Clinical Management Of Peri-Implantitis And Periodontitis: Are There Differences In Treatment Protocols?

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Introduction

Dental implants have evolved significantly over time. Four thousand years ago root form bamboo pegs were tapped into the jawbone to replace lost teeth, which changed to similarly shaped pegs made of precious metals or ivory in Egypt 2000 years ago. Ancient Mayans used pieces of shells shaped to resemble teeth in a similar fashion. In the 20th century, metals such as iridioplatinum and vitallium (cobalt-chromium-molybdenum alloy) were used.

In 1952, Dr. Per-Ingver Branemark stumbled upon titanium as a biocompatible material that "fuses" to bone - titanium currently represents the gold standard material for fabrication of implants worldwide.[1]

Other permutations including the blade implants **(Figs. 1A & B)** and other innovative tooth replacement techniques **(Fig. 2)** had poor long-term results. The Harvard Consensus Development Conference on Dental Implants in 1978 declared that no implant available at the time had a survival rate of 75 percent after five years.[2]

A seminal Toronto conference on osseointegration in clinical dentistry changed the field in 1982. Presently, osseointegrated dental implants are a popular and predictable treatment option used around the world.[3] During the 1980s, about 300,000 implants were installed worldwide annually[4] while just before the start of the millennium, the implant insertion rate had risen to more than one million per year.[5] It is important to reflect on these statistics to understand the magnitude of potential complications and the diversity of treatment scenarios associated with dental implants. The development and acceptance of successful treatment protocols require an understanding of the underlying biology of the inflammatory pathologies associated with natural teeth and implants. This paper proposes to address the similarities and differences between periodontal and peri-implant pathology.

Table 1 - Tooth and Implant Histological Comparison

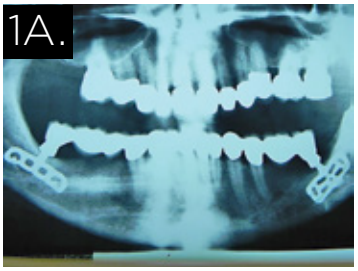
	Tooth	Implant
Connection	Cementum, bone, periodontal ligament (flexible)	Osseointegration, functional ankylosis (direct contact with bone/rigid)
Junctional Epithelium	Hemidesmosomes and basal lamina (lamina lucida, lamina densa zones)	Hemidesmosomes and basal lamina (lamina lucida, lamina densa, and sublamina lucida zones
Connective Tissue	Horizontal, oblique, vertical and perpendicular fibers More collagen with better adhesion and stronger seal	Parallel fibers (seal around implant is weak)
Probing Depth	≤ 3 mm in health	2.5-4.0 mm (dependent upon soft tissue depth)
Bleeding on Probing	More reliable	Less reliable

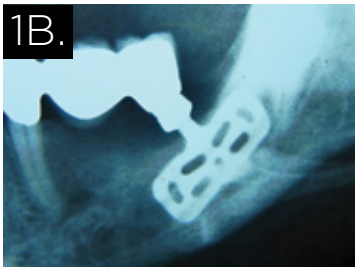
Adapted from Ikeda et. al

Peri-Implant and Periodontal Disease Associations

Peri-implant disease presents in two forms; peri-implant mucositis and peri-implantitis. Both are characterized by an inflammatory reaction in the tissues surrounding an implant.[6]

Peri-implant mucositis is described as a disease in which the presence of inflammation is confined to the soft tissues surrounding a dental implant with-





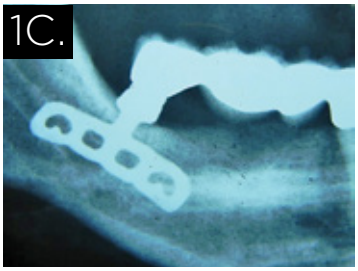
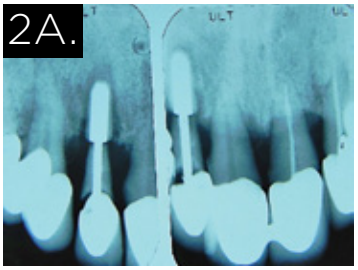


Figure 1A. Blade implants made of titanium were used in the 1970s and 1980s and had a very poor 10-year prognosis. The technique also required extensive surgical manipulation. This patient implants placed (not by author) in 1991 at aged 34, and required removal of them five years later.
Figures Band C. Close-up radiographs of bilateral blade implants demonstrating proximity to the mandibular nerve and radiolucencies suggesting rarefying osteitis (greater surgical risks were involved with this technique).



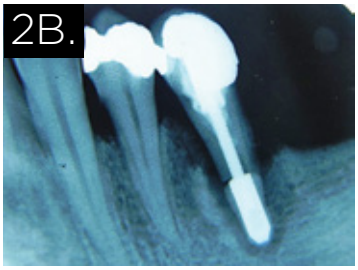


Figure 2. Innovative tooth re-implantation techniques were sometimes used to replace teeth. In these examples, "bullet post restorations" were retrofitted through the apex of the extracted roots with hopes of success. Both were lost in the same patient after severe tooth decay and periodontal bone loss difficulties (lasted less than eight years).

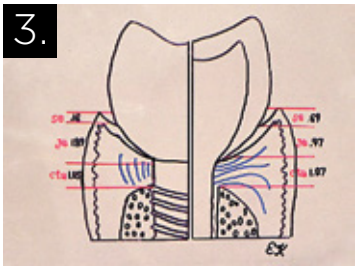


Figure 3. Implant versus tooth, cross-sectional comparative anatomy. SE – sulcular epithelium; JE – junctional epithelium; CTA – connective tissue attachment. Please refer to the text and Table 1 for classification. Peri-implant anatomy is more vulnerable when compared to the natural tooth system.

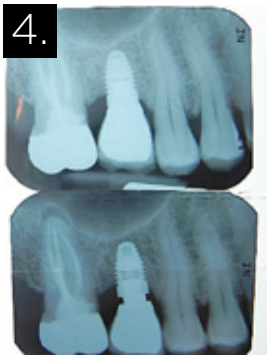


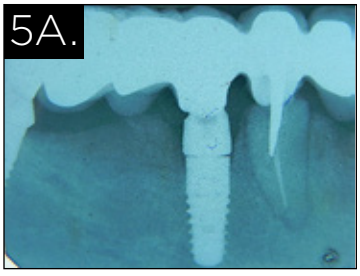
Figure 4. Post-treatment radiographs are beneficial to ensure proper seating of crowns on implant abutments. In this case, interproximal contacts required adjustment to allow for perfect seating of the crown (Nobel Biocare implant placed by author). Tooth 1.7 periapical pathology presently under re-treatment.

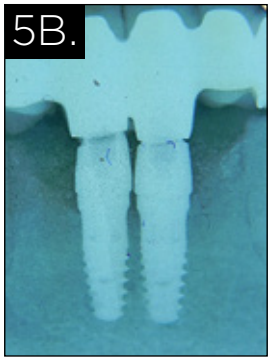
out loss of supporting bone beyond the initial bone remodelling during heeling. Peri-implant mucositis is reversible.

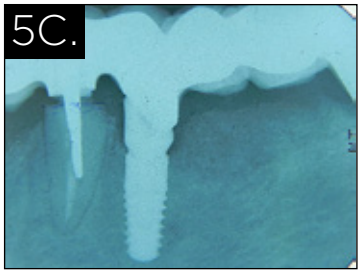
Peri-implantitis is characterized by an inflammatory process around an implant which includes both soft tissue and bone leading to a progressive loss of supporting bone beyond the initial biological remodelling.[7] This definition is similar to that given to established periodontitis characterized by deep pockets, inflammation and clinical attachment loss.[8]

Studies confirm an overall similarity in the composition of the microbiota associated with periodontitis and peri-implantitis.[9,10] Samples were selected from cases diagnosed as adult periodontitis (AP) and either refractory or recurrent periodontitis and implant derived samples.[9] The periodontitis and peri-implantitis bacterial species included mainly gram-negative aerobes.[10] These included *Porphyromonas gingivalis* (Pg), *Prevotella intermedia* (Pi) and *Actinobacillus actinomycetemcomitans* (Aa). Microbiota from failing implants consisted of a large proportion of gram-negative anaerobic rods, with black-pigmented bacteroides as well as *fusobacterium spp* as well as spirochetes.[11]

Anatomically, lesions of peri-implantitis and periodontitis from human

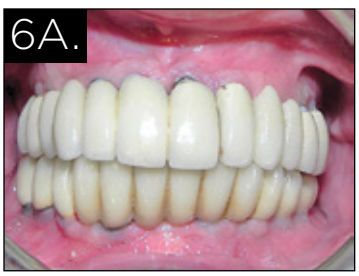






Figures 5A B and C. To save costs, flew abroad to get discounted implant dentistry performed to restore mandibular dentition. Peri-implant disease risk increased because the implants placed too close to each other. The suprastructure imperfectly attached to the natural dentition and adjacent implants leaving micrograps in various areas. Patient currently undergoing corrective restorative treatment in another clinic.

Figures 6 A-to-H. Ten years after Nobel Biocare implants were placed by author localized 8 mm pocketing and moderate peri-implant bone loss was identified in the mesial aspect of implant 21. Other implants in the maxillary arch had 4-5 mm pocketing. Conservative management resolved the condition (CIST A+B+C). Patient, presently 70 and a non-smoker, with improved oral hygiene practices.



Figures 6A. Labial view of moderate peri-implantitis affecting implant 21.

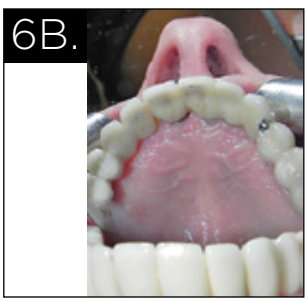


Figure 6B. Palatal view.

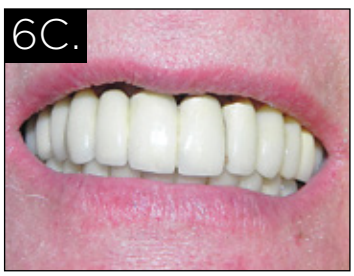


Figure 6C. Smile line is favorable for pocket reduction therapy.

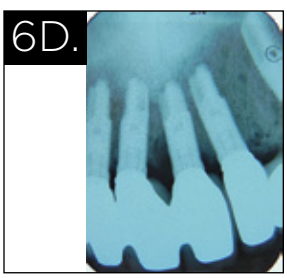


Figure 6D. Periapical demonstrating localized moderate bony defect.

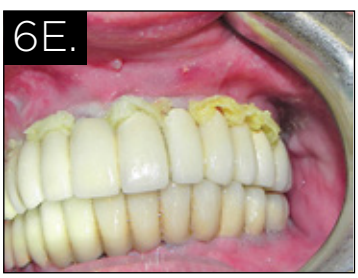


Figure 6E. Implant surface decontamination and detoxification.

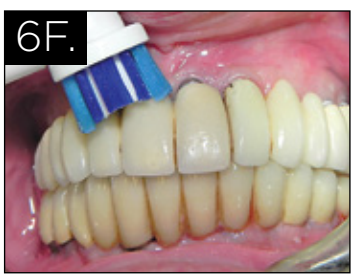


Figure 6F. Oral B-3D oral hygiene emphasizing "gum-line" technique.

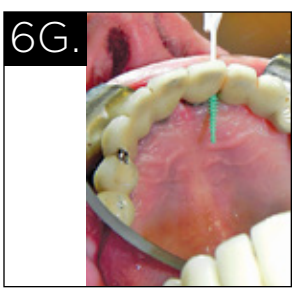


Figure 6G. Softpic™ interdental oral hygiene tool also demonstrating palatal healing.

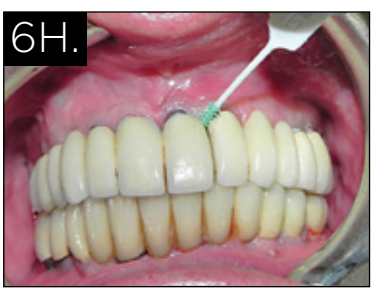
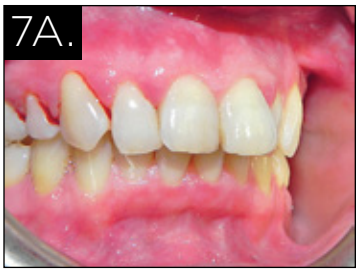
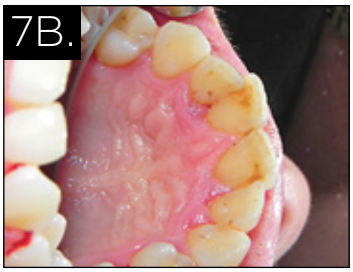
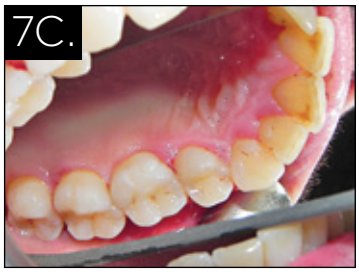


Figure 6H. Labial appearance of healed peri-implant mucosa and Softpic™ interdental placement.

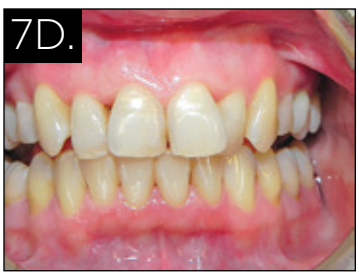
biopsies have many features in common.[12,13,14] The connective tissue (CT) adjacent to the pocket epithelium is infiltrated by inflammatory cells, with B-lymphocytes and plasma cells being the most dominating cell types. Also, basically similar markers are up-regulated between both peri-implantitis and periodontitis including pro-inflammatory cytokines such as interleukin

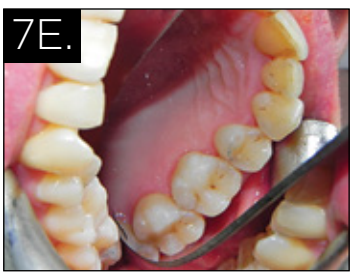






Figures 7A, Band C: Clinical presentation prior to conservative deep cleaning therapy.





Figures 7D and E. Excellent headlining following non-surgical periodontal therapy utilizing intense scaling and root planing, subgingival irrigation with hydrogen peroxide, oral antimicrobial modifications and oral hygiene measures. Similar success as with Figure 6.

Figures 7 A-E. 46 year old female non-smoker presented with generalized moderate and localized advanced adult periodontitis. Pockets ranged from 4-9 mm associated with bleeding on probing, suppuration and bone loss. Conservative deep cleaning similar to CIST A+B+C can help peri-implantitis as well as periodontitis situations.

(IL-1), IL-6, IL-8, IL-12 and tumour necrosis factor alpha (TNF-α).

Prevalence and Incidence

The frequency of peri-implant mucositis was estimated in 63.4 percent of patients and 30.7 percent of implants; peri-implantitis affected 18.8 percent of patients and 9.6 percent of implants as published in a systematic review paper encompassing 1,497 participants and 6,283 implants.[15] In another cross-sectional study involving 96 patients with 225 implants, the incidence of peri-implant mucositis was 48 percent of patients and 33 percent of implants; peri-implantitis occurred in 26 percent of patients and 16 percent of implants.[16] Basically, one in four-five patients and one in six-10 implants have peri-implantitis and one in two patients and up to one in three implants have peri-implant mucositis. Important statistics given that over one million implants are placed annually worldwide.[5]

The incidence of periodontal disease (PDz) at first glance appears to be much higher. A recent study spanning years 2009-2012 revealed that 46 percent of US adults representing 64.7 million people had PDz with 8.9 percent having severe periodontitis by or after 30 years of age. Prevalence varied 2 fold between lowest and highest levels of socioeconomic status whether defined by poverty or education.[17]

Interestingly, the incidence of peri-implantitis is actually comparable to that of periodontitis when parameters are changed. Eleven percent of individuals received the diagnosis of peri-implantitis when radiographic bone loss was ≥ 3 mm was set as a cut-off point versus 47 percent when peri-implantitis was defined as ≥ 2 mm bone loss.[18] That would make one of every two people suffering peri-implantitis, which is similar to periodontitis.[17] Many similarities appear to exist between these two disease entities.

Clinical Features

Clinical features of peri-implantitis are also similar to those of periodontitis as described by Mombelli et. al19 and include:

1. Radiographic evidence of vertical destruction of the crestal bone
2. Formation of a peri-implant pocket in association with radiographic bone loss
3. Bleeding after gentle probing, with or without suppuration
4. Mucosal swelling and redness
5. No pain, in most cases and in particular in less severe once

It becomes obvious that many similarities exist between peri-implantitis and periodontitis but one major difference is presented and described in the next section.

With respect to implants, an acceptable amount of bone loss in the past was defined as vertical bone loss of 1 mm during the first year of function followed by an annual loss of < 0.2 mm after the first year of service.[20] This was altered to 1.5 mm acceptable vertical bone loss after one year of service followed by an annual loss rate of 0.2 mm thereafter in 1993.[21] In

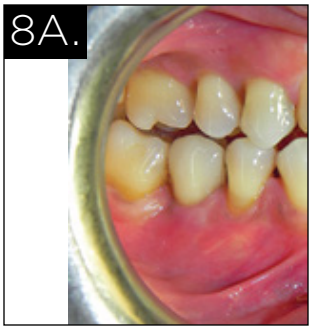


Figure 8A. 7 years after implant 46 was placed (not by the author), this non-smoking gentleman aged 54 developed mild peri-implantitis and 7 mm pocketing. 3 mm bone loss had occurred compared with earlier visit to the office five years earlier. CIST protocol A+B+C was instituted. Inflamed peri-implant tissues around implant 46.

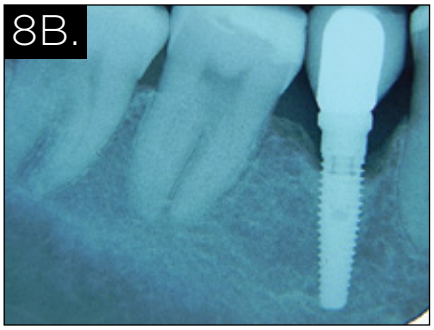
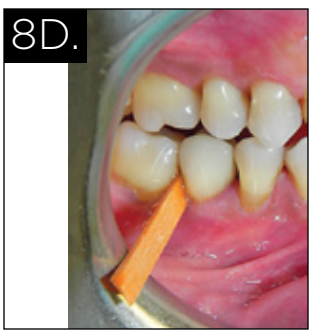


Figure 8B. Periapical showing localized "saucer-shaped" bone loss implant 46.

Figure 8C. Implant detoxification at time of CIST A+B+C protocol.



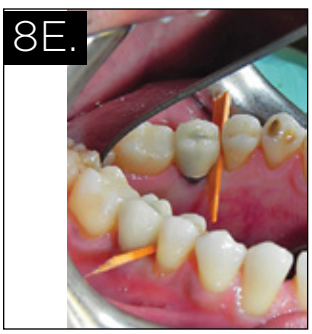


Figure 8D and E. Intense oral hygiene using interdental Stimudents™ angled towards the implant.

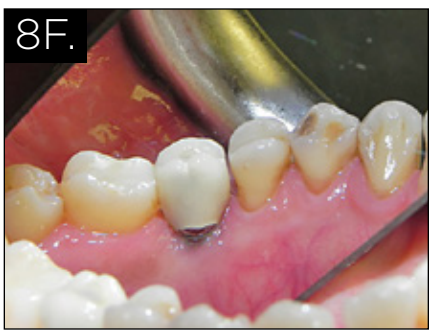


Figure 8F. Healed peri-implant tissues (less than 4 mm).

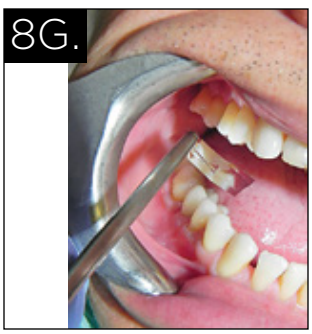


Figure 8G. Occlusion assessed with Shimstock to address undesirable occlusal interferences.

2013, normal bone loss is redefined as peri-implantitis if ≥ 2 mm bone loss occurs in addition to the presence of bleeding on probing or suppuration from at least one surface area of the implant and the presence of probing pocket depth of more than 4 mm.[22]

Implant-Tooth Interface (Anatomy And Histology)

The peri-implant mucosa provides a protective seal around teeth and implants from the contaminated environment of the oral cavity. The junctional epithelium (JE) forms an epithelial seal around teeth and implants maintaining continuity with the epithelial lining of the oral cavity. It contains adhesive structures such as hemidesmosomes but the internal basal lamina exists only in the lower region of the peri-implant interface[23] and is more vulnerable than in the tooth counterpart (Fig. 3 and Table 1).

The CT is very strong in the tooth system where collagenous fibres are directed perpendicularly to the tooth surface with direct anchorage to the tooth via cemental attachment. The connection also decreases further down-growth of the JE to ensure protection of the area. In sharp contrast, functionally oriented fibres are absent around implants. Only circular fibres are arranged in parallel orientation to the implant in an "O-ring" configuration.[24] Then it is highly suspected that the peri-implant mucosa is more vulnerable to bacterial infection. Once the infection takes hold it can lead to greater bone loss leading to circumferential bony defects around implants in variance with more defined bony lesions directed by "compartmentalization" of he functionally oriented fibres around natural teeth.

The biologic width (BW) has been established by Gargiulo et al[25] and Vacek et al[26], and although the gingival interface with implants is more vulnerable; Cochrane et al[27] found that the BW around implants was comparable to the dento-gingival tissues described by Gargiulo et al.[25] After 12 months of loading, the values were 0.16 mm for sulcus depth (versus 0.69 mm), 1.88 mm for JE (versus 0.97 mm) and

Figure 9A. Pre-treatment photo teeth 13-15 demonstrating periodontal abscess.

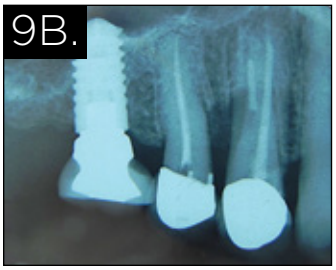
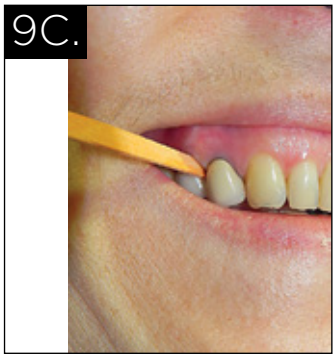
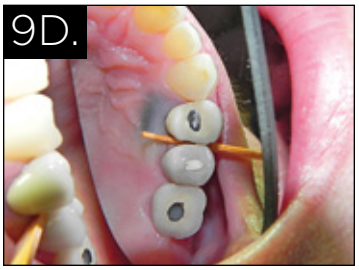


Figure 9B. Periapical of advanced osseous defect.





Figures 9C and D. Stimudent oral hygiene encouraged.

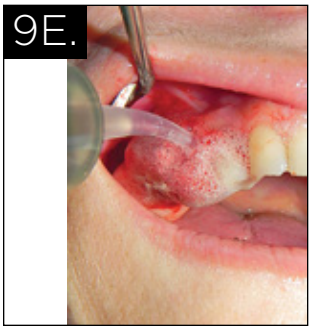


Figure 9E. Root detoxification in combination with scaling and root –planing.

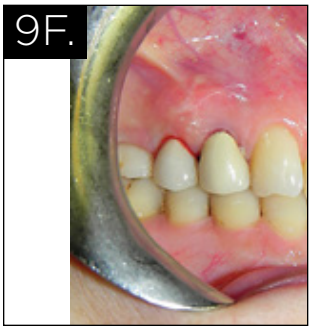


Figure 9F. Appearance after deep cleaning completed.

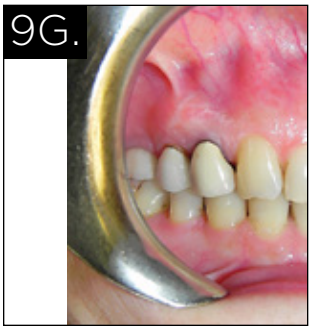


Figure 9G. Periodontal pocketing stabilized (no more than 4 mm) and periodontal health restored.

Figures 9 A-to-G. Localized moderate advanced adult periodontitis in a non-smoking 53-year-old patient, with 10 mm pocketing, questionable endodontic prognosis and purulence. Treatment sequence similar to Figure 8 with similar results.

1.05 mm CT attachment (versus 1.07 mm) when implants are compared to teeth (Fig. 3).

It seems the BW needs to form a seal both for teeth and implants. Wallace emphasized this significance and stated that "the fact that the ultimate location of the epithelial attachment following phase two surgery, will be on the implant body is of clinical significance to the implant surgeon since it will in part determine the amount of post-surgical bone loss".[28] Early implant bone loss is from the process of establishing the BW. The amount of bone loss and location of the BW may be associated with the thickness of soft tissue around implants, location of the junction between rough and polished implant surfaces in non-submerged implants and the location of the microgap in submerged implants. The implant interface is also influenced by gingival and osseous phenotype, type and width of implants and implant loading conditions.[23,24,29]

It has also been suggested that "punching-out" peri-implant soft tissue in the presence of a thin gingival biotype can completely remove the CT "O-ring" effect leading to insufficient stability of the peri-implant mucosa.[30]

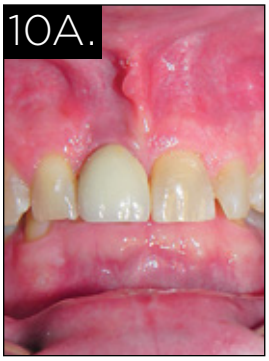
As described above, the bone can resorb 1.5-2 mm apically when implants are placed at or near the crest of bone, after the implant interface (BW) is established.[31,32]

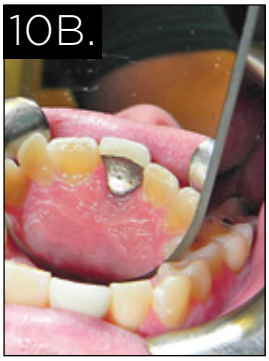
Risk Factors For Disease Development

Shared risk factors for periodontitis and peri-implantitis include smoking,[33] systemic diseases (e.g. diabetes and cardiovascular disease),[34,36] soft tissue defects (e.g. lack of attached gingiva),[35] genetic influences,[36] alcohol consumption[37] and, most importantly, poor oral hygiene practices.[38]

Additionally, risk factors towards the development of peri-implantitis include:

1. Bacterial leakage due to configuration and position of the implant-abutment microgap[39]
2. Localized inflammation at the implant-abutment interface[39] **(Fig. 3)**
3. Micro-movement of prosthetic components





Figures 10A and B. At age 31 (presently 51), a non-smoking male received a "new" downtown Toronto generated implant in 1995, at reduced cost. Presently asymptomatic, but 9 mm peri-implant pocketing and moderate bone loss facilitated referral for treatment of peri-implant disease. Pre-treatment presentation.

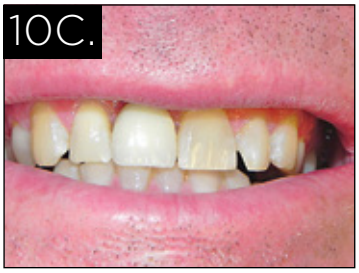


Figure 10C. Smile line encouraged conservative approach. Implantoplasty contraindicated due to design of implant-crown connection and "sharp-tooth" design of the implant.

Figure 10D. Aggressive oral hygiene encouraged with soft tooth brush (Butler 471). CIST A+B+C successful and peri-implant pocketing reduced to 4 mm.

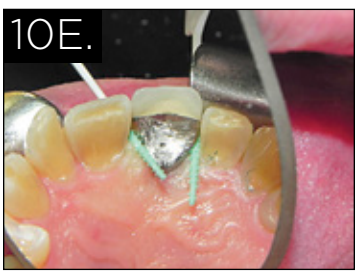


Figure 10E. Softpic™ oral hygiene encouraged interdentally.

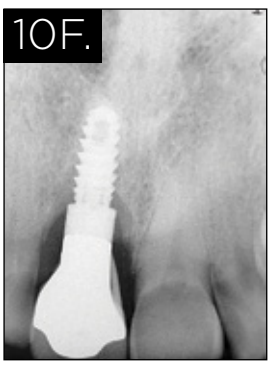


Figure 10E. Periapical demonstrating abrupt implant-abutment connection and sharp teeth of implant thread design.

4. Overloading of the implant[33,40]
5. Poor bone quality at the implant area[41]
6. Implant configuration and surfaces[42]
7. Residual cement[42]
8. Implants shorter than 10 mm have higher odds for early implant loss[44,45]
9. Miscellaneous considerations: implant 3-D position, extraction technique, location in aesthetic zone, implant design, surface properties, platform shift, emergence profile, restoration anatomy, occlusion (excessive loading), etc. **(Figs. 4 & 5)**

It becomes obvious that many other factors can contribute to peri-implantitis risk due to the fragility of the peri-implant interface. In a review, poor oral hygiene and compliance, history of periodontitis and cigarette smoking were found to be pivotal risk indicators for development of peri-implantitis with one study reporting that 78% of implants in smokers received a diagnosis of peri-implantitis.[46,34] A cross-sectional study demonstrated that smokers had an odds ratio of 3.8 for developing peri-implant mucositis and an odds ratio of 31.6 for developing peri-implantitis.[33] The presence of aggressive periodontitis itself increases susceptibility of peri-implantitis and late bone loss around implants.[47]

Since the peri-implant environment has a greater vulnerability towards development of peri-implantitis, greater vigilance is required for prevention and early detection of peri-implant disease. Preventive maintenance is associated with less occurrence of peri-implantitis.[50]

As an aside, recent research is suggesting the possibility of a foreign body reaction to titanium implants which might be related to marginal bone loss and potentially implant failure.[48]

Treatment Considerations And Maintenance

In general, the approach to treating peri-implantitis and periodontitis is similar and is detailed in an American Association of Periodontology (AAP) position paper.[49]

Once a proper diagnosis is made, all of the risk factors identified (medical and dental), prognosis and treatment plan established and occlusal (and implant) materials accounted for, periodontal treatment can commence, including attempts at habit modification (including smoking, compliance and oral hygiene influences).

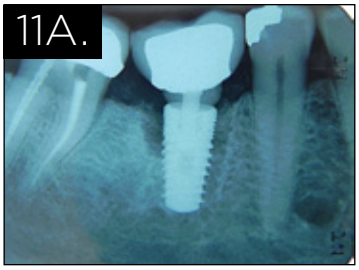


Figure 11A. Implant 46 placed five years earlier (by another practitioner) for a non-smoking gentleman aged 64. Crown 46 loosened (abutment screw) and 10 mm circumferential pocketing occurred with purulent exudate and obvious mobility of the crown. Patient seen for regenerative management of advanced peri-implantitis. Periapical film shows the extent of bone defect implant 46.

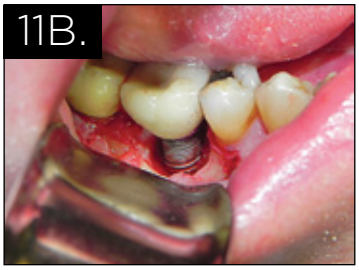


Figure 11B. Peri-implant surgery revealing extent of implant thread exposure and saucer shaped osseous defect.

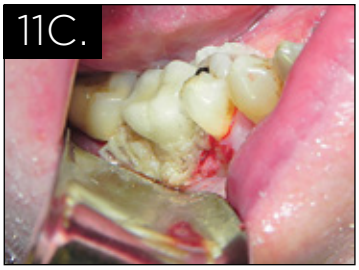


Figure 11C. Implant surface detoxification and decontamination (CIST A+B+C+D)

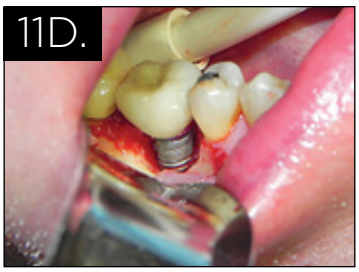


Figure 11D. Surface is clean following tetracycline implant surface cleansing.

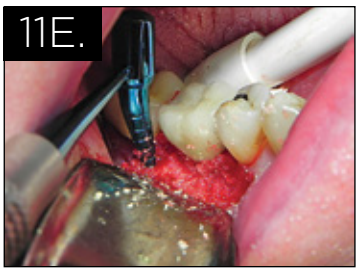


Figure 11E. Bioactive glass bone grafting material placed.

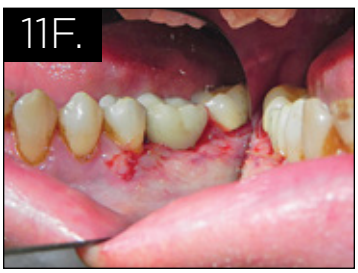


Figure 11F. Surgical site sutured (mirror-image).

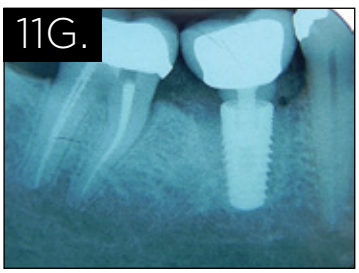


Figure 11G. Periapical radiograph demonstrating attempt at "overfill" of defect.

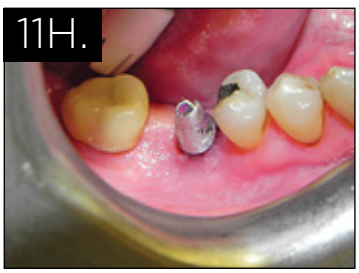


Figure 11H. Crown removed and implant abutment stabilized.

Figure 11I. Peri-implant tissues healed.

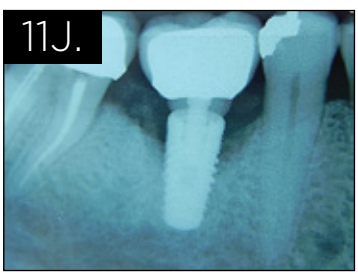


Figure 11J. Periapical radiograph demonstrating 70 percent bone fill after four months of healing.

The protocol for managing peri-implantitis is more complex because of the vulnerable peri-implant interface and the multitude of potential implant related risk factors.

The hallmark of successful surgical treatment of peri-implantitis depends on successful and meticulous surface decontamination of the contaminated implant surface. Mechanical choices include air abrasives (high pressure air powered abrasive containing mixture of sodium bicarbonate and water), curettes made of plastics, carbon graphite, titanium, and occasionally implantoplasty.[54]

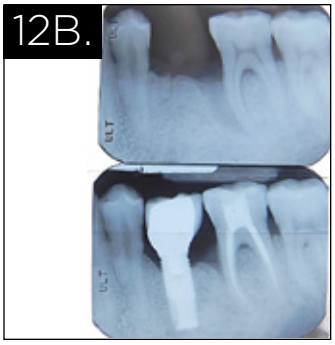


Figure 12A. Retained deciduous teeth can predispose to periodontal breakdown of adjacent permanent teeth. With removal, deep angular bony defects remain.
Figure 12B. In this case (non-smoking male aged 46), bioactive glass bone grafting provided and maintained integrity many years after Nobel Biocare implant placement, to replace extracted tooth 75 (75 extraction, bone graft and implant placement performed by author). Patient, presently aged 60, non-smoker.

Implantoplasty (filing down exposed implant threads) can predispose to implant fracture with narrow implants and heat generated with this procedure requires intense cold water lavage to avoid bone damage.[54] Chemical agents include chlorhexidine, cetylpyridinium chloride, citric acid, tetracycline, hydrogen peroxide and EDTA.[54]

No statistically significant difference existed when CO_2 and Er:YAG lasers were used to detoxify implant surfaces compared to the use of hand curettes.[55,56] Also, the use of steel curettes and the ultrasonic system proved to be totally unsuitable for cleaning titanium implants. These instruments can gouge the implant surface three to four times deeper than that of titanium curettes.[57]

To simplify treatment of peri-implantitis, Lang et. al developed the cumulative interceptive supportive therapy (CIST) protocol[58] **(Table 2)**. They felt it appropriate to apply periodontal parameters; plaque index, bleeding on probing (BOP), suppuration, pocket depth (PD) and radiographic bone loss, to the peri-implant tissues and to monitor their condition in a similar fashion as we do with periodontal tissues. The plaque and bleeding indices would gauge oral hygiene practices. Radiographic assessments were encouraged one-year following implant installation and repeated on a biannual basis. The implant patient would have regular recall and continuous diagnosis of the peri-implant tissues to provide adequate information for interceptive therapeutic measures. It also provides a template of what treatment actions to consider at various stages of peri-implant disease.

In health, (CIST A) only mechanical cleansing of the implant surface is required with maintenance classification 0 or I, perhaps yearly. In severe disease with suppuration, significant bone loss, BOP and > 5 mm PD treatment would include mechanical cleansing, anti-septic therapy, antibiotic therapy and surgical therapy (CIST A+B+C+D) and maintenance classification IV (3-monthly). CIST E reflecting poor prognosis and need for explantation, or removal of the implant, using specially designed instruments **(Figs. 6-10)**. Similar approach to the treatment for PD but much more intricate protocol.

Conservative Management (Figs. 6-10)

Conservative deep cleaning with the benefit of local anesthetic can be very effective when treating generalized chronic periodontitis and generalized aggressive periodontitis.[52] It is also preferable in moderate PD because it results in less clinical attachment loss in pockets between 4-6 mm.[52] Peri-implant mucositis can be successfully treated with conservative therapy but some authors feel non-surgical therapy is ineffective for the treatment of peri-implantitis.[53,59] A consensus report reviewing case controlled studies, however, has suggested that non-surgical treatment of peri-implantitis could be beneficial.[60]

Figures 7A – E demonstrate examples of successful conservative treatment in cases of peri-implant and periodontal diseases.

Surgical Management: Regenerative Or Resective Surgery (Figs. 11-13)

Surgical treatment of periodontitis can be very effective especially if pocket depth is > 7 mm.[52] Osseous surgery was more effective in reducing pocket depth than flap surgery without osseous recontouring. After 5 years, however, the difference between deep cleaning and osseous surgery treatment was not significant where periodontal pockets ranged between 4-6 mm.[52] The authors concluded that relapse was multi-factorial in this pocket range and encouraged the conservative option in pockets 4-6 mm. In anticipation of the variability of tissue response, sometimes periodontal surgery can be modified to obtain a desired effect.[61]

Overall, regenerative treatment of

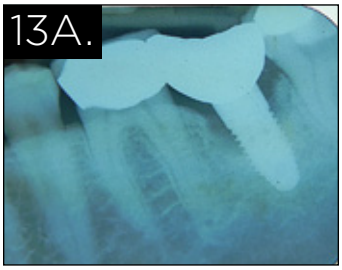


Figure 13A. Localized advanced peri-implantitis associated with implant 37, performed seven years earlier by another practitioner. 10 mm pocketing, purulent exudate and advanced peri-implant bone loss precipitated periodontal referral for correction for a non-smoking 69 year old male Periapical of area 37 demonstrating extent of osseous defect.

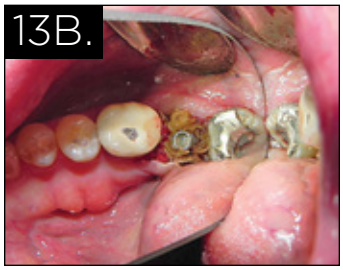


Figure 13B. During surgical protocol (CIST A+B+C+D), implant surface is decontaminated/disinfected/detoxified. Crown removal facilitated surgical access (this is not possible in cemented crowns and bridges).

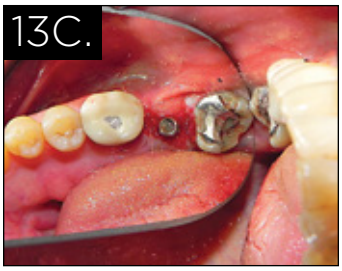


Figure 13C. Primary closure around implant abutment after bone graft material is placed.

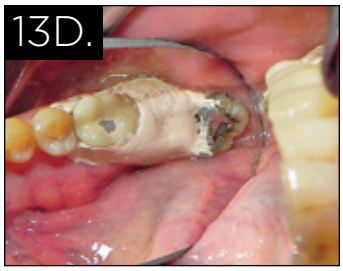


Figure 13D. Periodontal dressing is placed to protect the area.

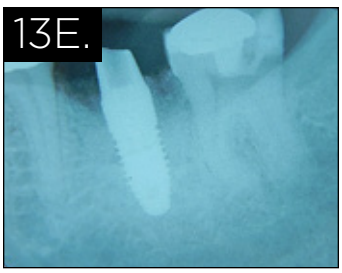


Figure 13E. Periapical radiograph demonstrating "overfill" of peri-implant defect.

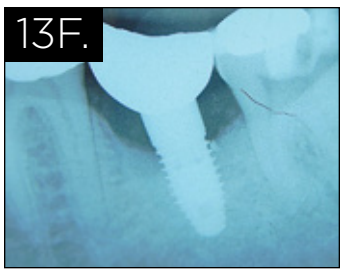
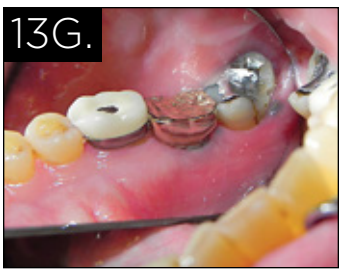
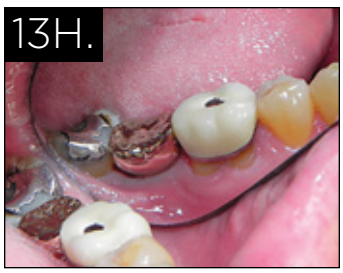


Figure 13F. Six-month post-operative radiograph demonstrating 85 bone fill of defect.





Figures 13G and H. Lingual and labial view of healed peri-implant tissues without evidence of inflammation.

infrabony defects is also associated with a relatively high degree of variability in clinical outcomes regardless of the therapeutic approach.[62]

Variability of therapeutic success is also found with surgical treatment of peri-implantitis. The more frequent surgery was flap surgery with osseous recontouring (49-58 percent) where bone loss was greater than one-third of the implant length.[63,64] Regeneration surgery was utilized in 81 percent of cases with severe peri-implantitis with a frequency of bone fill of 42 percent after a two-year follow-up. The authors concluded that the filling of bone defects did not seem to be a predictable treatment outcome.[64] Treatment results were not significantly different if the bone substitute was combined with a resorbable membrane.[64] Similarly, the success rate for surgical treatment of periodontitis can reach 69 percent[65] and in both peri-implantitis and periodontitis it was significantly lower for patients with poor compliance and unacceptable oral hygiene levels. In addition, compliance was significantly lower for smokers.[64] In one study, over 50 percent of treated cases of peri-implantitis relapsed. Most of the cases were related to poor compliance and smoking.[63] The study emphasized the need for regular maintenance and follow-up.

As can be seen the treatment of periodontitis and peri-implantitis both appear to have similar predictabilities and variabilities. The more the risk factors are controlled for both entities, the more success is achieved especially considering factors of compliance, oral hygiene and smoking.

Although PDz can increase risk of developing peri-implantitis, implant therapy can be very successful once periodontal disease is controlled in periodontally compromised patients.[66]

If peri-implantitis is extremely severe, implant removal or explantation may be required. **(CIST E; Figs. 14 & 15).**

Significance of Keratinized Mucosa (Figs. 16, 17, 18, 19 & 20)

There is a need for keratinized mucosa for maintenance of periodontal health.[67] The absence of adequate

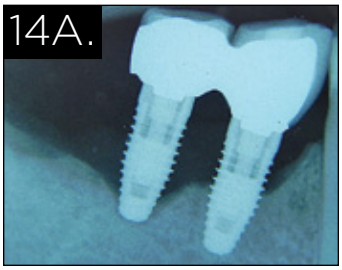


Figure 14A. At age 42, a non-smoking female had multiple implants placed 10 years previously by another practitioner and presented for management of moderate to advanced peri-implant disease. Implant 45 is basically supported by splinted implant 44. CIST protocol E would apply for removal of implant 45 which has bone loss to the apex.

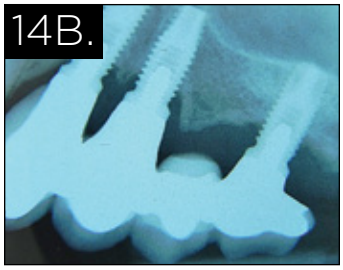


Figure 14B. Coincidentally, this same patient demonstrated peri-implant disease also in the second quadrant and would benefit from CIST protocol A+B+C+D for therapy due to 8 mm pocketing with purulence and angular bone loss.

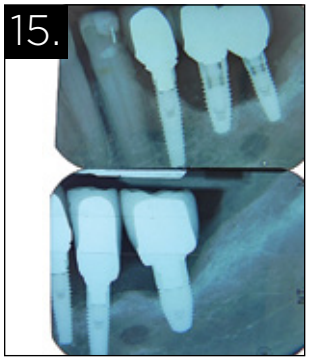
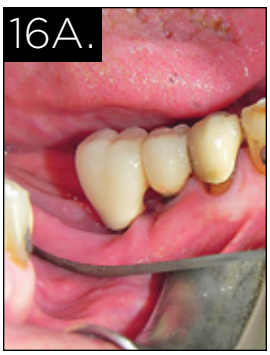


Figure 15. A patient aged 70 (smoker) had implants provided by another practitioner eight years earlier. Implants 35, 36 demonstrated advanced bone loss, 9 mm pocketing with gingival tenderness. After consultation, the original practitioner (not author) replaced implants 35 and 36 (CIST protocol E). With second effort, rebound peri-implantitis was able to be managed by the author with CIST protocol A+B+C. Note additional bone loss around implant 34 after re-implantation of implants 35 and 36, but before conservative peri-implantitis therapy.



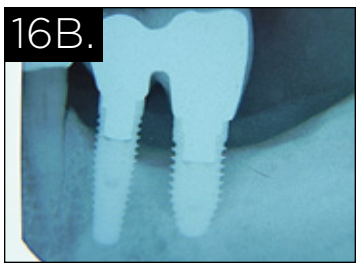


Figure 16A. I A 77 year old heavy smoker received nine dental implants in 2009 by another clinician. The patient was referred for management of peri-implant disease. CIST protocol A+B+C helped stabilize the condition in general, however, implant 36 had an extensive amount of bone loss, associated with major frenum involvement and lack of any attached gingiva. This case demonstrates the importance of keratinized mucosa with respect to implant stability. implants 35 and 36 were placed at the same time in this pack/day smoker. Note severe frenal pull without keratinized mucosa around implant 36, but not with implant 35.

Figure 16B. Periapical radiograph showing 90 percent bone loss around implant 36 (likely CIST protocol E), but 35 doing better in spite of smoking habit. It seems the lack of attached gingiva tipped the scale for peri-implant disease for this person on top of the smoking habit.

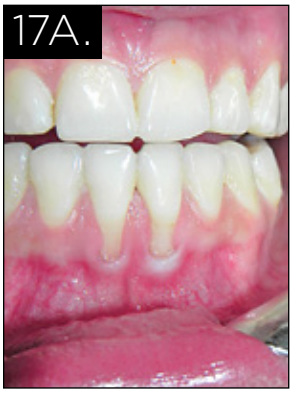
keratinized mucosa around endosseous dental implants, especially in the posterior segments, was associated with higher plaque accumulation and gingival inflammation[68] as well as attachment loss.[69] Without maintenance of this attachment, peri-implantitis can develop and predispose to significant bone loss. Successful mucogingival rehabilitation is shown in Figures 16-20, reflecting the periodontal and peri-implant environment.

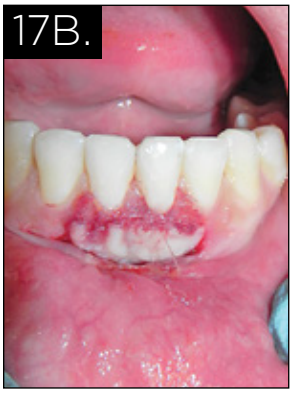
Conclusion

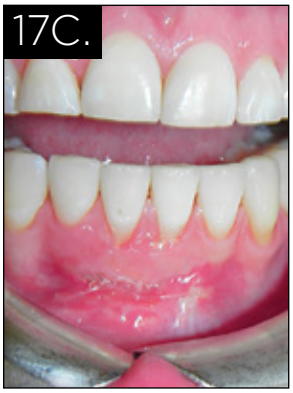
Although there are many similarities between peri-implantitis and periodontitis, the vulnerable peri-implant interface poses different challenges with respect to risk of development and subsequent treatment initiatives. The approach to treatment is similar in both diseases, but the protocol is more intense (CIST) with emphasis on IMPLANT surface decontamination and detoxification. The risk factors of smoking, poor oral hygiene and patient compliance can shift predisposition significantly in both diseases, but once all factors of risk are adjusted for, there is a high degree of success in managing both conditions. The earlier the diagnosis and intervention, the better the treatment outcome.

The greatest success with implant placement appears to be achieved in well-motivated, healthy, non-smoking patients with good bone quality, absence of periodontal disease, adequate attached gingiva, use of minimum 10 mm length implant, performed by an experienced practitioner.

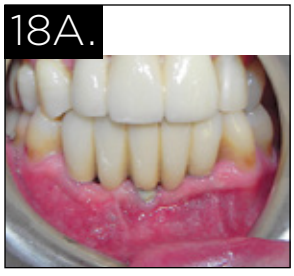
Since there is a relatively high occurrence of peri-implant diseases that can manifest and persist for years, informed consent for patients receiving implant treatment must include the need for such maintenance therapy.70 To end with a quote by Dr. Peter Fritz (periodontist, Fonthill, Ontario), "the patient often asks the question, how long do implants last? A long answer to this question is that implants will last as long as they are properly maintained – provided they were designed and manufactured properly, placed and restored properly and delivered to the right patient, by the right clinician."

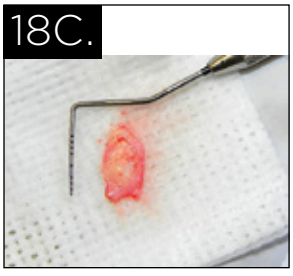


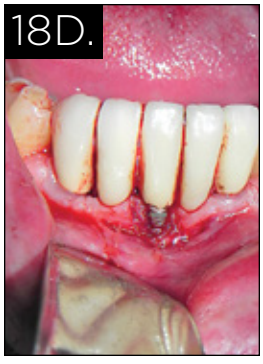


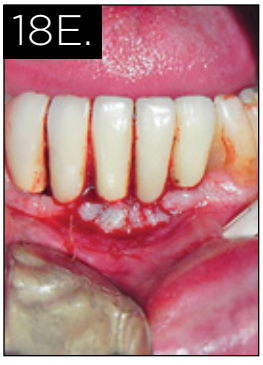


Figures 17A. A 25-year-old female with a smoking habit (5-8 cigarettes/day) also had a lower lip ornament for over five years which was associated with severe bone loss and severe gingival recession with major frenal pull and no attached gingiva. The lip ornament predisposed to this mucogingival defect. The patient was convinced to remove her ornament and a free gingival graft was placed to rehabilitate the area successfully. Pre-operative photograph. Notice that patient's intraoral manipulation of the lip ball connector also wore down the mesial-incisal edges of all central incisors to fit the diameter of the pin-attachment. The severe mucogingival defect predisposes to tooth loss of both teeth 41 and 31.
Figures 17B. Mucogingival graft is placed and stabilized (after root detoxification and decontamination with citric acid). This stage is similar to implant surgery as well.
Figures 17C. One-month post-operative appearance with more than 70 percent root coverage achieved. Harmony is re-established with only mild root exposure remaining.









Figures 18A. A 64-year-old non-smoking male experienced difficulty with implant 41. His implants were placed nine years earlier by another practitioner. Bone loss associated with severe frenum pull and lack of attached gingiva are accelerating peri-implant disease. Pre-operative clinical photograph showing severe mucogingival defect implant 41.
Figures 18B. Periapical radiograph showing 25 percent bone loss around implant 41 (4-5 threads exposed) with "saucer-shaped" osseous defect.
Figures 18C. Palatal tissue harvested.
Figures 18D. Initial surgical preparation releasing the frenum revealing exposed implant threads. This allows for chemical implant surface detoxification and decontamination.
Figures 18E. Mucogingival graft secured
Figures 18F. One-month post-operative healing demonstrating rehabilitation of the mucogingival junction with new keratinised mucosa and 3 mm of previously exposed implant threads are covered. Notice more graft shrinkage compared to Figure 17 because of mentalis muscle association. Mucogingival rehabilitation is harder with peri-implantitis than with the tooth counterpart. The mentalis muscle "fights" harder to regain its space deeper in the vestibule. A larger graft can compensate for this.

The wrong answer is the short answer, "forever".[71] **OH**

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Oral Health welcomes this original article.

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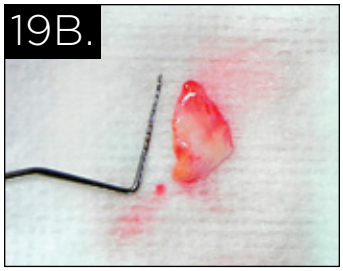

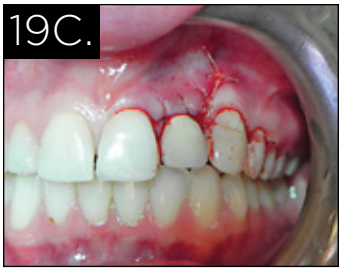

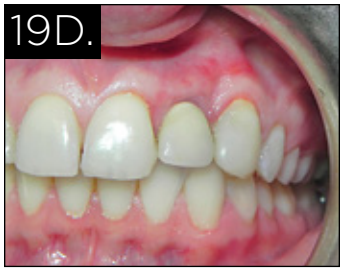

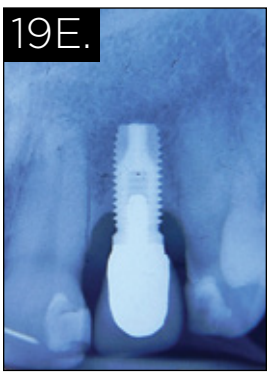

Figures 19A. 28-year-old non-smoking female had an implant placed at age 18 by another practitioner. Over time, labial gingiva collapsed associated with bone loss around the implant and a dark shadow appeared over implant 22 when the patient smiled. She found this to be disconcerting. Smile demonstrating cosmetic defect "shadow" over implant 22.
Figures 19B. Subepithelial connective tissue graft harvested from the palate to suit the area of involvement.
Figures 19C. After implant surface decontamination, the graft is stabilized utilizing papilla conservation with unilateral release technique.
Figures 19D. Attached gingiva has been "plumped-up" and cosmetic appearance is enhanced to patient satisfaction. Similar techniques are utilized with both implants and teeth with similar satisfaction (see Figure 20). With implants, it is harder because of the more fragile peri-implant interface and rough surface of implant threads.
Figures 19E. Periapical radiograph demonstrates three thread bone loss around implant 22 with saucer- shaped bone defect. In spite of significant circumferential bone loss, connective tissue grafting enhanced cosmetic improvement satisfying patient concerns.

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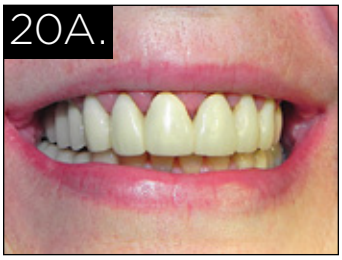
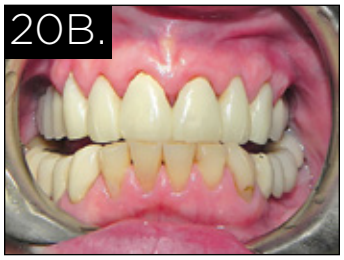
Table 2: Cumulative Interceptive Supportive Therapy (CIST)

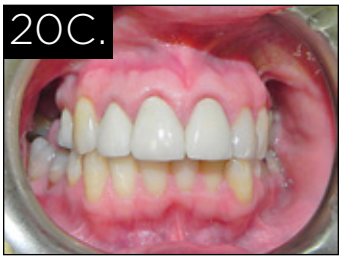
Clinical Parameters						
PII	BOP	Suppouration	PD mm	RX Defect	Maintenance Classification	CIST
±	–	–	<4	–	0	(A)
+	+	–	<4	–	I	A
+	+	±	4-5	+	II	A+B
+	+	±	>5	++	III	A+B+C
+	+	±	>5	+++	IV	A+B+C+D
+	+	±	>5	++++	V	E

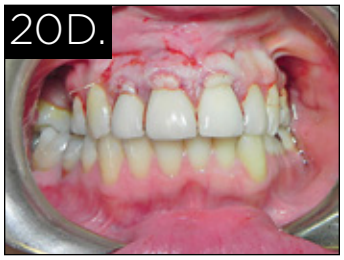
CIST modalities

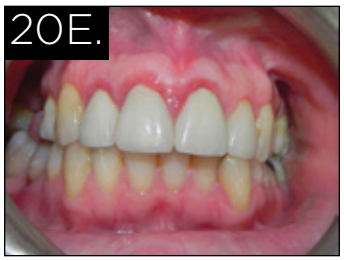
A. Mechanical cleansing using rubber cups and polishing paste, acrylic scalers for chipping-off calculus. Instruction for more effective oral hygiene practices.
B. Antiseptic therapy. Rinses with 0.1 to 0.2% chlorhexidine digluconate for 30 seconds using approximately 10ml, for 3 to 4 weeks supplemented by irrigating locally with chlorhexidine (preferably 0.2 to 0.5%) using a Leur syringe or local chlorhexidine gel application.
C. Antibiotic therapy
1. Systemic ornidazol (2 x 500mg/die) or metronidazole (2 X 250mg/die) for 10 days[63] or combination of metronidazole (500mg/die) plus amoxicillin (375mg/die) for 10 days.[77]
2. Local: Application of slow release devices for 10 days (25% tetracycline fibers).[78]
D. Surgical Approach
1. Regenerative surgery using abundant saline rinses at the defect, barrier membranes, close flap adaptation and careful post-surgical monitoring for several months. Plaque control is to be assured by applying chlorhexidine gels.
2. Resective surgery. Apically repositioning of the flap following osteoplasty around the defect.
E. Explanation using specially designed instruments.

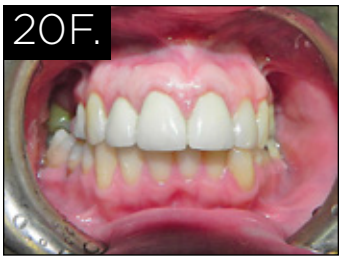












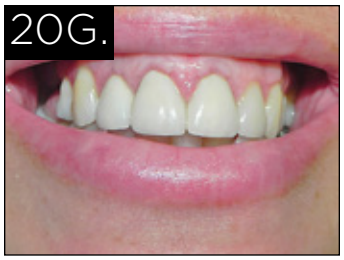


Figure 20A. A 41-year-old woman presented with a thin gingival biotype and an aggressive tooth brushing habit. New crowns 12, 11, and 21 were placed resulting in additional gingival recession and patient dissatisfaction when she smiled. Connective tissue grafts work well with implants or teeth when sufficient vestibular depth exists. Patient smile showing root exposure over recently cemented crowns 12, 11, and 21 revealing cosmetic impairment.
Figure 20B. Lips retracted revealing gingival profile.
Figure 20C. Modified brushing "paintbrush technique" encouraged. Photos show improvement but patient still not happy after one month.
Figure 20D. Subepithelial connective tissue grafts placed prior to final suturing and placement of a periodontal dressing. All grafts were 10mm x 10mm harvested from the posterior palate and introduced by papilla preservation pouch technique.
Figure 20E. One-month post-operative healing showing significant cosmetic improvement.
Figure 20F. Gingival margin restored (four months later) with mature enhanced gingival phenotype for teeth 12, 11, and 21.
20G. Patient's smile restored and uninhibited (much broader than Figure 20A). Patient very pleased with the result.

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