Health, Maintenance, and Recovery of Soft Tissues around Implants

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ABSTRACT

Background: The health of peri-implant soft tissues is one of the most important aspects of osseointegration necessary for the long-term survival of dental implants.

Purpose: To review the process of soft tissue healing around osseointegrated implants and discuss the maintenance requirements as well as the possible short-comings of peri-implant soft tissue integration.

Materials and Methods: Literature search on the process involved in osseointegration, soft tissue healing and currently available treatment modalities was performed and a brief description of each process was provided.

Results: The peri-implant interface has been shown to be less effective than natural teeth in resisting bacterial invasion because gingival fiber alignment and reduced vascular supply make it more vulnerable to subsequent peri-implant disease and future bone loss around implants. And we summarized common procedures which have been shown to be effective in preventing peri-implantitis disease progression as well as clinical techniques utilized to regenerate soft tissues with bone loss in advanced cases of peri-implantitis.

Conclusion: Due to the difference between peri-implant interface and natural teeth, clinicians and patients should pay more attention in the maintenance and recovery of soft tissues around implants.

KEY WORDS: osseointegration, peri-implant soft tissue, peri-implantitis, peri-mucositis

INTRODUCTION

When dental implants were first discovered and used to replace missing teeth, most clinicians focused primarily on implant stability as the biggest factor in and predictor for future success, and this trend has in part continued as the primary research criterion for implants. Osseointegration was and still is considered as the most important factor in maintaining implant stability, whereas the role of soft tissue healing and maintenance around implants has been somewhat neglected. More recently, evidence has demonstrated that the long-term survival of osseointegrated implants was also partly dependent on the transmucosal healing and stability around the implant collar, termed “peri-implant mucosa.” This attachment of the soft tissue to the coronal portion of an implant acts to provide a protective seal which prevents the development of bacterial invasion and future inflammation. Thus, the soft tissue seal is necessary for stable osseointegration and long-term survival of implants.

One of the key findings relating to peri-implant mucosa was the direction of gingival fibers compared with the natural tooth (Figure 1). This key difference explained the increased ability of bacteria to penetrate the epithelial layer and subsequent connective tissue thus increasing the breakdown of soft tissues around implants. Therefore, if patient compliance is not fully obtained and proper oral hygiene is not maintained, inflammatory changes in the soft tissues surrounding dental implants will develop. This inflammatory process in the peri-implant mucosa...
begins with reddening and swelling, and once bleeding on probing is initiated, the condition is then termed peri-implant mucositis. If this condition is left untreated, it may lead to progressive and irreversible destruction of implant-surrounding tissues including the loss of alveolar bone around dental implants and ultimately lead to implant failure.\textsuperscript{19} Thus, the structure and biological events that take place during osseointegration and soft tissue attachment are important components of implant survival and maintenance. This review article aims to describe the composition of soft tissues around implants, how to maintain their health and survival, and how to deal with peri-mucositis and peri-implantitis progression and its reversibility.

**BIOLOGICAL EVENTS DURING OSSEOINTEGRATION**

Osseointegration, which has also been called “functional ankylosis,”\textsuperscript{20} was initially defined as “a direct structural and functional connection between ordered, living bone and the surface of a load-bearing implant.”\textsuperscript{21} More recently, authors have modified the definition of osseointegration. In 2012, Zarb and Koka defined osseointegration as “a time-dependent healing process whereby clinically asymptomatic rigid fixation of alloplastic materials is achieved and maintained in bone during functional loading.”\textsuperscript{22} The definition explains in more detail that the stages of osseointegration are divided into three overlapping steps: early immune-inflammatory response, angiogenesis, and osteogenesis.\textsuperscript{23}

Interestingly, more recent evidence has demonstrated that a gap of about 60 μm between the implant surface and host bone is created,\textsuperscript{24,25} and it may extend to 100 to 500 μm over time.\textsuperscript{26} This gap typically contains a titanium oxide layer which comes into contact with blood plasma proteins and body fluids and is later adsorbed onto the implant surfaces immediately after implantation, forming a “conditioning film.”\textsuperscript{23,27} Several factors such as surface roughness\textsuperscript{28} as well as surface hydrophilicity\textsuperscript{29} are key determining factors influencing protein adsorption and are subsequently able to stimulate and induce cell attachment on the implant surface.\textsuperscript{30} The cell population which first occupies the implant surface is primarily composed of inflammatory cells, and many investigators refer to this original phase of implant healing as the “immune-inflammatory response.”\textsuperscript{23} Within 24 hours following implant insertion, neutrophils dominate the implant site. In the 2 to 4 days following, an increasing number of infiltrating macrophages and monocytes appear in the peri-implant gap. These cells are responsible for removing the debris, as well as secreting large quantities of cytokines and growth factors responsible for stimulating future mesenchymal cell recruitment and proliferation, angiogenesis, and collagen matrix deposition.\textsuperscript{31,32}

A simultaneous and equally important event that takes place during osseointegration is the formation of a clot and future stimulation of angiogenesis. On the first day after implantation, a blood clot is formed adjacent to the implant surface,\textsuperscript{23} and neovascularization begins within 24 hours. While infiltrating macrophages...
and monocytes migrate to the bone wound by day 4, the blood clot is gradually replaced by differentiating mesenchymal cells recruited from the bone marrow around newly developing blood vessels. These mesenchymal cells differentiate into osteoblasts which are influenced by both growth factors and surface topography where they begin to attach to the implant surface and deposit collagen matrix. New bone formation on the implant surface is observed in 5 to 7 days when calcification from the host bone onto the implant surface is also observed. By 4 weeks, new bone formation is observed on the implant surface (contact osteogenesis) connecting with bone formed on the host bone (distant osteogenesis). After 8 to 12 weeks, the peri-implant interface is completely replaced by mature lamellar bone in direct contact with the implant surface, thus completing the initial phase of osseointegration (Figure 2).

THE SHORTCOMINGS OF SOFT TISSUE HEALING AROUND IMPLANTS

One of the issues often overlooked during implant placement is the fact that implant insertion creates a wound in both hard and soft tissue. It is also noteworthy that soft tissues suffer more drastic changes than their bony counterparts. Typically, soft tissues need to regenerate a greater amount of tissue, and subsequent surgeries and/or temporary crown changes further aggravate soft tissues with a number of new adaptation periods during healing abutments, final abutments, imprints, and final restorations, offering numerous opportunities for inflammation to develop during each of these procedures. Immediately after dental implant insertion, the implant-mucosa interface also forms a blood clot that is infiltrated by incoming neutrophils. If bacterial invasion is not present, the initial mucosa begins forming a peri-implant seal by the fourth day postimplantation. This healing process takes 8 weeks to complete the perimucosa seal, whereby leucocytes are typically confined to the coronal portion of the implant, and collagen-producing fibroblasts are typically found in the apical portion of the peri-implant interface.

Histologically, the peri-implant mucosa is composed of a well-keratinized oral epithelium, sulcular epithelium, and a thin barrier epithelium facing the abutment equivalent to the junctional epithelium around teeth, termed the peri-implant junctional epithelium. The height of the peri-implant junctional epithelium is approximately 2 mm, and the connective tissue underlying this junctional epithelium is around 1.0 to 1.5 mm. Thus, the mean biological width (including the sulcus depth) may often exceed 3 mm. When biological width is reduced at any site of the peri-implant mucosa, marginal bone resorption is typically observed so that the biological width is adjusted to compensate for these changes. Many factors have been shown to influence biological width around implants.

Figure 2 Timeline for osseointegration of dental implants with respect to changes over time.
Various implant system (e.g., tissue-level vs bone level implants, one-piece vs two-piece), \(^{43-45}\) implant material (e.g., titanium, zirconium gold alloy), \(^{46}\) implant surface characteristics (macro design, topography, hydrophilicity, and surface coating), \(^{47,48}\) loading protocol (e.g., immediate vs early or delayed), \(^{49}\) and implant protocol (e.g., soft tissue flap design, flapless procedure) \(^{50,51}\) all have different effects on biological width. However, more in-depth research is needed to verify the significance of each of these factors.

With regard to the structure of the peri-implant mucosa, the amount and distribution of fibroblasts, collagen, and blood vessels is quite different from those of a natural tooth (Figure 1). In the supra-crestal soft connective tissue around implants, fewer fibroblasts and collagen fibers oriented in the axis of the implant are present than in natural teeth. Interestingly, mesenchymal cells with a high number of fibroblasts are found near the implant surface, typically interposed between collagen fibrils. \(^{52}\) One of the main differences among these collagen fibers is their orientation relative to natural teeth. In most cases, the collagen fibers are found oriented parallel or parallel-oblique to the smooth implant surface, whereas in natural teeth, the fibers are found perpendicular to the tooth embedded within cementum as Sharpey’s fibers. \(^{13,53,54}\) Some research has shown that rougher surfaces with either microgrooves or porous plasma-sprayed surfaces form collagen fibrils that are oriented slightly more to the perpendicular than their smooth surface counterparts. \(^{35-37}\) Although more perpendicular, these surfaces still fail to provide the protection barrier provided by natural teeth. Another discernible difference between soft tissue around natural teeth and that of dental implants is the number of blood vessels around both. Typically, few to no blood vessels are found in the zone adjacent to the implant surface. \(^{55}\) A clearing technique visualizing carbon-stained blood vessels showed that the vascular networks of the peri-implant mucosa are derived from the terminal branches of larger blood vessels originating from the periosteum of the bone at the implant site. \(^{38}\) Because of the numerous differences between soft tissues around natural teeth and around implant surfaces, several key components as listed below differ greatly between the systems.

Peri-Implant Probing

The importance of either tooth or peri-implant probing has been well documented in the literature. One of the first quantifiable differences between these two structures is the significantly deeper mean in probing depth at implant sites compared with tooth sites. \(^{39,60}\) This difference occurs irrespective of probing pressure, and it is also noted that the mean bleeding on probing percentage at implant sites is much higher than that of normal teeth. \(^{39,60}\) Furthermore, it has been documented that changes in probing depth forces around peri-implants are more sensitive to slight variations, making accurate analysis more difficult. \(^{39,60}\) This phenomenon may reduce the efficacy of peri-implant probing and provide less reliable evaluation of the inflammatory situation. Furthermore, slight increases in pressure may sometimes result in injury when the probe goes beyond the peri-implant seal. It is important, however, to note that not all investigators agree with the drastic changes of probing depth between implants and teeth, \(^{61}\) although the general consensus is that probing depth is increased in implant sites, BOP is also more sensitive to minor changes in pressure in implant sites.

Fiber Orientation and Distribution

As noted earlier, collagen fibers in natural teeth are perpendicularly oriented, attaching from the tooth cementum to the alveolar bone serving as a barrier to epithelial downgrowth and bacterial invasion. \(^{62}\) Since dental implants lack a cementum layer, collagen fibers typically orient themselves in a parallel manner to the implant surface, making them much weaker and more prone to periodontal breakdown and subsequent bacterial invasion. \(^{17,63}\) The lack of a proper periodontium is also a potential reason for faster inflammation progress, as discussed below. \(^{64}\)

Microbiota and Inflammatory Response

When implant surfaces began to show signs of periodontal plaque and biofilm accumulation, researchers began to determine the periodontal pathogens responsible for peri-implantitis. \(^{65,66}\) It is now well understood that the formation of a biofilm on an implant surface is influenced by the surface properties of the implant, such as chemical composition, roughness, and surface free energy. \(^{67}\) Studies have demonstrated that *Staphylococcus aureus* is common in deep peri-implant pockets closely linked to suppuration and bleeding on probing. \(^{68,69}\) Interestingly, *S. aureus* is not closely related to chronic periodontitis and seems to be more specific to implant surface contamination. \(^{70}\) Apart from this difference, the
bacteria species found in the subgingival microbiota in both natural tooth periodontal tissues and in peri-implant soft tissue are similar in terms of the occurrence and frequency of periodontal pathogens.71

Another important difference in the inflammatory response of soft tissues in natural teeth and implant surfaces is their different cellular responses. Inflammatory lesions in peri-implant sites are infiltrated with a high proportion of B cells and plasma cells, which is similar to chronic periodontitis and aggressive periodontitis.72–74 Although the development of peri-implantitis and periodontitis follows a similar pattern, the dynamics are quite different. Studies in human and animals show that there is very little difference in host response between natural and implant-supported teeth in the initial phase,75 but disease progression occurs more rapidly with subsequent bone loss in the peri-implant lesions than in natural teeth.64,76 The reason might lie in the lack of an intact supra-crestal connective tissue fiber compartment in peri-implant soft tissue77,78 which is able to wall off the lesions; the inflammatory cell infiltrates generally do not penetrate the alveolar bone marrow in periodontal tissues.79 Furthermore, the vascular supply as discussed below is reduced, decreasing the number of infiltrating neutrophils and B cells.

Vascular Supply

The vasculature of the periodontal soft tissues is derived from two sources. One source is the supra-periosteal vessels lateral to the alveolar process, and the other is the vessels of the periodontal ligament. When the natural tooth is extracted, the future implants have lost their vascular supply from the periodontal ligament.58 Furthermore, because of the dense fibers adjacent to the inner zone of the implant surface, there is also less vascular structure in the soft connective tissue directly adjacent to the implant surface compared with the natural tooth.52 As a sufficient vascular supply is necessary for wound healing and tissue repair by delivering numerous cell types and growth factors, the lack of an abundant blood supply has been suggested as one of the key reasons for the extensive progression of inflammation and lack of healing in soft tissues surrounding implants.80

MAINTENANCE AND RECOVERY OF SOFT TISSUE HEALTH AROUND IMPLANTS

Because of the vast differences between natural teeth and dental implants, their maintenance is of critical importance for the longevity of successful osseointegrated implants. A study which purposely banned oral hygiene around dental implants for a short period of time demonstrated a cause–effect relationship between the accumulation of bacterial plaque and the development of peri-implant mucositis.19 Recent studies have shown that bacterial colonization occurs within 30 minutes following implantation81 and becomes stable after a 2-week period.82,83 Thus, the primary objective of maintenance and recovery of any implant regimen is to remove the bacterial plaque and/or calculus.

MAINTAINING THE HEALTH OF PERI-IMPLANT SOFT TISSUE

Of course, the dental provider has a role in guiding implant stability following osseointegration; however, proper maintenance of the peri-implant soft tissue health is largely in the control of the patient’s own oral hygiene regimen. Patient self-management includes mechanical methods and chemical ways to control biofilm formation and subsequent plaque/calculus accumulation.

PATIENT SELF-MANAGEMENT

Mechanical Methods

Tooth Brushing. Manual and power brushes are both excellent and necessary means to remove dental plaque. These include manual squish grip brushes,84,85 sonic toothbrushes, ionic toothbrushes,86 and counter-rotational powered toothbrushes.87 Each is effective in plaque reduction and maintaining the gingival health of peri-implant soft tissues, and it is advised that patients brush a minimum of twice daily. Swierkot and colleagues88 found no significant difference between the various brushes utilized, but other investigators found that powered brushes were more effective in reducing plaque and gingivitis.89,90 Since the comparison of manual and powered toothbrushes does not show a definitive advantage of one type over another, it is typically advised that patients with good dexterity can choose either option, but as the patient ages, it is increasingly important to suggest powered toothbrushes.

Interdental Cleaning. Interdental cleaning devices are used to improve the efficacy of toothbrushes, especially in regions with small interspaces. Traditional string
floss and interdental brushes are useful devices, and the width of the interdental space determines which to use.\textsuperscript{91} Other methods to remove interdental plaque include using water floss; however, a lack of controlled clinical trials makes it difficult to speculate on its effectiveness.\textsuperscript{92}

**Chemical Methods**

Chemical agents may provide additional benefits to mechanical plaque control. Of course, it must be borne in mind that although they are not able to replace mechanical brushing, they may be utilized in combination approaches.

*Triclosan/Copolymer Toothpaste.* Triclosan (0.3%) together with methyl vinyl ether-maleic anhydride polymer (2.0%) in a sodium fluoride silica-based toothpaste has been demonstrated to reduce plaque and gingival inflammation.\textsuperscript{93,94}

*Fluoride-Containing Mouth Rinses.* Amino fluoride/stannous fluoride mouth rinses are an excellent choice as a mouthwash. In one study, it was shown that the results for plaque control were comparable with those for chlorhexidine gluconate mouth rinses, and the patients enrolled in the study preferred the taste.\textsuperscript{95} Furthermore, fluoride-containing mouth rinses are able to reduce pro-inflammatory molecules in peri-implant crevicular fluid.\textsuperscript{96}

*Essential Oils Mouth Rinse.* Listerine, Pfizer Inc, Manhattan, New York, United States, used twice daily for 30 seconds directly after standard oral hygiene mechanical brushing, has been shown to provide a reduction in plaque index, gingival index, and bleeding index.\textsuperscript{97} More recently, a variety of oils such as cinnamon oil and clove oil have been studied for their long-term antibacterial effect, although more research is necessary to characterize their beneficial effects on implants fully.\textsuperscript{98}

*Chlorhexidine.* Chlorhexidine gluconate (0.12%) is used to control plaque and maintain oral hygiene in postrestorative phases following implant placement.\textsuperscript{97,95} Furthermore, irrigation with 0.06% chlorhexidine using a powered oral irrigator (Water Pik, Water Pik Inc, Fort Collins, Colorado, United States) with a special subgingival irrigation tip has been shown to be more effective than chlorhexidine gluconate (0.12%) mouth rinses\textsuperscript{99}; however, very few studies have been conducted particularly on implants. Chlorhexidine is also available in gel form, which has also been demonstrated as effective.\textsuperscript{100}

**PROFESSIONAL MANAGEMENT**

It is important that patients receiving implants be placed on a strict recall programme so professional management can assess changes over time, which will help maintain the health of dental implants. Professional management may include mechanical debridement, application of phosphoric acid etching, or injections with chlorhexidine.

**Mechanical Debridement**

A qualified dentist is required to assess the state of the osseointegrated implant and make decisions on the mechanical debridement program most suitable for each placement. Typically, patients may be placed on recall every 6 to 12 months for supra and subgingival debridement of calculus and plaque around implants with carbon fiber curettes. In general, patients with a single tooth crown or bridge with two implant abutments should be on recall once per year with risk factors analyzed. Typically, if a bridge has more than two implants, it is advised to place the patient on a 6-month recall program which also includes patients with full arch bridgework retained by implants or implant-retained dentures. In patients with or at a higher risk of developing peri-implant disease caused by systemic factors such as smoking, personal factors such as bad oral hygiene, or genetic factors, the recall interval should be shorter (usually half of the suggested time). If, for some reason, peri-implant soft tissue becomes infected or shows signs of attachment loss, a much stricter regimen may then be necessary for implant health, typically with recall once every 12 weeks until soft tissue healing is observed. It is up to the healthcare provider to make optimal decisions for the long-term survival of implants.\textsuperscript{101}

**Phosphoric Acid Etching Gel**

The application of phosphoric etching gel in the peri-implant sulcus has been used as an alternate method for improving peri-implant soft tissue. Strooker and colleagues have demonstrated that application of a 35% phosphoric etching gel at pH 1 results in an instant reduction of colony-forming units, proving that it is an
effective way to fight against bacteria. Although long-term clinical studies are still lacking, the use of phosphoric etching has been demonstrated at least in part to counteract infected peri-implant soft tissues by decreasing bacterial counts and may be a viable option in already infected implant sites.

**Chlorhexidine**

Much like home mouth rinses with chlorhexidine, its application by a professional dental provider via local injections has also been demonstrated as a means to improve peri-implant soft tissue healing following peri-implant contamination. Groenendijk and colleagues showed that a 0.2% chlorhexidine injection to the inner part of implants at second-stage surgery demonstrated a significant inhibition of growth and acquisition of bacteria by the peri-implant interface.

**RECOVERING THE HEALTH OF PERI-IMPLANT SOFT TISSUE**

Because of the growing use of dental implants, more and more cases have now been reported as demonstrating signs of peri-implantitis and peri-mucositis. Of primary importance in the recovery of these implants is promoting the health of the peri-implant soft tissue by eliminating biofilm and calculus. Once again, this may be achieved through oral hygiene techniques at home, but more emphasis is now placed on the healthcare provider’s efforts at disease resolution. Therefore, patient self-management is identical to that mentioned in the section “maintaining the peri-implant soft tissue health”; however, we discuss next the professional means to improve peri-implant soft tissues once peri-implant soft tissue infection is present.

**PROFESSIONAL MANAGEMENT**

**Mechanical Debridement**

To recover periodontal soft tissues around implants following peri-implantitis, it becomes vital that the patient is followed up regularly. During these appointments, supra and subgingival debridement of the implant surface becomes vital, as the main goal is to remove the biofilm and calculus without altering the topography of the implant surface. Curettes and ultrasonic devices with polyether-etherketone-coated tips are the most common approaches, and patients are advised to be placed on a recall program every 12 weeks until implant inflammatory resolution is obtained.

**Curettes.** Typically, carbon curettes are advised again for the recovery of implants. Although metal curettes may be acceptable for zirconia implants, they leave too many scratches on the surface of titanium implants and thus should be avoided. Although titanium curettes are an option, and one study has shown less damage observed by scanning electron microscopy, nonmetal alternatives (carbon-fiber curettes, teflon curettes, plastic curettes) are the preferred option. It is noteworthy that a growing number of ultrasonic devices are being used for plaque and calculus removal in peri-implant tissues as discussed next and are therefore the preferred method for mechanical debridement once plaque and calculus are found subgingivally.

**Ultrasonic Devices.** A number of studies have shown the efficacy of using ultrasonic devices for reducing bacterial plaque and BOP scores. Renvert and colleagues demonstrated that plaque scores decreased from 73% to 53% \((p < .01)\), and bleeding scores were also significantly reduced following therapy \((p < .01)\). Park and colleagues demonstrated on implants with an SLA surface that metal scaler tips were more effective in eliminating bacteria and reducing bacterial adherence by smoothing the implant surface than plastic and carbon scaler tips. With regard to machined surfaces, the scratches caused by a metal scaler do not significantly affect the amount of biofilm that adheres, and it has been demonstrated that a smoother surface for soft tissue attachment is preferred since bacterial adhesion is reduced on these surfaces. It has been demonstrated that ultrasonic scalers do not show an increase in implant temperatures when the cooling system is used properly.

**Adjunctive Antibiotics**

When the microbial count increases during acute infections of peri-implantitis, it may become necessary to treat with antibiotics. Antibiotics are used to enhance the effect of the mechanical debridement and prevent the future recolonization of bacteria. Minocycline microspheres are used because of their sustained release of antibacterial ingredients up to 12 months, and studies have shown that they are effective in reducing plaque, and probing pocket depth as well as BOP. Amoxicillin, metronidazole, and their combination are usually delivered as local antibiotic applications. The combination of amoxicillin and metronidazole showed significant inhibition on the growth of adherent
Streptococcus sanguinis and Porphyromonas gingivalis and much lower bacterial resistance. Furthermore, systemic antibiotics may be used to increase the antimicrobial level in the peri-implant crevicular fluid to support the effect of mechanical debridement, but no sound scientific basis has been found for the use of systemic antibiotics.

**HOW TO RECONSTRUCT THE IMPLANT SOFT TISSUE FOLLOWING PERI-IMPLANTITIS**

Peri-implantitis is accompanied with crestal bone loss, bleeding on probing, and the possibility of suppuration. It is very difficult for the clinician to manage such a disease as the resulting implant surface has lower blood supply than the natural surface, and if the disease is left untreated, it may well lead to implant failure. Various studies have revealed that the prevalence of peri-implantitis ranges from 2.7% to 47.1% and that as implants become more popular, the necessity for effective strategies to reconstruct peri-implant tissues will be equally important.

**NONSURGICAL DECONTAMINATION AND INFECTION CONTROL**

As peri-implantitis is a bacterial infectious disease, the first thing to do before reconstructive therapy takes place is to control infection. To this end, and prior to surgical intervention, the peri-implantitis site should demonstrate no bleeding on probing and exhibit no suppuration. The mechanical decontamination methods for peri-implantitis include mainly curettes, air-abrasive devices, ultrasonic devices, and lasers (Table 1).

**Air-Abrasive Systems**

Air-abrasive systems are based on the air spray of powders made from a variety of materials including sodium bicarbonate, sodium hydrocarbonate, calcium phosphate, erythritol-chlorhexidine, and amino acid glycine. They have been shown to be effective in eliminating biofilms and calculus both in vivo and in vitro. Typical devices include a specially designed nozzle, which is used for horizontal exit of air powder mixture. It is recommended that the nozzle be...
moved circumferentially around the implant surface in equal fashion in order to decontaminate the implant surface.\textsuperscript{128}

One of the main drawbacks of the air-polishing technique is that it increases implant surface roughness, which in turn increases bacterial adhesion. The standard powdered air-abrasive system (sodium-carbonate) proved unsuitable for implant instrumentation because of the high abrasiveness revealed by SEM examination, whereas low-abrasive amino-acid glycine powder is recommended for debriding implant surfaces as it does no damage to hard or soft tissues.\textsuperscript{129} Petersilka and colleagues compared a low abrasive air-polishing powder with hand instruments and found that the powder resulted in a significantly greater reduction in mean CFU (log 1.7 ± 0.98 and log 0.61 ± 0.79, respectively; \( p < .05 \)) from pockets of 3 to 5 mm depth.\textsuperscript{126} Another study showed that air-abrasive powders produced a 0.8 to 0.5 mm reduction in periodontal depth and a reduction in bacteria (\textit{Pseudomonas aeruginosa}, \textit{Staphylococcus aureus} and \textit{Peptostreptococcus anaerobius} ) at 1 month.\textsuperscript{130} Drago and colleagues constructed a new formulation consisting of erythritol and chlorhexidine powders, and its in vitro antimicrobial and antibiofilm effects on bacteria (\textit{S. aureus}, \textit{Bacteroides fragilis}, and \textit{Candida albicans}) were stronger than the standard glycine powder used in air-polishing devices.\textsuperscript{124} Apart from abrasiveness, another potential disadvantage of air powder abrasive systems is the remnants. A study by Tastepe and colleagues showed that although the powder was effective in biofilm removal, powder particle remnants were observed on and impacted on the titanium surface.\textsuperscript{123} In the HA and HA + TCP group, a calcium content varying between 2% and 5% was observed.

Lasers

Since their first application in dentistry in 1989,\textsuperscript{131} lasers have gained popularity in different aspects of dentistry and have been utilized in peri-implant decontamination. There are a variety of options for decontamination of implant surfaces including semiconductor diode lasers, the solid state laser Nd:YAG, Er:YAG lasers, and gas lasers such as CO\textsubscript{2} lasers.\textsuperscript{132–134}

It was found in an in vitro study that the 980-nm diode laser caused little or no damage to implant surfaces\textsuperscript{135} while still being useful for bacterial reduction of \textit{P. gingivalis}-contaminated implants.\textsuperscript{136} This result has since been verified in clinical testing with a similar 810-nm diode laser.\textsuperscript{137} The CO\textsubscript{2} laser as well as the Nd:YAG laser have been used to a limited extent in dentistry. The CO\textsubscript{2} laser has lower penetration depth in soft tissues than the Nd:YAG lasers.\textsuperscript{138} In contrast, CO\textsubscript{2} lasers at an energy density of 100 J/cm\textsuperscript{2} can destroy microbial colonies including \textit{S. sanguinis} and \textit{P. gingivalis} without damage to the tooth root surface.\textsuperscript{139} Furthermore, in another study, Deppe and colleagues found that CO\textsubscript{2} laser-assisted therapy of ailing implants did not cause excessive titanium accumulation in tissues, thus making it a suitable and safe method for implant decontamination.\textsuperscript{140} Although CO\textsubscript{2} is relatively stable for titanium decontamination, its combination with Nd:YAG lasers can produce the undesirable result of extensive melting in irradiated areas and damage to the microstructure of the implant as confirmed by SEM examination.\textsuperscript{135}

Promising results have also been observed with the Er:YAG laser in the treatment of peri-implantitis, and it is the most popular choice at present. Kreisler and colleagues demonstrated that the Er:YAG laser has a high bactericidal potential regarding common implant surfaces, even at low energy densities.\textsuperscript{141} A study by Matsuyama and colleagues showed that an Er:YAG laser at 30 mJ/pulse and 30 Hz with water spray was capable of effectively removing plaque and calculus without injuring the implant surfaces.\textsuperscript{142} Schwarz and colleagues observed that the Er:YAG laser resulted in a statistically significant higher reduction of BOP than mechanical debridement with plastic curettes and antiseptic therapy.\textsuperscript{143} Takasaki and colleagues demonstrated that degranulation and implant surface debridement were obtained effectively and safely by the Er:YAG laser and that a favorable formation of new bone was observed on the laser-treated implant surface histologically.\textsuperscript{144}

Kreisler and colleagues performed an extensive study of numerous lasers including Nd:YAG, Ho:YAG, Er:YAG, CO\textsubscript{2}, and diode lasers for implant decontamination.\textsuperscript{138} It was found that Nd:YAG and Ho:YAG were not suitable for implant surface decontamination because of partial melting, cracking, and crater formation on implant surfaces irrespective of the power output. The Er:YAG and CO\textsubscript{2} lasers were recommended for use at low power settings so as to avoid surface damage. However, the diode laser did not cause surface changes. Thus, despite their being popular choices, care must be taken when implants are decontaminated with either CO\textsubscript{2} or Er:YAG lasers to avoid temperature...
increases above the critical threshold (10°C) after 10 seconds of continuous irradiation.\textsuperscript{145,146}

**SURGICAL TECHNIQUES**

Typically, surgical techniques and reconstructive procedure are more effective but limited to moderate to severe peri-implantitis. Below is a list of various techniques used for the treatment of peri-implantitis.

**Apically Positioned Flaps**

Apically positioned flap surgery is aimed at decontaminating the implant surface and exposing the affected part of the implant to the oral cavity for better self-managed oral hygiene,\textsuperscript{147} often accompanied by osteoplasty.\textsuperscript{148} This technique is very similar to apically positioned flaps for natural teeth and enables reduced pocket depths, facilitating patient hygiene. This technique has clear drawbacks and is only recommended for nonaesthetic regions, however.\textsuperscript{149}

**Access Flap Surgery**

Access flap surgery is a surgical way to decontaminate the implant surface while maintaining the soft tissues around the affected implant. The aim of this surgery is to maintain the soft tissue around the implant neck, and it is recommended when bone loss is minimal. Following the access flap, the technique may be combined with various other methods such as curettes, air-abrasive devices, ultrasonic devices, and lasers to enhance cleaning efficacy.\textsuperscript{150}

A regenerative technique is mainly utilized to support the tissue dimensions to avoid mucosa recession. After decontamination of the implant surface, a graft may be placed around the implant, filling the peri-implant defect. The graft commonly used is either autologous bone\textsuperscript{151} or bone substitutes,\textsuperscript{152} and combined with or without a resorbable or nonresorbable membrane.\textsuperscript{151,153} Furthermore, the use of a connective tissue graft with a bone graft may be advantageous aesthetically.\textsuperscript{154}

The long-term goal of a regenerative procedure should be the re-adhesion of the peri-implant soft tissue and further enhancement of bone regeneration around the implant surface. The change in probing depth has also been compared for various bone grafting materials. Aghazadeh and colleagues demonstrated that xenografts are better than autogenous grafts in reducing the probing depth,\textsuperscript{155} and others have demonstrated that synthetically fabricated hydroxyapatite performed worse than xenografts.\textsuperscript{152} Although the data comparing bone grafting materials for the treatment of peri-implantitis remain very limited, it has been observed in numerous studies that a xenograft in combination with a resorbable membrane increases clinical attachment level.\textsuperscript{152,153,156–158}

**ADDITIONAL CAUSES OF PERI-IMPLANT INFLAMMATION**

Although the aim of this review article was to examine differences between soft tissues found in natural teeth and dental implants as well as their related health and maintenance programs, it is also vital to state that many additional factors and etiologies are constantly being investigated for their possible roles in peri-implantitis. For example, recent reports have suggested that cement-retained crowns have been more prone to peri-implantitis when compared with their screw-retained counterparts.\textsuperscript{159–161} Some reports suggest that when surgical flaps were raised to treat peri-implant bone loss, over 70% of cases presented excessive cement in cement-retained crowns.\textsuperscript{162} As a result from over-contour of cement, oral bacteria accumulation and a greater inflammatory response leading to eventual bone loss pose a greater risk for peri-implantitis.\textsuperscript{163} For these reasons, it has become more mainstream to use screw-retained implants when possible or to modify cementing techniques to avoid excess cement in peri-implant soft tissues.\textsuperscript{164} Another area of research which has gained tremendous awareness over the last decade is the effect of micro-gaps between implant components and their effect on bacterial microleakages.\textsuperscript{165,166} Some investigators have reported that these gaps have been reported as large as 70 um implant-abutment interface, and implant design may affect the potential risk for invasion of oral microorganisms into the fixture-abutment interface microgap under dynamic loading conditions.\textsuperscript{165,167–170} Further research to improve the implant to abutment connection will further decrease the likelihood of soft and hard tissue inflammation around implants.

**SUMMARY AND CONCLUSIONS**

Osseointegration is a special kind of bone healing process, and the intact peri-implant seal plays an important role in protecting the alveolar bone from bacterial invasion in the oral cavity. However, because of the structural differences between implants and natural
teeth, there are drawbacks in peri-implant soft tissue healing compared with natural teeth such as deeper probing depth, weaker connective tissue attachment, faster inflammatory expansion, and reduced vascular supply, making the implant more vulnerable to bacterial accumulations and other external stimulations. Thus, it is clear that implant surfaces necessitate more attention to the maintenance of peri-implant soft tissue health. It is also vital that patient self-management is rigorously employed, and this should be stressed at each dental consultation. Dentists have a variety of means to promote soft tissue health around implants, such as using both mechanical and chemical methods to remove plaque and calculus. However, when the implant surface is contaminated with peri-implantitis, the healthcare professional must utilize methods of decontamination, re-osseointegration, and re-adhesion of soft tissues around implant collars. Both nonsurgical and surgical techniques exist to facilitate this attachment; however, a great deal of research is still necessary in terms of comparing the different options available to dentists to generate long-term predictable results. Future research should better define the most predictable methods for decontamination of implant surfaces and characterize the effects of surface material and surface topography on the various implant systems. An ever-increasing number of implant companies offer various soft tissue connections either to the abutments in bone-level implants or to the implant collar in tissue level implants. Although new implant companies frequently demonstrate the effects of their surface topography on bone-forming osteoblasts, much research on the most effective transmucosal attachment system is necessary to prevent further peri-implant tissue inflammation. Direct comparisons between the various implant systems as well as cell behavior need to be conducted which offer more evidence on the best peri-implant tissue attachment at the connective tissue and epithelial levels. The field of peri-implantitis faces many upcoming challenges to meet the large number of dental implants that is now placed every year, and more importance should be given to the health and maintenance of soft tissues around implants.

REFERENCES


