Current Status of Dental Implants: A Periodontal Perspective

Perry R. Klokkevold¹/Michael G. Newman, DDS²

The success of osseointegrated dental implants has revolutionized dentistry. The ability to permanently replace missing teeth with a function and appearance close to that of the natural dentition has never been greater. With more than 3 decades of evidence to support the clinical use of osseointegrated dental implants, it is possible to confidently resolve that implants are predictable and provide patients with long-term functional tooth replacement.¹⁻⁶ This is a remarkable accomplishment, considering the many challenges and stresses that the oral environment and forces of mastication present for dental implants. The success of dental implants has transitioned dentistry into an entirely different approach to treatment compared to just 20 years ago.

CHANGED PHILOSOPHY AND PRACTICE OF PERIODONTICS

Perhaps more than any other dental specialty, the current success of dental implants has dramatically changed the philosophy and practice of periodontics. Many of the "rules" of periodontal therapy have been forever changed, with a paradigm shift from the practice of saving teeth at all costs toward one that will consider the extraction of "maintainable" teeth to improve esthetics, function, and long-term success of dental implant restorations. Prior to the current era of predictability with dental implants, periodontal patients, along with their dentist and/or periodontist, would strive to maintain periodontally compromised teeth. Many times, the goal was to preserve their "natural" teeth to avoid a removable prosthesis.

Surgical periodontal therapy, although beneficial for improving periodontal health and maintaining compromised teeth, is often destructive to the tooth (root removal, tooth hemisection) and supporting structures (pocket reduction surgery, osseous resective surgery). Each of these treatment modalities has the potential to compromise form, function, and esthetics. Because of periodontal therapy, patients who are struggling to keep their teeth often suffer from root sensitivity, increased interdental spaces, poor esthetics, and limited function. When teeth have compromised periodontal support, they often have increased mobility and may become (subjectively) uncomfortable or painful in function.

Splinting is another treatment used to help maintain periodontally compromised teeth and overcome the discomfort of mobility. Compromised teeth are splinted to adjacent teeth to gain support, functional stability, and comfort, and to potentially protect against additional bone loss. The ultimate form of tooth splinting therapy is the periodontal prosthesis, which typically involves full-crown restoration and splinting of many if not all of the remaining teeth.

With these forms of periodontal treatment and good plaque control, it is possible to maintain severely periodontally compromised teeth for long periods of time without additional loss of tissue attachment or bone support, but the periodontal condition of the tooth rarely if ever improves over time, regardless of treatment. At best, the tooth is kept in place and may or may not provide a significant functional purpose for the individual.

The predictability of dental implants has changed the perspective on periodontal therapy and the ability to provide reconstructive treatment for patients who suffer from the destruction of periodontal disease. The "maintainance" of periodontally compromised

¹Associate Professor and Clinical Director, UCLA School of Dentistry, Division of Associated Specialties, Section of Periodontics, Los Angeles, California.

²Adjunct Professor, UCLA School of Dentistry, Division of Associated Specialties, Section of Periodontics, Los Angeles, California; Medical Science Systems, Newport Beach, California.

Reprint requests: Dr Perry Klokkevold, UCLA Periodontics and Implant Surgery, 63-022A CHS-Dental, Los Angeles, CA 90095-1668.

teeth to avoid tooth loss is no longer necessary. In fact, removal of severely periodontally compromised teeth and replacement with implants will usually enhance the overall function, esthetics, and comfort of the definitive implant-supported or implantassisted dental prosthesis. Whereas compromised teeth with severe attachment loss, moderate to severe bone loss, and mobility have a very limited capacity to regain natural periodontal form, function, and esthetics, implants placed in conjunction with tissueregenerative procedures can restore not only the missing teeth but in some cases the surrounding tissues as well.

The last 20 years have been significant in periodontics, not only because of the success of osseointegrated dental implants, but also because of an improved understanding of periodontal disease, the host response to periodontal disease, and the requirements for guided tissue regeneration. A great deal has been learned from experience with implants. Initial implant success and surgical protocols were established primarily in a completely edentulous patient population. The implants and the armamentarium were initially designed for the edentulous patient. The simple transfer of the protocols of the Brånemark implant successes with edentulous patients did not automatically result in the same level of success with partially edentulous patients. It has become evident (sometimes painfully) that the partially edentulous patient is not the same as the edentulous patient. Many factors unique to the partially edentulous patient make their treatment success or failure rate different than completely edentulous patients.

The purpose of this article is to provide an overview of the current status of implants as related to the practice of periodontics.

RESTORING FORM, FUNCTION, AND ESTHETICS

The success of dental implants in the completely edentulous patient quickly led to applications in the treatment of the partially edentulous patient (Table 1). In the early 1980s, when osseointegrated dental implants became an accepted mode of therapy, the surgical goal of therapy was the placement of an implant in the available bone. The prosthetic position was not thought to be critical to the success of implants, and therefore it was not a major consideration. Resorption of alveolar bone is common following tooth loss, and sometimes it is so severe that the only remaining bone is basal bone. As a result, implant positioning in bone was too far palatal (maxilla) or too far labial.

One of the most gratifying aspects of implant dentistry in periodontics is the ability to replace missing teeth (and supporting tissues) in an esthetic and predictable manner. Restoration of the form and function of missing teeth and, whenever possible, supporting hard and soft tissues is critical to the esthetics of any implant case. This is especially true for the partially edentulous patient, since the remaining teeth and supporting tissues serve as a visual reference point to the normal dimensions of the periodontium. Any deficiency in soft tissue anatomy around the ideally positioned and contoured tooth restoration is noticeable. Because of the esthetic demands of patients, implant therapy has become increasingly focused on prosthetically driven implant position. Several new evidence-based guidelines emerged from prosthetically driven implant positioning, including the need to reconstruct hard and soft tissues that had been lost. In other words, if there was a lack of bone in the area that was planned to receive an implant, then a bone augmentation or bone regenerative procedure had to be considered. The combination of complex biologic processes and sophisticated technical procedures has improved the quality of life for millions of patients.

The advances seen with the development and progress of dental implants coincide with an equally important advancement in the understanding and ability to regenerate lost periodontal tissues. The concepts of guided tissue regeneration were then adopted for guided bone regeneration. Hard tissue regeneration made it possible to restore form and function to the edentulous and partially edentulous patient. As a result, surgical treatment planning for implant cases has become increasingly complex and demanding, because both the patient and clinician have greater expectations and demands as compared to earlier years (the 1980s). Peri-implant soft tissue management has also become critical to the creation of maximum esthetics, and it has become more and more apparent that hard tissue regeneration is essential to soft tissue esthetics.

The biologic principle of different tissue contributions to periodontal wound healing, originally described by Melcher in 1976,⁷ was used to define the essential elements of guided tissue regeneration in a series of studies by Nyman, Karring, and coworkers.^{8–10} Subsequently, guided tissue regeneration procedures and techniques were adapted advantageously to exploit the concept of separating soft tissues from bone so as to selectively favor the formation of new bone. This concept, termed "guided bone regeneration," is ideal for generating increased bone volume for implant placement. Several studies in animals and humans have demonstrated the

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER.

Dental/periodontal condition	Conventional treatment	Comments	Implant alternative	Comments
Single missing tooth	3-unit fixed prosthesis; Maryland (resin-bonded) prosthesis; removable prosthesis	Requires preparation of adjacent teeth; many patients do not like removable prostheses	Implant restoration; depending on amount and location of existing tissue, may need bone and/or soft tissue aug- mentation	No preparation of adjacent teeth; solid " natural" look and feel of tooth replace- ment; long-term suc- cess is good
Periodontally com- promised teeth with moderate to severe bone and attachment loss	Periodontal surgery (extent of treatment depends on severity of disease); periodontal regenerative surgery to improve bone fill and attachment levels; peri- odontal splinting	Teeth can be maintained long-term; regenerative success depends on type of defect as well as other factors; splinted teeth "maintained" but not "improved"	Extraction; may require bone regenerative/ augmentation proce- dure; implant restoration	After bone and soft tissues establish steady state, bone loss is minimal; long- term success is good
Moderate to severe furcation-involved tooth	Periodontal surgery to reduce pocket depth and increase access for oral hygiene; periodontal regenerative surgery to improve bone fill and attachment levels	Prognosis diminished; risk of further bone loss and attachment loss	Extraction; possible bone regenerative procedure; implant restoration	Eliminates furcation problem; after bone and soft tissues establish steady state, bone loss is mini- mized; long-term suc- cess is good
Multiple missing teeth, eg, unilateral distal extension	Removable partial denture	Partial moves	Multiple unit fixed implant-supported restoration; depending on amount and location of existing tissue, may need bone and/or soft tissue augmentation	No preparation of adjacent teeth; solid "natural" look and feel of tooth replace- ment; long-term suc- cess is good
Edentulous arch	Complete denture	Removable; bone resorption and tissue changes continue	Implant-assisted overdenture; implant- supported fixed denture	Patients are more confident with their implant restoration; bone resorption and tissue changes mini- mized; long-term suc- cess is good

Table 1 Comparison of Conventional Dental Treatment Options with Implant Treatment for Selected Dental/Periodontal Problems*

*Note regarding implant treatment options: Not all patients are candidates for implants. Some require bone and soft tissue grafting to achieve the desired results.

viability of generating new bone with a barrier membrane, space-making devices, bone graft materials, or a combination of these materials.^{11–13}

The incorporation of implants into periodontal practice has changed many of the day-to-day systems and procedures. The techniques and measures that implant dentistry requires demand an increased need for advanced bone augmentation approaches. Many periodontists are incorporating advanced surgical skills that enable them to achieve better and better results. For example, the use of onlay grafts, particulate grafts, and sinus elevation surgery allows placement of implants into more ideal and less compromised positions. Other advances include the harvesting of autogenous bone; the use of barrier membranes, tacks, or screws for fixation; and modification of flap management. Although the concept and success of guided bone regeneration is widely accepted, the achievement of predictable results depends on the care and precision of the operator. Guided bone regeneration techniques are technically more demanding and less forgiving than other surgical procedures. New bone growth requirements include space maintenance, adequate blood supply, osteoblasts or osteoprogenitors, and wound stability. If any of these basic requirements for guided bone regeneration are lacking or inadequate, the success of bone regeneration will be compromised (Table 2).

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER.

Table 2 Requirements for Successful Bone Regeneration Biologic Technical				
Biologic requirements	requirements	considerations		
Good blood supply to bone graft site	Perforation of cortical bone to create "bleed-	Systemic conditions (eg, diabetes) can alter wound healing and might impair regenera- tion		
Space maintenance for new bone	ing" points for blood supply and source of osteoblasts and osteo-			
Separation of soft tissues from bone	progenitors	Some medications (eg, immunosuppressive drugs) can impair heal- ing and regeneration Habits (eg, smoking) may impair wound healing and potential		
Migration and prolifera- tion of osteoblasts and	Use of barrier mem- branes; important to establish intimate con- tact with bone and to stabilize with screws or tacks			
osteoprogenitors Bone (graft) stabilization to prevent any				
micromovement	Adequate blood supply	for bone regeneration		
Primary wound closure and stabilization:	maintained by good flap management	Any pressure (eg, pros- thesis) placed over the		
important to achieve blood clot stability	Bone graft stabilization with screw fixation (may include use of fixation plate or mesh)	surgical site can cause wound dehiscence and impair potential for bone regeneration		
	Flap management designed to achieve primary closure over increased volume with- out tension; important to release periosteum			

PERI-IMPLANT PATHOLOGY AND IMPLANT FAILURE

Peri-implant pathology, or more specifically periimplant bone loss, has been attributed to several different factors, including poor surgical management, failure to achieve osseointegration, premature loading, biomechanical overload, peri-implant infection, and impaired host response. Poor surgical placement, premature loading, and implants that fail to achieve initial osseointegration are all related to early implant failures and will not be discussed as part of this review. The most significant factors that contribute to bone loss and implant failure in the otherwise healthy patient include biomechanical overload14 and bacterial infection (periimplantitis).15 Peri-implant infection and biomechanical overload are etiologic factors that can contribute to progressive bone loss after implants have been in function. They require early recognition and treatment.

Osseointegrated dental implants, unlike implants and hardware used elsewhere in the body, have different environmental and functional stresses because

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER. of the transmucosal nature of their tissue position. The peri-implant tissue arrangement is unique to dental implants. No other implants or implanted materials placed in any other anatomic locations of the body face the challenges that dental implants face in the oral cavity. The transmucosal communication of the implant (abutment) material with the bacterial flora of the oral cavity presents a host of potential problems to be overcome for the success of dental implants. This implant/abutment transition through the oral soft tissues creates an opportunity for bacterial and soft tissue invasion of the bone-implant interface. Hence, a mucosal tissue seal around the coronal aspect of the implant is critical to the preservation of osseointegration and ultimately to the success of the implant. Whether the implant is a 1- or a 2-stage design, the oral soft tissues must establish a seal around the abutment and/or implant to prevent invasion by bacteria into the underlying connective tissues and bone. This soft tissue seal is established within the first week after second-stage surgery (abutment connection) or at the time of implant placement for 2-stage and single-stage implant designs, respectively.

PERI-IMPLANT TISSUE ANATOMY FROM THE PERIODONTAL PERSPECTIVE

There are some distinct differences in the structure and function of soft tissues around osseointegrated dental implants, as compared to gingival soft tissues around teeth. The periodontal tissues that surround a tooth are often discussed with respect to their anatomic location and their functional attributes. These tissues include the periodontal ligament, the connective tissue attachment, the long junctional epithelium, the sulcular epithelium, and the masticatory or gingival epithelium. The primary distinguishing features of periodontal tissues, as compared to peri-implant tissues, are the periodontal ligament and the supra-bony connective tissues. The tooth is attached to the alveolar bone and the supra-bony gingival connective tissues via periodontal ligament and connective tissue fibers. The connective tissue fibers are attached to the cementum (and dentin) of root surfaces via perpendicular collagen bundles or Sharpey's fibers. This network of collagenous fibers suspends the tooth within the alveolar bone and provides a unique resilient sling that allows for physiologic tooth movement.

Osseointegrated dental implants do not have any connective tissue fiber attachments. There are no suspending or otherwise interposed connective tissues between the bone and the implant. As a result, implants have no mobility. Osseointegrated dental implants by definition do not have any soft tissues intervening between the implant surface and the bone. There are no collagen fibers attached to the implant surface. There is a connective tissue network of fibers around the implant coronal to the level of supporting bone. The supra-bony connective tissue surrounding the implant is made up of circumferential fibers that run parallel to the implant surface.^{16,17} Similar to teeth, soft tissues surrounding implants form an epithelial attachment, a sulcular epithelium, and, depending on the nature of the surrounding tissue, may also have masticatory mucosa.

BIOLOGIC DIMENSION OF SOFT TISSUES AROUND IMPLANTS

Several investigators have evaluated healthy periimplant tissues and determined the connective tissue dimension to be 1 to 1.5 mm in height.^{16,18} This zone of connective tissue was found to be collagenrich and cell-poor. Berglundh and Lindhe¹⁸ used a beagle dog model to measure the peri-implant soft tissue dimensions. Regardless of the type of implant used and the soft tissue dimensions at the time of placement, the authors found that a 2-mm-long junctional epithelium and a 1-mm zone of connective tissue were consistently established.¹⁸ It is interesting to note that at sites where the mucosa was thinned to 2 mm or less, bone resorption and soft tissue growth occurred to re-establish a mucosa-implant attachment that was approximately 3 mm. Hence, similar to the finding by Gargiulo et al¹⁹ of a biologic width of connective tissue and epithelial attachment around teeth, implants appear to have a minimum requirement for connective tissue attachment dimension. This determination is consistent with the initial bone loss pattern seen in 2-stage, Brånemark-type implants. Bone loss occurs around the coronal aspect of the implant to re-establish biologic dimension.

PERI-IMPLANTITIS

Peri-implantitis is defined as an inflammatory process affecting the tissues around an osseointegrated implant in function that results in loss of supporting bone, while peri-implant mucositis is an inflammatory process distinguished from periimplantitis by the lack of bone loss.²⁰ Peri-implant mucositis is believed to be a reversible condition, similar to gingivitis. Bone loss associated with periimplantitis is typically circumferential or "saucer" shaped, as opposed to periodontal bone loss, which is localized to one side. The other interesting finding with peri-implant bone loss, as compared to bone loss around natural teeth, is that the shape of the bony defects appears to be influenced by the macroscopic shape of the implant. Screw-type implants tend to exhibit more of a flat (horizontal) defect, while cylindric implants exhibit deep angular (vertical) defects.²¹ Implant surface characteristics may also influence the shape of the bony defect. Implants with surface coatings have surface characteristics that can harbor and perpetuate infections, causing bone loss in a vertical direction. Implants with peri-implantitis can remain stable (no mobility) until osseointegration is completely lost, regardless of the amount or severity of inflammation, bleeding, and pocket depth.

Microbiology

There is ample evidence to support the relationship of bacterial plaque to peri-implant disease, similar to the cause-and-effect relationship between bacterial plaque and periodontal disease. The causative role of anaerobic bacteria on peri-implant mucositis and peri-implantitis has been documented from several different lines of research, including experimentally induced mucositis,^{22,23} documentation of different microorganisms at healthy versus diseased sites,^{24–27}

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER. improvement of peri-implantitis with antimicrobial therapy,^{28,29} and evidence that good oral hygiene enhances long-term implant success.³⁰

Dental implants with probing depths greater than 6 mm have been associated with a higher percentage of anaerobic, gram-negative bacteria. The pathogens identified are similar to those found in periodontal disease sites. DNA probe analysis identified moderate levels of Actinobacillus actinomycetemcomitans, Bacteroides (Prevotella) intermedius, and Bacteroides (Porphyromonas) gingivalis at failing implant sites.³¹ On the other hand, healthy sites in patients with both diseased and healthy sites had smaller amounts of bacteria dominated by facultative grampositive cocci and rods.32 It is suggested that periimplant tissues behave very similarly to periodontal tissues and that peri-implantitis lesions should be considered as site-specific infections harboring a high number of periodontal pathogens, mainly gram-negative anaerobic rods.

Danser and coworkers³³ evaluated 20 edentulous implant patients with past history of periodontal disease (reason for extraction of remaining teeth included extreme mobility). Clinical and microbiologic examination revealed healthy peri-implant tissues with a mean probing implant pocket depth of 3.6 mm and a healthy composition of bacteria. The results indicate that when periodontally diseased teeth are extracted prior to implant therapy, the subsequent peri-implant microbiota are composed of bacteria associated with periodontal health or gingivitis.³³ This finding strongly suggests that the elimination of periodontal pathogens in the subgingival environment by periodontal therapy or extraction of diseased teeth would have a beneficial effect on the microflora around implants.

Inflammatory Response

Similar inflammatory responses to bacterial plaque around teeth and implants have been shown.^{34,35} Inflammatory cell infiltrates were consistently found in the connective tissues adjacent to the long junctional epithelium in both gingiva and peri-implant mucosa. In a beagle dog study, Berglundh et al²² demonstrated that the masticatory mucosa around implants and the gingiva around teeth responded similarly to de novo plaque formation with the development of an inflammatory lesion. The magnitude and composition of the lesions in both tissues had common features. It was concluded that the mucosa around implants and the gingiva around teeth had a similar potential to respond to early plaque formation.²²

Despite all the similarities in periodontal and peri-implant soft tissues, it has been suggested that implants are resistant to peri-implant tissue destruction. The differences in the anatomic features of tissues surrounding implants and teeth suggest differences in function and may result in different susceptibility to breakdown by inflammatory disease. Wilson and Nunn have recently reported³⁶ that there was no correlation between the interleukin-1 genotype, previously associated with severe periodontitis, and early implant failure. One possible explanation is the absence of periodontal ligament cells, which are associated with inflammatory mediators.³⁶ The frequency of peri-implantitis is estimated to be as low as 4% to as high as 15% with various implant systems.^{37,38}

Peri-implant Soft Tissues

The question of whether a zone of keratinized attached mucosa surrounding dental implants is important to the health of peri-implant tissues has not been determined. In a primate study designed to evaluate the susceptibility of implants, with and without a zone of keratinized attached mucosa, to peri-implantitis, the implants with movable, nonkeratinized mucosa appeared to be more susceptible to the progression of peri-implantitis.³⁹ However, clinical studies fail to support the concept that a lack of keratinized attached tissue leads to an increased progression of peri-implantitis.⁴⁰ It is difficult to conclude, with a lack of evidence to support or refute the need for a zone of keratinized attached tissue, whether soft tissue grafting is indicated in patients with minimal or no attached tissue. Logic would suggest that a firm, relatively avascular zone of keratinized attached tissue could offer more resistance to injury and disruption of its seal around the implant. Numerous techniques have been described to increase the zone of attached tissue, either at the time of second-stage surgery or subsequent to restoration.^{41,42} The patient's ability to perform oral hygiene is improved as compared to having movable, non-keratinized tissue.

TREATMENT OF PERI-IMPLANTITIS

The most important aspect of treatment for periimplantitis is to stop the progression of bone loss by controlling bacterial infection. Because of ethical considerations and because peri-implantitis does not occur frequently, only a limited number of longitudinal studies have been conducted to evaluate different peri-implantitis treatment modalities. Most studies involve animal models designed to simulate the occurrence of peri-implantitis. Of particular interest is the finding that peri-implantitis–induced bone loss

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER.

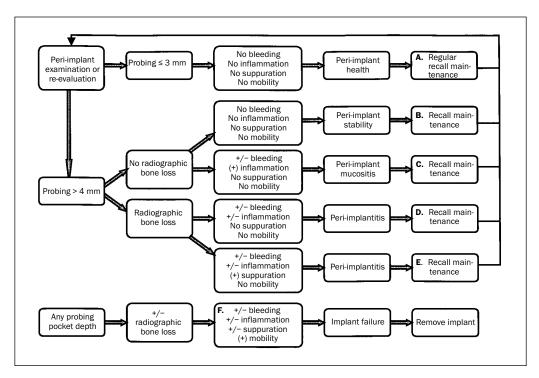


Fig 1 Algorithm for the diagnosis and treatment of peri-implantitis. A. Regular recall maintenance includes oral hygiene, plaque and calculus removal at intervals appropriate for the individual patient. B. A. plus consider increasing recall frequency. May consider surgical reduction of pockets \geq 5 mm. C. A. and B. plus consider topical rinse, irrigation, or use of antimicrobial controlled-release device to control inflammation. D. A. through C. plus consider surgical correction of osseous defect. E. A. through D. plus consider systemic antibiotics to control infection. Check for foreign material in peri-implant pocket (eg, cement). F. Check the fit and integrity of restorative components.

can be induced only with ligatures.⁴³ In another study, Klinge found that bone loss proceeds more slowly around implants than around teeth.⁴⁴

Similar to conventional periodontal therapy, initial treatment is non-surgical and consists of plaque control and removal of calculus deposits (Fig 1). Any other contributing factor should be addressed as well, including adjustment of occlusal forces. In more advanced cases, surgical therapy may be indicated. As with periodontal therapy, the goal of surgical therapy is complete debridement of the defect, decontamination of the implant surface, and possibly removal of any porous implant surface coatings.

Once the inflammatory disease process is controlled, it is possible to attempt regenerative procedures. There are some reports documenting successful treatment of peri-implant defects. However, histologic evidence of the reestablishment of osseointegration following contamination of the implant surface is lacking. In one dog study, Jovanovic and coworkers observed some re-osseointegration following treatment of peri-implantitis.⁴⁵ In another dog study, Persson and coworkers attempted to regenerate bone around peri-implantitis–induced defects.

62 Volume 15, Number 1, 2000

They found only dense connective tissue around the previously contaminated implant surfaces.⁴⁶ The lack of re-osseointegration of contaminated implant surfaces is most likely the result of an inability to completely decontaminate the implant surface and restore its original "out of the package" characteristics.

Biomechanical Overload

In addition to bacterial infection, biomechanical overload has been shown to contribute to periimplant bone loss. Bone loss associated with biomechanical overload is most frequently found to be localized around the coronal aspect of the boneimplant interface. The subgingival microbial composition of implants under excessive stress was found to be comparable to that of healthy sites.³² Some investigators have suggested that biomechanical overload causes microfractures in the bone around the coronal aspect of the bone-implant interface.⁴⁷ The loss of bone allows soft tissue invasion into the space between the bone and the implant. If stresses continue to be excessive or if a bacterial infection is present, bone loss can continue to progress similar to the situation in natural teeth.

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER.

Implant Monitoring and Maintenance

While the long-term success rates for dental implants are very good, most clinicians and researchers agree that monitoring and maintenance of implants and the peri-implant supporting tissues are important. It is equally as important to periodically evaluate and clean dental implants as it is for teeth. The frequency of dental implant monitoring and maintenance visits will vary from one individual to another, based on their ability to perform oral hygiene and their individual response to the peri-implant bacterial challenge. It is generally agreed that clinical examination parameters adopted from periodontal examination, such as probing depth, tissue attachment level, bleeding on probing, suppuration, mobility, plaque, and gingival inflammation, are important, but their clinical significance for determining future breakdown or success of implants has been questioned.

Radiographs

Radiographs should be taken periodically to evaluate potential loss of bone. It has been well established that the amount of bone loss around Brånemark-type implants is approximately 1 to 1.5 mm in the first year after loading and 0.1 mm annually thereafter.48 The only way to effectively monitor bone loss is to document with standardized digital or conventional radiographs the bone level around implants at baseline (time of implant placement and/or time of restoration) and at regular intervals over time. However, standardized radiographs are not practical for dental practices and are usually used only in research settings. Nonetheless, the use of radiographs, albeit not standardized, to monitor bone levels around implants is an important part of the documentation. Severe horizontal bone loss, peri-implant radiolucencies, and loss of implant osseointegration can be detected with periodic radiographs.

Microbiologic Monitoring

In a review of the peri-implantitis literature,²⁰ Mombelli and Lang concluded that too little is known about the relationship between specific bacteria associated with peri-implantitis and progression of bone loss to be able to assess the benefit of microbiologic testing as a tool for monitoring patients and determining future risk of breakdown.²⁰

Maintenance

At regular intervals (every 3 to 6 months), implant abutment/restoration surfaces should be debrided of plaque and calculus accumulations. Scratching of titanium implant surfaces may result in increased plaque accumulation and corrosion and decreased cellular attachment.^{49,50} Because of this concern, several

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER. instrumentation materials have been evaluated. Nylon brushes, rubber cups, and plastic scalers appear to be the safest, as they do not alter the titanium implant surface. Metal scalers, on the other hand, significantly scratch and gouge the titanium surface.⁵¹

EXISTING KNOWLEDGE DEFICIENCIES AND NEED FOR MORE INFORMATION

The ability to measure individual patient "host" response to implant therapy is essentially unavailable.³⁶ Current guidelines suggest avoiding the use of implants in individuals with bone metabolic disease, immune compromise, etc. There is evidence to suggest that implant therapy can be successful in individuals who have suffered from refractory or recalcitrant periodontal disease.52 A total of 309 implants was placed in 10 edentulous maxillae, 11 edentulous mandibles, 33 partially edentulous maxillae, and 27 partially edentulous mandibles. Only 3 maxillary and 4 mandibular implants failed over a period of 7 years of follow-up, for a survival rate of 98%. It is important to note that these patients were maintained on a regular recall interval of as often as every 3 months but never less frequent than every 5 months. Some bone loss around implants did occur in these patients. Many patients had 1 or more implants with radiographically evident bone loss to the first or second thread. A few implants (4 maxillary and 3 mandibular) lost bone to the fourth thread, but all of them stabilized and did not lose additional bone after the first year.

Jemt et al reported a 98.7% success rate for 876 implants placed in 244 patients.⁵³ Similar success rates have been reported by other investigators. Jaffin and Berman reported that implants placed in Type IV bone (poor-quality bone) had significantly lower success rates.⁵⁴ Lower success rates in poorquality bone have been reported by others as well.

It is advisable to avoid placing implants in individuals with parafunctional habits, such as bruxism. Similar caution must be considered and patients warned of the risk that smoking causes to implant success rates.^{55,56} Except for smoking, the relationship of various risk factors to implant failure is not well understood. These guidelines are based on the premise that if an individual were susceptible to bone disease, then their implant success rates would be diminished.

CONCLUSION/FUTURE DIRECTIONS

The last 20 years of incorporating implants into periodontal education and practice have transformed

periodontology. Comprehensive and sophisticated treatment plans can be developed, and highly skilled clinicians can carry out the proposed treatment with degrees of success that are as good or better than many conventional procedures. Good-quality evidence has been generated about many aspects of biology and practice that result in predictable and worthwhile outcomes. The range of treatment options now available to patients is more comprehensive than at any time in history. Currently, a great deal of biomaterial research is being conducted in an attempt to determine factors or substances that can improve the quality of bone-to-implant contact. Specifically, biomaterial alterations of the implant surface57-60 or biomaterial substances within the healing tissues⁶¹⁻⁶³ can have a bone-inducing effect, and the use of genetically engineered tissue factors will likely be available in the near future.

The combination of in-depth biologic knowledge together with extensive surgical training has given the periodontist the skills to provide the highest quality care to patients around the world.

REFERENCES

- Adell R, Eriksson B, Lekholm U, Brånemark P-I, Jemt T. Long-term follow-up study of osseointegrated implants in the treatment of totally edentulous jaws. Int J Oral Maxillofac Implants 1990;5(4):347–359.
- Lekholm U, Gunne J, Henry P, Higuchi K, Lindén U, Bergström C, et al. Survival of the Brånemark implant in partially edentulous jaws: A 10-year prospective multicenter study. Int J Oral Maxillofac Implants 1999;14:639–645.
- Spiekermann H, Jansen VK, Richter EJ. A 10-year followup study of IMZ and TPS implants in the edentulous mandible using bar-retained overdentures. Int J Oral Maxillofac Implants 1995;10(2):231–243.
- Albrektsson T, Zarb GA, Worthington P, Eriksson AR. The long-term effficacy of currently used dental implants: A review and proposed criteria of success. Int J Oral Maxillofac Implants 1986;1(1):11–25.
- Zarb GA, Schmitt A. The longitudinal clinical effectiveness of osseointegrated dental implants: The Toronto study. Part II: The prosthetic results. J Prosthet Dent 1990;64(1):53–61.
- Lazzara R, Siddiqui AA, Binon P, Feldman SA, Weiner R, Phillips R, Gonshor A. Retrospective multicenter analysis of 3i endosseous dental implants placed over a five-year period. Clin Oral Implants Res 1996;7(1):73–83.
- Melcher AH. On the repair potential of periodontal tissues. J Periodontol 1976;47:256–260.
- Karring T, Nyman S, Lindhe J. Healing following implantation of periodontitis infected roots into bone tissue. J Clin Periodontol 1980;7:96–105.
- Karring T, Nyman S, Lindhe J, Stirirat M. Potential for root resorption during periodontal healing. J Clin Periodontol 1984;11:41–52.
- Nyman S, Karring T, Lindhe J, Planten S. Healing following implantation of periodontitis-affected roots into gingival connective tissue. J Clin Periodontol 1980;7(5):394–401.
- 64 Volume 15, Number 1, 2000

- Becker W, Becker BE, Handlesman M, Celletti R, Ochsenbein C, Hardwick R, Langer B. Bone formation at dehisced dental implant sites treated with implant augmentation material: A pilot study in dogs. Int J Periodontics Restorative Dent 1990;10(2):92–101.
- Nyman S, Lang NP, Buser D, Brägger U. Bone regeneration adjacent to titanium dental implants using guided tissue regeneration: A report of two cases. Int J Oral Maxillofac Implants 1990;5(1):9–14.
- Buser D, Ruskin J, Higginbottom F, Hardwick R, Dahlin C, Schenk RK. Osseointegration of titanium implants in bone regenerated in membrane-protected defects: A histologic study in the canine mandible. Int J Oral Maxillofac Implants 1995;10(6):666–681.
- Quirynen M, Naert I, van Steenberghe D. Fixture design and overload influence marginal bone loss and fixture success in the Brånemark system. Clin Oral Implants Res 1992; 3(3):104–111.
- Newman MG, Flemmig TF. Periodontal considerations of implants and implant associated microbiota. J Dent Educ 1988;52(12):737–744.
- Berglundh T, Lindhe J, Ericsson I, Marinello CP, Liljenberg B, Thomsen P. The soft tissue barrier at implants and teeth. Clin Oral Implants Res 1991;2(2):81–90.
- Abrahamsson I, Berglundh T, Wennstrom J, Lindhe J. The peri-implant hard and soft tissues at different implant systems. A comparative study in the dog. Clin Oral Implants Res 1996;7(3):212–219.
- Berglundh T, Lindhe J. Dimension of the peri-implant mucosa. Biological width revisited. J Clin Periodontol 1996; 23(10):971–973.
- Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. J Periodontol 1961;32:261–267.
- Mombelli A, Lang NP. The diagnosis and treatment of periimplantitis. Periodontol 2000 1998;17:63–76.
- Spiekermann H. Histopathology. In: Spiekermann H, Donath K, Hassell T, Jovanovic S, Richter EJ (eds). Implantology. New York: Thieme Medical Publishers, 1995.
- Berglundh T, Lindhe J, Marinello C, Ericsson I, Liljenberg B. Soft tissue reaction to de novo plaque formation on implants and teeth. An experimental study in the dog. Clin Oral Implants Res 1992;3(1):1–8.
- Pontoriero R, Tonelli MP, Carnevale G, Mombelli A, Nyman SR, Lang NP. Experimentally induced peri-implant mucositis. A clinical study in humans. Clin Oral Implants Res 1994;5(4):254–259.
- Becker W, Becker BE, Newman MG, Nyman S. Clinical and microbiologic findings that may contribute to dental implant failure. Int J Oral Maxillofac Implants 1990;5(1): 31–38.
- George K, Zafiropoulos GG, Murat Y, Hubertus S, Nisengard RJ. Clinical and microbiological status of osseointegrated implants. J Periodontol 1994;65(8):166–170.
- Mombelli A, Lang NP. Microbial aspects of implant dentistry. Periodontol 2000 1994;4(Feb):74–78.
- Salcetti JM, Moriarty JD, Cooper LF, Smith FW, Collins JG, Socransky SS, Offenbacher S. The clinical, microbial, and host response characteristics of the failing implant. Int J Oral Maxillofac Implants 1997;12(1):32–42.
- Ericsson I, Persson LG, Berglundh T, Edlund T, Lindhe J. The effect of antimicrobial therapy on peri-implantitis lesions. An experimental study in the dog. Clin Oral Implants Res 1996;7(4):320–328.
- Mombelli A, Lang NP. Antimicrobial treatment of periimplant infections. Clin Oral Implants Res 1992; 3(4):162–168.

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER.

- Lindquist LW, Rockler B, Carlsson GE. Bone resorption around fxtures in edentulous patients treated with mandibular fixed tissue-integrated prostheses. J Prosthet Dent 1988; 59:59–63.
- Becker W, Becker BE, Newman MG, Nyman S. Clinical and microbiologic findings that may contribute to dental implant failure. Int J Oral Maxillofac Implants 1990;5(1): 31–38.
- 32. Sanz M, Newman MG, Nachnani S, Holt R, Stewart R, Flemmig T. Characterization of the subgingival microbial flora around endosteal sapphire dental implants in partially edentulous patients. Int J Oral Maxillofac Implants 1990; 5(3):247–253.
- Danser MM, van Winkelhoff AJ, van der Velden U. Periodontal bacteria colonizing oral mucous membranes in edentulous patients wearing dental implants. J Periodontol 1997;68(3):209–216.
- 34. Lillenberg B, Gualini F, Berglundh T, Tonetti M, Lindhe J. Composition of plaque-associated lesions in the gingiva and the peri-implant mucosa in partially edentulous subjects. Clin Periodontol 1997;24(2):119–123.
- Tonetti MS, Imboden M, Gerber L, Lang NP. Compartmentalization of inflammatory cell phenotypes in normal gingiva and peri-implant keratinized mucosa. J Clin Periodontol 1995;22(10):735–742.
- Wilson TG Jr, Nunn M. The relationship between the interleukin-1 periodontal genotype and implant loss. Initial data. J Periodontol 1999;70(7):724–729.
- Weber HP, Buser D, Fiorellini JP, Williams RC. Radiographic evaluation of crestal bone levels adjacent to nonsubmerged titanium implants. Clin Oral Implants Res 1992; 3(4):181–188.
- Quirynen M, Naert I, van Steenberghe D, Schepers E, Calberson L, Theuniers G, et al. The cumulative failure rate of the Brånemark system in the overdenture, the fixed partial and the fixed full prostheses design. J Head Neck Pathol 1991;10:43–53.
- Warrer K, Buser D, Lang NP, Karring T. Plaque-induced peri-implantitis in the presence or absence of keratinized mucosa. An experimental study in monkeys. Clin Oral Implants Res 1995;6(3):131–138.
- Wennstrom JL, Bengazi F, Lekholm U. The influence of the masticatory mucosa on the peri-implant soft tissue condition. Clin Oral Implants Res 1994;5(1):1–8.
- Alpert A. A rationale for attached gingiva at the soft tissue/implant interface: Esthetic and functional dictates. Compendium 1994;15(3):356,358,360–362 passim; quiz 368.
- 42. Han TJ, Klokkevold PR, Takei HH. Strip gingival autograft used to correct mucogingival problems around implants. Int J Periodontics Restorative Dent 1995;15(4):404–411.
- Lang NP, Brägger U, Walther D, Beamer B, Komman KS. Ligature-induced peri-implant infection in cynomolgus monkeys. I. Clinical and radiographic findings [published erratum appears in Clin Oral Implants Res 1993 Jun;4(2): 111]. Clin Oral Implants Res 1993;4(1):2–11.
- Klinge B. Implants in relation to natural teeth. J Clin Periodontol 1991;18(6):482–487.
- 45. Jovanovic SA, Kenney EB, Carranza FA Jr, Donath K. The regenerative potential of plaque-induced peri-implant bone defects treated by a submerged membrane technique: An experimental study. Int J Oral Maxillofac Implants 1993;8(1): 13–18.
- 46. Persson LG, Ericsson I, Berglundh T, Lindhe J. Guided bone regeneration in the treatment of peri-implantitis. Clin Oral Implants Res 1996;7(4):366–372.

COPYRIGHT © 2000 BY QUINTESSENCE PUBLISHING CO, INC. PRINTING OF THIS DOCUMENT IS RESTRICTED TO PERSONAL USE ONLY. NO PART OF THIS ARTICLE MAY BE REPRODUCED OR TRANSMITTED IN ANY FORM WITH-OUT WRITTEN PERMISSION FROM THE PUBLISHER.

- Rangert B, Thorsten J, Jörneus L. Forces and moments on the Brånemark implant connected to a natural tooth: An in vitro study. Int J Oral Maxillofac Implants 1989;4:241–247.
- Albrektsson T, Lekholm U. Osseointegration: Current state of the art. Dent Clin North Am 1989;33(4):537–554.
- Fox SC, Moriarty JD, Kusy RP. The effects of scaling a titanium implant surface with metal and plastic instruments: An in vitro study. J Periodontol 1990;61(8):485–490.
- Dmytryk JJ, Fox SC, Moriarty JD. The effects of scaling titanium implant surfaces with metal and plastic instruments on cell attachment. J Periodontol 1990;61(8):491–496.
- Rapley JW, Swan RH, Hallmon WW, Mills MP. The surface characteristics produced by various oral hygiene instruments and materials on titanium abutment surfaces. Int J Oral Maxillofac Implants 1990;5:47–52.
- Nevins M, Langer B. The successful use of osseointegrated implants for the treatment of the recalcitrant periodontal patient. J Periodontol 1995;66(2):150–157.
- Jemt T, Lekholm U, Adell R. Osseointegrated implants in the treatment of partially edentulous patients: A preliminary study of 876 consecutively placed fixtures. Int J Oral Maxillofac Implants 1990;4:211–217.
- Jaffin RA, Berman CL. The excessive loss of Brånemark fixtures in type IV bone: A 5-year analysis. J Periodontol 1991; 62(1):2–4.
- Bain CA, Moy PK. The association between the failure of dental implants and cigarette smoking. Int J Oral Maxillofac Implants 1993;8(6):609–615.
- 56. Bain C. Influences of smoking on the periodontium and dental implants. Dent Update 1997;24(8):328–330.
- 57. Klokkevold PR, Nishimura RD, Adachi M, Caputo A. Osseointegration enhanced by chemical etching of the titanium surface. A torque removal study in the rabbit. Clin Oral Implants Res 1997;8(6):442–447.
- Kieswetter K, Schwartz Z, Hummert TW, Cochran DL, Simpson J, Dean DD, Boyan BD. Surface roughness modulates the local production of growth factors and cytokines by osteoblast-like MG-63 cells. J Biomed Mater Res 1996; 32(1):55–63.
- 59. Buser D, Nydegger T, Oxiand T, Cochran DL, Schenk RK, Hirt HP, et al. Interface shear strength of titanium implants with a sandblasted and acid-etched surface: A biomechanical study in the maxilla of miniature pigs. J Biomed Mater Res 1999;45(2):75–83.
- Lohmann CH, Sagun R Jr, Sylvia VL, Cochran DL, Dean DD, Boyan BD, Schwartz Z. Surface roughness modulates the response of MG63 osteoblast-like cells to 1,25(OH) (2)D(3) through regulation of phospholipase A(2) activity and activation of protein kinase A. J Biomed Mater Res 1999;47(2):139–151.
- 61. Boyne PJ. Animal studies of application of rhBMP-2 in maxillofacial reconstruction. Bone 1996;19(suppl 1):83–92.
- Cochran DL, Schenk R, Buser D, Wozney JM, Jones AA. Recombinant human bone morphogenetic protein-2 stimulation of bone formation around endosseous dental implants. J Periodontol 1999;70(2):139–150.
- Lynch SE, Buser D, Hernandez RA, Weber HP, Stich H, Fox CH, Williams RC. Effects of the platelet-derived growth factor/insulin-like growth factor-I combination on bone regeneration around titanium dental implants. Results of a pilot study in beagle dogs. J Periodontol 1991;62(11): 710–716.