DOI: 10.1111/prd.12335

REVIEW ARTICLE

Periodontology 2000 WILEY

Breaking the wave of peri-implantitis

Jia-Hui Fu[1] | Hom-Lay Wang[2]

[1]Discipline of Periodontics, Faculty of Dentistry, National University of Singapore, Singapore, Singapore

[2]Department of Periodontics and Oral Medicine, University of Michigan School of Dentistry, Ann Arbor, USA

Correspondence
Hom-Lay Wang, Department of Periodontics and Oral Medicine, University of Michigan School of Dentistry, 1011 North University Avenue, Ann Arbor, MI 48109-1078, USA.
Email: homlay@umich.edu

1 | INTRODUCTION

Dental implants are reported to have high long-term cumulative mean implant survival and success rates of 94.6% ± 6% and 89.7% ± 10.2% after mean postfunctional loading periods of 13.4 years and 15.7 years, respectively.[1] The number of implants placed per year has increased exponentially and will probably continue to rise as treatment protocols become more predictable and successful over time. However, there is a steep learning curve involved in the execution of the implant-related surgical and prosthetic treatment plans. It is thus important that one grasps the fundamental biologic principles to avoid or minimize the risk factors involved in implant therapy. Otherwise, unwanted mechanical and biologic complications can be expected.[2,3] Therefore, this paper reviews recent evidence of factors that could predispose implants to peri-implantitis and measures to prevent it.

2 | DEFINITIONS AND PREVALENCE OF PERI-IMPLANT DISEASES

An implant is considered healthy when there is no visible clinical sign of inflammation around the implant; ie, the peri-implant soft tissues are coral pink in color and of firm consistency, with no bleeding and/or suppuration on probing, no deepening of probing depths, and no marginal bone loss beyond the initial physiological bone remodeling process.[4] Implants affected by peri-implant mucositis, on the other hand, will display overt clinical signs of inflammation, such as erythematous, soft, swollen, and shiny peri-implant soft tissue, bleeding and/or suppuration on probing, and deepening of probing depths with no signs of progressive marginal bone loss of beyond 2 mm since the installation of the prosthesis (Figure 1).[4] If peri-implant mucositis is left untreated, the marginal soft tissue inflammation may worsen, resulting in an irreversible condition termed peri-implantitis. In peri-implantitis, pathological inflammation of the peri-implant tissues occurs. The peri-implant soft tissue will appear inflamed with bleeding and/or suppuration on probing and an increase in probing depths, typically extending to 6 mm or deeper. There is also progressive peri-implant marginal bone loss beyond initial physiological bone remodeling (Figure 1). However, in situations where no baseline radiographs were available to assess the bone level changes over time, it was recommended that bone levels extending at least 3 mm apical to the implant platform be considered as diseased.[5,6]

These case definitions have facilitated the determination of the prevalence of peri-implant diseases. A recent meta-analysis estimated that the weighted mean prevalence of peri-implant mucositis and peri-implantitis to be 43% and 22% across Europe and South and North America, respectively.[7] A prospective study revealed that peri-implantitis at patient and implant levels was slightly reduced in China, at 19% and 11.2%, respectively.[8] Also, the prevalence of peri-implantitis was found to increase as a function of time.[7] Yet, the treatment success of this condition was at best favorable in the short term,[9] with 75% of the cases unresolved or recurred after 5 years[10]; hence, it would probably be best to avoid peri-implantitis at all costs.

3 | FACTORS THAT PREDISPOSE IMPLANTS TO PERI-IMPLANTITIS, AND PREVENTIVE MEASURES

Experimentally induced peri-implant mucositis studies in humans established that bacterial plaque is the etiological agent that causes peri-implant diseases,[11] and its removal leads to the reinstitution of peri-implant tissue health.[12] A recent review lists risk factors of developing peri-implantitis, including poor plaque

FIGURE 1 Clinical presentation of peri-implant mucositis at maxillary right lateral incisor and peri-implantitis at maxillary left central incisor. Notice the erythematous and edematous peri-implant soft tissues at both sites with the mucosal recession at the distal of the maxillary left central incisor [Colour figure can be viewed at wileyonlinelibrary.com]

control, failure to have regular periodontal maintenance, a history of periodontitis, poor spatial positioning of the implant fixture, overcontoured suprastructures, the presence of excess cement, and a lack of keratinized mucosa.[5] All these factors hinder an individual's ability to remove plaque. Current literature has also identified other factors with conflicting evidence that could increase a site's susceptibility to peri-implantitis. Figure 2 summarizes and broadly categorizes all factors into five groups: (1) patient, (2) implant design, (3) implant site, (4) prosthesis, and (5) clinician-related factors. It is important to understand that these factors may have a synergistic effect on the overall host's response to bacteria plaque at the implant sites.

3.1 | Patient-related factors

3.1.1 | Poor plaque control

A site-level analysis revealed that implants in patients not practicing proper oral hygiene were 3.8 times more likely to be affected by peri-implantitis than those with proper oral hygiene.[13] Multilevel logistic regression models also showed that, as plaque accumulated, the odds of having peri-implant mucositis increased.[14] Clinical trials have also established an association between poor plaque control and peri-implantitis (odds ratios of 3-14).[5] Therefore, it is not surprising that both patient administered plaque control (eg, manual toothbrushing[15] with either interdental brushes or floss) and professionally administered plaque control (eg, mechanical debridement using hand or powered instruments) will reduce the clinical inflammation in the peri-implant soft tissue[16] (Figure 3). Though such measures may not lead to complete clinical resolution (only 38% of affected implants had complete resolution[17]), reduction of the overall microbial burden within the periodontal pocket could be achieved if meticulous plaque control was delivered in the long run.[18] Adjunctive therapies, like systemic and locally delivered antibiotics, antiseptics, and air-abrasive devices, however, did not increase the efficacy of plaque removal.[16]

While providing personalized oral hygiene instructions to a patient is the first step in empowering him or her to make lifestyle changes and maintain good oral hygiene habits, it is often insufficient. Studies have demonstrated that most patients have difficulty achieving complete and consistent plaque removal[19] by remaining adherent to effective oral hygiene practices over time.[20] Factors such as stress, lack of knowledge, fear, and perceived indifference of the dentist could prevent a patient's adherence to oral hygiene

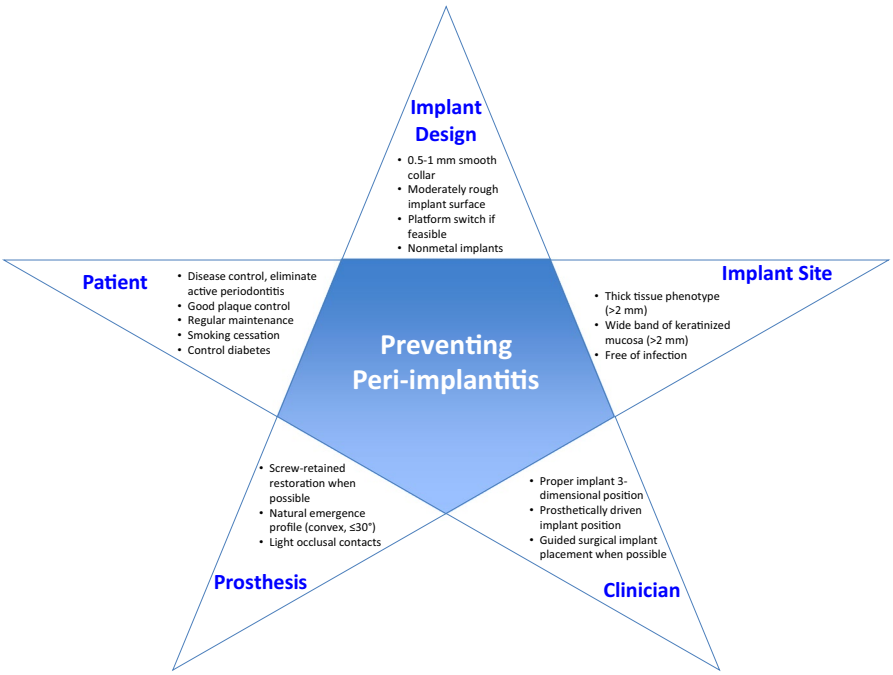

FIGURE 2 Summary of the factors to control for avoiding peri-implantitis [Colour figure can be viewed at wileyonlinelibrary.com]

FIGURE 3 Patient with poor plaque control around his screw-retained provisional fixed partial prosthesis. The risk of having peri-implantitis was reduced because there was a thick band of keratinized mucosa around the machined collar implants and the patient was compliant with a 3-monthly maintenance program. A, Buccal and, B, lingual views of the screw-retained provisional fixed partial prosthesis with calculus at the implant collar and, C, after prophylaxis was completed [Colour figure can be viewed at wileyonlinelibrary.com]

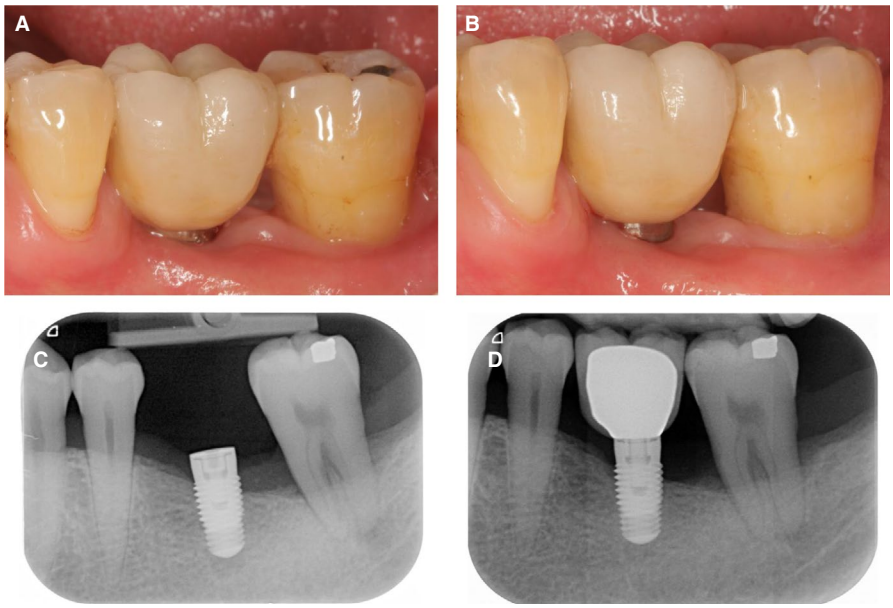

FIGURE 4 Patient with a history of severe periodontitis with a poorly placed implant at the mandibular left first molar. The progression of peri-implant marginal bone loss was reduced because the machined collar implant reduced plaque accumulation, the patient had excellent plaque control, and the patient was compliant with a 3-monthly maintenance program. Buccal view of the cemented implant crown at, A, 1 week and, B, 5 years postdelivery. Periapical radiographs of the implant site at, C, 6 months and, D, 5 years postplacement [Colour figure can be viewed at wileyonlinelibrary.com]

practices.[21] Therefore, several concepts, such as the social cognitive theory,[22] self-efficacy theory,[23] and health action process approach theory,[24] were investigated to increase patients' adherence. It was also reported that repeated reminders would help to maintain oral hygiene practices.[25] In addition, the use of digital technology was found to promote positive behavioral changes in patients with regard to adherence to oral hygiene practices.[24,26] For example, videos were reported to be an engaging and effective way to improve and reinforce a patient's oral health knowledge and maintenance of good oral habits.[27] Therefore, clinicians could create personalized oral hygiene instructional videos for their patients to encourage them to maintain good oral hygiene habits over time.

Conclusion: There is strong evidence supporting poor plaque control as a risk factor/indicator for peri-implantitis.

3.1.2 | Enrollment into a regular periodontal maintenance program

The periodontal literature has demonstrated increased tooth loss and recurrence or progression of periodontitis in successfully treated patients who did not adhere to a periodontal maintenance regimen.[28-33] Similarly, it was recently reported that the prevalence of peri-implantitis and implant loss escalated in patients who did not adhere to a regular maintenance program.[34-37] For instance, the prevalence of peri-implantitis doubled in patients who did not have any dental visits in the first 5 years post implant therapy compared with those who were seen at least once every year.[38] A plausible explanation was that the overall bacterial load and proportions of *Porphyromonas gingivalis*, *Treponema denticola*, and *Fusobacterium nucleatum* were increased in

nonadherent patients.[39] Consequently, one in five patients would thus have peri-implantitis.[40] Furthermore, a recent clinical trial demonstrated that, over 6 years, implants placed in high-risk patients performed just as well as implants placed in healthy patients if a regular periodontal maintenance program was followed.[41] Hence, it was obvious that all patients with implant restorations should have regular maintenance care to prevent peri-implantitis (Figure 4).

Determining an effective periodontal maintenance interval involves a detailed evaluation of the patient and his or her risk of having periodontal tissue breakdown. As such, it is difficult to define a standard optimal dental recall interval for prevention of a chronic and dynamic inflammatory condition like periodontitis or peri-implantitis,[42,43] since each patient displays a host of unique factors that influence disease activity. Thus, a personalized periodontal maintenance regimen appropriate for each patient's therapeutic needs and risks should be formulated.[44] The periodontal risk assessment helps the clinician determine the proper maintenance interval based on the risk of disease recurrence.[45] Interestingly, recolonization of bacteria within the peri-implant pockets after mechanical debridement has not been investigated. However, one can draw conclusions from classic periodontal literature whereby subgingival microorganisms generally recolonize the subgingival pockets within 6-12 weeks post mechanical debridement[46-48] depending on the treatment rendered, the distribution of periodontal pathogens, and the quality of the self-administered plaque control.[49] In addition, regular periodontal maintenance contributes to periodontal stability by keeping the levels of periodontopathogenic microbial load[50] and proinflammatory cytokines[51] low. Therefore, patients with dental implants should be seen at regular intervals (eg, once every 3 months[52] or 5-6 months[35]) for periodontal maintenance.

Conclusion: There is strong evidence suggesting that lack of regular maintenance is a risk factor/indicator for peri-implantitis.

3.1.3 | History of active periodontitis

Despite the ambiguity in case definitions for periodontitis and peri-implantitis, longitudinal and cross-sectional studies have repeatedly reported a positive association between peri-implantitis and patients with either active periodontitis or a history of periodontitis. The odds ratio ranged from 2.2 to 19.0[5] with a risk ratio of 9 after a 10-year follow-up period.[53] A recent meta-analysis revealed that periodontally susceptible patients had a 2.3-increased risk of having peri-implantitis compared with periodontally healthy patients.[54] It might be that those with a history of periodontitis were found to be less adherent (erratic and noncompliant) than periodontally healthy individuals were.[55,56] Moreover, it was estimated that implants replacing periodontally involved teeth had approximately 0.5 mm more marginal bone loss after 5 years.[53] It was found that periodontopathogens such as *Aggregatibacter actinomycetemcomitans*, *Prevotella intermedia*, *P. gingivalis*, *T. denticola*, and *F. nucleatum* might be transmitted from natural teeth to the adjacent implants.[57] Hence, the presence of residual probing depths of 5 mm or deeper appeared to indicate a significant risk for development of peri-implantitis,[36,58] especially when it involved more than 10% of all sites.[8] These observations are not surprising in periodontally susceptible patients who might have more pathogenic bacteria, higher bacterial load, or a host response unable to cope with bacterial insults.[59] In addition, periodontitis might have resulted in a residual ridge that was compromised in terms of bone quality and quantity,[8] predisposing the site to peri-implantitis.

Periodontitis is the sixth most prevalent global disease.[60] The latest National Health and Nutrition Examination Survey reported that approximately 42% of Americans above the age of 30 years old had periodontitis and 7.8% had the severe form.[61] Therefore, it was not surprising that periodontal patients would need dental implants in their prosthetic rehabilitation. In order to avoid peri-implantitis, it would be best that all patients received periodontal assessment and management prior to implant placement so that pockets of 6 mm or more were eliminated or controlled prior to implant placement[62] (Figure 4).

Conclusion: There is strong evidence suggesting that a history of or active periodontitis is a risk factor/indicator for peri-implantitis.

3.1.4 | Smoking

Studies on the effect of smoking on peri-implant health are inconclusive. It is clear that the systematic effects caused by smoking adversely affect wound healing. For example, studies using animal models show that smoking resulted in reduced peri-implant bone mineral density[63-65] and bone to implant contact.[65] Both effects may cause a higher incidence of bone loss that increases a site's susceptibility to peri-implantitis. Some studies showed that smokers generally had significantly higher proinflammatory cytokine levels,[66] probing depths, suppuration, and bleeding and plaque scores at implant sites compared than nonsmokers did.[67-69] Analysis of the peri-implant microbiome also revealed that smoking resulted in an increase in *Fusobacterium*, *Tannerella*, and *Mogibacterium*, which were largely associated with tissue inflammation.[70] Furthermore, smokers displayed poor oral hygiene and were less adherent to supportive periodontal therapy.[55] Therefore, smoking was thought to be associated with a higher prevalence of peri-implantitis[71] with an odds ratio of 1.7[72] to 2.57[40] and a prevalence ratio of 6.59.[58]

However, several systematic reviews have reported weak evidence for an effect of smoking on peri-implant health.[73-77] A cross-sectional study that examined 239 patients with dental implants with a mean follow-up period of 5 years found that the overall prevalence of peri-implantitis was 15% and smokers were not at a higher risk of having peri-implantitis.[78] These contrary results may be explained by the possibility that smoking habits were mainly self-reported, and the dosing and duration of smoking varied considerably from study to study. In addition, its effect might be confounded by other factors, such as good oral hygiene,[79] adherence to periodontal maintenance,[80] and susceptibility to periodontitis[16,81] (Figure 5).

Conclusion: Evidence suggesting that smoking is a risk factor/indicator for peri-implantitis is inconclusive.

3.1.5 | Diabetes

The International Diabetes Federation estimates 415 million adults (20-79 years old; 8.8% of the global population) suffered from diabetes mellitus or type 2 diabetes in 2015. This number will drastically

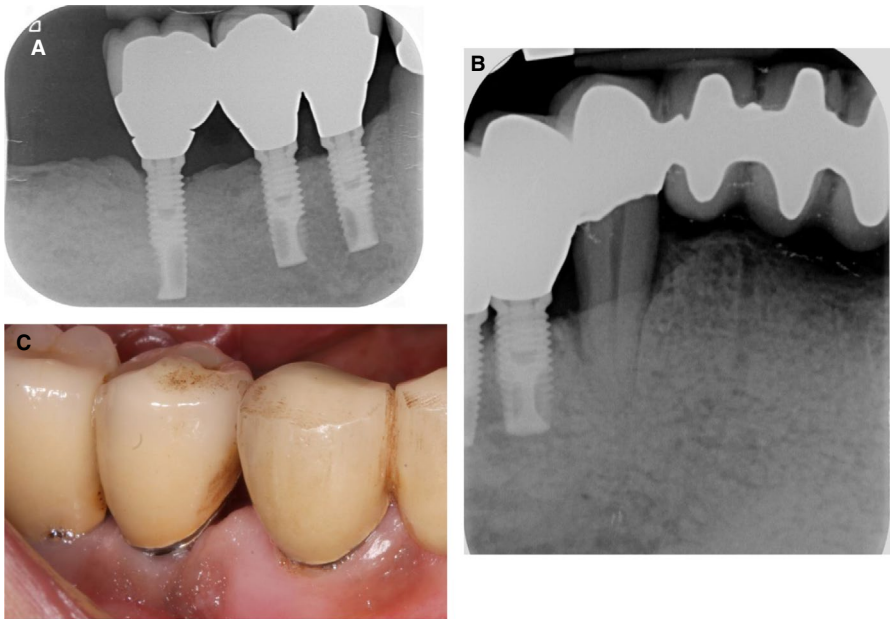

FIGURE 5 Patient is a heavy smoker who lost his mandibular right first premolar due to clenching. The risk of peri-implantitis was reduced because the machined collar implant reduced plaque accumulation and the patient was compliant with a 6-monthly maintenance program. Periapical radiograph of implant at, A, crown delivery and, B, 6 years postloading. C, Buccal view of the cemented implant crown at 6 years postloading [Colour figure can be viewed at wileyonlinelibrary.com]

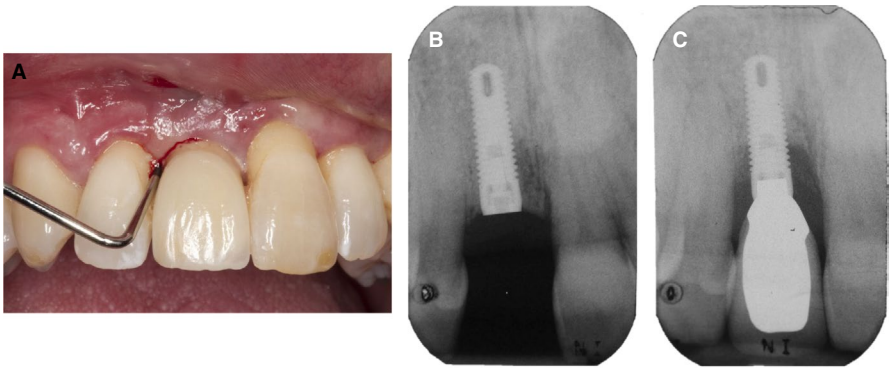

FIGURE 6 Peri-implantitis at implant placed at maxillary right central incisor position in patient with poorly controlled diabetes mellitus. His glycemic control was HBA1c < 8% prior to implant placement and it fluctuated between 8% and 9% after implant placement. A, Clinical presentation of screw-retained implant crown at 1 year postloading, showing erythematous and edematous peri-implant tissues with deep probing depths circumferentially. Periapical radiographs of implant, B, prior to crown delivery and, C, at 1 year postloading [Colour figure can be viewed at wileyonlinelibrary.com]

increase to 642 million adults (10.4% of the global population) by 2040. Likewise, the number of adults estimated to have impaired glucose tolerance (pre-diabetes) will increase from 318 million (6.7%) in 2015 to 481 million (7.8%) in 2040.[82] In the oral environment, a hyperglycemic state triggers an increase in inflammation, oxidative stress, apoptosis, and altered polymorphonuclear function.[83] There is an upregulation and release of proinflammatory cytokines, which is a similar response that microbial plaque can elicit within the periodontal tissues. The dysregulated and hyperinflammatory responses that fluctuate with glycemic control are key processes influencing the clinical changes that are observed within the periodontal tissues as well.[84] These immunological processes supported the clinical findings, whereby significantly higher mean levels of advanced glycated end products in hyperglycemic patients are associated with increased plaque index, bleeding on probing, probing depths, marginal bone loss, and implant stability compared with healthy patients.[85-88]

One study showed that the prevalence rate for peri-implantitis was 24% in a sample population of individuals on anti-hyperglycemic medication or presented with fasting blood sugar levels of 126 mg/dL at the final clinical examination. In contrast, only 7% of healthy individuals were found to have peri-implantitis.[89] Similarly, several systematic reviews report a positive association between type 2 diabetes and peri-implantitis with an odds ratio of 1.89[90] to 2.50.[72] Those with type 2 diabetes had a 3.39 times higher risk of having peri-implantitis compared with healthy individuals when the confounder smoking was excluded from the analysis[90] (Figure 6).

Other cohort studies reported contradictory analysis where hyperglycemia adversely altered peri-implant bone healing and implant stability but did not affect implant survival rates.[40,76,91,92] Several systematic reviews also showed no strong association between hyperglycemia and peri-implantitis.[5,93,94] The inconclusive outcomes might be partially due to unclear distinction between controlled and uncontrolled type 2 diabetes and patient-reported glycemic control. Nonetheless, patients should be encouraged to lead an active lifestyle, have healthy diets, regular medical follow-ups, and practice good oral hygiene[95] to reduce their susceptibility to peri-implant diseases.

Conclusion: Evidence suggesting that diabetes is a risk factor/indicator for peri-implantitis is inconclusive.

3.2 | Implant-related factors

3.2.1 | Implant surface characteristics

The evolution of dental implant surfaces from the original designs that used a machined surface to the current roughened surface has enabled faster osseointegration in the early wound healing phase and immediate implant placement and loading.[96] A review of the longitudinal studies evaluating implant surface roughness and implant survival rates have reported a high mean weighted implant survival rate of 98.4% with a mean marginal bone loss of 1.01 mm,[97] when implants were properly placed and maintained.[96] The meta-analysis revealed a significant difference in mean marginal bone loss between moderately and minimally rough implant surfaces (1.01 mm vs 0.86 mm), but confounders such as a history of periodontitis, underlying systemic diseases, and smoking might have a stronger effect on peri-implantitis than implant surface roughness.[97,98] In a recent meta-analysis, it was found that implants with a moderately rough surface were found to be associated with lower prevalence of peri-implantitis (5.4% at implant level and 5.9% at patient level) compared with the minimally rough and rough surfaces.[99] Despite the existence of reports drawing different conclusions,[96,97,100] it remains that exposed rough implant surfaces are extremely plaque retentive and prone to peri-implantitis.[101] Therefore, the key to avoidance of peri-implantitis is to abide by the rules of proper case selection, treatment execution, and maintenance. Furthermore, it may be advisable to use implants with polished (smooth) collars (of 0.5-1 mm) to minimize plaque retention (Figure 4).

Conclusion: Characteristics of the implant surface are not an established risk factor/indicator for peri-implantitis because the available evidence is controversial. However, it is clear that rough implant surfaces that are exposed to the oral cavity are difficult to keep free of plaque and disease.

3.2.2 | Implant collar/platform designs

Different implant collar designs, ranging from machined to rough collars, microthreaded to laser microtextured, straight to scalloped, and butt joint to platform switched, have been introduced over the years. Predictive nonlinear models were used on 72 patients with 237 implants to determine clinical features of implants that increase their susceptibility to peri-implantitis. Machine-surfaced implants were found to have 5.2 mm of marginal bone loss or remodeling after 16-20 years of function in patients with irregular maintenance regimens.[102] Several studies found that roughened titanium surfaces promoted osseointegration and maintained peri-implant marginal bone[103-105] by reducing the stress concentrations in the crestal bone region.[106] Recent systematic reviews and meta-analyses of these designs found that rough or laser microtextured collars had significantly less marginal bone loss if placed subcrestally compared with machined collars.[107,108] Similarly, the platform switching concept was favored as more peri-implant marginal bone was preserved,[109-114] but thickness of soft tissues might still influence the amount of crestal remodeling.[115] However, these results must be interpreted with caution, as the studies were very heterogeneous in design and the actual differences in the marginal bone levels between the designs (eg, weighted mean difference of 0.43-0.77 mm) were clinically irrelevant. In general, rough collared implants with platform switching were preferred in the anterior esthetic areas (Figure 7); in periodontally susceptible patients, however, minimally rough or machined collar implants might be a wise alternative, as proven by their lower prevalence of peri-implantitis and crestal bone loss[40,116] (Figures 3-5).

Conclusion: Evidence suggesting that design of implant collar/platform is a risk factor/indicator for peri-implantitis is inconclusive.

3.2.3 | Titanium dissolution products

Several situations might trigger the release of titanium dissolution products into the peri-implant tissues. For example, during implant placement or dental prophylaxis, when corrosion is present at the implant surface, frictional wear occurs at the implant-abutment interface at sites with malpositioned implant, excess cement, or peri-implantitis.[117] These dissolution products influence the development of peri-implantitis as they stimulate inflammation and elicit foreign body reactions within the peri-implant tissues.[118,119] Additionally, ribonucleic acid sequencing techniques detected that corrosion of titanium modified the peri-implant microbiome, favoring the colonization by *Veillonella* instead of *Streptococcus*, *Prevotella*, and *Haemophilus*, which typically promoted health.[120] Cross-sectional studies, too, had reported a significant increase in the level of dissolved titanium in the submucosal plaque at implants with peri-implantitis compared with those without (0.85

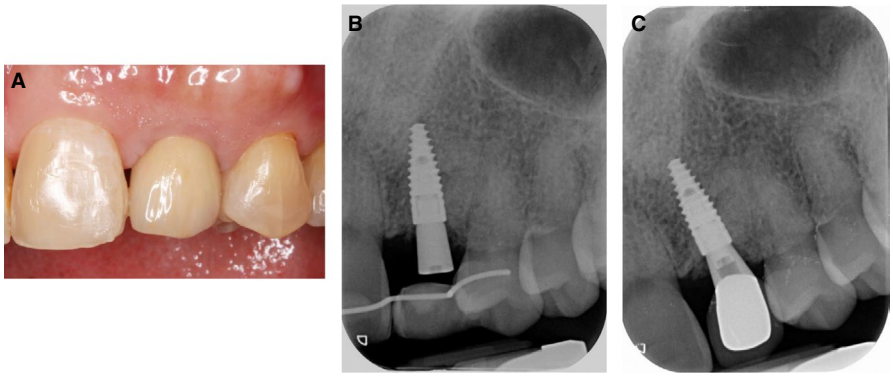

FIGURE 7 Implant with rough collar and in-built platform switch design at the maxillary left lateral incisor. A, Clinical presentation; periapical radiographs at, B, 4 months after implant placement and, C, 2 years postloading [Colour figure can be viewed at wileyonlinelibrary.com]

peri-implantitis vs 0.07 healthy).[120,121] Though evidence points to an association between titanium dissolution products and peri-implantitis, it is not strong enough to support a causal relationship between them.

To prevent the possible impact of titanium dissolution products, nonmetal (eg, zirconia) implants may be used. A benchtop study demonstrated an estimated 10-fold reduction in release of dissolved titanium at sites with a platform-switching concept compared with platform-matched ones.[122] Zirconia implants were also found to be comparable[123] to titanium implants, as histologic human specimens showed that the former had a mean bone to implant contact of 76.5% after 4 years of loading.[124] More prospective longitudinal human trials are needed to further investigate the efficacy of this implant surface.

Conclusion: Evidence suggesting that titanium dissolution products is a risk factor/indicator for peri-implantitis is limited. More research is needed to evaluate of the role of titanium or metal particles in the pathogenesis of peri-implant diseases.

3.3 | Implant site–related factors

3.3.1 | Tissue phenotype

Mucosal thickness and amount of attached keratinized mucosa are key components of the peri-implant mucosal tissue phenotype (Figure 8) and their value in the maintenance of peri-implant tissue stability is one of the most contested topics in implantology. Tissue biotype was thought to play a role in the maintenance of peri-implant health,[125] as it affected the ease of performing oral hygiene measures and maintenance of peri-implant marginal bone stability. Yet, in recent years, it was more evident that implants placed in sites with thick soft tissues (thickness of more than 2 mm) had two to five times lower crestal bone resorption than in sites with thin tissues.[126,127] Several systematic reviews also reported a positive association between soft tissue thickness and preservation of peri-implant marginal bone,[125,128,129] where sites with thick tissues had a mean 0.8 mm more bone than sites with thin tissues.[129] However, a recent meta-analysis found no significant difference between thin and thick tissues in the preservation of crestal bone level. This observation could be attributed to the use of only two studies in the analysis and most of the studies included in the systematic review had an unclear or high risk of bias.[130] Hence, in order to avoid bone remodeling at the implant platform in sites with thin soft tissues, autogenous soft tissue grafts (either subepithelial or free connective tissue graft) or soft tissue substitutes (eg, acellular dermal matrix or collagen matrix) could be used to thicken the tissues around the implants.[131-136] Thickening of the soft tissue phenotype would allow patients to perform better oral hygiene and, therefore, prevent the incidence of peri-implantitis (Figure 9).

The majority of the human clinical trials reported that the presence of a wide band of keratinized mucosa (2 mm or more) significantly reduced plaque accumulation, tissue inflammation, and probing depths, because patients had less discomfort when brushing. There was also a protective effect against mucosal recession and crestal bone loss[137-141] (Figure 10). A 10-year longitudinal study showed that almost half of the subjects (43%) reported discomfort while performing oral hygiene when there was no keratinized mucosa around the implants. As such, implants surrounded by only alveolar mucosa had significantly more plaque (16%) than those with keratinized mucosa.[142] A prospective study also reported that patients preferred having keratinized mucosa around their implants, as it made the restorations significantly more esthetic.[143] Several systematic reviews concurred with this observation.[125,128,129,144] As a preventive measure, especially in those who did not adhere to a maintenance protocol,[145] it would be wise to increase the band of keratinized mucosa around dental implants.

Considering that the lack of keratinized mucosa makes a site more susceptible to peri-implantitis,[146] an apically positioned flap with a free gingival graft is the gold standard for increasing the

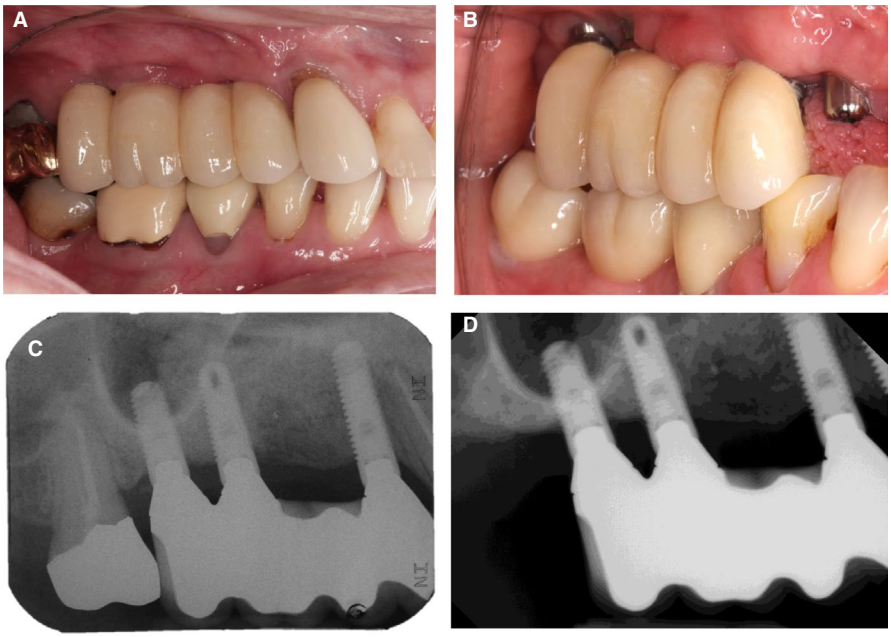

FIGURE 8 Progressive peri-implant bone loss around the implants placed in a site with an inadequate band of keratinized mucosa. A, Clinical presentation at prosthesis delivery. B, Clinical presentation at 5-year follow-up. C, Periapical radiograph at prosthesis delivery. D, Periapical radiograph at 5-year follow-up [Colour figure can be viewed at wileyonlinelibrary.com]

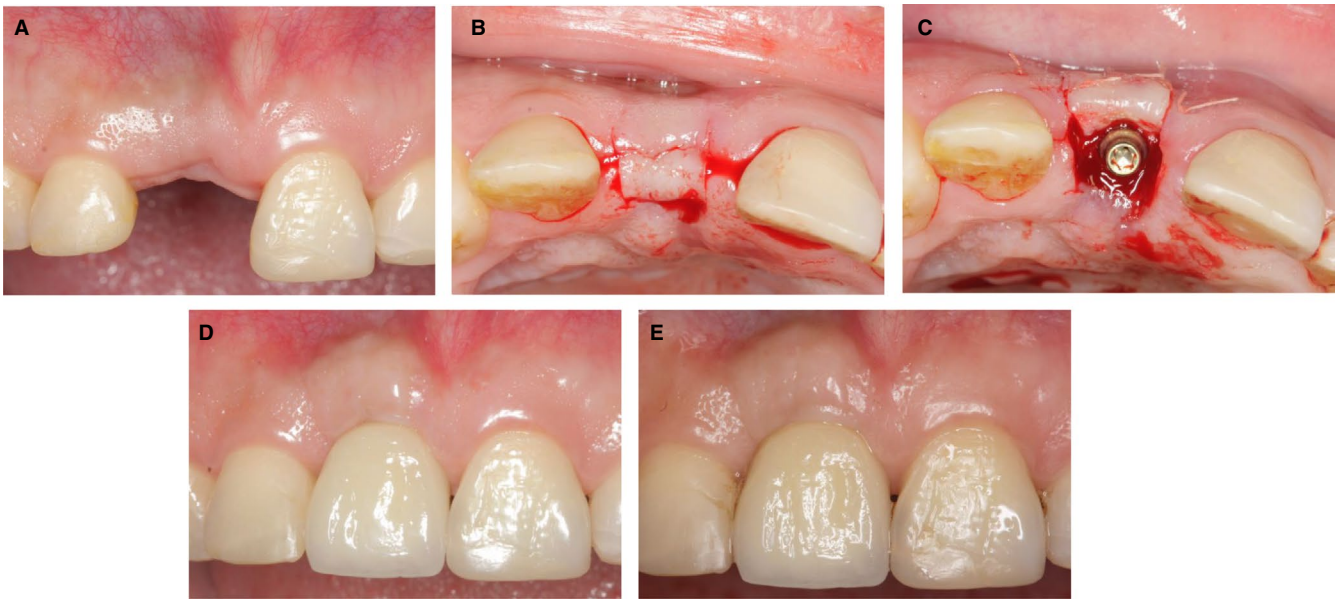

FIGURE 9 Thickening of the buccal peri-implant soft tissue by rolling the crestal tissues to the buccal side. A, Preoperative presentation; B, split-thickness flap design; C, crestal tissues rolled underneath the buccal flap; D, 6 months postloading; E, 2 years postloading [Colour figure can be viewed at wileyonlinelibrary.com]

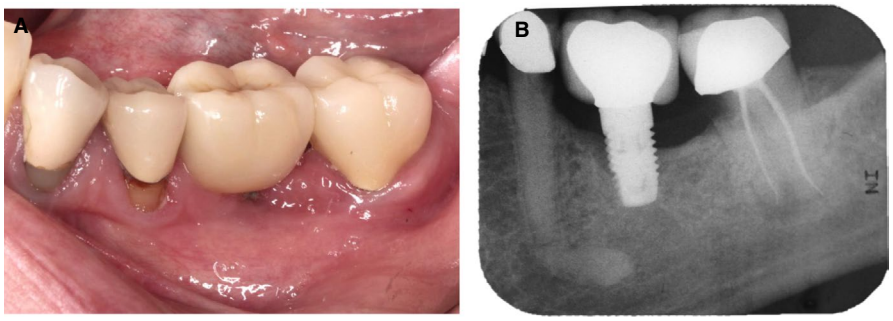

FIGURE 10 Implant supporting an overcontoured crown at the mandibular left first molar was placed in a site with no keratinized mucosa: A, baseline; B, 2-year follow-up [Colour figure can be viewed at wileyonlinelibrary.com]

band of keratinized mucosa.[125,147,148] Soft tissue substitutions could also be utilized, as they increased patient satisfaction by reducing the treatment duration and postoperative morbidity (Figure 11). However, they have not demonstrated superior long-term clinical outcomes compared with the autogenous grafts.[125,132]

Conclusion: There is moderate evidence suggesting that tissue phenotype (either mucosal thickness or amount of attached keratinized mucosa) is a risk factor/indicator for peri-implantitis.

3.3.2 | Residual infection

Retrograde peri-implantitis is defined as localized periapical radiolucency around the implant fixture, which may or may not be associated with clinical signs of inflammation. It was found to be closely related to the presence of endodontic pathoses from adjacent teeth.[5] Also, if a tooth was removed due to an endodontic infection, it was reported that a peri-apical radiolucency would occur in 8.2% to 13.6% (odds ratio 7.2) of the cases.[149] Hence, it is important to meticulously debride the implant site or extraction socket to remove any pathological tissues, especially when placing immediate implants, as the residual infection may spread to the implant and result in periapical bone loss.[150] It is also acceptable to wait for the infection to clear prior to implant placement, allowing clinicians to choose to perform early or delayed implant placement.[151]

Conclusion: There is evidence suggesting that residual endodontic infection is a risk factor/indicator for developing retrograde peri-implantitis.

3.4 | Prosthesis-related factors

3.4.1 | Excess cement

One major disadvantage of cemented implant restorations is the possibility of residual cement persisting in the peri-implant tissues,[152] which unfortunately is extremely difficult to detect clinically and radiographically (only 7.5%-11.3% of cement remnants can be detected).[153,154] The residual cement layer not only acts as a foreign body but is also plaque retentive, resulting in 81% of cemented restorations having signs of peri-implant inflammation.[155] The amount of undetected excess cement increases linearly with the apical positioning of the implant platform[153,156] and concave emergence profile abutments.[156] It also had a worse effect in periodontally susceptible patients, where all cemented

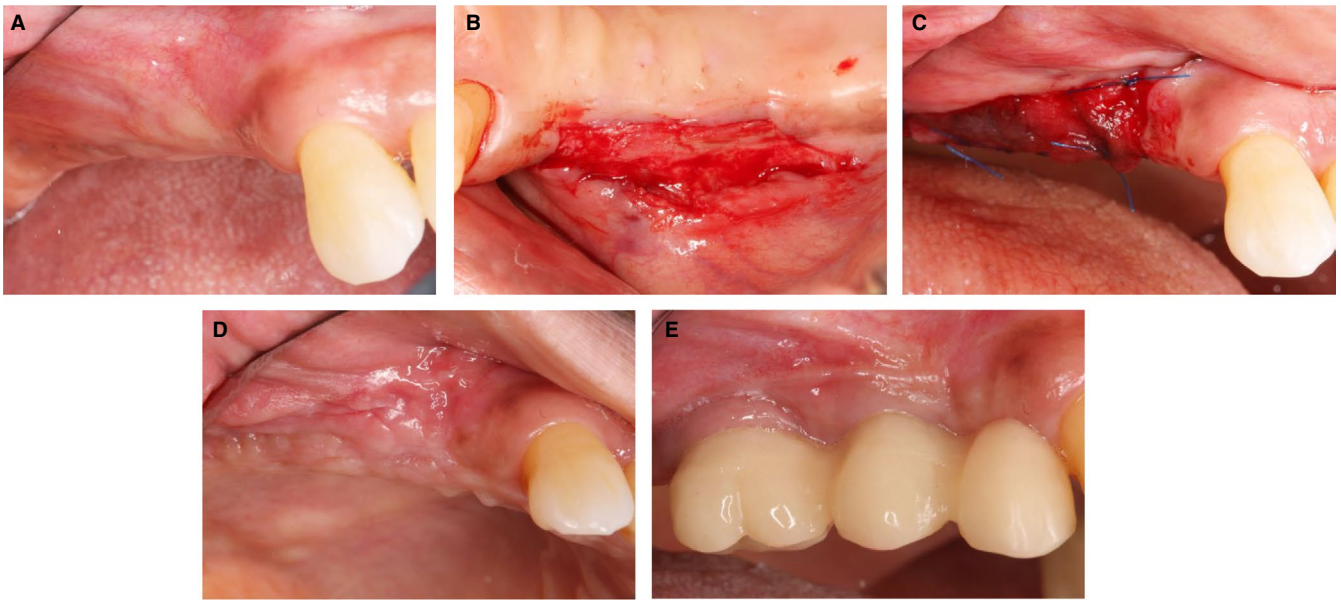

FIGURE 11 Increasing the band of keratinized mucosa using a collagen matrix prior to crown installation. A, Preoperative presentation. B, Split-thickness flap design. C, Collagen matrix secured with cross mattress sutures. D, At 1 month postsurgery. E, At 3 months after delivery of provisional crowns [Colour figure can be viewed at wileyonlinelibrary.com]

restorations developed peri-implantitis in approximately 2 years (range: 10-48 months) compared with 8.8% in periodontally healthy patients (median 3.5 years; range: 14-85 months).[157] A recent systematic review also agreed that 33%-100% of cemented restorations with peri-implantitis had excess cement (Figure 12). Therefore, it was suggested to use screw-retained restorations whenever possible; or for cemented restorations, it was suggested to bring the crown margin to the level of the peri-implant mucosa for easy removal of excess cement, to cement the suprastructure after maturation of the soft tissue cuff, and for early follow up after delivery of the restoration.[158] Also, it is relatively easier to remove zinc phosphate cement from titanium surfaces than it is other luting agents (eg, resin cement).[159,160]

Conclusion: There is a strong evidence suggesting that residual or excess cement is a risk factor/indicator for developing peri-implantitis.

3.4.2 | Overcontoured suprastructures

A study found that overcontoured restorations (Figure 13) with an emergence angle of more than 30° had a two times greater prevalence of peri-implantitis than restorations with narrower emergence angles did (31% vs 15%; P = .04), especially in the roughened surface implant.[161] This could be in part due to a malpositioned implant or larger emergence angle (greater than 30°), leading to difficulty in accessing

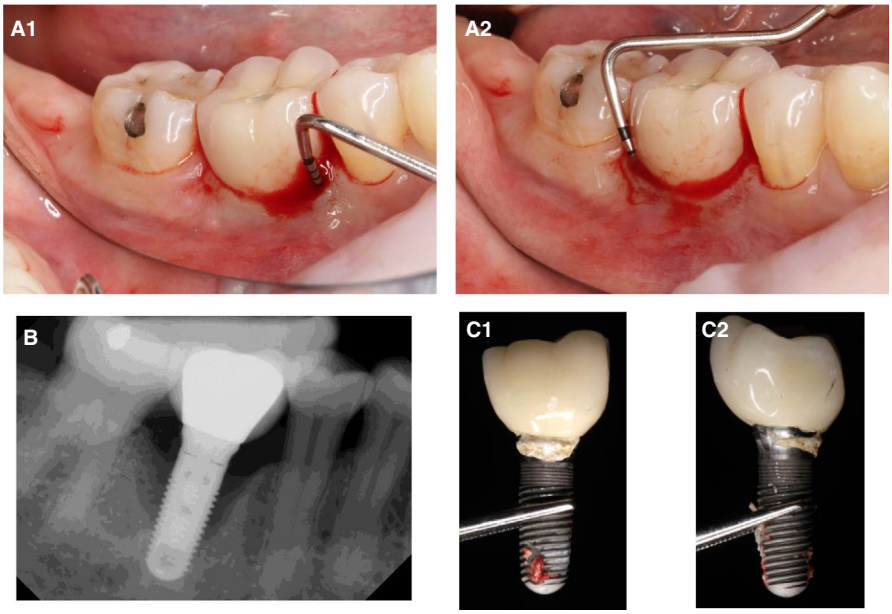

FIGURE 12 Implant at the mandibular right first molar after 2 years of function. A, Clinical presentation showing deep probing depths (7-8 mm) with bleeding on probing interproximally. B, Periapical radiograph showing severe bone loss around the implant. C, Excess cement was left around the implant collar [Colour figure can be viewed at wileyonlinelibrary.com]

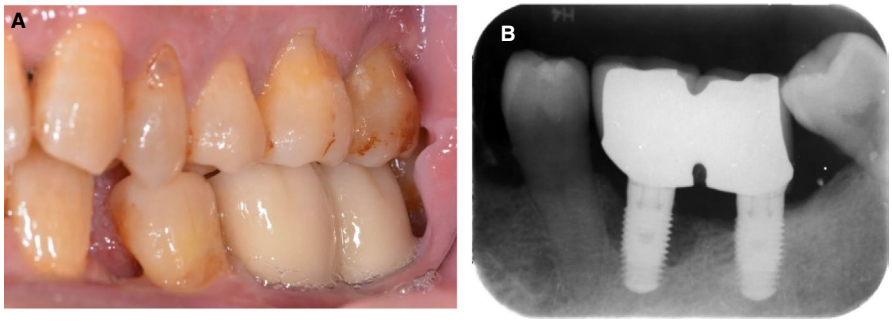

FIGURE 13 Overcontoured splinted implant crowns replacing mandibular left first and second molars with peri-implant bone loss. A, Clinical and, B, radiographic presentations [Colour figure can be viewed at wileyonlinelibrary.com]

the implant platform for plaque control. Thus, it is important to place the implant in a prosthetically driven position, with a less than 30° emergence angle to provide access for hygiene. Thereafter, the restoration should have an emergence profile that mimics natural tooth contours and wide enough embrasures for interdental cleaning.[162]

Conclusion: There is moderate evidence suggesting that overcontoured restoration is a risk factor/indicator for developing peri-implantitis.

3.4.3 | Occlusal overloading/interproximal opening

It is difficult to establish the effect of occlusal overloading in humans because interventional studies would violate human ethics standards. Therefore, studies have been mainly observational and retrospective and thus confounded by the influence of plaque control and maintenance care.[163,164] Nevertheless, the use of an advanced data mining model showed that implants under occlusal overloading were at risk of peri-implantitis, having an odds ratio of 18.70.[165] Recent human case reports demonstrated the loss of osseointegration when implants were excessively loaded, and reosseointegration occurred once the occlusal load was removed.[166,167] It was demonstrated in an animal model that occlusal overloading accelerated peri-implant bone loss in the presence of plaque and inflammation[168] but not in peri-implant mucosal health.[169] Systematic reviews also found positive associations between occlusal overloading and loss of peri-implant marginal bone, especially when peri-implant inflammation was present.[164,170,171] The potential cause of occlusal overloading could be due to the opening of interproximal contacts between the implant crown and adjacent teeth creating eccentric contacts in maximum intercuspation and excursive movements (Figure 14). It was reported that the occurrence was 18%-66% and 37%-54% in the maxilla and mandible, respectively.[172] It is evident from these studies that regular review of the occlusal contacts to ensure light contacts on the implant restorations to avoid occlusal overloading is important.[171] In addition, delivery of an occlusal guard may be helpful in maintaining the positions of the teeth in the arch.[172]

An implant is ankylosed and thus unable to move within bone. Teeth, on the contrary, do move coronally and mesially throughout life, as the result of normal physiological function. As such, the continued eruption of teeth in young adults often creates esthetic disharmony, especially in the maxillary anterior region.[173,174] Masticatory forces tend to cause interproximal wear, specifically on the mesial tooth surfaces, resulting in teeth drifting mesially and interproximal contacts opening

FIGURE 14 Loss of interproximal contacts resulting in food trap and peri-implant bone loss

as a compensatory mechanism[175-177] in almost 43% of patients who had implant restorations.[176,178] As a result of the loss of interproximal contact, food traps may occur, leading to infrabony defects at the proximal surfaces of teeth and also the adjacent implants.[175-177] Therefore, having screw-retained restorations allows easy retrieval of the prosthesis when seeking ways to correct the open interproximal contacts if needed. Likewise, having an occlusal guard might be beneficial in maintaining interproximal contacts between teeth and restorations in the arch.[172]

Conclusion: Evidence suggesting that occlusal overloading or opening of interproximal contacts is a risk factor/indicator for peri-implantitis is limited.

3.5 | Clinician-related factors: Spatial positioning of implants

The spatial position of a dental implant within bone impacts the long-term function and esthetics of the implant restoration. It influences the preservation of peri-implant hard and soft tissues, emergence profile, prosthetic contour, the angle at which occlusal forces hit the fixture, the ability to use retrievable screw-retained restorations, and, most importantly, facilitates effective plaque control to prevent peri-implant mucosal inflammation.[162,179,180] A malpositioned implant has a high probability of leading to peri-implantitis (odds ratio of 48.2).[165] This is because a malpositioned implant not only violates physiological hard

and soft tissue boundaries, it also results in poorly contoured restorations that are impossible to clean (Figure 15). The accumulation of plaque will result in significantly greater submucosal microbiome dysbiosis, consequently increasing the site's susceptibility to peri-implantitis.[181]

Advances in technology have enhanced the accuracy and precision of surgical implant placement so that clinicians can place the implants in prosthetically driven positions. With digital workflows, patients can have digital impressions, three-dimensional imaging and implant treatment planning, milled or three-dimensional printed surgical guides, and the implant placement surgery all done in one visit. The implant surgeries can be performed in a more precise and accurate manner with static or dynamic guides in partially or fully guided surgeries compared with freehand surgeries.[180] Fully guided surgeries with a flapless approach are minimally invasive and hence reduce patient morbidity and increase patient satisfaction. This protocol also had the greatest accuracy[182] with the least horizontal apical deviation and angular deviation.[183] However, more studies are needed to further validate this approach because potential errors can occur during the acqusition, transfer, and superimposition of the images and also during the fabrication of the guides.[184,185]

A recent randomized controlled trial compared the accuracy of implant placement in pilot drill–guided, fully guided, and free-handed surgeries in partially edentulous cases. The study showed that fully guided surgeries were significantly more accurate as the apical global deviation was 0.97 mm (1.43 mm for pilot drill–guided surgery and 2.11 mm for free-handed surgery). The time spent on treatment planning was comparable between pilot drill and fully guided surgeries (23.73 minutes pilot drill guided vs 21.40 minutes fully guided), and approximately 18 minutes of surgical time was saved with guided surgeries compared with the free-handed surgery. The cost analysis revealed that fully guided surgeries were the most efficient after considering time invested and surgical accuracy.[186,187] A meta-analysis of 20 clinical trials using static guides revealed a total mean error of 1.2 mm and 1.4 mm at entry and apical point with an angular deviation error of 3.5°, proving the accuracy of such guides in implant placement.[188] In addition, guided surgery minimizes the effect of the surgeon's experience[189] to ensure optimal placement of the implant.

FIGURE 15 Poor implant positions resulted in unseated crowns and excess cement [Colour figure can be viewed at wileyonlinelibrary.com]

As surgical protocols continue to be refined, dynamic navigation during implant placement might positively impact implantology as it provides real-time feedback and adaptability to clinical situations. Nonetheless, it is important to recognize that dynamic navigation is surgically challenging and technically demanding, with advanced training needed prior to its use.[190]

Conclusion: There is a strong evidence suggesting that poor spatial positioning of a dental implant is a risk factor/indicator for peri-implantitis.

4 | CONCLUSIONS

Bacterial plaque is a known etiological agent of peri-implantitis. The microbial insult must be prevented so that peri-implant inflammation is minimized. In order to do that, the following measures are recommended.

Individuals with dental implants should:

1. Perform effective plaque control daily via toothbrushing and interdental cleaning aids (preferably using an interdental brush).
2. Have professional mechanical debridement performed at regular intervals (eg, once every 3 months) to reduce the submucosal microbial load.
3. Live a healthy lifestyle by maintaining good glycemic control and avoiding smoking.

Clinicians should:

4. Ensure periodontal health by removing active periodontal inflammation prior to implant therapy and control aberrant host response by smoking cessation and encouraging a healthy lifestyle to patients as necessary.
5. Create a band of thick, keratinized, and attached peri-implant tissue through soft tissue grafting when indicated to protect the underlying crestal bone and facilitate plaque removal.
6. Have an implant with inbuilt platform switching placed in a prosthetically driven position with proper implant treatment planning and guided surgical protocols to facilitate the fabrication of a cleansable screw-retained implant restoration that mimics natural tooth contours (emergence angle of 30° or less) with light occlusal contacts.

CONFLICT OF INTEREST

The authors reported no conflicts of interest related to this work.

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How to cite this article: Fu J-H, Wang H-L. Breaking the wave of peri-implantitis. *Periodontol 2000*. 2020;84:145-160. https://doi.org/10.1111/prd.12335