin Periodontol* 1990; 17: 174–78.

Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL Jr. Microbial complexes in subgingival plaque. *J Clin Periodontol* 1998; 25: 134–44.

Stillman PR. The management of pyorrhea. *Dental Cosmos* 1917; 59: 405–14.

Van Dyke TE. The management of inflammation in periodontal disease. *J Periodontol* 2008; 79: 1601–08.

Wilson M, Moore J, Kieser JB. Identity of limulus amoebocyte lysate-active root surface materials from periodontally involved teeth. *J Clin Periodontol* 1986; 13: 743–47.