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REVIEW ARTICLE

Peri-implantitis—Is it mainly a clinician-initiated complication of implant therapy?

Stephen T. Chen[1,2] | Alberto Monje[3,4,5] | Franz J. Strauss[6,7,8] | Frank Schwarz[9] | Andrea Roccuzzo[10,11] | Irena Sailer[12] | Dennis Tarnow[13] | France Lambert[14,15] | Sascha Jovanovic[16,17] | Daniel Buser[18,19]

Correspondence
Stephen T. Chen, Melbourne Dental School, Faculty of Medicine, Dentistry and Health Sciences, The University of Melbourne, Vic 3010, Australia and Melbourne Periodontal Specialists, Balwyn, Vic 3103, Australia.
Email: schen@periomelbourne.com.au

Abstract

Aim: The high prevalence of peri-implantitis is concerning, with a growing consensus that the majority of cases are complications initiated by clinician-related errors rather than classic pathology. A primary predisposing factor for peri-implantitis is exposure of the micro-rough implant surfaces to the peri-implant sulcus after treatment.

Objectives: To identify surgical/prosthetic factors causing micro-rough surface exposure and advocate for prevention and evidence-based protocols.

Materials and Methods: This paper reviews evidence linking surgical and prosthetic errors to micro-rough surface exposure to the sulcus and subsequent peri-implantitis development.

Results: Surgical factors for surface exposure include malposition, avascular necrosis, and incomplete bone regeneration of peri-implant defects. Prosthetic factors include cement remnants and wide prosthetic emergence angles. Clinician-influenceable co-factors—including patient compliance, history of periodontitis, uncontrolled systemic factors/habits, lack of keratinized mucosa, prosthetic misfit, overcontoured/uncleansable prostheses, failure to detect early bone loss or mucosal changes, and inadequate maintenance—contribute to the initiation and progression of peri-implantitis once micro-rough surfaces are exposed.

Conclusions: Most peri-implantitis cases are preventable complications. Professional education and research must prioritize identifying clinician-related errors and adherence to foundational treatment principles. This requires a paradigm shift toward a complication-based model of peri-implantitis.

Clinical Relevance: To reduce the risk of peri-implantitis, clinicians should avoid clinical errors that lead to exposure of the micro-rough implant surface. Hybrid surface designs and subcrestal micro-rough surface placement should be considered as safety buffers. Success depends on meticulous diagnostics and planning, proper

For affiliations refer to page 24.

surgical/prosthetic execution, cleansable prosthesis design, and proactive maintenance to detect early bone loss and soft tissue changes.

KEYWORDS
clinician-related errors, hybrid implant design, iatrogenic complications, micro-rough implant surfaces, peri-implantitis, prosthodontic errors, surgical errors

1 | INTRODUCTION

Peri-implantitis is a plaque related pathological condition occurring in the tissues surrounding a dental implant, characterized by inflammation of the peri-implant mucosa and bone loss that commences in the crestal area, with a tendency to progress over time.[1] The presenting clinical signs include erythema, swelling, bleeding, suppuration, and deepened peri-implant pockets. It is a significant dental pathology, causing chronic ulceration of the pocket wall of the peri-implant mucosa, a densely infiltrated connective tissue zone in the adjacent mucosa, and resorption of the crestal bone.[2] The crestal bone shows active signs of resorption[3] and a lympho-plasmocytic infiltrate is found within the bone marrow.[2] Peri-implantitis can ultimately lead to loss of the implant. Radiographically, the bone defects have a semilunar or crater-like shape around the neck of the implant.[4]

The general consensus is that peri-implantitis is initiated by inflammatory processes caused by microbial biofilm accumulation on an implant surface within the peri-implant sulcus.[1] Another school of thought is that peri-implantitis is initiated by a foreign body reaction to the titanium material of the implant when there is loss of an immune-mediated foreign body equilibrium.[5] Irrespective of the initiating factor, the clinical presentation is an infection of the peri-implant tissues associated with a submucosal plaque composed of a mixed and variable microflora dominated by gram-negative anaerobic bacteria.[6]

The reported prevalence of peri-implantitis is alarming. Notwithstanding the arguments and controversies in establishing agreement on diagnosing peri-implantitis, the 2017 World Workshop arrived at a consensus on diagnostic criteria that can be used in clinical practice and epidemiological research.[1] Adopting the criteria of the 2017 World Workshop, a recent systematic review reported that the prevalence of peri-implantitis was 25.0% (CI: 21.1–29.3%) of patients and 18.0% (CI: 15.8–20.5%) of implants.[7]

These prevalence figures are unacceptably high, and seem to be tolerated or even normalized by the dental community. One reason for this may be the way in which peri-implantitis has been framed. Since the late 1990s, peri-implantitis has been characterized as a disease entity driven predominantly by patient-related risk factors. This has shaped the dental community's perception of peri-implantitis and has driven the direction of research. An alternative perspective is that, rather than a disease, peri-implantitis should be viewed primarily as a complication of implant therapy, and therefore a "man-made" condition due mainly to human error.[8]

In a recent retrospective study, it was reported that plaque-induced and clinician-triggered peri-implantitis are different entities associated with distinctive predictive profiles.[9] This study identified 207 healthy implants and 125 implants affected by peri-implantitis in 56 patients. The triggers for peri-implantitis were reported to be clinician-related surgical (40.8%) and prosthetic (30.4%) factors. Thus, the majority of cases of peri-implantitis (71.2%) were identified to have been a result of clinician-related factors. Only a minority of implants with peri-implantitis (28.8%) were purely plaque-induced.[9] Recent scientific consensus increasingly points to clinician-related factors as a significant driver for peri-implantitis.[10–12]

The authors of this review contend that the majority of cases of peri-implantitis arise from clinician-related errors. In these cases, peri-implantitis should be regarded as a complication of therapy rather than a disease in the classical sense. This conceptual shift does not dispute the role of microbial or host-related mechanisms, but reframes peri-implantitis within an iatrogenic context. A primary predisposing factor is the exposure of the micro-rough implant surface to the peri-implant sulcus, which facilitates biofilm contamination.[13] Given the increasing evidence linking errors in diagnosis, surgery, and prosthetic management to early crestal bone loss,[10] this paper aims to identify the surgical and prosthetic factors that cause the exposure of the micro-rough surface, with a primary focus on the role of human error.

2 | PERI-IMPLANTITIS—A DISEASE OR A LATE COMPLICATION FOLLOWING IMPLANT THERAPY?

For over three decades, peri-implantitis has been regarded as a disease with clinical features similar to periodontitis. The term peri-implantitis is purported to have first been used in the dental literature in 1987,[14] and was then formally adopted at the first European Workshop on Periodontology.[15] The term "peri-implant disease" has been widely used and formally adopted in numerous consensus conferences, classification workshops and treatment guidelines, for example, the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases,[1] the Prevention and Treatment of Peri-implant Diseases—The EFP S3 Level Clinical Practice Guideline,[16] and the Risk for Peri-implant

Diseases and Defects: Report of Workgroup 1 of the Joint AO/AAP Consensus Conference.[17]

Thus, "peri-implant disease" is now a well-established term in the dental implant scientific lexicon. Peri-implantitis is undoubtedly an inflammatory condition that results in diseased peri-implant tissues; however, regarding it as a "classic" disease carries implications for how dentists and patients perceive the condition. Words and terms have a framing effect on the preferences, perceptions and attitudes of individuals.[18] When a condition is framed as a disease, the tendency is to attribute the risk factors primarily to the patient. For example, type II diabetes mellitus (T2DM) is a chronic disease with a strong hereditary basis. Prevention and management strategies focus on identifying and mitigating patient-related risk factors such as obesity, diet and lifestyle issues. Practitioner-related factors or health system-related barriers leading to therapeutic inertia are often not considered when discussing effective management of T2DM.[19,20] Similarly, when peri-implantitis is framed as a disease, the perception of most dentists and patients is that it is a condition primarily related to patient risk factors, such as smoking, diabetes, periodontitis and poor homecare. Human error and the role of the dentist are often overlooked, despite current guidelines emphasizing that primordial prevention strategies should incorporate clinician-related factors including implant positioning and prosthesis design.[16] Compounding this is the relatively long lag between initial implant therapy and the manifestation of peri-implantitis[21] which can lead to an information/memory gap or lack of access to treatment data,[22] potentially obscuring the role of clinician-related factors that may have predisposed the development of peri-implantitis.

On the other hand, a complication is an undesirable and unintended event that arises as a direct result of an operation.[23] Even if the complication develops months or years after the procedure, it may still be regarded as a complication if it can be established that the processes were initiated during the original treatment. Framing peri-implantitis as a complication rather than a disease requires clinicians to evaluate whether there were factors established at the time of implant therapy that predisposed the patient to later developing peri-implantitis. This transition to a complication-based model of peri-implantitis requires a paradigm shift in the mindset of clinicians.

It seems that framing an infection of the tissues surrounding an implanted device as a disease may be peculiar to dentistry. In orthopedics, for example, prosthetic joint infection (PJI) is universally regarded as a complication[24] and rarely described as a disease.[25] Infective conditions associated with percutaneous devices such as bone anchored hearing aids, external fixation devices, and catheters are also considered complications and not disease entities.[26–28]

In a consensus conference in 2012, implant and clinician-related factors that may contribute to crestal bone loss (CBL) around implants were identified, such as surgical and prosthetic experience, skills, materials, implant surface characteristics and design and foreign body reactions caused by corrosion byproducts and excess cement in soft tissues.[29] Since then, increasing attention has been directed toward human factors, such as clinician-related errors, as well as device or implant-related factors, as important considerations when discussing the etiology of peri-implantitis.[10–13,30]

3 | A PRIMARY PREDISPOSING FACTOR—THE MICRO-ROUGH IMPLANT SURFACE EXPOSED TO THE PERI-IMPLANT SULCUS

A central and often overlooked risk factor for peri-implantitis is the exposure of the micro-rough implant surface to the peri-implant sulcus, which the authors of this review consider a primary predisposing factor for this condition. Bacterial biofilm that forms on a micro-rough surface exposed to the peri-implant sulcus invokes an inflammatory response in the peri-implant mucosa.[31] Compared to machined surfaces, micro-rough surfaces have significantly greater bacterial adhesion[32] and trigger a stronger immune response.[33] Once the exposed micro-rough implant surface becomes contaminated with biofilm, a susceptible individual may subsequently develop peri-implantitis. The pathogenesis was demonstrated in a recent preclinical study, which investigated the impact of plaque accumulation on exposed micro-rough implant surfaces in a buccal dehiscence defect. Compared to the non-dehiscence controls, there was clinically more peri-implant mucosal inflammation, and histomorphometrically more CBL and greater apical extension of the inflammatory infiltrate in the dehiscence group.[34]

In the following sections, the factors causing exposure of the micro-rough implant surface to the peri-implant sulcus will be discussed (Figure 1). Subsequently, the role of co-factors such as patient susceptibility and compliance, prosthesis design, and maintenance protocols will be discussed in relation to their contribution to the development and progression of peri-implantitis when a micro-rough surface is exposed.

3.1 | Implant surfaces and hybrid implant designs

An exposed micro-rough implant surface to the peri-implant sulcus is a primary predisposing factor for peri-implantitis. The surface characteristics of the implant and the location of the roughened implant surface on the body of the implant are therefore important considerations.

3.1.1 | Implant surfaces

The discussion of implant surface characteristics goes back to the early days of modern implant dentistry (for review, see Buser et al. 2017)[35] with the two pioneers of modern implant dentistry, Prof. P.I. Brånemark from the University of Gothenburg, Sweden and Prof. André Schroeder from the University of Bern, Switzerland. The original Brånemark implants (Nobel Biocare Services AG, Kloten, Switzerland) from the late 1960s were made of commercially pure (cp) titanium and had a machined implant surface.[36] They were

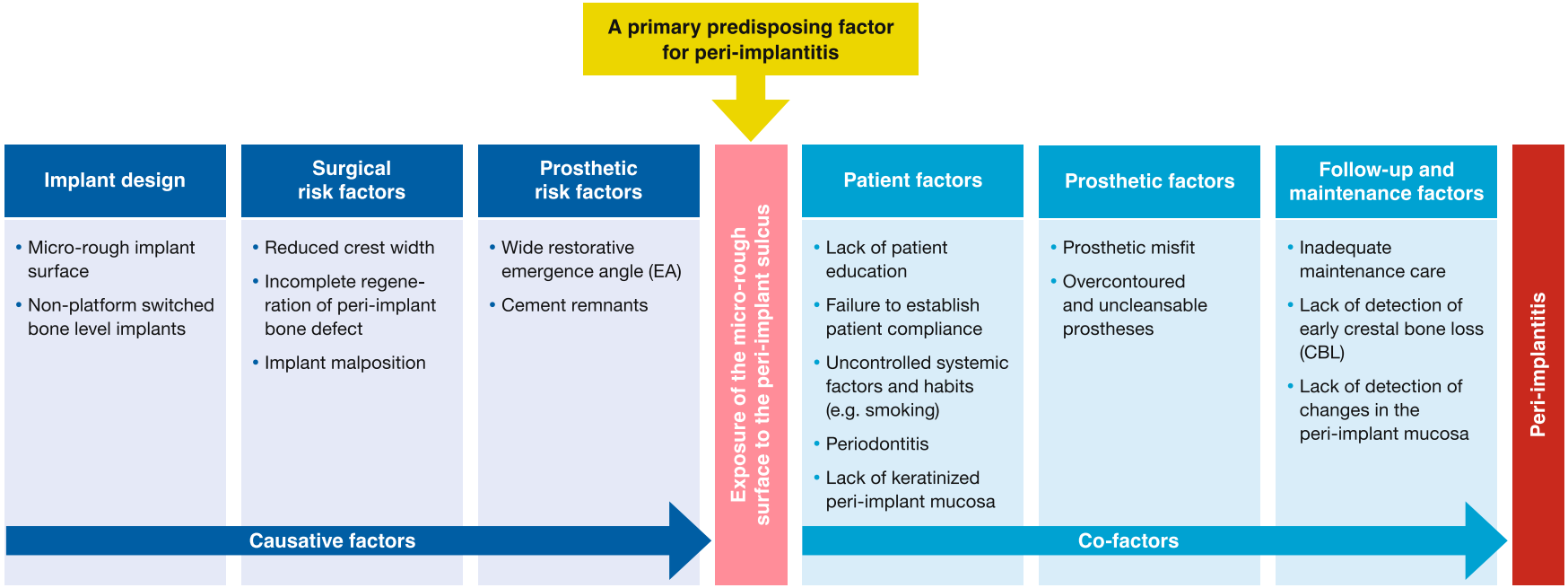

FIGURE 1 Factors that may cause exposure of the micro-rough implant surface to the peri-implant sulcus, and co-factors for peri-implantitis.

primarily used for the rehabilitation of fully edentulous patients, with the implants placed into healed alveolar ridges.[37,38] Interestingly, in their first reports of up to 15 years of experience, the authors did not report on peri-implant infections causing peri-implant bone loss. In contrast, the original implants by the team of Schroeder tested cp titanium implants with a roughened surface in preclinical studies[39,40] and in fully edentulous patients.[41,42] These implants are often referred to today as the first-generation Straumann implants (Straumann Group, Basel, Switzerland). These 1-piece implants featured a micro-porous titanium plasma-sprayed surface (TPS) extending up to the implant shoulder, resulting in a rough surface within the transmucosal area. In addition, they were only placed in healed sites utilizing a non-submerged healing approach. The clinical experience in the early 1980s showed that many patients developed peri-implant soft tissue infections, often combined with progressive bone loss[14]; a pathology which today would be referred to as peri-implantitis. In 1986, the Schroeder team and Straumann AG developed the second generation of implants, which were two-piece implants with a tulip shape in the shoulder region. To avoid the risk of peri-implant infection, the micro-porous TPS surface was only used in the endosteal portion of the implant for bone anchorage, whereas a Brånemark-type machined surface of 2.8 mm in height was utilized in the supracrestal transmucosal area.[43,44] Later, these Straumann implants were referred to as Tissue Level (TL) implants. In the early 1990s, several preclinical studies examined new micro-rough implant surfaces to improve osseointegration and to accelerate the bone healing process.[45–48] These studies lead to a paradigm shift toward micro-rough implant surfaces in the dental implant market.

3.1.2 | Implants with a hybrid surface design

The shift toward micro-rough implant surfaces gained traction throughout the 1990s and 2000s. In 1993, Dennis Tarnow published a paper that supported the shift to micro-rough implant surfaces, but he argued that a micro-rough implant surface all the way up to the implant shoulder would cause an increased risk for the development of peri-implantitis. He recommended a 'hybrid design' for future implants, featuring a micro-rough surface in the endosteal portion for improved bone anchorage and a smooth machined surface in the transmucosal portion of the implant,[49] similar to the concept developed for the TL implants. Around the millennium change, all major implant companies had shifted to implants with a micro-rough implant surface, with either a hybrid or non-hybrid surface design.

The prediction of Tarnow was confirmed in a 9-year study based on data from the Swedish implant registry.[21] The early and late failure rate analysis showed the lowest failure rate of 1.2% was for implants with a hybrid surface design (TL implants of Straumann AG), whereas implants with a non-hybrid surface design (Astra Tech, Mölndal, Sweden and Nobelbiocare, Gothenburg, Sweden) showed higher failure rates of 3.7% and 4.0%, respectively. This difference was also demonstrated in two 10-year studies.[50,51] Both studies not only analyzed the failure rate, but also the prevalence of peri-implantitis. The 10-year study of 1482 non-hybrid, Astra Tech implants in fully and partially edentulous patients showed a failure rate of 5.3% and a peri-implantitis prevalence of 11.8%. Hence, the overall 10-year complication rate for these non-hybrid implants was 17.1%.[50] The authors concluded that early (CBL) during healing exceeding 0.5 and 1.0 mm leads to the exposure of the micro-rough implant surface to the peri-implant sulcus, which was considered by the authors a predictor for the development of peri-implantitis. In contrast, a 10-year study by Buser and co-workers of 303 patients with 511 hybrid Straumann TL implants, all inserted in partially edentulous patients, showed a failure rate of 1.2% and a peri-implantitis prevalence of 1.8%. Hence, the overall 10-year complication rate for these hybrid TL implants was 3.0%.[51] Consequently, the success rate was 97.0%, and the survival rate 98.8%, applying the well-established success criteria utilized at the University of Bern for all long-term clinical studies with dental implants.[52] This observation is further supported by two long-term retrospective studies. In the first study of 382 original Brånemark implants with a machined implant surface and a follow-up time between 13 and 32 years, the reported failure rate of 2.3% and the prevalence of peri-implantitis of 1.2% were both very low.[53] In contrast, a study with 223 non-hybrid implants with an anodized, micro-porous surface showed a failure rate of 4.5% and a peri-implantitis prevalence of 33% after an average follow-up time of 10.4 years (range 5 to 17 years).[54]

In a currently unpublished follow-up of the study of Buser and co-workers in 2012,[51] 159 of the original patients with 252 TL implants were available for a 25-year examination (submitted for publication).[55] The clinical and radiographic examination resulted in a success rate of 90.8% and a survival rate of 94.0% at 25 years of follow-up. Two findings were most interesting. Firstly, 60% of the additional implant failures showed a loss of osseointegration without signs of peri-implant infection, most probably due to occlusal overload. Secondly, despite a high prevalence of implants with peri-implant mucositis, these implants did not develop into peri-implantitis between the 10-year and 25-year examinations. This confirms again the low risk for the development of peri-implantitis in TL implants with a hybrid surface design, when other surgical factors are under control, such as correct implant positioning, predictable bone regeneration of peri-implant bone defects and the presence of keratinized mucosa (KM). In these two studies, the implants were inserted with the micro-rough SLA implant surface (Straumann Group, Basel, Switzerland) slightly below the bone crest (subcrestally) up to 1 mm, to reduce the risk of exposure of the SLA implant surface during early bone healing. Later, this insertion technique became the standard of care not only for TL, but also for non-hybrid, bone-level (BL) implants.[56] The excellent long-term stability of hybrid, TL implants has been confirmed in several other long-term studies.[57–59]

Based on these observations over many years, the authors of this review strongly recommend that implant surgeons consider utilizing dental implants with a hybrid surface design. Every effort needs to be made to avoid exposure of the micro-rough implant surface to the peri-implant sulcus to reduce the risk for the development of peri-implantitis.

3.1.3 | Implants with platform-matching and platform-switching designs

The original Brånemark implants and the first and second generation Straumann implants both had platform-matching prosthetic connections, meaning that the implant restoration was directly on the implant shoulder. Bone remodeling with a CBL of 2–3 mm was routinely seen with these implants. This phenomenon was often called bone saucerization[60] caused by circumferential bone resorption at the neck of the implant. This phenomenon was explained by two potential causes. Firstly, a microgap was present at the implant-abutment interface. Since the epithelium always showed a proliferation apical to this microgap, there was resorption of the crestal bone to provide space for the connective tissue with its blood vessels. The dimension of this biologic reaction was referred to as the biologic width and was examined in various preclinical studies.[61–63] This microgap harbored bacteria and caused a chronic infiltrate at the interface level due to bacterial microleakage.[64,65] Secondly, the stability between the implant and the restoration plays an important role as well. The external hex connection of the original Brånemark implant was associated with micromovement and CBL.[66,67]

In the early 2000s, a new implant-abutment interface was introduced for bone-level implants—the platform-switching (PS) concept,[68] with the goal of reducing the amount of CBL. PS refers to the use of an abutment that is smaller than that of the platform of the implant. It has been shown in a preclinical study[69] and in systematic reviews that PS, combined with a stable internal conical abutment connection and tight abutment fit, can minimize CBL during healing and function.[69–72] When using bone-level implants, clinicians should select PS systems specifically designed to minimize CBL.

3.2 | Surgical risk factors

From a surgical perspective, the therapeutic goal is to achieve a circumferential anchorage of the implant within the alveolar bone at completion of bone healing. This should not only provide sufficient osseous anchorage to sustain the anticipated functional load of the implant during masticatory function, but also provide a protective mechanism against bacterial contamination of the micro-rough implant surface.[73] The following section will discuss surgical factors that can lead to early CBL, including reduced crest width, incomplete bone regeneration, and implant malposition.

3.2.1 | Reduced crest width

When a dental implant is placed into an alveolar ridge that is reduced in width buccolingually, the vascular supply to the cortical bone in the crestal region may be compromised, particularly in open-flap surgical approaches. Implants placed in healed sites must have an adequate lingual and buccal bone wall thickness (BBT) to ensure that the implant is circumferentially embedded in bone at completion of bone healing. Most often, the buccal aspect is the critical side of the alveolar ridge, since post-extraction ridge alterations lead primarily to ridge atrophy on the buccal side.[74] Once initial bone healing and remodeling have taken place, the entire micro-rough implant surface must be osseointegrated and circumferentially covered by vital bone as outlined above. A seminal clinical study with re-entry data showed that the buccal bone was intact at second-stage surgery when the BBT was ≥1.8 mm at the time of implant placement. On the other hand, when BBT was approximately 1.2 mm at implant placement, >3 mm of vertical bone loss was noted at surgical re-entry.[75] Similarly, Nohra et al. showed that implants presenting BBT <2 mm at baseline exhibited 8x and 12x greater vertical CBL at the proximal and buccal aspects, respectively, when compared to implants displaying BBT ≤2 mm.[76] Monje et al. reported in a preclinical model that avascular necrosis occurred when the BBT following preparation of the osteotomy was less than 1.5 mm.[77] This is a phenomenon that occurs as a consequence of damage to the alveolar bone.[78,79] The alveolar process is composed of cortical and cancellous bone in the outer and inner portions, respectively. The cortical bone receives its blood supply from the outside through the blood vessels of the periosteum and from the inside through the endosteum.[80] Therefore, when an implant is inserted with an open-flap procedure, the blood supply from both sources is disrupted.[79] Following implant placement, avascular necrosis starts 12 h after disruption of the blood supply, when the hematopoietic cells that are particularly sensitive to low oxygen levels die. This may lead to the death of bone cells, leading to more noticeable osteoclastic activity.[81] CBL and mucosal recession are thus attributable to this process. This hypothesis was further supported by another preclinical study that demonstrated that thin buccal bone walls (<1 mm) were associated with greater vertical bone loss and implant surface exposure, whereas thick walls (>1.5 mm) preserved the buccal bone better and protected the implant surface.[82]

Clinically, progressive CBL may be radiographically detected during the healing phase, usually in the presence of healthy peri-implant mucosa (Figure 2A–C). The risk is that the exposed implant surface within the supracrestal soft tissues may be susceptible to biofilm accumulation and the development of peri-implant infection (Figure 3A–C). As will be discussed in a later section, buccally positioned implants are prone to avascular necrosis and CBL if the buccal bone wall is <1.5 mm at the time of placement (Figure 4A–C). Typically, the pattern of the peri-implant bone defect as a consequence of avascular necrosis, CBL, and subsequent peri-implantitis is a dehiscence of the facial bone wall and angular bone defects mesially, distally, and palatally (Figure 5A,B). The remaining buccal bone in the apical region of the implant is usually thin. If the lingual bone is <1.5 mm at the time of placement, there will also be a dehiscence of the lingual bone wall as well (Figure 6).

In the opinion of the authors of this review, avascular necrosis resulting in early CBL is a significant risk factor for peri-implantitis. Placing implants in ridges with insufficient crestal width without appropriate augmentation represents a high-risk surgical decision

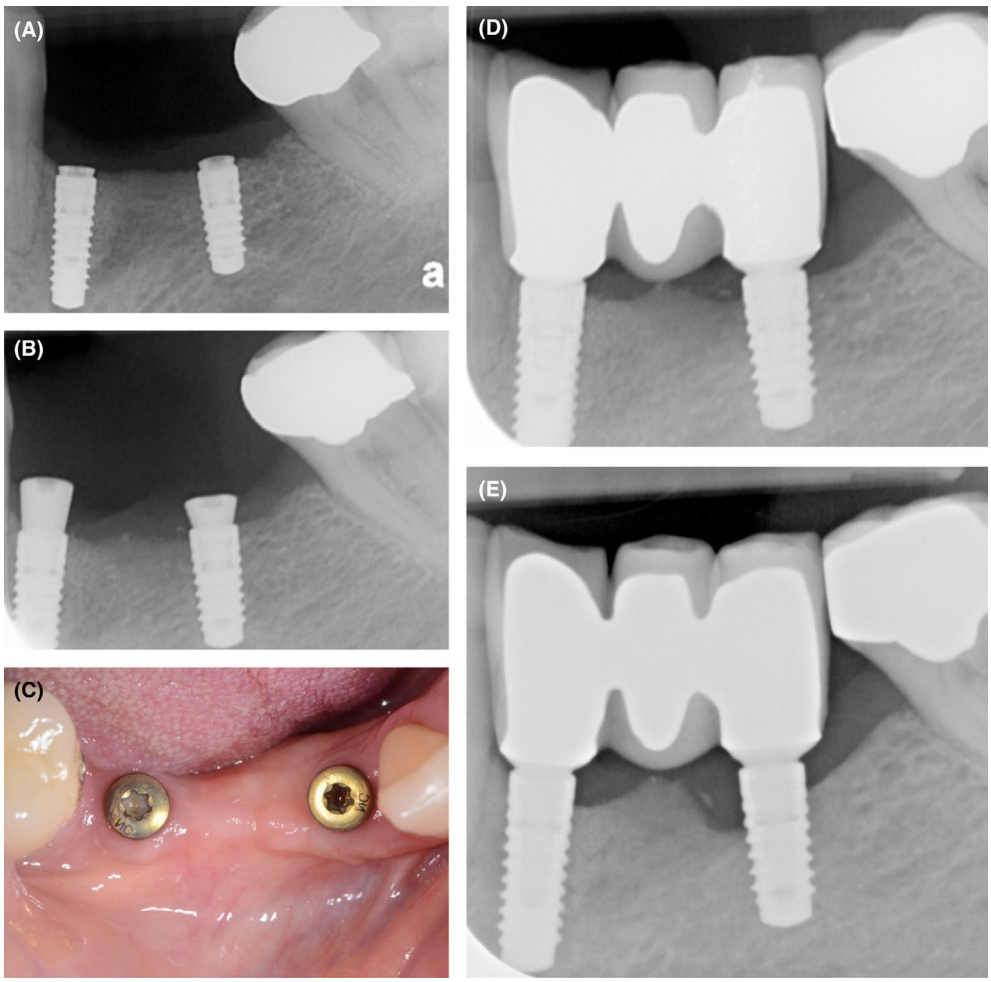

FIGURE 2 (A) The immediate post-operative radiograph of two reduced-diameter bone-level implants placed in a mandibular ridge with reduced crest width (mandibular left canine and second premolar sites). (B) Post-operative radiograph of the implants after connection of transmucosal healing abutments 3 months later. Note the crestal bone loss already apparent on the mesial aspect of the second premolar implant. (C) Clinical image (mirror view) of the two implants 4 weeks after connection of the transmucosal healing abutments. Note the healthy peri-implant mucosa. The reduced crest width at the distal implant in the second premolar site is apparent. (D) Radiograph of the implants after restoration (7 months after surgical placement). Notwithstanding the prosthetic design with the wide emergence angle of the abutments, further crestal bone loss has occurred in the absence of clinical signs of inflammation of the peri-implant mucosa. (E) Two years later, additional crestal bone loss has occurred around the necks of both implants. The patient had been seen for regular supportive maintenance care and maintained good plaque control. The peri-implant tissues were free of inflammation. The progressive crestal bone loss in the ridge with reduced crest width may be attributed primarily to avascular necrosis.

associated with an increased likelihood of early crestal bone loss. This is often a well-intentioned but erroneous decision made to minimize the surgical burden on the patient by avoiding a simultaneous or staged bone augmentation procedures.

3.2.2 | Various bone grafting procedures to regenerate bone defects in implant sites

Dental implants are placed either in post-extraction sites or in sites that have healed. These sites may have adequate bone volume for implants, or exhibit varying degrees of horizontal and/or vertical bone atrophy.

In the pioneering phase of modern implant dentistry that spanned the 1970s and 1980s, implants were primarily inserted into healed sites. The protocol was to place implants after a healing period of 6 months to allow the alveolar ridge to heal. Many cases presented for treatment years after the initial extraction. Bone atrophy was very often present at these sites, most frequently in a horizontal direction on the buccal aspect of the ridge, but in some cases, it was combined with vertical ridge atrophy.

In the late 1980s, various surgical techniques for post-extraction implant placement were developed to shorten the treatment time between extraction and implant placement, and to overcome the problem of advanced resorption of the alveolar ridge. Post-extraction implant placement began increasingly to dominate daily practice

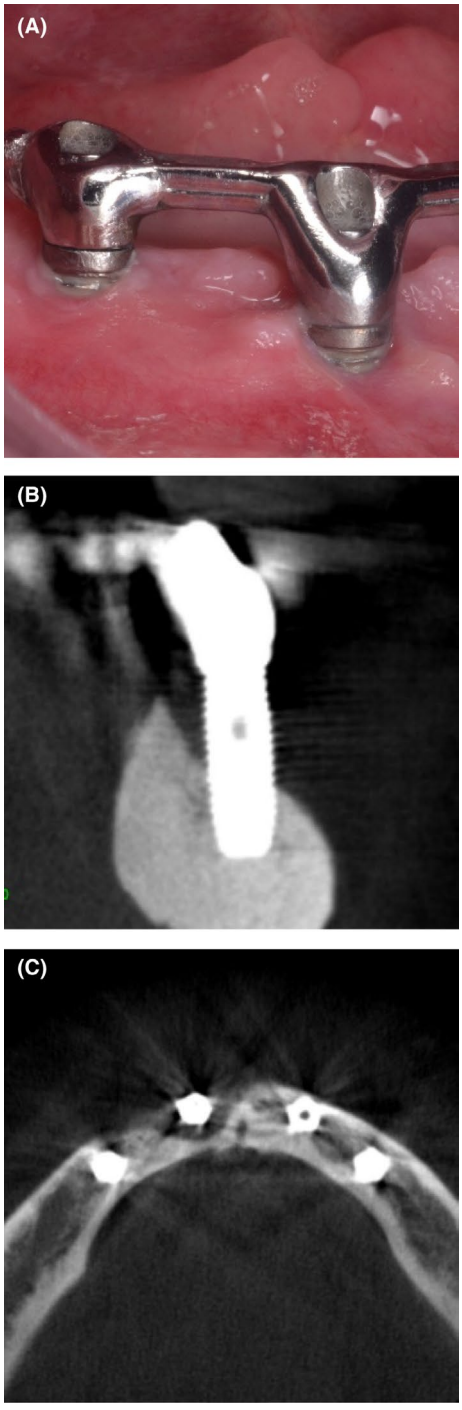

FIGURE 3 (A) One of the mandibular implants presented with pocketing, inflammation, and suppuration consistent with peri-implantitis. (B) The sagittal view of the CBCT shows the bone loss on the buccal and an angular bone defect on the lingual. (C) The corresponding axial view of the CBCT shows the relatively buccal position of the affected implant in the line of the arch, resulting in a thin buccal bone wall which subsequently underwent avascular necrosis.

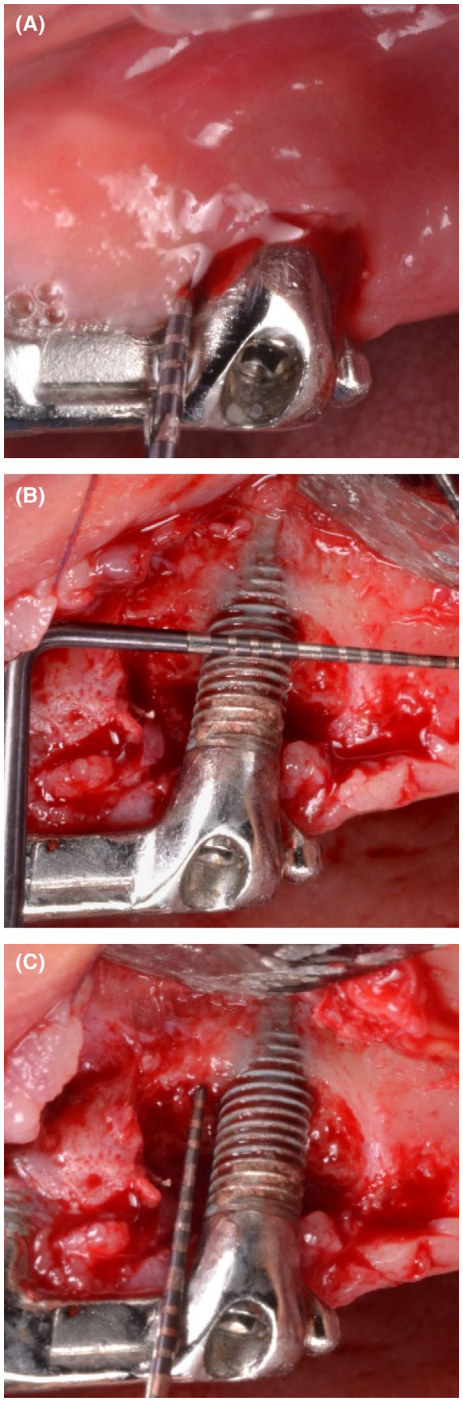

FIGURE 4 (A) This implant presented with the clinical signs of peri-implantitis—deep probing pockets, inflammation, and suppuration. (B) Following flap reflection, the pattern of bone loss can be seen. There is a dehiscence of the buccal bone wall of about 8 mm in width. (C) Bone loss on the mesial and distal has created broad crater defects. The bone loss extends to the palatal side. However, the height of the palatal bone wall was retained.

since the millennium change (for review see Buser et al.).[35] In these healing sites, a bone defect is almost always present.

Thus, bone defects are frequently encountered with implant placement, irrespective of the timing of placement after tooth extraction. Bone grafting procedures to regenerate bone in these defects are therefore an essential part of surgical implant therapy.

In all these clinical scenarios, the implant surgeon must first perform a detailed clinical and radiographic examination, most often with

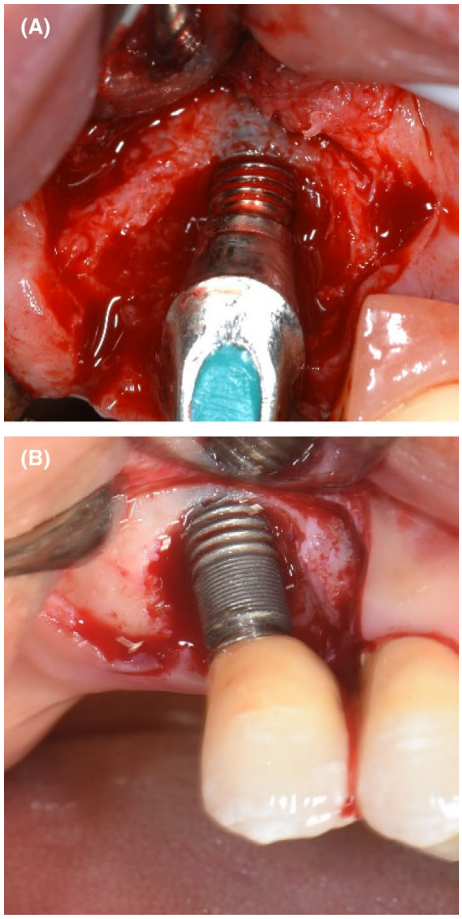

FIGURE 5 (A) The pattern of bone loss as a consequence of avascular necrosis, CBL and subsequent peri-implantitis on this implant with a machined surface is a dehiscence of the facial bone wall, and angular bone defects mesially, distally, and palatally. The palatal bone wall was present. The remaining facial bone in the apical half of the implant was thin. (B) In this implant with a micro-rough surface, the pattern of bone loss due to peri-implantitis is a dehiscence of the facial bone wall. The palatal bone wall is present. There are angular bone defects mesially, lingually, and palatally. The residual facial bone is thin.

a 3-dimensional CBCT, to characterize the local bone and soft tissue anatomy. Based on this, a surgical strategy based on the prosthetic goal is then selected to achieve complete bone regeneration of the bone defect with high predictability. Failure to completely regenerate the bone defect leads to exposure of the implant surface. If this surface is micro-rough, the risk of biofilm contamination is high, which in turn increases the risk of peri-implantitis in the future. Therefore, incomplete bone regeneration combined with exposure of the micro-rough implant surface represents a significant risk for the development of peri-implantitis. This situation must be avoided by all means. In the following sections, the various bone grafting procedures will be briefly presented in post-extraction sites and in healed sites.

Implant placement into post-extraction sites

In the past 25 years, major progress has been made in the field of post-extraction implant placement based on (a) a much better

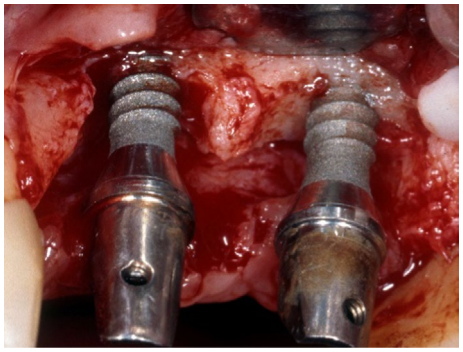

FIGURE 6 These implants with a titanium plasma-sprayed surface were placed in a ridge with reduced crest width. There is circumferential bone loss, which includes loss of both the buccal and palatal bone walls. Peri-implantitis developed secondary to CBL as a result of avascular necrosis.

understanding of the tissue biology with hard and soft tissue alterations post-extraction as shown in the landmark study of Araujo & Lindhe in 2005 describing bundle bone resorption,[83] (b) accurate 3-dimensional radiographic examination of the local anatomy by means of cone beam computed tomography (CBCT)[84] and (c) the development of digital implant dentistry allowing precise computer-assisted implant surgery,[85,86] which is important for a correct 3D positioning of the implant. A widely accepted classification proposed by the ITI—based on the ITI Consensus Conference in 2003—described immediate implant placement at the time of extraction (type 1), early implant placement with soft tissue healing (type 2), early implant placement with partial bone healing within the socket (type 3), and late implant placement (type 4).[87]

The first option is immediate implant placement (type 1), which today is a well-accepted approach based on strict case selection criteria. These selection criteria have been thoroughly discussed at two subsequent ITI Consensus Conferences in 2013 and 2023.[88–90] An important criterion is an intact buccal bone wall, and if possible, with a thick buccal bone wall phenotype (≥1 mm). A buccal gap of at least 2 mm between the shoulder of the implant and the buccal bone wall is recommended. Immediate implant placement is very appealing for patients, especially in the aesthetic zone. It is usually performed with flapless extraction of the tooth, followed by computer-aided implant surgery to place the implant and internal grafting of the gap between the implant and internal buccal and proximal socket walls with a particulate bone substitute. If the implant achieves sufficient primary stability, a provisional crown can be attached for immediate restoration.[89] This is appealing to patients as there is usually minimal post-operative pain and it reduces the number of surgical procedures by combining extraction with implant placement. However, it is a challenging procedure to undertake and is classified as a complex procedure according to the SAC classification.[91]

Many clinicians make the assumption that the gap between the implant and socket walls will completely regenerate with bone. However, two studies with re-entry data following immediate implant placement

and internal grafting with deproteinized bovine bone mineral reported that defect fill is unpredictable (Figure 7). The studies reported that the proportion of sites with complete defect fill and implant surface coverage ranged from 21% to 57%.[92,93] This unpredictability may be attributed to the relatively rapid bundle bone resorption in the case of a thin buccal bone,[94] which typically creates a dehiscence-type defect with an inverted V-shape configuration.[95] The particulate graft is then no longer protected by the buccal bone wall and may not fully regenerate with new bone. The result is crestal bone resorption, loss of support of the mucosa and risk of developing peri-implantitis (Figure 8A–C). This often presents clinically as mucosal recession, as shown in a prospective 5-year study with immediate implants, in which 47% of the implants developed a recession of 1 mm or more.[96]

The second option is early implant placement (type 2), in which the tooth is extracted, usually with a flapless approach, and the socket is left to heal undisturbed. The implant is then placed about 4 to 8 weeks later with an open-flap procedure. At this time point, the ridge presents typically with an inverted V-shape dehiscence-type defect on the buccal aspect.[97] When the implant is placed, the residual peri-implant defect typically has a 2-wall defect configuration with two intact proximal bone walls mesially and distally in the crestal region. Occasionally, the buccal bone wall is intact, which results in a 3-wall defect when the implant is placed. The regenerative potential is high when there are 2- and 3-walls present.[73,98] The defect is managed with a 2-layer composite graft consisting of autogenous bone chips locally harvested that are placed directly onto the exposed implant surface, followed by a layer of a low-substitution bone filler. The graft is protected with a resorbable collagen membrane and the flap advanced for primary closure.[56] The implant is then accessed for connection of a transmucosal healing abutment 8 to 12 weeks later, usually with a small opening using a "punch" technique.

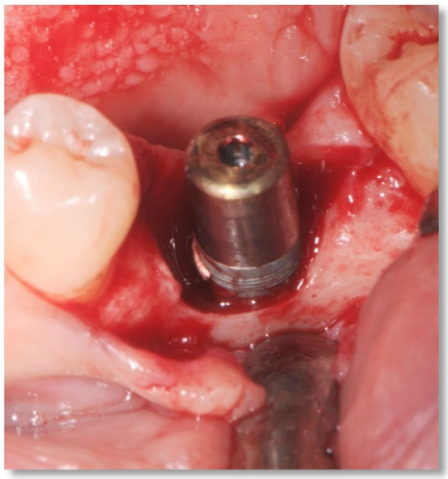

FIGURE 7 This case demonstrates that the gap between an immediate implant and the socket walls may not completely regenerate with bone. This implant developed peri-implantitis. It was placed into an extraction socket and restored with a cemented crown after integration of the implant. Note the cement remnant on the mesial aspect which is located on the endosseous surface of the implant. If bone had regenerated completely, the cement could not have been retained on the endosseous part of the implant.

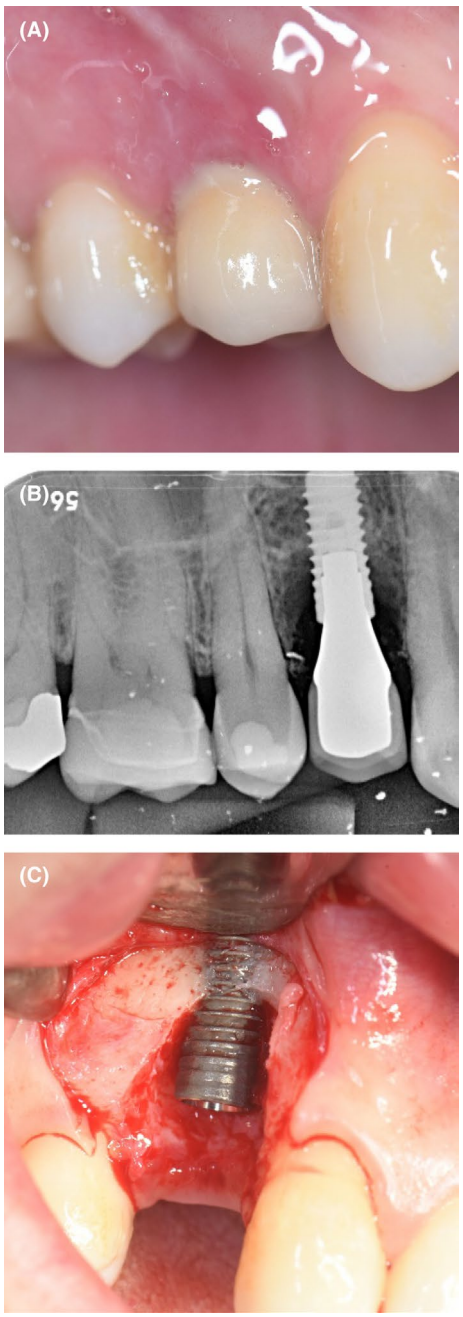

FIGURE 8 (A) The 14 implants developed peri-implantitis 6 years after placement. The implant had been placed immediately in a flapless procedure with conventional loading (Type 1C). The marginal gap had been grafted with deproteinized bovine bone mineral. (B) The periapical radiograph provided showed bone loss on the proximal aspects of the implant, and a reduction in mineral density in the bone adjacent to the defect. (C) Surgical exposure of the implant illustrated the morphology of the peri-implant bone defect. The resorption of the buccal bone wall is likely to have occurred soon after implant placement. The particulate graft was then no longer protected by the buccal bone wall and did fully regenerate, leaving the micro-rough implant surface exposed to the sulcus and subsequent peri-implant infection.

Early implant placement has been classified as an advanced procedure according to the SAC classification.[91] Long-term studies have demonstrated the efficacy of this technique to regenerate the missing bone on the facial of the implant.[59,99] However, errors with this technique can lead to incomplete bone regeneration. Firstly, there needs to be at least two intact bone walls for space maintenance to contain and protect the graft and to provide the osteogenic progenitor cells which are derived from the proximal bone walls. If only one bone wall remains, the regenerative potential is significantly reduced (Figure 9).[73] If this situation arises at the time of surgery, it is invariably the result of clinician errors resulting in an incorrect diagnosis of the dimensions of the ridge and/or not anticipating the degree of resorption likely to occur post-extraction, or inserting an oversized implant which extends buccally beyond the bone walls.

In recent years, socket grafting for alveolar ridge preservation (ARP) followed by delayed implant placement 4 to 6 months later has gained a lot of interest as a third option.[100–104] With this approach, the tooth is extracted with a flapless approach, and the socket is grafted with a low-substitution bone filler. The graft is then protected with a resorbable collagen plug or membrane or an autologous connective tissue graft.[105,106] Preclinical and clinical studies have shown that ARP is able to reduce the amount of ridge atrophy, but not completely, since bundle bone still resorbs.[100,107] The objective of this approach is not only to minimize post-extraction resorption of the ridge, but also to simplify the subsequent placement of the implant, often allowing flapless implant placement without bone grafting. This approach is therefore very appealing in posterior, non-aesthetic sites and in elderly patients. It is classified as a straightforward procedure according to the SAC classification.[91] Studies have shown that socket grafting is effective in maintaining sufficient bone for subsequent implant placement, although additional grafting at the time of placement is sometimes necessary.[108] The socket grafted alveolar ridge has been shown to be dimensionally stable over time.[109]

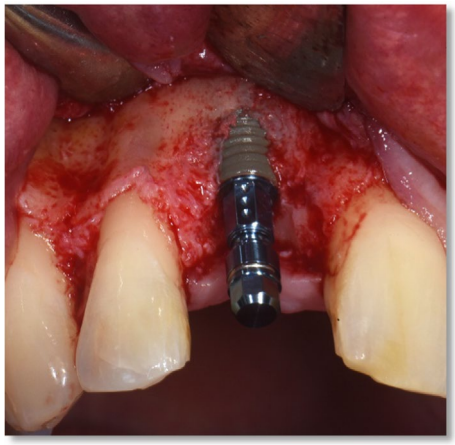

FIGURE 9 In this case of early implant placement in a resorbed ridge, the standard diameter implant extends buccally beyond the bone walls. The lack of bone walls significantly reduces the bone regenerative potential and risks incomplete bone regeneration and exposure of the micro-rough implant surface after bone healing.

Complications associated with socket grafting include post-operative infection characterized by swelling, tenderness, and suppuration, membrane exposure, and extravasation of bone graft materials which can compromise the treatment outcome.[110] There does not appear to be an increased risk of peri-implantitis in grafted compared to non-grafted sockets per se.[111] However, sockets with significant soft and hard tissue defects grafted for ARP may be at greater risk of peri-implantitis. In a recent multicentre cross-sectional study, extraction sockets were classified based on hard and soft tissue characteristics as adequate, compromised, and deficient. Deficient sites had an increased risk of peri-implantitis compared to type I and II sites.[112] Clinicians should therefore exercise caution when managing significantly compromised extraction sites with ARP procedures.

Implant placement into healed sites with various degrees of horizontal and/or vertical atrophy

Implant placement in healed sites is often referred to as late implant placement (type 4) and takes place following healing of the ridge, often years after extraction.[87] Most often, these healed sites are atrophic, most commonly in the horizontal plane, but sometimes also in the vertical plane. The surgical approach depends upon the local ridge anatomy. The most important consideration is the anticipated morphology of the bone defect if an implant is placed. Three-wall defects do not exist in healed ridges; they have either a 1-wall or a 2-wall defect morphology.[73] Vertical bone defects are always 1-wall defects, whereas horizontal bone defects can have 1-wall or 2-wall defect morphologies. The defect morphology must be assessed during pre-operative examination using a three-dimensional CBCT, since 1-wall defects have a low regenerative potential and are most challenging for the surgeon. The morphology of horizontal bone defects can be positively influenced by reducing the ridge height to increase the crest width, which is often used in posterior sites and in fully edentulous patients. Another option is the utilization of diameter-reduced implants that are placed within the bony envelope.[113,114] By doing so, a typical crater-like defect on the buccal aspect can be maintained that has a 2-wall defect morphology (Figure 10A,B).

The defect morphology is also decisive for the selection of the appropriate surgical approach, either a simultaneous or staged bone augmentation procedure. For the patient, a simultaneous approach is more attractive since it reduces the surgical morbidity and the overall treatment time, and it also reduces the treatment costs. However, the simultaneous approach should only be used when the defect morphology allows a regenerative outcome with high predictability.

In 2-wall defects, a simultaneous bone augmentation can be combined with implant insertion. The best documented and widely utilized is the guided bone regeneration (GBR) technique,[115,116] utilizing nonresorbable e-PTFE membranes[117] or resorbable collagen membranes supported by a composite graft, either as a 2-layer or a mixed composite graft.[56,118,119] In borderline cases, the sausage technique is often utilized with tacks to stabilize the membrane and, hence, improve bone filler stability.[120,121]

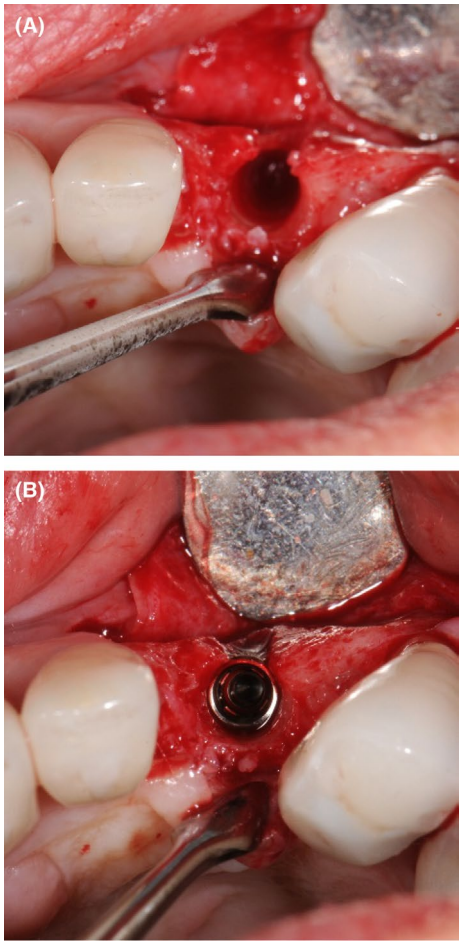

FIGURE 10 (A) In this maxillary canine site, the alveolar ridge is reduced in bucco-palatal width. The osteotomy was prepared with a 2.8 mm diameter twist drill. Note the narrow dehiscence of the buccal bone wall. If the next twist drill with a diameter of 3.5 mm was used for a standard diameter implant, the buccal fenestration would have enlarged and the buccal aspect of the implant would have been positioned outside the confines of the ridge. (B) Rather than a standard diameter implant, a reduced diameter implant (Straumann NC Bone-Level implant, Straumann AG, Basel) with a body diameter of 2.8 mm and external thread diameter of 3.3 mm was inserted. Note that the resultant buccal bone defect had intact mesial and distal bone walls (2-wall defect configuration). This defect configuration is amenable to the guided bone regeneration (GBR) technique to reconstruct the missing bone wall.

In 1-wall defects, a staged approach is most often required with horizontal and/or vertical ridge augmentation as a first step, followed by implant placement into the augmented bone in a second surgery after 6 to 9 months of bone healing (Figure 11). Various surgical techniques have been recommended such as GBR with block grafts,[122–124] GBR with reinforced or non-reinforced e-PTFE membranes,[125–128] GBR with perforated PTFE membranes,[129] customized titanium meshes[130] or the Khoury technique.[131,132] These techniques are all complex and require extensive training, surgical talent, and good clinical experience by the surgeon to achieve regenerative outcomes on a predictable basis. A significant advantage of bone augmentation with a staged approach is that the regenerative outcome can be assessed

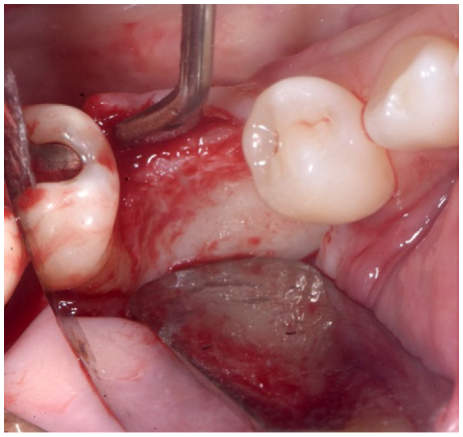

FIGURE 11 In the mandibular first molar site, buccal resorption has resulted in significant narrowing of the crest width in the coronal third of the alveolar ridge. If an implant was placed, it would be lingually positioned and the resultant peri-implant bone defect on the buccal side would have no proximal bone walls (i.e. a 1-wall defect configuration would result).

during the subsequent open-flap surgery before the implant is placed, and if necessary, can be combined with a second simultaneous GBR procedure.

In summary, the critically important pre-requisites for successful bone augmentation are a thorough pre-operative examination, the selection of the most appropriate surgical technique, and precise surgical performance[116] to ensure the ultimate goal of bone augmentation is met—achieving complete circumferential bone anchorage of the micro-rough implant surface in bone. Implants with a hybrid surface design, which incorporate a machined surface at the implant neck, provide a safety buffer for cases when bone regeneration is not fully achieved. Incomplete bone regeneration represents a failure of the bone augmentation technique and is a critical surgical error. It results in exposure of the micro-rough implant surface to the peri-implant sulcus and risks the subsequent development of peri-implantitis.

3.2.3 | Implant malposition

Malpositioned implants are due to clinician error and are associated with an increased risk of peri-implantitis.[13] At a recent joint consensus meeting of the American Academy of Periodontology and the Academy of Osseointegration, it was reported that implant malposition was a major factor which increased the risk of peri-implantitis.[11,133]

Buccal malposition resulting from lack of prosthetic planning is the most common implant positioning error,[134] and is frequently associated with dehiscences of the buccal bone and exposure of the implant surface at the shoulder region of the implant (Figure 12A–C).[135] Buccal malposition is also associated with thinning and recession of the buccal peri-implant mucosa, often causing severe aesthetic complications due to mucosal recession.[136] Due to exposure of the

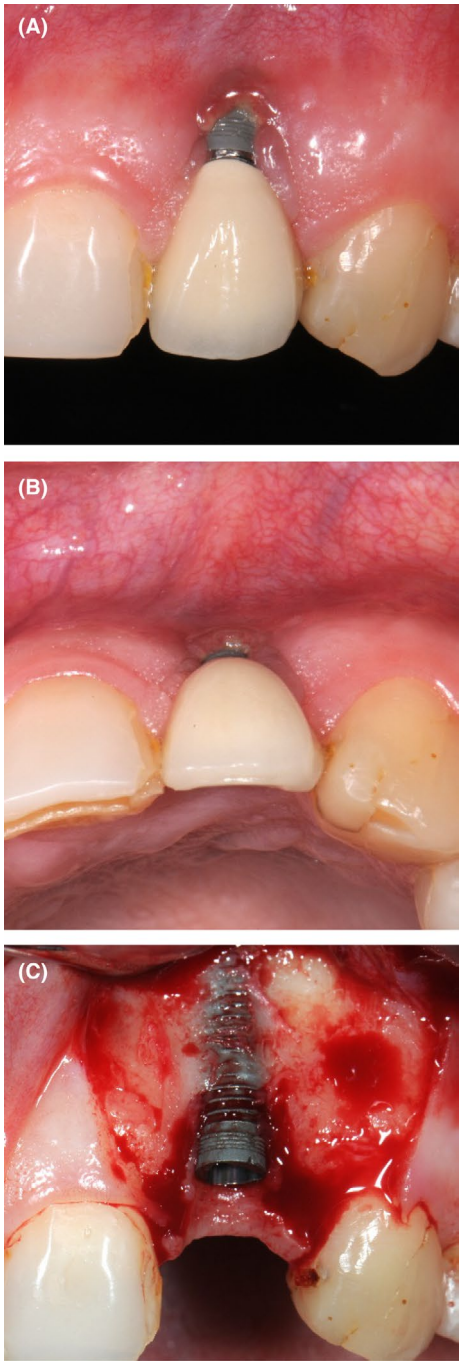

FIGURE 12 (A) The maxillary left lateral incisor implant has been placed in a buccal malposition. Note the soft tissue dehiscence, exposure of the implant surface, and inflammation of the facial mucosa. (B) Occlusal view of the implant, highlighting the buccal malposition of the implant. (C) On flap reflection, there was crestal bone loss and thinning of the residual buccal bone in the apical two-thirds of the implant. Buccally malpositioned implants are frequently associated with crestal bone loss.

micro-rough surface to the peri-implant sulcus, implant malposition in the buccolingual direction is often associated with peri-implantitis.[137] More recently, it was demonstrated that implants, when placed too far buccally either within the alveolar ridge or outside of the skeletal

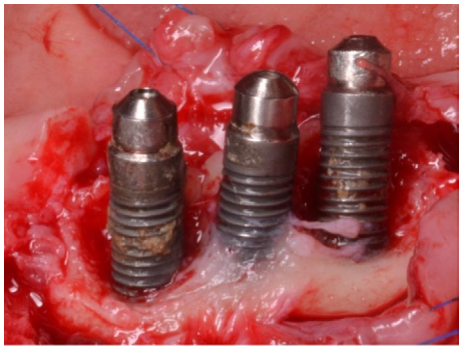

FIGURE 13 Bone loss following improper implant positioning and implant proximity leading to early bone loss and subsequent peri-implant infection.

bony envelope, have a 3x and 8x greater peri-implantitis risk when compared to the correct prosthetically-guided implant position, respectively.[30]

Implant malposition in a mesiodistal dimension can also be a risk factor for peri-implantitis.[13] A seminal study demonstrated that implants placed in close proximity to each other (<3 mm) were 8.6x more exposed to having peri-implantitis when compared to implants placed ≥3 mm apart.[138] This finding is most likely caused by vertical bone loss initiated by surgical trauma and compromised blood supply, leading to an exposed micro-rough implant surface (Figure 13). This phenomenon was also noted with implants in close proximity to natural teeth, and the bone loss may also compromise the height of the adjacent periodontal support, resulting in a reduction of the papilla height.[139] Accordingly, narrow diameter implants may be a good alternative for adjacent implants with limited mesiodistal distance, or when close to adjacent teeth. An alternative strategy for limited mesiodistal space is the use of implant-supported cantilever prostheses.

Implants malpositioned too far apically (i.e., too deep) can contribute to peri-implant mucositis[140] and peri-implantitis as well (Figure 14A,B).[141] A retrospective analysis of non-splinted restorations demonstrated that implants placed in an apical malposition ≥6 mm from the adjacent cementoenamel junctions had an 8.5x higher risk of being diagnosed with peri-implantitis.[141] In fact, a landmark study showed that the implant-abutment interface dictates the intensity and location of peri-implant inflammatory cell accumulation, as a subcrestally positioned implant-abutment microgap promoted a significantly greater invasion of neutrophils than did supracrestal interfaces.[65]

As such, implant connections with large implant-abutment microgaps may be predisposed to microleakage due to the lack of complete sealing, and whenever placed subcrestally, may trigger an inflammatory response that may ultimately initiate peri-implantitis. To overcome these situations, either the use of implants with a hybrid surface design or bone-level implants with tight implant-abutment connections that reduce the dimension of the microgap may assist in preventing inflammation and bone loss. Whenever possible, with bone-level implants, the use of transmucosal abutments

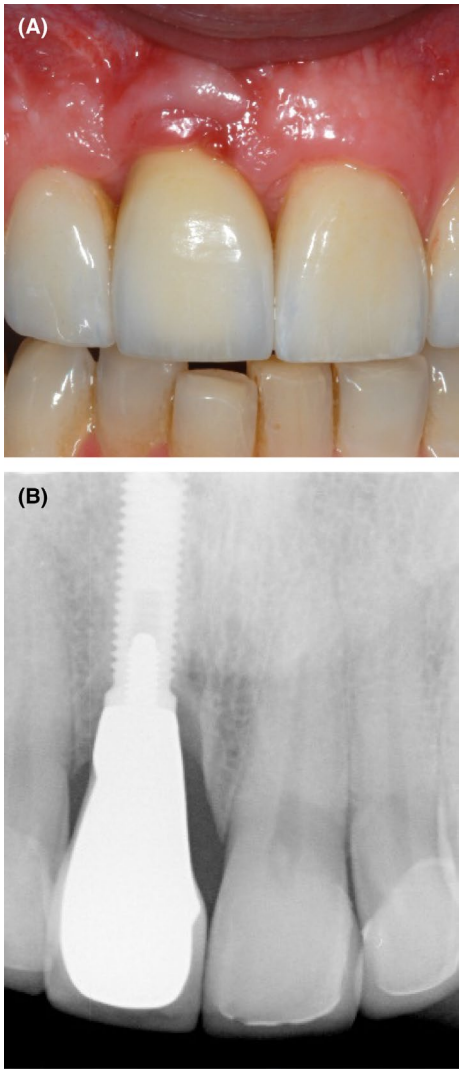

FIGURE 14 (A) This implant replacing the maxillary right central incisor has been placed in an apical malposition, that is, it has been too deeply placed. There is a persistent inflammation of the mucosa which could not be eradicated despite the patient's efforts at plaque control and regular supportive maintenance therapy. (B) The corresponding periapical radiograph shows the apical position of the implant shoulder. The implant has a machined surface with an external hex abutment connection.

≥2 mm allows the peri-implant soft tissue seal to be established from the day of surgery and will elevate the restorative material away from the bone crest.[142]

Implants placed in a coronal malposition (i.e., too superficial) may not have the micro-rough surface of the implant inserted within bone in the first instance. This is a consequence of surgical errors in planning and osteotomy bed preparation (Figure 15A–C). Implants placed in a coronal malposition may also have insufficient vertical restorative space to adequately restore the implant. This often occurs with overeruption of opposing teeth, or a collapse in the occlusion and loss of the vertical dimension of occlusion. If there is insufficient vertical restorative space, restoration of the implants usually requires the use of short abutments with wide restorative EA and ridge lap designs. As mentioned previously, abutments with wide EA are associated with early CBL. Ridge lap designs impede access for home care and maintenance and increase the risk of plaque-induced inflammation of the peri-implant tissues.

Another aspect of implant positioning is where to locate the micro-rough implant surface in relation to the bone crest during surgery. As already outlined above, flap-elevation causes an interruption of the periosteal blood supply followed by crestal bone resorption due to avascular necrosis (for review see Monje et al. 2023).[143] This varies from patient to patient, and hence, we strongly recommend positioning the micro-rough surface at least 1 mm subcrestally during insertion of bone-level and tissue-level implants (Figure 16A,B). This surgical approach was first introduced in the late 1990s[144] and was utilized in the patients serving for the long-term study at the University of Bern with hybrid TL implants, which showed at 10 years of follow-up a minimal risk success and survival rates at 97.0% and 98.8%, respectively.[51] Subcrestal placement of the micro-rough surface may act as a "safety buffer" to minimize an exposure of the micro-rough surface to the peri-implant sulcus during healing.

Malpositioned implants are a major cause of CBL and peri-implantitis and are a consequence of clinician errors resulting from inadequate diagnosis and planning and improper surgical placement.

3.3 | Prosthetic risk factors

The long-term biological stability of dental implants is directly influenced by the restoration, where prosthetic design factors such as the restorative emergence angle, emergence profile, prosthetic fit, prosthesis contour, prosthesis cleansability, and mode of crown retention (cement vs. screw retained) influence peri-implant tissue health and maintenance of crestal bone.[145] There needs to be sufficient restorative space to establish a physiologic supracrestal tissue attachment[146] or biologic width of 3 to 4 mm.[62]

The contour of an implant restoration may be characterized in two ways: the emergence angle and the emergence profile. According to the Glossary of Prosthodontic Terms, the restorative emergence angle (EA) is defined as the angle formed between the average tangent of the transitional contour and the long axis of the implant and is measured at the shoulder of the implant.[147] EA relates primarily to bone-level implants. The EA of tissue-level implants is "built in" to the design of the transmucosal neck of the implant. In bone-level implants, an EA >30° to 40° is considered to be wide, although there is no well-defined threshold.[148] The restorative emergence profile (EP) is the contour of the restoration as it emerges from the soft tissues[147] and can be described as having a convex, concave, or straight profile. The EA and EP are closely related in bone-level implants—EA tends to be larger for convex EP compared to concave or straight profiles. EA is influenced by the restorative space and depth of the implant shoulder, with EA increasing from deeper to more superficial shoulder placement.[149] EA is also influenced by the relative differences in the dimensions of the desired restoration compared to the implant. For example, a maxillary central incisor that is triangular

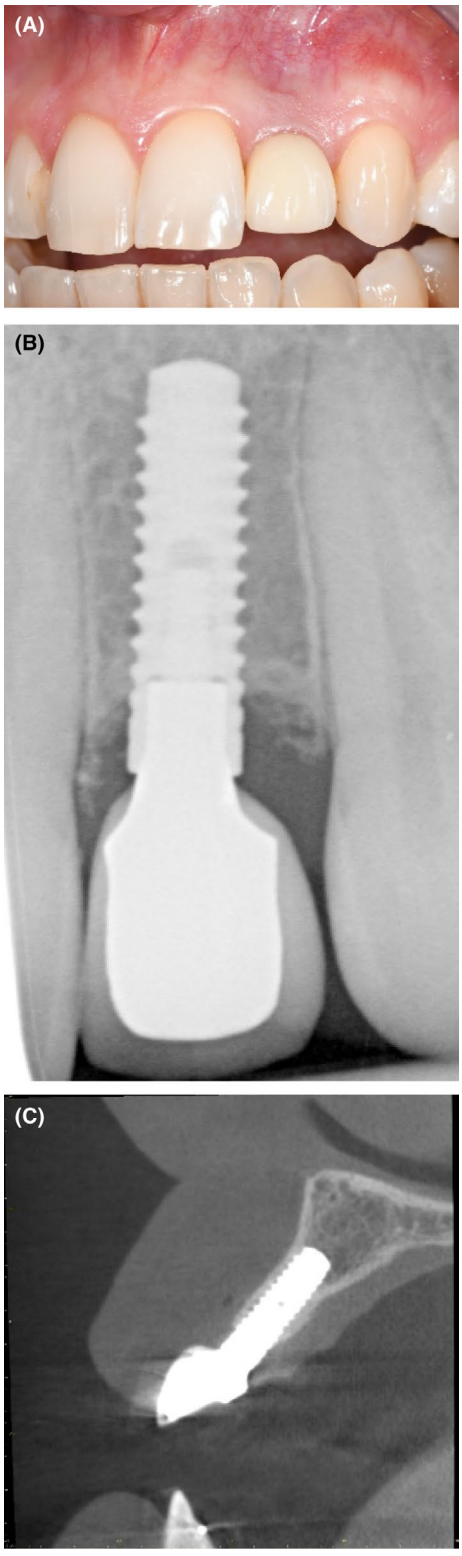

FIGURE 15 (A) The implant in the maxillary left lateral incisor site is in a coronal malposition, that is, it has been placed too superficially. (B) The periapical radiograph shows crestal bone loss and an overcontoured crown. (C) The corresponding parasagittal CBCT view of the implant shows crestal bone loss on the buccal and palatal aspects of the implant.

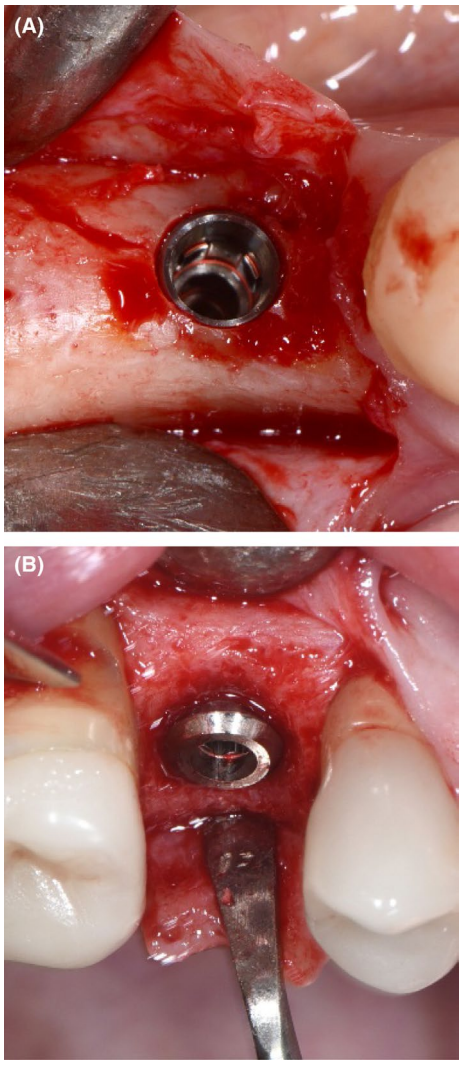

FIGURE 16 (A) Clinical image showing a bone-level implant with subcrestal placement of the implant shoulder. (B) This tissue-level implant with a hybrid design (1.8 mm smooth collar) has been placed with the rough-to-smooth junction 1 mm subcrestally.

in shape requires sufficient restorative space to compensate for the discrepancy between the narrower implant platform and the larger, differently shaped emergence profile of the crown.

In the following section, cementation of the prosthesis and the influence of EA will be discussed as having the potential to cause early CBL and the subsequent development of peri-implantitis. The EP of the implant restoration will be discussed in a later section in relation to the cleansability of the restoration.

3.3.1 | Restorative emergence angle

There is a significant association between wide EA and early CBL,[148,150,151] and prevalence of peri-implantitis in bone-level

implants.[149] It has been reported that early CBL exceeding a threshold of 0.5 to 1 mm may result in biofilm formation on exposed micro-rough surface and the risk of peri-implantitis.[50] A prospective study demonstrated that EA significantly predicts both the probability and extent of early CBL.[148] It should be noted that the association of EA and CBL on the incidence of peri-implantitis is almost exclusively related to bone-level implants. Katafuchi and co-workers found a significant association between peri-implantitis and EA, and the combined effect of EA and EP for bone-level implants, but not for tissue level implants.[149] The subsequent discussion therefore relates to bone-level implants.

The effect of EA on the crestal bone appears to be modulated by transmucosal abutment height, with abutments >2 mm reducing the bone loss otherwise associated with steep angles.[152,153] Controlled preclinical evidence further reinforces these findings. A recent preclinical study using CAD-CAM abutments with standardized angles (20°, 40°, 60°, 80°) demonstrated a clear dose–response relationship, with the widest angles producing the greatest bone loss.[148] This association was confirmed across mesial, distal, and, for the first time, buccal sites using histology, micro-CT, and synchrotron imaging. In susceptible individuals, implants with early CBL due to wide EA may be a predisposing condition for the subsequent development of peri-implantitis.

Several biologic mechanisms may explain these observations. Shallow implant placement may limit the space available for establishing an adequate supracrestal tissue attachment height, leading to the need for wide EA and EP to create the appropriate aesthetics of the prosthesis,[62,154,155] thereby promoting CBL, exposure of micro-rough surfaces, and plaque retention. Conversely, narrower angles appear to improve cleansability and reduce inflammation. Supporting this, a recent study using intraoral scanning and radiographs found that increasing restorative angles was strongly associated with greater plaque accumulation and bleeding on probing.[156]

From a biologic standpoint, increasing the vertical and horizontal distance between the restorative margin and the crestal bone—whether through narrower emergence angles or greater transmucosal height—helps maintain inflammatory infiltrates in a more coronal position. Inflammatory lesions located farther from the bone crest are less likely to trigger osteoclastic activity,[157] consistent with Waerhaug's classic "radius of action" concept, which proposes that plaque-induced inflammatory destruction is confined to an approximate radius of 1 mm, with tissues beyond this distance largely unaffected.[158]

As will be discussed later, restorations with a wide EA are often observed when the restorative space is limited, thereby forcing the laboratory technician to fabricate restorations with convex EP and short abutments. Clinically, CBL associated with wide EA may often be seen radiographically soon after connection of the prosthesis (Figure 17A,B). It may also be possible to reverse the CBL associated with a wide EA by remaking the prosthesis with a taller abutment, as demonstrated in this clinical case (Figure 18A–D).

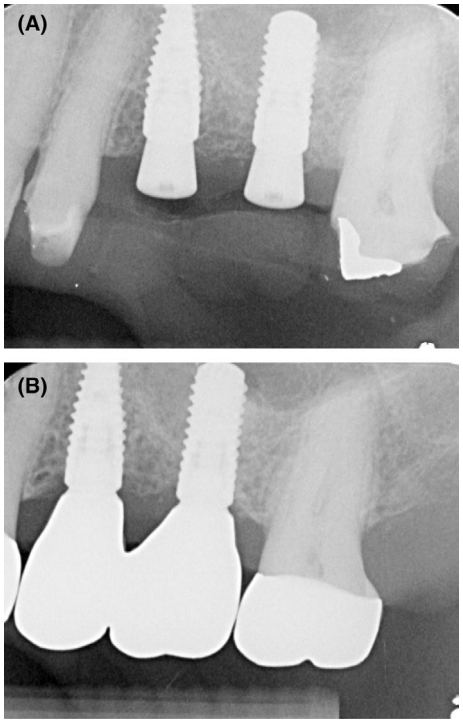

FIGURE 17 (A) This periapical radiograph was taken 3 months after the placement of 2 bone-level type implants in the maxillary left posterior segment. The implants were placed subcrestally and with a transmucosal healing protocol. Note the bone wall in proximity to the healing abutments. (B) The follow-up periapical radiograph taken 2 months after the prosthesis was connected shows early crestal bone loss (CBL) of 1 to 1.5 mm at both implants. Note the short abutments and wide restorative emergence angles (EA).

Clinicians should aim for restorative EA of <30–40° to reduce the risk of early CBL. In line with this, the most recent EFP clinical practice guidelines emphasize that optimal restorative design, particularly careful control of the EA, is essential for the primary prevention of peri-implantitis.[16]

3.3.2 | Cement remnants

Cement remnants retained on an implant surface within the sulcus can cause peri-implantitis, with a recent systematic review reporting that 33%–100% of cemented restorations with peri-implantitis had retained cement remnants.[159,160] A recent clinical study reported on 109 single-unit implant crowns designed with screw access channels to allow crown retrieval.[161] The crowns were cemented on the abutments intraorally, and excess cement was removed. On unscrewing the abutment-crown complex, 72.5% of the crowns were found to have cement remnants. The authors reported that the incidence of cement remnants was significantly greater for crowns with submucosal margins (60.22% and 61.4% of the 0.5 mm submucosal and ≥1 mm submucosal margin groups, respectively) compared to equimucosal (26.6%) and supramucosal (23.7%) crowns. It may be concluded that

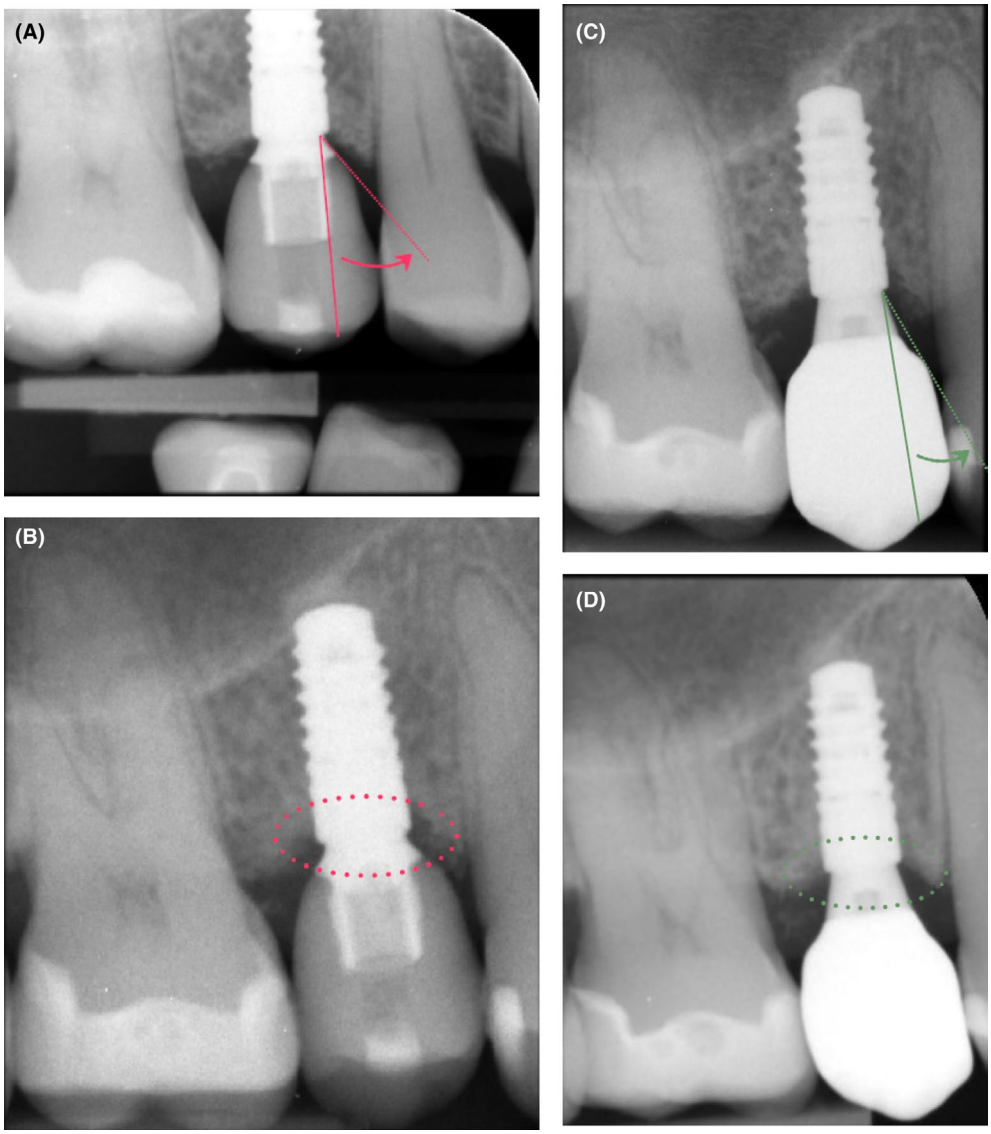

FIGURE 18 (A) This is a case of an implant in a maxillary second premolar site with a wide restorative emergence angle (EA) at crown delivery. The red dotted lines indicate the width of the angle. (B) After 4 years, the marginal bone loss can be observed (area within the red dotted line). (C) The crown was subsequently remade with a longer abutment, which effectively reduced the width of the restorative angle (green dotted lines). (D) A follow-up radiograph after 12 months shows remineralization and a radiographic gain in crestal bone height (area within the green dotted line).

cement remnants frequently occur with cemented restorations. The retained cement acts as a foreign body and is plaque retentive,[162] and invokes an inflammatory response in the peri-implant mucosa.[163] An endoscopic study reported that the cement is often found on the buccal and lingual surfaces of the implant and therefore undetectable on radiographs.[164] The depth of the restorative margin is a key factor, with the amount of cement remnants increasing as the restorative margin deepens.[165] In the presence of an exposed implant surface due to a deficient bone augmentation procedure, cement remnants can be found on the micro-rough implant surface.[166] The combination of cement remnants retained on a micro-rough surface deep within the peri-implant tissues will undoubtedly cause a significant peri-implant infection (Figure 19A–C). Periodontitis patients are particularly susceptible to developing peri-implantitis in the presence of cement remnants.[167]

Screw-retained restorations are generally preferred when prosthetically feasible. If cement-retained restorations are unavoidable, margins should be placed at or above the mucosal level. Radiographic verification should be performed, and retrievability strategies should be incorporated. Clinicians should avoid cemented implant crowns whenever possible,[159] particularly in periodontal patients and at sites that have undergone bone augmentation procedures.

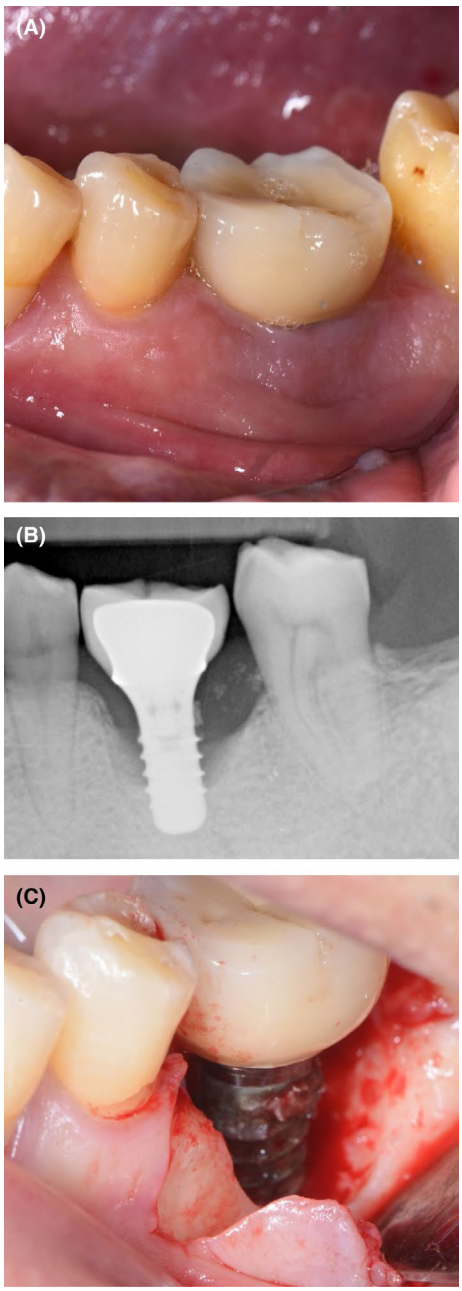

FIGURE 19 (A) Peri-implantitis was diagnosed on this implant replacing the mandibular left first molar. Note the diffuse inflammation of the mucosa on the buccal aspect of the implant. The crown was cement-retained. (B) The corresponding radiograph highlighted the circumferential bone loss and the presence of retained cement on the distal aspect. (C) The surgical view of the implant following flap reflection. Note the retained cement on the micro-rough surface of the implant, which confirms that the endosseous part of this tissue-level implant was not completely embedded in bone at the completion of bone healing.

4 | CO-FACTORS FOR PERI-IMPLANTITIS

A primary predisposing factor for peri-implantitis is exposure of the micro-rough implant surface to the peri-implant sulcus. In the following section, the role of co-factors will be discussed in relation to their contribution to the development and progression of peri-implantitis, particularly in the presence of an exposed micro-rough surface to the sulcus.

4.1 | Patient factors

Clinicians have a duty of care to their patients. Clinicians are responsible for identifying the factors that may increase their patient's risk of complications, and to develop strategies to manage and mitigate these risks. Ultimately, it is the clinician who determines if a patient is suitable or not to receive dental implant therapy.[168] The standard of care dictates that clinicians should not proceed to treat patients without the establishment of comprehensive risk mitigation strategies.

4.1.1 | Patient education and compliance

Long-term success with implant therapy requires a commitment from patients to achieve oral health prior to having implants placed, and subsequently to adhere to a personalized recall and supportive maintenance program.[169] Patients who comply with daily homecare and attend regularly for supportive maintenance therapy have a lower incidence of peri-implantitis.[170,171] Therefore, an important element at the beginning of the patient care pathway is for dental professionals to educate patients on the risks, potential complications, and long-term maintenance requirements. However, the proportion of patients who do not comply with maintenance recalls is highly variable, with a recent systematic review reporting that the proportion of full compliers and non-compliers ranged between 3.3%–86.8% and 1.69%–64.4%, respectively.[172] Clinicians should therefore make every effort to educate their patients who are being prepared for implant therapy to understand their responsibility to comply with oral hygiene measures and to return for regular supportive maintenance. Treatment should not commence if there are doubts about the patient's commitment to long-term follow-up care.

4.1.2 | Systemic factors and habits

Patients who smoke have an increased risk of developing peri-implantitis.[173,174] They should be made aware of the risks and be offered smoking cessation counseling with appropriately trained professionals.

It has been estimated that patients with diabetes mellitus have a 50% higher risk of peri-implantitis compared to healthy individuals.[175] The risk diminishes with controlled diabetes, where success rates may be similar to healthy patients.[176] Dental implant therapy should not proceed in the presence of uncontrolled diabetes and poor compliance, and if there are concerns about the patient's understanding of the risks and commitment to long-term recall and maintenance.

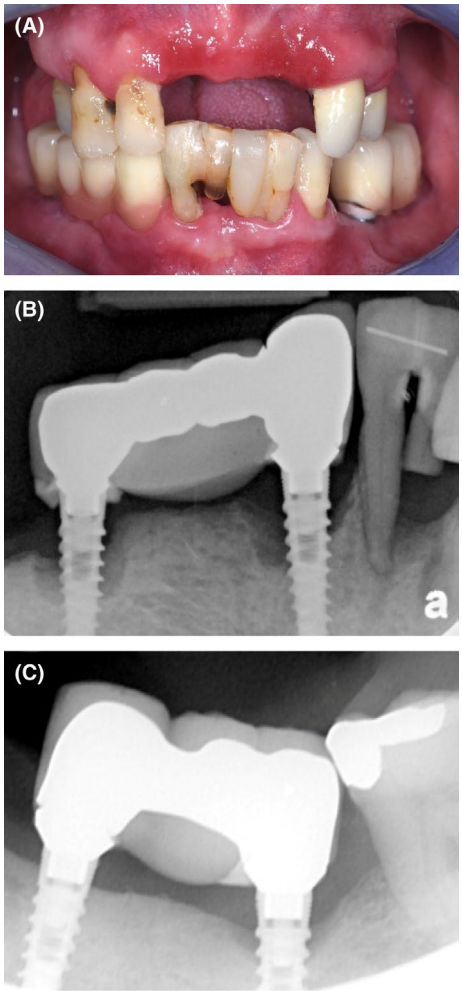

FIGURE 20 (A) Initial presentation of a patient referred for management of peri-implantitis. The patient had generalised periodontitis which had not been properly managed. (B) Periapical radiograph of implants in the mandibular right posterior segment, showing bone loss around both implants due to peri-implantitis. (C) Periapical radiograph of implants in the mandibular right posterior segment, showing bone loss due to peri-implantitis around the distal implant.

Complex cases requiring significant bone augmentation and extensive surgical management should be approached with caution due to the risk of complications and suboptimal outcomes.

4.1.3 | Periodontitis

There is unequivocal evidence that patients with a history of periodontitis have a higher risk of developing peri-implantitis than patients without periodontitis.[177] Furthermore, periodontal patients with poor plaque control[170] and those with residual periodontal pockets of ≥5 mm have an elevated risk of developing peri-implantitis (Figure 20A–C).[178] Many leading dental organizations have published guidelines for managing periodontal patients with dental implants. For example, the International Team for Implantology

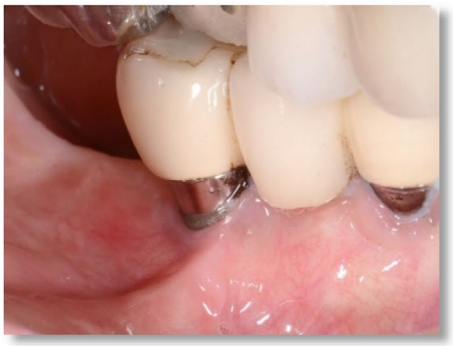

FIGURE 21 The implant in the mandibular right molar region developed peri-implantitis. Note the recession of the marginal mucosa, lack of keratinized tissue, and a frenal attachment nearby.

(ITI) has recommended that active periodontal therapy should be completed with the aim of improving home care and compliance, and eliminating periodontal pockets with bleeding after probing before proceeding with implant placement. Patients should then be enrolled in a personalized supportive periodontal maintenance program that includes monitoring of peri-implant tissue health.[169] Implants should not be placed in patients with periodontitis until the disease is properly managed, and patient compliance is established. Clinicians should employ strategies to minimize the risk of exposure of the micro-rough implant surface to the sulcus in patients with periodontitis.

4.1.4 | Site factors—Lacking peri-implant keratinized mucosa

Lack of peri-implant KM has been reported to be associated with the risk of peri-implantitis.[179,180] Implants that lack KM, defined as <2 mm of keratinized tissue, were significantly more prone to plaque accumulation, peri-implant mucosal inflammation, mucosal recession, marginal bone loss, and even greater patient discomfort during oral hygiene.[179,181–183] This minimum KM width has been reported to be beneficial for long-term peri-implant health.[16,184]

In the presence of an exposed micro-rough surface to the peri-implant sulcus, the risk of developing peri-implantitis in the absence of KM is significant. Although lack of KM is a patient-related site factor, the clinical error is to place implants without maintaining or reconstructing KM around the implants, or addressing adjacent frenal attachments (Figure 21). When peri-implant sites exhibit deficient KM in conjunction with inflammation, soft-tissue augmentation such as free gingival grafts or xenogeneic substitutes should be considered to increase the keratinized tissue zone.[185] Free autogenous grafts are considered the gold standard for widening the keratinized zone, with studies reporting that substitute graft materials can achieve similar improvements.[186]

Clinicians need to be mindful of the importance of KM around implants, and have the necessary training and surgical skills to maintain KM around implants when performing implant surgery, and to

undertake soft tissue augmentation procedures when KM is deficient. Contemporary consensus recommendations emphasize the importance of evaluating the width of KM as part of the initial assessment, as well as during post-treatment implant maintenance care.[185]

4.2 | Prosthetic factors

4.2.1 | Prosthetic fit

Modern implant systems today are designed with well-fitting abutment connections to minimize micromovements under functional load. Micromovements alter load transmission and may promote wear, screw loosening, and component fatigue under cyclic functional loading.[187,188] However, errors in handling and fabrication of the prosthesis by the laboratory and subsequent clinical handling of prosthetic components can damage the abutment connection.[189] Prosthetic misfit creates gaps that allow accumulation of bacterial biofilm. In the presence of a micro-rough surface exposed to the sulcus, prosthetic misfit increases the risk of peri-implantitis.

Laboratory errors which can damage the abutment connection include inadvertent damage of the abutment connection interface during manufacturing procedures,[190] contamination of connection surfaces and abutment screws with debris,[191] inaccuracies in casting and soldering,[192] incorrect tolerances in CAD/CAM manufacturing,[193] and inaccuracies when milling components.[194] Clinical errors include multiple connections and disconnections of abutments which can cause permanent deformation at the implant-abutment interface,[189] improper seating of abutments, incorrect tightening torques,[195] and non-passivity in fit, especially in multi-unit prostheses.[196]

Prosthetic misfit can also cause biological complications secondary to mechanical failure. Inaccurate component alignment can induce uneven stresses and strains within the prosthetic complex, leading to mechanical complications such as screw fracture, framework damage, or implant fracture.[152,197] These mechanical complications have the potential to cause biological complications, including peri-implantitis. Clinical evidence underscores a direct association between poorly fitting prostheses and increased rates of complications and treatment failure.[198]

Clinicians should avoid using non-original dental implant components, including abutments, titanium bases, and prosthetic screws, which are often selected to reduce treatment costs or increase prosthetic options.[199] However, these potential savings, whether clinical or financial, may be accompanied by greater uncertainty regarding component fit, mechanical reliability, and long-term performance.[200] Unlike original components, which are designed and validated for specific implant systems, non-verified non-original components may differ in geometry and tolerances. Such discrepancies can impair interface accuracy, resulting in inadequate sealing,[201] rotational instability,[202] microleakage, and reduced connection stability.[199] Over time, these factors may increase the risk of mechanical and biological complications, including screw loosening,[203] component fracture, or implant failure.[204]

Finally, the increasing integration of intraoral scanners into implant workflows introduces an additional dimension to prosthetic fit.[205] The accuracy of digital impressions depends not only on scanner technology but also on the clinician's ability to recognize and interpret scanning errors.[206] As with conventional impressions, digital scans are susceptible to operator-related errors—such as missing data, stitching defects, soft-tissue capture, unreliable scan areas, surface noise, and implant scan body distortions—as well as patient-related factors, for instance moisture[207] and restricted access. If these errors remain undetected, they may compromise marginal accuracy, surface detail, and implant positioning, while corrective rescanning may itself reduce accuracy depending on the scanner system and workflow. Therefore, the systematic evaluation of digital impressions, analogous to the assessment of conventional impressions, remains essential.

4.2.2 | Prosthesis contour and cleansability

A direct consequence of the contour of the prosthesis is its cleansability. Dental implant prostheses that are not cleansable risk development of peri-implantitis of the supporting implants (Figure 22A,B),[208] particularly in the presence of an exposed micro-rough surface to the sulcus. Implant restorations must be designed to be accessible for biofilm removal by the patient and by the dental team when providing supportive maintenance therapy.[12] Examples of prosthetic designs that hamper access for cleaning include deeply positioned restorative margins,[209] restorative EP that are overcontoured or convex,[210] ridge lap designs,[211] flanges that overlap the buccal aspect of the ridge,[12] concave intaglio surfaces on extended or full-arch bridges,[212] insufficient embrasure space between prosthetic units,[213] poorly polished and rough prosthesis surfaces,[214] and misfit at implant-abutment or prosthesis-abutment interfaces when using non-original or damaged implant components.[199]

As discussed previously, the EA has a significant influence on the maintenance of crestal bone. In addition to this, the transmucosal EP of the restoration, typically described as concave, convex or straight, has a decisive influence on cleansability[215] and peri-implant soft-tissue behavior.[210,216,217] This contour affects aesthetics, mucosal thickness and height, cleansability and the risk of biological complications, and has therefore become a key consideration in prosthetically driven implant therapy and maintenance of peri-implant tissue health.

There is a growing body of evidence that concave emergence profiles have a biological advantage.[218–222] Concave forms increase the horizontal volume available for soft-tissue maturation, creating additional "running room" that facilitates coronal migration (or soft-tissue creeping) and helps stabilize the position of the midfacial mucosal margin. Histologically, concave or narrow EA profiles are associated with a structured, cuff-like peri-implant mucosal seal, characterized by a continuous junctional epithelium and dense connective tissue.[148]

In contrast, convex profiles reduce the available soft-tissue space. Several studies have reported that convex contours are associated

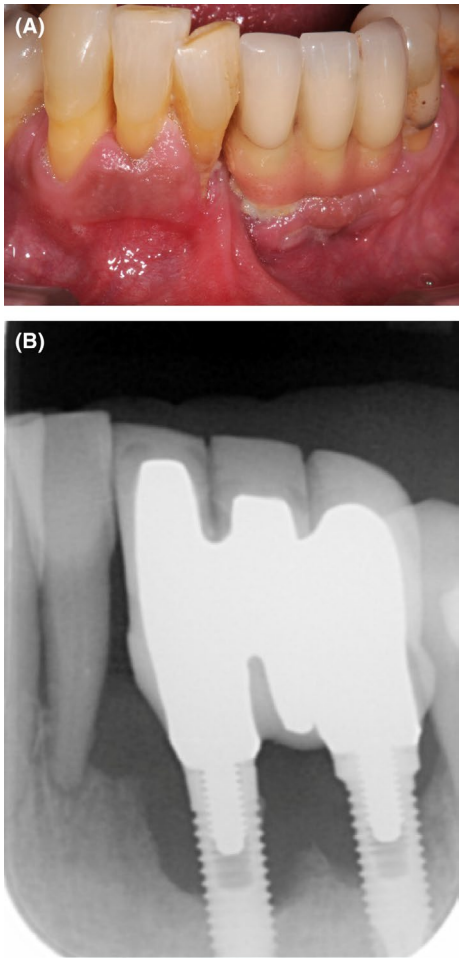

FIGURE 22 (A) A 3-unit mandibular anterior implant bridge had a prosthetic design that was uncleanable by the patient. (B) The corresponding periapical radiograph showing bone loss as a consequence of peri-implantitis.

with higher rates of midfacial recession, increased plaque accumulation, and increased bleeding on probing, particularly in the presence of wide EA.[156,210,217,223] When there is a pronounced convexity, the restorative margin is located closer to the crestal bone, which in turn narrows the biologic space for establishment of the supracrestal tissue attachment. This may place the soft-tissue seal under tension. These factors can hinder coronal migration and bring inflammatory infiltrates closer to the bone crest.

Recent preclinical and clinical studies have provided further evidence for the adverse effect of convex restorative profiles on the cleansability of the prosthesis and therefore maintenance of peri-implant tissue health.[224,225] A recent randomized clinical trial reported a trend toward increased bleeding on probing around convex restorative profiles.[210] Two cross-sectional studies found higher odds of bleeding on probing at sites restored with convex EP (>40°).[156,223] These observations are in concordance with in vitro findings that concave crown contours are more cleansable compared to straight or convex designs,[215] reinforcing the concept that prosthetic morphology significantly influences biofilm retention and the inflammatory response. In a recent in vivo study, it was reported that steeper emergence angles (>40°) may compromise self-cleansability and promote plaque accumulation.[226] In this study in a canine model without oral hygiene, inflammatory infiltrates increased in a dose-dependent manner with wider angles. Buccal sites showed consistently higher levels of inflammation than lingual sites.[226] Wider restorative contours may further predispose to food trapping,[12] especially when implant position limits the ability to reproduce natural anatomic crown morphology.

The EP also affects clinical monitoring and diagnostic accuracy.[227] Patient discomfort or pain during probing[228] may further limit probing depth penetration and compromise measurement reliability. These challenges are particularly pronounced around convex profiles, especially with shallow implant placement and crown contours that restrict access for periodontal probing. In this context, flexible plastic probes may improve probing accuracy by better adapting to curved transmucosal surfaces than rigid metal probes.[227]

Nevertheless, convex contours remain necessary in certain clinical situations arising from clinical errors. They may be indicated when implants are placed palatally or lingually, or when there is insufficient restorative space. Convex contours may be necessary when shaping the most coronal component of the transmucosal zone to achieve proper aesthetic contouring or alignment with adjacent teeth.[229] In such circumstances, biologic risks may be mitigated by using strategies such as increasing transmucosal abutment height or maintaining narrow emergence angles to preserve soft-tissue volume and distance from the crestal bone.

Over the last two decades, there has been a shift toward milled zirconia restorations for implants due to the strength, durability, and biocompatibility of the material. However, Zhang and co-workers reported a high proportion of zirconia multi-unit restorations exhibiting convex contours which are uncleansable and significantly associated with biological complications (Figure 23A,B).[230] Although adjustment of the prosthesis may improve hygiene access, chairside recontouring of milled zirconia restorations is technically challenging and may increase surface roughness, induce microcracks, and compromise aesthetics.[231,232] Clinicians should therefore be actively involved in the design phase, understanding the material thickness requirements and limitations in CAD/CAM manufacturing for zirconia restorations, and not accept overcontoured and uncleansable restorations.

In addition, the basal and submucosal surfaces of monolithic implant restorations should ideally be polished rather than veneered (Figure 24), as surface characteristics and material type significantly influence peri-implant soft tissue attachment and biological stability.[233,234] Experimental evidence demonstrates that veneering ceramics, including glazed surfaces, result in reduced mucosal attachment compared with non-veneered materials, indicating that such surfaces are less favorable for establishing a stable soft tissue seal.[233]

Furthermore, both titanium and zirconia abutments have shown superior soft tissue compatibility and comparable healing responses, performing significantly better than gold-platinum alloys in the formation of a healthy mucosal barrier.[234,235]

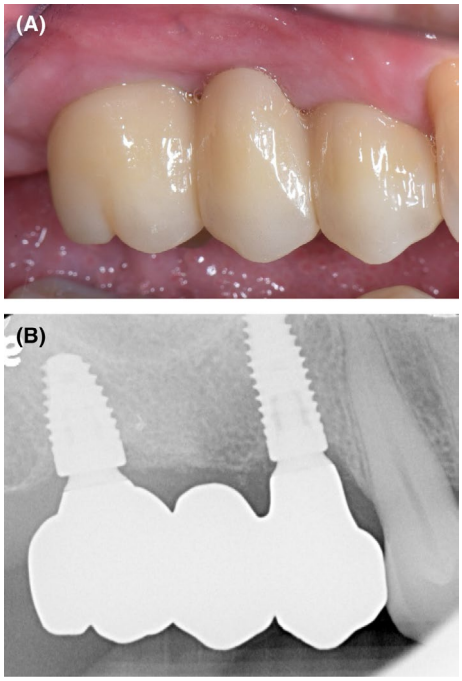

FIGURE 23 (A) A milled zirconia 3-unit prosthesis has been connected to the implants in the maxillary right first premolar and first molar sites. The prosthesis is overcontoured and uncleanable by the patient. (B) The periapical radiograph of the prosthesis.

Laboratory studies suggest that surface finishing methods influence fibroblast behavior, and although both polishing and glazing can promote fibroblast adhesion, the negative clinical findings associated with veneered surfaces support highly polished titanium or zirconia as the preferred material–surface combination for the submucosal region.[31,233,236,237] Whenever possible, polishing should be performed under controlled laboratory conditions, as laboratory protocols achieve smoother and more consistent surfaces than chairside polishing systems.[214]

Collectively, these insights demonstrate that prosthetic design—including both EA magnitude and transmucosal EP—influences not only plaque accumulation and inflammatory burden but also the clinician's ability to perform accurate peri-implant monitoring. It should be noted that compromised prosthetic designs are often a consequence of surgical errors in implant position/malposition,[238] with the restorative dentist and laboratory technician "doing their best" to restore the implant they are presented with. Clinicians must understand the profound impact of implant position on the final restoration, ensuring that the surgery and prosthetic phases of treatment fulfill the prosthetic goal.

The corono-apical position of the implant shoulder and the available restorative space are closely related to the EA and EP. Insufficient restorative space will almost always result in wide EA and a convex EP. In cases of limited restorative space, subcrestal implant placement with/without crestal bone reduction may be necessary, and often in conjunction with increasing the vertical dimension of occlusion. Thus, proper pre-surgical planning is essential to achieve the ultimate

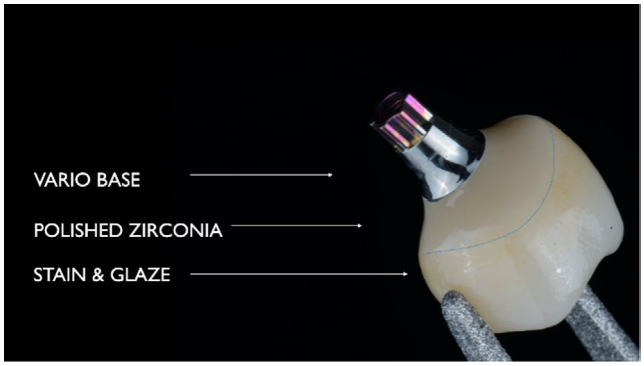

FIGURE 24 Image of a zirconia crown showing the Variobase abutment and the submucosal region of the crown which should be polished only. The supramucosal region (junction delineated with a line) can be stained and glazed.

restorative goal of a prosthesis with EA <30–40° and flat/concave EP that is cleansable and minimizes CBL.

4.3 | Follow-up and maintenance

There is compelling clinical and scientific evidence that long-term and effective dental implant therapy extends far beyond the surgical placement and prosthetic restoration, requiring a comprehensive and long-lasting commitment to supportive care, especially considering the increasing documented prevalence of peri-implantitis.[10,239] Recent evidence has demonstrated that individualized risk-based supportive peri-implant care (SPIC) protocols substantially enhance clinical outcomes and implant longevity compared with standard fixed-interval maintenance schedules or the absence of supportive therapy altogether. Mojaver et al. have shown that tailoring maintenance intervals and interventions according to individual patient risk profiles leads to superior preservation of peri-implant tissues and reduced incidence of biological complications.[240] These findings are of pivotal importance and represent a paradigm shift from a "one-size-fits-all" approach to a "personalized/tailored care" approach, supporting the clinical experience that patients' needs vary significantly from one to another. Although the intervals for SPIC are widely published, they do not provide details of patients' adherence to the recommended recall frequency. In addition, it should be noted that a patients' risk profile is dynamic and can change over time.[241]

The implementation of risk-stratified maintenance protocols requires clinicians to conduct a thorough assessment of numerous factors that can compromise peri-implant health, thereby enabling the development of individualized interventions.[242] This process starts at the end of active treatment and is aimed at detecting early peri-implant hard and soft-tissue changes. Whenever early CBL occurs, whether secondary to avascular bone necrosis, excessive restorative emergence angles or suboptimal bone augmentation procedures, the exposure of the micro-rough implant surface to

the peri-implant sulcus represents an important predisposing factor for biofilm accumulation and the potential subsequent onset of peri-implant tissue inflammation and destruction.[82,143] Similarly, early detection of changes in peri-implant soft tissue phenotypes (i.e., KM thickness and width) necessitates closer surveillance, as such deficiencies may compromise long-term peri-implant health as a consequence of suboptimal plaque control. Patients should be informed if CBL is detected or if there are alterations to the peri-implant mucosa that may compromise health. Patients should be recalled more frequently to facilitate monitoring, and in cases of soft tissue anomalies, consideration of corrective soft-tissue grafting procedures.[243]

Among the documented risk factors for the development of peri-implant disease, a history of periodontitis represents the most significant one, with marked evidence demonstrating an increased risk of developing peri-implantitis in patients with previous severe periodontal disease compared to patients not affected by periodontitis.[58,244,245] Therefore, an accurate assessment of periodontal conditions (i.e., full-mouth plaque and bleeding scores) followed by an adequate treatment of sites displaying recurrence of periodontal disease (i.e., probing depths ≥5 mm + bleeding of probing) should be regularly performed.[178] Among systemic conditions, diabetes mellitus represents a critical factor, as both controlled and poorly controlled diabetic patients exhibit altered immune function, impaired wound healing, and modified inflammatory responses that can compromise the delicate balance required for maintaining healthy peri-implant tissues and osseointegration.[246] Beyond systemic conditions, behavioral factors such as smoking significantly elevate risk levels, as tobacco use impairs microcirculation, reduces oxygen delivery to tissues, alters immune cell function, and creates an environment that favors pathogenic bacterial colonization.[247,248] The current evidence strongly demonstrates that smokers are associated with low compliance levels with SPIC, further compounding their risk profile.[172] Along with smoking, inadequate oral hygiene habits represent the most modifiable yet frequently encountered risk factor, with poor plaque control consistently identified as a major contributor to peri-implant disease development.[239]

As mentioned previously, certain implant-supported prostheses designs and configurations, such as deep restorative margins, presence of ridge lap designs, concave intaglio surfaces, wide emergence profiles, and poor-fitting restorations represent an ideal microenvironment for plaque and debris accumulation, not allowing for proper patient oral hygiene procedures.[149,249] Clinicians should routinely check prosthesis fitting and cleansability and proceed with adjustments whenever needed. In particular, uncleanable prostheses or full-arch reconstructions may require the prosthesis to be removed regularly for assessment of peri-implant soft tissue conditions and to adequately carry out SPIC procedures.[250]

It is important to note that even when properly treated with GBR, the alveolar ridge will undergo physiological dimensional changes over many years. The surrounding peri-implant soft tissues may also undergo changes, so it is essential to continuously monitor peri-implant soft tissue conditions. In patients displaying difficulties in proper plaque control and/or reporting brushing discomfort, soft-tissue augmentation procedures are recommended.[251]

In conclusion, clinicians should routinely inform patients receiving implant therapy on the importance of SPIC as a pivotal step of therapy. SPIC sessions, whose frequency should be tailored to patient need and continuously adapted through time, should encompass the evaluation of patients' and implant/prosthesis-specific features, followed by appropriate preventive interventions to ensure early detection of peri-implant inflammation to minimize the risk of peri-implantitis.

5 | CONCLUSIONS AND RECOMMENDATIONS

The prevalence of peri-implantitis has reached concerning levels. There is a growing consensus that the majority of cases of peri-implantitis arise from clinician-related errors in implant therapy, and in these cases, peri-implantitis should be regarded as a complication of implant therapy rather than a disease in the classical sense. Peri-implantitis is therefore not a homogeneous condition, but rather a pathology driven by diverse patient-related and clinician-related factors. A primary predisposing factor for peri-implantitis is exposure of the micro-rough implant surface to the peri-implant sulcus. The exposed micro-rough implant surface may subsequently become contaminated with bacterial biofilm, eliciting an inflammatory response in the peri-implant mucosa. This elevates the risk of developing peri-implantitis in susceptible individuals, particularly in those with a history of periodontitis. The exposure of the micro-rough implant surface often occurs at the time of surgery or soon after surgery or prosthetic treatment. The causes are frequently iatrogenic, due to errors in the diagnostic, treatment planning, surgical, and prosthetic phases of treatment.

5.1 | Recommendations

1. To reduce the risk of peri-implantitis, clinicians should make every effort to avoid exposure of the micro-rough implant surface to the peri-implant sulcus by adhering to fundamental principles of diagnosis, surgery, prosthetics, and follow-up care and maintenance.
2. From a surgical perspective, early CBL causing exposure of the micro-rough implant surface can be caused by implant malposition and implant proximity, and by avascular necrosis when implants are placed in sites with reduced crestal bone width. These errors stem from inadequate diagnosis and planning and are avoidable errors.
3. Failure to completely regenerate bone within peri-implant defects due to errors in grafting techniques will result in exposure of the implant surface at the outset. Implant surgeons need to have a sound understanding of tissue biology and training in grafting techniques to ensure proper diagnosis, case selection, and execution of treatment to achieve predictable bone regeneration outcomes.

4. When bone-level implants are restored, prostheses with wide emergence angles should be avoided as they can cause early CBL and increase the risk of peri-implantitis. To prevent this, meticulous pre-surgical planning is required to ensure that the implants are positioned according to the prosthetic plan and that there is sufficient restorative space to allow for longer abutments with narrower emergence angles to be used.
5. Cemented restorations should be avoided as they are associated with a high risk of peri-implant infection.
6. Clinicians should be cognisant of the co-factors that may contribute to the development and progression of peri-implantitis in the presence of an exposed micro-rough surface—including patient susceptibility and lack of compliance, site factors such as lack of peri-implant KM, prosthesis factors resulting in prosthesis misfit, over-contour and non-cleansability, improper patient follow-up/maintenance, and late detection of crestal bone loss and changes to peri-implant KM thickness and width. These factors fall within the clinician's sphere of influence and may be modifiable through appropriate diagnosis, treatment planning, surgical execution, prosthetic design, and maintenance protocols.
7. In the follow-up and maintenance phase of therapy, clinicians should actively look for early CBL and/or alterations to the peri-implant KM thickness and width. If changes are detected, patients should be advised of their increased risk of developing biological complications, and recall intervals should be shortened to allow more frequent monitoring.
8. If the design of the prosthesis and/or suboptimal implant positioning make it difficult to clean and maintain, patients should be seen for SPIC more frequently. Uncleanable prostheses should be adjusted whenever possible, and if necessary, removed regularly for assessment of peri-implant soft tissue conditions and to adequately carry out SPIC procedures.
9. The introduction of implants with micro-rough surfaces in the 1990s has significantly improved the success rate of implants in the maxilla, facilitated the utilization of shorter implants, reduced healing periods and treatment times, and has enhanced the predictability with various loading protocols. However, these implants have also increased the risk of peri-implantitis when the micro-rough implant surface becomes exposed to the peri-implant sulcus. Clinicians should strongly consider utilizing implants with a hybrid surface design which provides a machined implant surface in the transmucosal area to mitigate the risk of micro-rough surface exposure. This is particularly relevant in patients presenting with elevated systemic and site-related risk profiles. The placement of the micro-rough implant surface approximately 1 mm subcrestally is strongly recommended as a safety buffer.

5.2 | Education and research

If we accept that iatrogenic factors create the conditions for most cases of peri-implantitis, then professional education must become the central focus to minimize this complication.[13] Providers of dental implant education, whether university or privately based, must stress the fundamentals of implant therapy: proper patient examination and risk assessment, establishing a prosthetic goal with meticulous prosthetic diagnostics and implant positioning, using least risk treatment approaches, and evidence-based protocols. Too often, dental implant courses focus on complex cases and procedures without adequately covering foundational principles.

From a research perspective, a more nuanced approach is required. Studies must move beyond the documentation of patient-related risk factors to include the identification of clinician-related predisposing and co-factors for peri-implantitis. Such differentiation is critical if therapeutic strategies, currently based on the potentially erroneous premise that all peri-implantitis cases are homogenous, are to be effective. The transition to a complication-based model of peri-implantitis may well represent a paradigm shift in how it is diagnosed and clinically managed.

All stakeholders in implant dentistry—from clinicians and researchers to educators and implant manufacturers—share a collective obligation to lower the prevalence of peri-implantitis.

AFFILIATIONS

[1]Melbourne Dental School, Faculty of Medicine, Dentistry and Health Sciences, The University of Melbourne, Melbourne, Victoria, Australia

[2]Melbourne Periodontal Specialists, Balwyn, Victoria, Australia

[3]Department of Periodontology and Oral Medicine, University of Michigan, Ann Arbor, Michigan, USA

[4]Department of Periodontology, Universitat Internacional de Catalunya, Barcelona, Spain

[5]Department of Periodontology, University of Bern, Bern, Switzerland

[6]Clinic of Reconstructive Dentistry, Center of Dental Medicine, University of Zurich, Zurich, Switzerland

[7]Discipline of Periodontics, School of Dentistry, Faculty of Medicine and Health, The University of Sydney, Sydney, New South Wales, Australia

[8]Faculty of Dentistry, Universidad Finis Terrae, Santiago, Chile

[9]Department of Oral Surgery, Implantology and Oral Medicine, Goethe University, Frankfurt, Germany

[10]Shanghai Perio-Implant Innovation Center, Institute of Integrated Oral, Craniofacial and Sensory Research, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine, College of Stomatology, Shanghai Jiao Tong University, National Center of Stomatology, National Clinical Research Center for Oral Diseases, Shanghai Key Laboratory of Stomatology, Shanghai, China

[11]Department of Periodontology, School of Dental Medicine, University of Bern, Bern, Switzerland

[12]Division of Fixed Prosthodontics and Biomaterials, University Clinic of Dental Medicine, University of Geneva, Geneva, Switzerland

[13]Implant Education, Columbia University College of Dental Medicine New York, New York, New York, USA

[14]Department of Periodontology and Oro-Dental Surgery, Faculty of Medicine, University of Liège, Liège, Belgium
[15]Dental Biomaterial Research Unit, University of Liège, Liège, Belgium
[16]giDE Dental Institute, Losa Angeles, California, USA
[17]giDE Dental Implant Center, Los Angeles, California, USA
[18]School of Dental Medicine, University of Bern, Bern, Switzerland
[19]Centre for Implantology Buser and Frei, Bern, Switzerland

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ORCID

Stephen T. Chen https://orcid.org/0009-0007-3303-0344
Alberto Monje https://orcid.org/0000-0001-8292-1927
Franz J. Strauss https://orcid.org/0000-0002-5832-7327
Frank Schwarz https://orcid.org/0000-0002-5873-9903
Andrea Roccuzzo https://orcid.org/0000-0002-8079-0860
Irena Sailer https://orcid.org/0000-0002-4537-7624
Dennis Tarnow https://orcid.org/0000-0002-0805-5802
France Lambert https://orcid.org/0000-0002-1018-2544
Daniel Buser https://orcid.org/0000-0003-3920-4257

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