



Management of peri-implantitis

R. Gilbert Triplett, DDS, PhD^{a,*}, J. Adam Andrews, DDS, MD^a,
William W. Hallmon, DDS^b

^a*Department of Oral and Maxillofacial Surgery and Pharmacology, Baylor College of Dentistry,
TAMUSHSC and Baylor University Medical Center, 3302 Gaston Avenue, Dallas, TX 75246, USA*

^b*Department of Periodontics, Baylor College of Dentistry, TAMUSHSC, 3302 Gaston Avenue, Dallas, TX 75246, USA*

Failure of osseointegrated dental implants is a frustrating problem for the patient and dentist. Peri-implantitis and occlusal overload are the most common causes of implant failure after osseointegration, and they often require removal of the involved implant. A single failed implant can result in complete prosthetic failure when load-sharing mechanics of the prosthesis depend on the health and integrity of each individual implant. Peri-implantitis, which is an inflammatory process around an osseointegrated dental implant in function with resulting bone loss, affects approximately 5% to 10% of osseointegrated implants [1]. Peri-implant mucositis refers to reversible inflammation of the peri-implant soft tissues without bone loss [1,2]. Accurate diagnosis and appropriate intervention are essential if implant salvage techniques are to be successful in preventing implant failure. This article focuses on the methods available for diagnosis and treatment of peri-implantitis that involve various implant systems.

The role of bacteria in the development of peri-implantitis

The experimental gingivitis model of L oe and Silness elegantly displays the interaction between bacterial plaque and gingivitis in a human model [3]. This landmark study provided the foundation for

the indisputable evidence that linked bacteria to periodontal disease and was later repeated in several experimental mucositis/implantitis models [1]. The evidence compiled from these well-designed studies identified bacteria as the primary culprit in the development of peri-implantitis. Large numbers of gram-negative anaerobic bacteria (*A. actinomycetemcomitans*, *P. gingivalis*, *P. intermedia*) tend to be found around implants with objective signs of peri-implantitis, whereas healthy implants are most often colonized with flora dominated by gram-positive cocci [1]. Endotoxins produced by gram-negative bacteria have the capability to adhere to the implant surface and produce inflammation and resulting bone loss around implants in a similar fashion to periodontitis [1]. Several studies have demonstrated the ability of periodontal pathogens to infect peri-implant tissues in partially edentulous patients (Fig. 1). This observation may account for the higher implant success rates reported in several studies when implants were placed in edentulous mouths versus partially edentulous mouths.

The convincing evidence that suggests a bacterial cause of peri-implantitis provokes several interesting clinical scenarios regarding the optimal restorative treatment of the partially edentulous patient. Extrapolation of research data, which suggest that implant failures may be significantly higher in partially edentulous patients with a history of periodontitis, may lead to the possible recommendation of extraction of questionable teeth before implant placement. A possible alternative is treatment of the periodontally diseased tissues before implant placement in persons with a history of severe periodontal prob-

* Corresponding author.

E-mail address: gtriplett@tambcd.edu (R.G. Triplett).

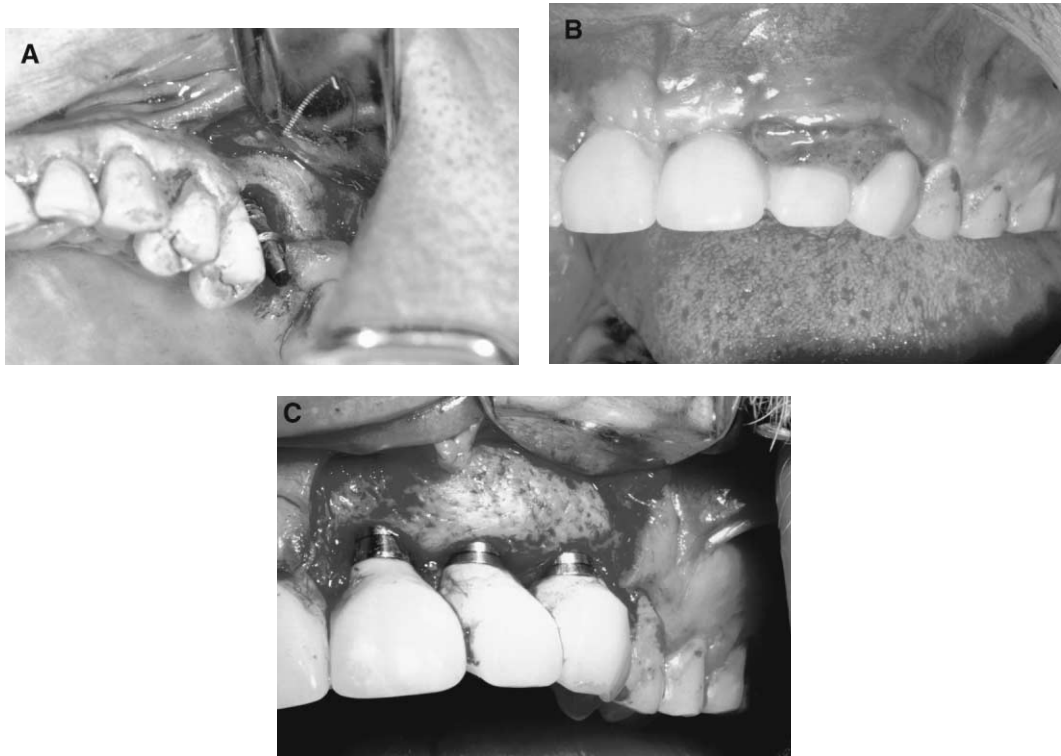


Fig. 1. (A) Peri-implantitis in the partially edentulous patient. (B,C) Peri-implant mucositis demonstrated by soft tissue inflammation and increased probing depths without bone loss.

lems. Edentulous mouths tend to host a more benign flora because of the lack of deep pockets and crevices that commonly harbor gram-negative anaerobes and spirochetes in patients with periodontal disease. Restoration of a more benign oral microbiota seems to be a logical goal before implant placement if long-term implant and prosthetic success is to be achieved. Several protocols for local and systemic decontamination of periodontal tissues have been published; however, the overall long-term benefit depends on many variables and is often unpredictable [4].

Titanium implant surface characteristics and the relationship with implant success and failure

The implant market has been inundated with various systems that use different materials, surface coatings, and manufacturing processes. Implants can be categorized by differences in macrostructure (eg, cylindrical, threaded, screw design) and microstruc-

ture (rough versus smooth, commercially pure titanium versus titanium alloy). It seems evident, based on sound clinical and experimental data, that the same surface implant characteristics that enhance osseointegration may possibly increase the risk of peri-implantitis when placed under certain unfavorable conditions.

Patient selection is as important as the type of implant used if favorable long-term success rates are to be expected. Implant selection should be based on a combination of patient-dependent clinical and biologic considerations. Several recent studies have concluded that rough surface implants demonstrate a significantly higher percentage of bone-to-implant contact and faster and stronger osseointegration when compared to machined surface titanium implants [5–7]. A study by Trisi et al [5] evaluated differences in the rate of osseointegration between smooth and rough surface implants in low-density human jaw bone. At 12 months, the implant-to-bone contact rates for smooth and rough surface titanium implants were noted to be 6.7% and 76.75%, respectively. The study

concluded that although a rough surface may enhance the rate of osseointegration, it is not able to improve bone density [5].

The surface oxide layer on commercially pure titanium and titanium alloy (Ti-6Al-4V) determines the biocompatibility of a particular implant because it is the only portion of the implant in contact with host tissues [6]. The same surface oxide layer has been shown to exist on rough titanium surfaces, which may contribute to enhanced osseointegration when compared to machined surfaces of the identical material [6]. Several other studies have reported significantly better bone anchorage with titanium-oxide-blasted (TiO₂-blasted) screw implants than with machined implants of similar composition and higher torque removal for implants with increased surface roughness [6]. Schwartz et al demonstrated that as the surface roughness of titanium implants increases, the cytokine and growth factor production by host osteoblast-like cells also increases and could account for improved bone formation around the roughened implant surface [6,8]. Osteoblasts also have been found to display a more mature phenotype when grown on rougher surfaces [6]. Studies that compared different types of roughened titanium surfaces concluded that rougher titanium plasma-sprayed surfaces, corundum-blasted surfaces, and sandblasted surfaces alone are inferior to surfaces that are treated with a combination of sandblasting and acid-etching [5,6,9].

Despite the convincing evidence that rough titanium surfaces may enhance osseointegration and implant stability, several studies have suggested that a rough surface may contribute to plaque formation and higher implant failure rates from peri-implantitis. Tillmanns et al [9] experimentally induced peri-implantitis in a canine model with three different types of implant surfaces (smooth, blasted, and hydroxyapatite coated). The results after 3 and 6 months revealed plaque accumulation and the same amount of peri-implant bone loss among all implant surfaces studied. The study concluded that the three implant surfaces are equally susceptible to ligature-induced peri-implantitis [9]. The conclusion is in direct contrast to most experimental results that compared implant surfaces. The finding seems to be well supported that bacterial plaque formation depends on the surface properties of the implant material. A surface roughness (R_a) more than 0.2 μm facilitates early plaque formation in an experimental model but may be favorable to soft tissue sealing around transmucosal abutments. Anything below this R_a value (smoothing) seems to be ineffective in reducing the amount of plaque formation, but it

prevents soft tissue attachment to the implant surface [10]. Recently published results that evaluated different surface coatings on rough surface implants concluded that titanium nitride or zirconium nitride can reduce the accumulation of plaque by coating the underlying more reactive titanium surface (independent of surface roughness), which may reduce peri-implant mucositis and peri-implantitis [10].

Considerations with hydroxyapatite-coated titanium implants

Hydroxyapatite is a bioactive material with osteoconductive properties [11]. Hydroxyapatite-coated titanium alloy implants were first introduced in 1984 for use in restoring partially or totally edentulous maxillas and mandibles [12]. Plasma-sprayed hydroxyapatite-coated implants demonstrate greater tolerance to unfavorable healing conditions and promote bone growth into gaps that measure less than 1 mm [11]. Early reports of faster osseointegration (biointegration), a stronger bone-to-implant interface (compared to titanium surfaces), and vertically directed bone growth along the implant surface were met with optimism among the implant community [12,13]. Short- and long-term studies have demonstrated higher implant-to-bone contact in hydroxyapatite implants at 6 weeks when compared to titanium implants. At 12 weeks, however, the titanium implants displayed superior implant-to-bone contact compared to hydroxyapatite-coated implants and had an increase in total implant-to-bone contact surface area after 1 year. The hydroxyapatite-coated implants showed a reduction in the implant-bone contact area over the same period with a significant decrease in shear strength [12,14,15].

A combination of clinical observation and prospective research suggests that hydroxyapatite-coated implants may be more prone to enhanced plaque growth and peri-implantitis because of the questionable long-term quality of the hydroxyapatite-to-bone bond and the affinity of microorganisms for the hydroxyapatite surface [12]. Johnson reported sudden and rapid bone loss around hydroxyapatite-coated implants “after an initial period of apparent success” (Fig. 2A) [12]. Several clinical reports claim that failure of hydroxyapatite-coated implants involves “significant morbidity and permanent destruction of bone tissue,” whereas titanium implants tend to fail with minimal loss of bone volume and typically regenerate to the original dimension of the alveolus (Fig. 2B) [12]. Despite the claims of higher long-term failure rates with hydroxyapatite-coated implants,

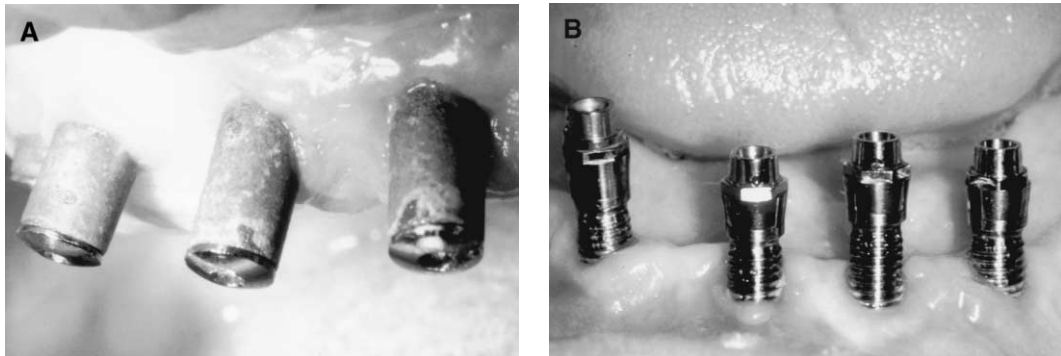


Fig. 2. (A) Hydroxyapatite-coated implants with significant bone and attachment loss. (B) Threaded, machined surface titanium implants with significant attachment and bone loss.

many clinicians have found them valuable in situations that involve unfavorable bone quantity or quality [16]. Recommendations and special considerations regarding the salvage of infected hydroxyapatite-coated implants are discussed in this article.

Retrograde peri-implantitis

Radiolucencies around the apical aspect of dental implants have been attributed to several causes, including contamination of the implant surface, overheating of bone, occlusal overload, preexisting bone pathology, presence of residual root fragments or foreign bodies in implant sites, lack of biocompatibility, placement of the implant in poor quality bone, and drilling through the inferior border of the mandible or lingual cortex [17]. The term “retrograde peri-implantitis” was first used to describe radiographic periapical bone loss around a dental implant without evidence of peri-implant soft tissue inflammation [16]. It was proposed that a radiolucent lesion found on plain film radiography was caused by traumatic or premature implant loading that resulted in microfractures of the peri-implant bone and subsequent resorption. Because the microflora around implants that suffered from retrograde peri-implantitis have been shown to be similar to the microflora around healthy implants, one can assume that infective failure is not experienced with this type of implant lesion [3,16]. Because bacteria do not seem to be a causative factor in this type of implant failure, normal probing depths without bleeding are often observed, and resolution of the periapical radiolucency may be observed if traumatic loading of the implant is relieved.

Diagnosis of the failing implant and peri-implantitis

Much debate exists regarding the definition of implant failure. The endeavor to define implant failure should be preceded with a definition of success. The First European Workshop on Periodontology defined success as absence of implant mobility, an average radiographic marginal bone loss of less than 1.5 mm during the first year of function and less than 0.2 mm annually thereafter, and absence of pain and paresthesia [2]. Because this definition was based on the mean marginal bone loss around Brånemark implants, it seems presumptuous to conclude that different implants would behave in a similar manner. Much attention has been given to the terms “ailing” and “failing” when referring to implant health. It has been proposed that an “ailing” implant demonstrates radiographic evidence of bone loss and probing depths more than 5 mm that are stable when reevaluated at 3 to 4 months. A “failing” implant demonstrates increasing probing depths, suppuration or bleeding when probed, and progressive bone loss [18]. A failed implant no longer is osseointegrated or never achieved osseointegration. These implants display peri-implant radiolucency caused by fibrous tissue encapsulation, are clinically mobile, and demonstrate dullness to percussion. Failed implants must be removed to prevent chronic bone loss and the possibility of osteomyelitis (Fig. 3) [18].

Esposito et al [19] concluded that radiographic examination and mobility testing were the most reliable parameters in determining the prognosis of osseointegrated implants. Marginal bone loss around the neck of the implant can be evaluated radiographically and by peri-implant probing. Reproducing the

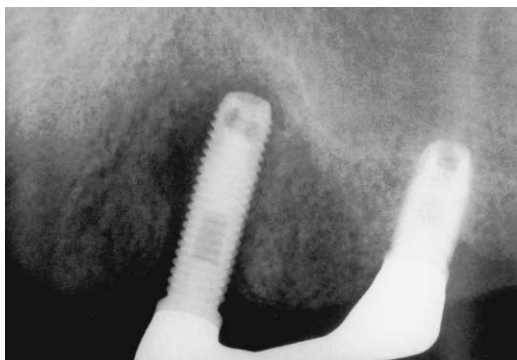


Fig. 3. Failed implant with fibrous tissue encapsulation and clinical mobility.

exact radiographic exposure geometry is difficult and can lead to clinically significant variability. Despite the latter finding, it seems that serial radiographs are more reliable in monitoring peri-implant conditions than probing, particularly in the setting of inflamed peri-implant tissues and bony defects [19]. Digital subtraction radiography eventually may prove to be superior to traditional radiographic techniques in evaluating subtle changes in peri-implant bone density, and it is highly recommended, if available [19,20]. If no clinical evidence of inflammation is present, radiographs should be obtained 1 year after implant placement and not more than every 2 years thereafter [1]. Radiographs should be taken more frequently if clinical evidence of peri-implant inflammation (increased probing depths) raises suspicion of peri-implantitis [1].

Differences among implant systems make the task of quantitatively defining the normal amount of marginal bone loss difficult. Several studies have demonstrated that marginal bone loss and biologic width are determined largely by the implant system and the host response. Gargiulo [21] demonstrated that natural teeth have a normal biologic width of 2.73 mm. Hermann et al [22] evaluated the anatomic location of the biologic width around various types of dental implants and determined that the one-piece (single-stage) nonsubmerged implant with a rough/smooth border placed at or 1 mm apical to the alveolar crest resulted in a biologic width that most closely resembled that of natural teeth (2.84 mm). They also concluded that the presence of a microgap (component interface) in two-piece implant systems significantly affects the level of crestal bone and soft tissue dimensions. The presence of a microgap in two-stage implant systems may play an important role in the development of peri-implantitis because

bacterial contamination of this interface has been demonstrated [22].

Peri-implant probing may provide valuable information regarding implant health or progression of disease. Healthy implants generally have probing depths less than 4 mm, with interproximal probing depths normally 0.5 to 1 mm more than the buccal and lingual probing depths [20]. Peri-implant pockets of 5 mm or more should be considered an indicator of peri-implantitis because deep pockets have been shown to harbor a microflora consistent with inflammation and bone loss [1].

There has been much debate over the location of the probe tip and the effect on the peri-implant tissues during peri-implant probing in disease and health. Several studies suggest that there is a resilient soft tissue collar in peri-implant health and mucositis. The tip of the probe may travel without impedance to the alveolar crest in peri-implantitis, however [20]. It has been demonstrated experimentally that the probe tip penetrates apically to the laterally displaced junctional epithelium with Brånemark implants, resulting in the probe tip approaching the alveolar crest [18]. This is in contrast to the findings around ITI dental implants, which demonstrate the probe tip location at the apical termination of the junctional epithelium (0.05 mm in healthy sites and 0.02 mm in diseased sites) [19]. Issues regarding the practicality and reproducibility of probing depths have brought the entire practice of peri-implant probing into question. Several studies have concluded that peri-implant probing damages the peri-implant soft tissues, but the magnitude and long-term effects have yet to be determined [19].

It can be said that there is a positive correlation between peri-implant probing depths and the degree of peri-implant mucosal inflammation, but not necessarily bone loss [19]. More important than a single measurement at a single point in time is documentation of progressive peri-implant bone loss as evidenced by increasing probing depths. It is recommended that baseline probing depths be acquired at the time of prosthetic reconstruction to account for the predictable marginal bone loss during the first several months after implant placement. Assuming that probing has been accepted by the surgeon as a method of providing beneficial clinical information that exceeds the potential for harming the peri-implant tissues, the frequency and interval must be tailored to each individual patient based on compliance, oral hygiene, and other risk factors. Probing attachment levels relate probing depths to a fixed reference point on the implant or abutment and provide valuable information regarding attachment loss over time. Probing attachment level

increases of 2 mm or more should be interpreted as marginal bone loss [19].

In addition to providing information regarding attachment level and bone loss, peri-implant probing can demonstrate peri-implant inflammation clinically. Bleeding on probing is another controversial issue regarding the diagnosis of peri-implantitis. No correlation has been shown between bleeding and the histologic or radiographic changes associated with peri-implant mucositis or peri-implantitis around smooth surface threaded implants [19]. Another study demonstrated that the absence of bleeding on probing around ITI implants was associated with implant health, whereas bleeding on probing correlated highly with peri-implant mucositis and peri-implantitis [19]. Because the reason for these differences may be caused by inconsistent probing forces or other factors, bleeding on probing is not scientifically supported as a method of diagnosing peri-implantitis [19]. Several authors still maintain that “probing depth measurements related to a fixed landmark on the implant and examination of the bleeding tendency of the peri-implant tissues seem to be well-suited for the longitudinal monitoring of peri-implant stability” [1]. It has been recommended that nonmetallic probes (eg, plastic) with a calibrated constant probing force be used for more reliable, less traumatic measurements. A probing force of 0.25 N has been recommended by several authors to fulfill the previously mentioned criteria [1,20]. Mombelli and Lang [1] recommend the use of standardized probes, such as the Audio Probe, titanium plasma-sprayed probe, or the HAWE Click Probe, for consistent measurements.

Implant mobility in a previously healthy implant should be considered a sign of failure. Even implants that show significant bone loss usually are immobile if any direct implant-to-bone contact remains. Several devices and methods have been proposed for evaluating implant mobility. Periotest (Siemens AG, Bensheim, Germany) is a device used for measuring the damping effect of the supporting tissues to a standardized force as an indicator of slight changes in implant mobility. Although several studies have reported its success in detecting subtle changes in the bone-to-implant interface, its usefulness and accuracy are still being evaluated [1,18]. A torque wrench that delivers a set amount of force is another method for determining implant osseointegration. An implant is considered to be osseointegrated if a torque of 10 to 20 Ncm is applied to the implant without resulting mobility [18]. Although not scientifically supported, the percussion test is reported to be a simple and sensitive method of determining osseointegration. The implant abutment interface is percussed with the blunt

end of an instrument and the sound is interpreted. A “ringing” sound is considered favorable for osseointegration, whereas a “dull” sound suggests fibrous tissue encapsulation [18]. Finally, OSSTELL (Integration Diagnostics, Inc., Sävedalen, Sweden) is a new Food and Drug Administration–approved device that uses resonance frequency analysis to assess implant stability. The reliability and usefulness of the device in implant dentistry still are being evaluated.

A systematic approach should be used when evaluating implants for possible disease. It is often more prudent to evaluate the implant by assuming disease and proving health because this mode of reasoning tends to eliminate the possibility of false-negative screening results. If probing is to be performed at the recall visit, the new probing depths should be compared to baseline measurements and the overall trend observed. Attachment levels also should be recorded because peri-implant pockets potentially can remain normal as marginal bone loss progresses and the attachment and marginal tissue level follows. If no attachment loss is evident and probing depths are normal, one can assume that the implant is associated with a nonpathogenic microflora and that the peri-implant tissues are not clinically inflamed [1].

Implant salvage

The type of intervention for implant salvage depends largely on clinical findings and implant characteristics. Peri-implant mucositis is a reversible process that often responds well to conservative, noninvasive treatment because increased probing depths are usually caused by soft tissue inflammation and not crestal bone loss. Implants that show evidence of mucosal inflammation, plaque, and accumulation of calculus but lack suppuration and probing depths more than 3 mm are often treated effectively with mechanical débridement. Special nonmetallic instruments should be used for débridement to minimize surface defects and the theoretical possibility of galvanic corrosion. Special rubber cups and implant polishing paste can be used to remove plaque [20]. Subgingival chlorhexidine irrigation may be added to the regimen for cases in which probing depths have increased to 4 to 5 mm and inflammation and plaque/calculus deposits are noted. Generally, treatment for 3 to 4 weeks with chlorhexidine as a daily rinse or gel is required to achieve the desired result [20].

Lang et al [20] recommend antibiotic treatment in addition to mechanical débridement and antiseptic

treatment for peri-implant probing depths more than 6 mm. Tetracycline has been used for many years in treating periodontally involved teeth because of its antibiotic properties and facilitating effect on fibroblastic growth and attachment on root surfaces [16]. The decision to use tetracycline in implant therapy may depend on the type of implant surface because of the possibility of tetracycline altering the composition of the hydroxyapatite coating [16]. Treating titanium implant surfaces with topical tetracycline (50 mg/mL) for 3 minutes before regenerative techniques has been reported as successful [23]. Actisite (Alza, Palo Alto, CA) is a local delivery system that consists of nonresorbable tetracycline fibers for local use around periodontally involved teeth. It has been supplanted largely by resorbable delivery systems. Several case reports have documented the effectiveness of the tetracycline fibers around infected dental implants [20].

Some authors advocate systemic antibiotics before and during implant salvage techniques. Three recommended regimens are (1) clindamycin, 150 mg orally three times a day, (2) doxycycline hyclate, 100 mg orally twice daily, and (3) amoxicillin with or without clavulonic acid, 500 mg four times a day. Some authors recommend the addition of metronidazole if amoxicillin is selected for systemic treatment. All regimens should be started 2 days before implant salvage treatment and continued for 10 days after [18].

Peri-implantitis has been shown to be an endotoxin-mediated host response that progresses to implant failure if not treated. Differences among implant surface characteristics are important when selecting appropriate interventional techniques for implant salvage. Zablotsky et al [24] published a landmark study in 1992 that compared the abilities of various chemotherapeutic modalities to detoxify endotoxin-contaminated implant surfaces (grit-blasted titanium alloy and grit-blasted titanium alloy with a hydroxyapatite plasma spray coating). The study concluded that detoxification of hydroxyapatite-coated implants is best accomplished with anhydrous citric acid reconstituted to a 40% pH 1 (supersaturated) solution that is applied to the implant surface for 30 seconds to 1 minute [24]. This effect is caused by a demineralization of the superficial hydroxyapatite layer. The titanium grit-blasted surface was effectively decontaminated by burnishing with saline or citric acid for 1 minute. Air powder abrasives, such as sodium bicarbonate mixed with sterile water, also have been shown to be effective in decontaminating implant surfaces [25]. Stannous fluoride treatment seems to result in significantly greater levels of

endotoxin on both types of implant surfaces when compared with controls, whereas treatment with chlorhexidine gluconate, tetracycline HCl, hydrogen peroxide, and chloramine T is less effective at decreasing levels of endotoxin than saline burnishing alone. Charge interactions have been proposed as the reason for endotoxin having a greater affinity for hydroxyapatite-coated surfaces than grit-blasted titanium alloy surfaces [24]. Studies also have shown that chlorhexidine and stannous fluoride can result in the binding of endotoxin to the hydroxyapatite implant surface because of the inherent charge characteristics of these two compounds [16].

Surgical treatment should be considered for any implant that displays radiographic evidence of progressive crestal bone loss that still has adequate residual bony anchorage. If surgical treatment is to be initiated, the patient first should be placed on antibiotics, then a mucoperiosteal flap is elevated to expose the defect, and the implant surface is decontaminated by one of the methods described previously. Any granulation tissue should be removed with instruments that do not scratch or contaminate the titanium surface so that it is more favorable for regenerated tissue or osseointegration. Hydroxyapatite implants should be inspected for evidence of surface pitting, cracking, or color changes. If the hydroxyapatite shows wear or contamination, the entire layer should be removed mechanically and the underlying titanium surface decontaminated in the manner described previously.

Guided bone regeneration (GBR) also can be used to treat osseous defects around failing implants (Fig. 4) [16]. Decontamination of the diseased implant surface is paramount if this technique is to be used. The process for GBR involves placing a resorbable or nonresorbable membrane over an osseous defect to permit new bone growth into the defect while inhibiting soft tissue infiltration [26]. GBR has been performed around peri-implant osseous defects with and without grafting; however, bone fill and attachment gain seem to be better achieved when a grafting material is used in conjunction with a barrier membrane [16]. Several different materials, including demineralized freeze-dried bone, autologous bone, and resorbable bovine-derived hydroxyapatite, have been used in various forms as grafting material in GBR. It has been proposed that an alloplast, such as nonresorbable hydroxyapatite or bioactive glass, be used if the implant surface is difficult to decontaminate because of the presence of vents, holes, or tortuous osseous defects [18]. Alloplasts do not achieve biologic healing in these cases but are effective in filling bony defects and minimizing peri-implant

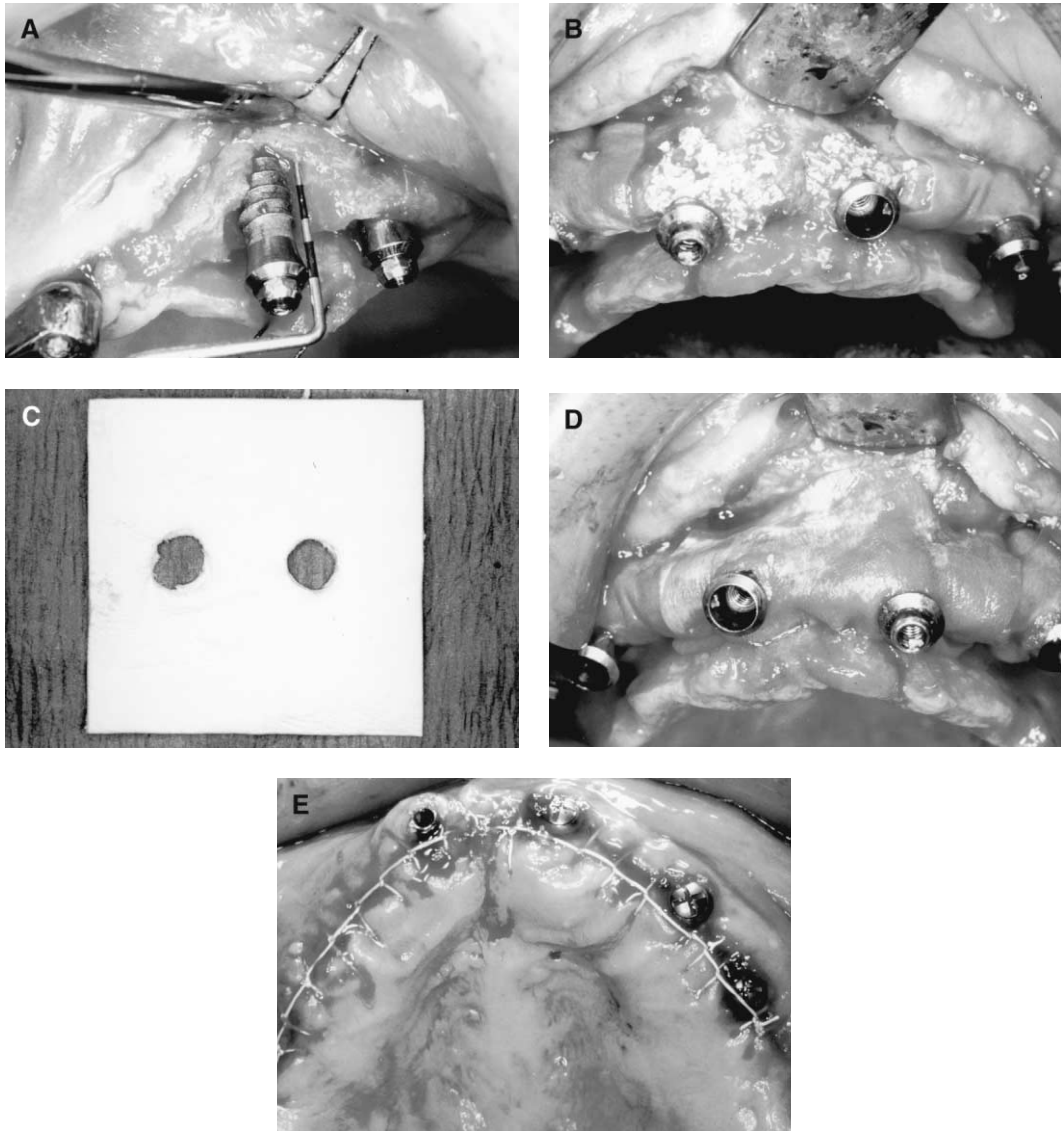


Fig. 4. Guided bone regeneration around a failing implant. (A) Large bony defect of a rough surface implant with increased probing depths and radiographic evidence of bone loss. (B) Bony defect covered with graft material after surface débridement and decontamination with tetracycline HCl paste. (C) Resorbable collagen membrane prepared to cover the graft material and necks of implants. (D) Collagen membrane in place. (E) Water-tight closure around implants and surgical site (some authors advocate burying single-stage implant systems after this procedure). (Courtesy of Dr. Tinou Roncone.)

pockets [18]. Although studies have demonstrated that GBR is effective around implants, questions have been raised regarding the ability of the regenerated bone to “re-osseointegrate” with the implant surface. Persson et al [27] published a study investigating whether “re-osseointegration” could be accomplished after the treatment of peri-implantitis. The study con-

cluded that “re-osseointegration” could not be achieved with a smooth (turned) surface but was consistently successful with a decontaminated sand-blasted, large grit, acid-etched surface [27]. If bone loss has progressed to the apical one third of the implant, removal is indicated because there is little chance of successful salvage [25].

The membrane selected for GBR should be bio-compatible and easy to place, maintain its original shape, and be capable of functioning as a barrier for at least 6 weeks [16]. A commonly used nonresorbable material, expanded-polytetrafluorethylene, has provided good results for many years. Several resorbable membranes recently have gained popularity, however, because of a reduced number of complications from infection or exposure and because they do not require retrieval surgery. Although it is acceptable to leave a membrane exposed during immediate implant placement in fresh extraction sites, primary closure over the membrane is necessary in implant salvage to prevent contamination and infection of the involved area. If GBR is to be used as a salvage procedure for implants, it is imperative that a nonresorbable membrane remain covered for as long as possible because premature removal decreases the chances of success and results in less-than-optimal bone fill [16]. Many authors recommend a minimum of 6 weeks before removal of a nonresorbable membrane [23]. If a resorbable membrane is to be used, it should provide barrier function for at least 6 weeks. It has been suggested that all patients remain on an antiseptic mouth rinse, such as chlorhexidine gluconate 0.12%, for 6 weeks or until the membrane is retrieved to minimize the chance of membrane infection [28]. If the membrane becomes prematurely exposed, however, immediate removal is indicated to prevent contamination of the regenerating tissues [16]. Single-stage implant systems ideally should be submerged during GBR salvage procedures to minimize contamination of the membrane and regenerating tissues.

Summary

Peri-implantitis is a treatable disease that affects functioning osseointegrated implants. Although unfavorable mechanical loading may play a contributing role, peri-implantitis seems to be mediated primarily by the endotoxins from gram-negative bacteria and the host response around the implant site. Many patients who were previously considered unfavorable candidates for implant therapy are being treated successfully when certain treatment considerations and implant maintenance programs are implemented.

It is essential that implant surgeons have a firm understanding of the favorable aspects of implant design and the potential liabilities when these systems are placed in unfavorable clinical conditions. Studies have demonstrated that the same potential benefits of implant materials and surface characteristics also may

lead to increased failure rates when these implants are affected by peri-implantitis. In the presence of adequate apical osseointegration, compromised implants that present with peri-implantitis must undergo thorough débridement and be decontaminated before any attempt at GBR. GBR may be accomplished successfully with many different types of membranes and grafting materials if the implant surface is thoroughly decontaminated before regenerative therapy. Implant salvage is an important, yet often ignored, component of clinical practice that can prevent implant and prosthetic failure if the principles of decontamination, biomodification, and guided tissue regeneration are understood and followed.

References

- [1] Mombelli A, Lang N. The diagnosis and treatment of peri-implantitis. *Periodontology* 2000;17:63–76.
- [2] Albrektsson T, Isidor F, et al. Consensus report of session IV. In: Lang NP, Karring T, editors. *Proceedings of the First European Workshop on Periodontology*. London: Quintessence; 1994. p. 365–9.
- [3] Løe H, Morrison E. Epidemiology of periodontal disease. In: Genco RJ, Goldman HM, Cohen DW, editors. *Contemporary periodontics*. St. Louis: CV Mosby Co.; 1990. p. 106–16.
- [4] Hammond BF, Genco RJ. Sensitivity of periodontal organisms to antibiotics and other antimicrobial agents. In: Genco RJ, Goldman HM, Cohen DW, editors. *Contemporary periodontics*. St. Louis: C.V. Mosby Co.; 1990. p. 161–9.
- [5] Trisi P, Rao W, Rebaudi A. A histometric comparison of smooth and rough titanium implants in human low-density jawbone. *Int J Oral Maxillofac Implants* 1999; 14:689–98.
- [6] De Leonardis D, Garg A, Pecora G. Osseointegration of rough acid-etched titanium implants: 5-year follow-up of 100 Minimatic implants. *Int J Oral Maxillofac Implants* 1999;14:384–91.
- [7] De Leonardis D, Garg A, Pecora G, et al. Osseointegration of rough acid-etched implants: one-year follow-up of placement of 100 Minimatic implants. *Int J Oral Maxillofac Implants* 1997;12:65–73.
- [8] Schwartz Z, Kieswetter K, Dean DD, Boyan BD. Underlying mechanisms at the bone-surface interface during regeneration. *J Periodont Res* 1997;32:166–71.
- [9] Tillmans HW, Hermann JS, Tiffée JC, et al. Evaluation of three different dental implants in ligature-induced peri-implantitis in the beagle dog. Part II. Histology and microbiology. *Int J Oral Maxillofac Implants* 1998;13:59–68.
- [10] Größner-Schreiber B, Griepentrog M, Haustein I, et al. Plaque formation on surface modified dental implants: an in vitro study. *Clin Oral Implants Res* 2001;12: 543–51.

- [11] Strnad Z, Strnad J, Povysil C, et al. Effect of plasma-sprayed hydroxyapatite coating on the osteoconductivity of commercially pure titanium implants. *Int J Oral Maxillofac Implants* 2000;15:483–90.
- [12] Johnson BW. HA-coated dental implants: long-term consequences. *CDA Journal of the California Dental Association* 1992;20:33–41.
- [13] Meffert RM, Block MS, Kent JN. What is osseointegration? *International Journal of Periodontics and Restorative Dentistry* 1987;7:9–21.
- [14] Cook SD, Kay JF, et al. Interface mechanics and histology of titanium and hydroxyapatite-coated titanium for implant applications. *Int J Oral Maxillofac Implants* 1987;2:15–22.
- [15] Gottlander M, Albrektsson T. Histomorphometric studies of hydroxyapatite-coated and uncoated CP titanium threaded implants in bone. *Int J Oral Maxillofac Implants* 1991;6:399–404.
- [16] Meffert RM. Periodontitis vs. peri-implantitis: the same disease? The same treatment? *Crit Rev Oral Biol Med* 1996;7:278–91.
- [17] Scarano A, Di Domizio P, Petrone G, et al. Implant periapical lesion: a clinical and histologic case report. *J Oral Implantol* 2000;26:109–13.
- [18] Martin RM, Carter JB, Barber HD. Surgical implant failures. In: Fonseca R, Powers MP, Barber HD, editors. *Oral and maxillofacial surgery: reconstructive and implant surgery*. Philadelphia: W.B. Saunders Co.; 2000. p. 275–308.
- [19] Esposito M, Hirsch J-M, Lekholm U, et al. Biological factors contributing to failures of osseointegrated oral implants (I). Success criteria and epidemiology. *Eur J Oral Sci* 1998;106:527–51.
- [20] Lang NP, Wilson TG, Corbet EF. Biological complications with dental implants: their prevention, diagnosis and treatment. *Clin Oral Implant Res* 2000;11:146–55.
- [21] Garqinto AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. *Journal of Periodontology* 1961;32:261–7.
- [22] Hermann J, Buser D, Schenk RK, et al. Biologic width around one- and two-piece titanium implants: a histometric evaluation of unloaded nonsubmerged and submerged implants in the canine mandible. *Clin Oral Implants Res* 2001;12:559–71.
- [23] Mellonig JT, Griffiths G, Mathys E, et al. Treatment of the failing implant: case reports. *International Journal of Periodontics and Restorative Dentistry* 1995;15:385–95.
- [24] Zablotsky MH, Diedrich DL, Meffert RM. Detoxification of endotoxin contaminated titanium and hydroxyapatite-coated surfaces utilizing various chemotherapeutic and mechanical modalities. *Implant Dentistry* 1992;1:154–8.
- [25] Jovanovic SA. The management of peri-implant breakdown around functioning osseointegrated dental implants. *J Periodontol* 1993;64:1176–83.
- [26] Rominger JW, Triplett RG. The use of guided tissue regeneration to improve implant osseointegration. *J Oral Maxillofac Surg* 1994;52:106–12.
- [27] Persson LG, Berglundh T, Sennerby L, et al. Re-osseointegration after treatment of peri-implantitis at different implant surfaces: an experimental study in the dog. *Clin Oral Implant Res* 2001;12:595–603.
- [28] Lehmann B, Bragger U, Hammerle CHF, et al. Treatment of an early implant failure according to the principles of guided tissue regeneration (GTR). *Clin Oral Implant Res* 1992;3:42–8.