The long-term success and predictability of dental implants has been well documented in the dental literature for more than 40 years. As their use continues to become more common in contemporary restorative and surgical dental practices, the choice of dental implants in treatment plans now ranges from single tooth replacement to full arch/full mouth reconstruction.

Considerations for Implant Success
Ancillary procedures such as bone grafting, soft tissue replacement, and more advanced surgical techniques have become necessary to restore the proper peri-implant environment, which must consist of an acceptable amount of attached keratinized gingival tissue to protect the alveolar structures and the implant/abutment connection.

Many factors can affect the short- and long-term success of dental implants. A patient’s health status, pre-existing dental infections, and social habits can all play a significant role in the success of dental implants for natural tooth replacement. Patient selection and evaluation, proper pretreatment planning, and effective communication among all members of the implant team (surgeon, restorative dentist, and laboratory technician) also are positive contributors to success. Additionally, success is also dependent to some extent on the proficiency of the surgeon (whether dental specialist or restorative clinician) and the restorative practitioner.

Implant design is also an important factor in success. Surface alterations can enhance the integration process and the soft tissue seal at the coronal aspect of the implant and reduce bacterial adherence, whereas abutment continuity is essential for the success of the implant.

A Protocol for Treatment of Peri-Implantitis

<table>
<thead>
<tr>
<th>Step</th>
<th>Procedure</th>
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| 1.   | Administration of antibiotic
|      | • Augmentin 875 mg; 20 tablets; 1 tab every 12 hr, start the day prior to procedure |
|      | • If allergic to penicillin derivatives, Levaquin 500 mg; 10 tablets; 1 each day until gone |
| 2.   | Preparation of platelet-rich plasma (PRP) |
|      | • 20 cc whole blood obtained prior to procedure is centrifuged to provide a platelet concentrated gel (minimum 1 million platelets/µl) |
| 3.   | Degranulation of peri-implant infected tissue |
| 4.   | Removal of contaminated implant surface with piezoelectric scaler (mechanical cleaning) |
|      | • Brush exposed surface of implant threads 3 minutes, rinse |
| 5.   | Decontamination with citric acid (pH 1) (antiseptic chemical treatment) |
|      | • 5-watt power settings over exposed implant thread surface |
| 6.   | Decontamination of bacteria and inflammatory debris with Nd:Yag laser |
| 7.   | Application of PRP to defect |
|      | • Prepared platelet concentrated gel is used over entire exposed surface and exposed threads |
| 8.   | Placement of PRP/bone graft complex |
|      | • Mineralized cancellous 1-mm to 2-mm partial size bone graft |
| 9.   | Application of PRP membrane over graft complex |
| 10.  | Closure |
designs can minimize their removal, preventing repeated trauma to the hemi-desmosomal seal formed between the implant/abutment complex and surrounding soft tissues. Other implant designs are best for incorporation into immediate restoration and/or loading protocols. Making the right choice for an individual clinical scenario can contribute to more rapid case completion and continued success with dental implants for tooth replacement.

Confronting Peri-Implantitis

Despite the high success rates observed with dental implants in the contemporary dental practice, complications and implant loss can occur. An important complication to dental implant treatment is a pathologic process called peri-implantitis.

Peri-implantitis is a condition caused by peri-implant tissue changes that can have flora present similar to that of periodontitis, causing a mild to aggressive inflammation mimicking that found in cases of mild-moderate to advanced periodontitis. The disease state is generally thought to be caused primarily by plaque and biofilm, which has been shown to accumulate on implant surfaces as well as teeth. Tissue inflammation, bleeding upon probing, purulence, and advanced bone loss is often observed around a dental implant affected with peri-implantitis.

Peri-implantitis has been linked to cases with implant loss. Looking at survival rates of implant-supported single crowns, Jung and colleagues observed that peri-implantitis and soft tissue complications were seen in approximately 10% of cases reviewed.

“As implant procedures become more prominent, dental practitioners will be confronted with the challenge of treating peri-implantitis.”

Additional factors that can contribute to peri-implantitis include a lack of hygiene, which can increase inflammation and bone loss levels, excessive excessive occlusal load, and, as previously mentioned, inadequate zones of attached keratinized tissue. The effects of tobacco use on implant surfaces have also been shown to negatively impact implant success rates. Baig and Rajan found that in implant patients who smoke, significantly greater marginal bone loss occurs after implant placement and the occurrence of peri-implantitis increases.

Genetic factors have been correlated with occurrence of peri-implantitis. Studies have shown a direct linkage between the IL-1 gene polymorphism and peri-implantitis. Plagnat and colleagues demonstrated that a similarity existed in the inflammatory processes of tissue surrounding implants and natural teeth, and that elastase and alkaline phosphatase activity could be important markers for bone loss around dental implants.

Corrosion can also play a role in peri-implantitis, particularly if a base metal allergy is observed in direct connection to the titanium implant. This could directly contribute to the deconstruction of osseous tissue, resulting in peri-implantitis.

Poor surgical technique can also be a cause of peri-implantitis. Bone overheating during the implant site preparation process can lead to bone necrosis and implant surface contamination during implant placement can lead to the development of an apical lesion; these can contribute to the loss of bone as well as inflammation of the peri-implant tissues.

As implant procedures become more prominent in treatment plans and implant patients present for routine maintenance visits, dental practitioners will most likely be confronted with the challenge of treating peri-implantitis cases. The following case reports outline a protocol to treat peri-implantitis (Table 1) and reverse advanced bone loss around dental implants in occlusal function for greater than 5 years.

Case 1

A 64-year-old nonsmoking man presented for treatment of a failing implant at the mandibular right first molar (Figure 1 and Figure 2). The patient had a 4.7-mm x 13-mm Tapered Screw-Vent titanium plasma-sprayed surface implant (Zimmer Dental, www.zimmer-dental.com) placed 8 years prior. Probing
depths in excess of 10 mm circumferentially were noted, with bleeding on probing and purulence present. No mobility to the implant structure was present.

The mandibular right second premolar was diagnosed with a guarded-hopeless prognosis, and was slated for removal and replacement with a dental implant by the immediate extraction, placement, and restoration protocol. The patient had wished to keep the implant at the No. 30 position; therefore, the decision was made to attempt repair at the No. 30 site simultaneously with the immediate restoration procedure at tooth No. 29. The patient was given a prescription for 20 tablets of Augmentin (amoxicillin clavulanate) 875 mg to be taken every 12 hours to start the day prior to the surgical therapy.

After administration of an appropriate local anesthetic, an intrasulcular incision was made from the distal of the first molar area to the mesial line angle of the mandibular right canine. A full thickness muco-periosteal flap was elevated, and the granulation tissue surrounding the affected implant was removed (Figure 3). The exposed contaminated implant surface, including each exposed thread, was then debrided with a piezoelectric scaler with water irrigation. Mechanical debridement and cleaning of the contaminated surface was achieved. Tooth removal and debridement of the second molar site was then accomplished (Figure 4).

Following tooth removal, citric acid (pH 1) was applied to the exposed implant surface. Initially, the surface was burnished and rinsed with sterile water and then citric acid–saturated cotton pellets were placed around the exposed implant surface for a total of 3 minutes (Figure 5), followed again by rinsing with sterile water. Figure 6 shows the appearance of the exposed implant surface after antiseptic chemical treatment using citric acid.

A 4.0-mm x 11.5-mm ETTIII implant (Hiossen, www.hiossen.com) was then placed in the No. 29 position to appropriate depth, followed by the placement of a stock titanium abutment (Figure 7) and completion of the depths in excess of 10 mm circumferentially were noted, with bleeding on probing and purulence present. No mobility to the implant structure was present.

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immediate provisional restoration (Figure 8). Decontamination of bacteria and inflammatory debris on the implant surface was further treated by surface contact with a neodymium-yttrium-aluminum garnet (Nd:Yag) laser at the 5-watt power setting. The laser beam was in contact with the exposed implant surface, including all exposed threads (Figure 9). Figure 10 shows the appearance of the implant surface post-laser treatment.

A platelet-rich plasma (PRP) gel was then applied to the implant surface (Figure 11) prior to placement of a PRP/grafting complex into the peri-implant defect (Figure 12), followed by application of a PRP membrane over the grafted site (Figure 13). A
continuous sling suturing technique with 5-0 MONOCRYL™ Sutures (Ethicon Inc., www.ethicon360.com) was employed to approximate and close the surgical site (Figure 14). The patient was instructed to finish the antibiotic administered presurgically, and prescribed chlorhexidine rinse 2 times per day for 10 days postsurgically.

Figure 15 shows the immediate post-operative digital periapical radiograph of the No. 29 and No. 30 sites. A 1-month postoperative digital periapical radiograph can be seen in Figure 16. Figure 17 and Figure 18 show the 2-month post-repair clinical view and periapical radiograph, respectively. Note the post-repair maintenance and band of keratinized attached tissue. A 1-year post-repair periapical radiograph can be seen in Figure 19; bone fill has occurred in the peri-implant structures. Probing depths were reduced to 3 mm or less circumferentially, and the absence of purulence and bleeding upon probing was observed at the 1-year post-repair followup.

Case 2
A 66-year-old nonsmoking man presented for treatment of a large peri-implant defect at the mandibular left second molar site (Figure 20). A 4.7-mm x 15-mm coated Tapered Screw-Vent HA implant was had been placed 7 years prior to the surgical technique outlined in Table 1, the repair process was completed. Figure 21 shows the 1-year post-repair periapical radiograph of the implant in the mandibular left second molar site. Comparing Figure 20 and Figure 21, it is observed that bone fill has occurred following the repair process. At the 1-year post repair evaluation, no bleeding upon probing or purulence was noted. Additionally, probing depths were reduced to 3 mm circumferentially, with the exception of the reading at the distal-buccal of the molar, which registered 5 mm.

Discussion
Bone and tissue loss around dental implants over their life span, placed into occlusal function, will be a problem with which restorative and surgical clinicians will be faced in recall and maintenance programs. As previously outlined in the text of this article, the causes of peri-implantitis can vary from poor surgical technique to genetic predisposition of the host response to periodontal pathogens similar to periodontitis. To achieve resolution of any type of dental infection, the etiologic cause of the problem must first be identified and corrected before any regenerative/reconstructive procedures are attempted. Otherwise, the breakdown of the peri-implant structures would likely recur.

A technique for the treatment of peri-implantitis and subsequent loss of the peri-implant structures has been presented above. The technique, when strictly adhered to, has been shown to replace deep circumferential-type peri-implant defects in more than 20 treated sites, with observation of the successful replacement of bone and soft tissues for more than 1.5 years post-treatment. The author recommends additional clinical trials to substantiate the surgical technique outlined and the clinical findings presented in the case reports.

References
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