BULLETIN OF STOMATOLOGY AND MAXILLOFACIAL SURGERY
Volume 21, Issue 8

DOI: 10.58240/1829006X-2025.21.8-159

REVIEW ARTICLE

UNDERSTANDING, PREVENTING, AND MANAGING INITIAL BONE REMODELING AROUND DENTAL IMPLANTS: A REVIEW OF CURRENT EVIDENCE

Teresa Lombardi DDS[1], Fabio Bernardello MD, DDS[2], Claudio Stacchi DDS, MSc, FEBOS[3], Antonio Rapani DDS, MSc[4], Sergio Spinato DDS, PhD[5].

[1]Department of Medical and Surgery Specialties, Radiological Sciences and Public Health, University of Brescia, 25123 Brescia, Italy. drteresalombardi@libero.it; ORCID #0000-0002-0334-6029

[2]Private Practice, 37045 Terranegra di Legnago (VR), Italy. fabio.tredici@libero.it; ORCID #0000-0001-6432-1549

[3]Department of Medical, Surgical and Health Sciences, University of Trieste, 34100 Trieste, Italy. claudio@stacchi.it; ORCID # 0000-0003-4017-4980

[4]Department of Medical, Surgical and Health Sciences, University of Trieste, 34100 Trieste, Italy. rapani.antonio@gmail.com; ORCID # 0000-0002-9767-4401

[5]Private Practice, 41049 Sassuolo (MO), Italy. albispina@tiscali.it; ORCID #0000-0002-9014-8732

***Corresponding Author:** Claudio Stacchi DDS, MSc, FEBOS Department of Medical, Surgical and Health Sciences, University of Trieste, 34100 Trieste, Italy. claudio@stacchi.it; ORCID # 0000-0003-4017-4980

***Received:** Jul 28, 2025; **Accepted:** Aug 28, 2025; **Published:** Sep 7, 2025*

ABSTRACT

Initial bone remodeling (IBR) around dental implants is a multifactorial process influencing long-term success. While historical criteria allowed up to 2 mm marginal bone loss in the first year, recent evidence indicates that resorption >0.5 mm within 6–12 months significantly increases long-term peri-implantitis risk. IBR, defined as dimensional bone changes from implant placement through one year of loading, is non-infective in origin but may predispose to bacterial colonization if implant treated surface becomes exposed.

Key anatomical determinants include bone density, crestal bone width, and supracrestal tissue height. High-density cortical bone, insufficient buccal/palatal bone envelope, or thin supracrestal mucosa correlate with greater marginal bone resorption. Implant characteristics such as internal conical connections, platform switching, optimized crest module geometry, and favorable transmucosal profiles can reduce microbial leakage, optimize load distribution, and promote long-term crestal bone stability.

A critical surgical factor is apico-coronal placement of the implant according to mucosal vertical thickness, ensuring bone remodeling during supracrestal tissue adhesion occurs coronal to the platform. Depth varies with tissue phenotype and requires precise assessment. Additional measures include controlling intraosseous temperature and avoiding excessive insertion torque to limit cortical compression.

In the prosthetic phase, risk factors include repeated abutment dis/reconnections, abutment height <2 mm in bone-level implants, excess cement, and excessively convex emergence profiles. Mitigation strategies involve "one abutment–one time" protocols or the use of tissue-level implants, screw-retained or cement-free retention, abutments >2 mm in height, and emergence profiles that promote soft tissue stability and facilitate plaque control.

Integrating biology-driven site preparation, accurate apico-coronal positioning, selection of macro- and micro-designs with documented bone-preserving properties, and evidence-based prosthetic protocols can minimize IBR. This multidisciplinary approach shifts the objective from accepting early marginal bone loss to its prevention, improving the predictability, longevity, and biological stability of implant-supported rehabilitations.

***Keywords**:* dental implant, implant therapy, marginal bone levels, implant functional loading

INTRODUCTION

The stability of marginal bone levels has always been considered among the key parameters for assessing the long-term success of implant therapy. Traditionally, radiographic marginal bone loss of up to 1.5–2.0 mm during the first year of functional loading, followed by a maximum annual loss of 0.2 mm, was widely accepted as an indicator for implant success[1-4]. However, advances in implant design, surface modification, prosthetic connections, and clinical protocols have significantly improved outcomes in implant dentistry, suggesting that these traditional thresholds may no longer represent the most reliable criteria for success in contemporary practice.

Recent evidence highlights the critical role of maintaining stable marginal bone levels during the healing phase after implant placement and throughout the first year of functional loading.

Galindo-Moreno et al. identified peri-implant bone loss exceeding 0.44 mm at six months post-loading as a predictor of progressive bone loss over time[5]. In a 10-year prospective study, Windael et al. reported that marginal bone resorption ≥ 0.5 mm after one year of function was associated with a 5.43-fold increase in the odds of developing peri-implantitis, with the risk further amplified when early bone loss exceeded 1–2 mm or occurred in combination with smoking and/or a history of periodontitis[6] (Fig. 1). More recently, a radiographic marginal bone loss threshold of 0.5 mm at six months following prosthetic loading has been proposed as a potential objective criterion for defining the success of osseointegrated implants[7].

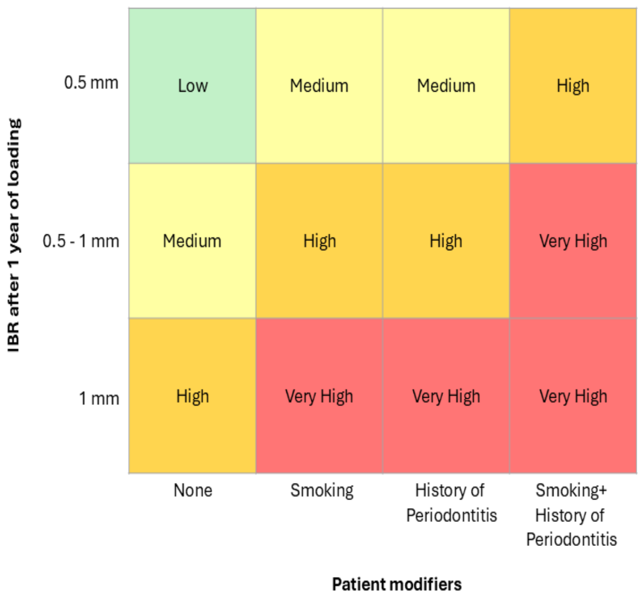

Figure 1. Risk matrix for peri-implantitis likelihood according to Initial Bone Remodeling (IBR) after 1 year and patient-related modifiers (smoking and history of periodontitis).

As the prevalence of peri-implantitis is quite high (10.3%) also in pristine bone[15], representing the major cause of late implant failure, its prevention is crucial to enhance long-term success of implant therapy. Nowadays, a better knowledge of the biological principles underlying peri-implant hard and soft tissue healing, together with the selection of appropriate implant features and sound clinical protocols, may permit to minimize and manage the initial breakdown of the bone-implant interface at crestal level.

The present paper aims to summarize the possible causes of IBR and suggest a comprehensive clinical strategy to minimize initial marginal bone resorption around dental implants. In particular, the influence on IBR of site-specific characteristics, macro- and micro-implant design, surgical protocols, and prosthetic procedures will be reviewed.

1. SITE-SPECIFIC CHARACTERISTICS

1.1 Bone Density

Bone architecture at planned implant site should be carefully evaluated and surgical technique has to be adapted to the anatomical conditions. Cortical bone provides mechanical support to the implant and allows to obtain high primary stability, whilst trabecular bone is the main source for mesenchymal progenitor cells and blood supply necessary for osseointegration. These two bone components present distinct mechanical and biological properties, heavily influencing their response to surgical trauma and functional loading. Among them, mean Young's modulus of trabecular bone (10.4 ± 3.5 GPa) was demonstrated to be significantly lower than that of cortical bone (18.6 ± 3.5 GPa)[16], indicating a different elastic response of the two tissues. This factor is reflected by their biological response: cortical bone, due to its low deformation properties, is much more susceptible than trabecular bone to stress and strain application. At crestal level, where cortical is usually the main bone component, excessive stress and strain may create microfractures promoting osteocyte apoptosis and subsequent bone resorption. Apoptotic osteocytes play a crucial role in osteoclastogenesis through a direct pathway (production of chemotactic factors recruiting osteoclasts and increasing of RANKL levels)[17]. This biological mechanism is also amplified by the fact that areas with apoptotic osteocytes do not produce osteoclast inhibitory signals[18]. Moreover, limited blood supply and poor cellularity typical of cortical bone reduce the availability of osteogenetic cells in the bone remodeling area. In some situations, the minimal density of osteoblastic cells necessary for bone formation may not be reached, creating areas where only bone resorption can occur[19]. Therefore, all clinical procedures should be always conducted with the aim to limit surgical trauma and avoid occlusal overload to preserve cortical bone and prevent IBL. In particular, when high-density bone is present at implant site, special attention should be paid in surgical and prosthetic planning.

1.2 Bone Crest Width

Horizontal dimension of the edentulous bone crest plays a pivotal role on peri-implant bone stability. An adequate bone envelope surrounding implant at placement may effectively prevent IBR by counteracting the action of the various factors which negatively influence the stability of peri-implant bone levels. Nonetheless, there is no agreement in the literature on the minimal buccal and palatal bone thickness necessary to preserve peri-implant bone after implant placement. Belser and co-workers suggested to maintain at least 1 mm of buccal bone at implant positioning[20], Spray et al. recommended buccal plate thickness ≥1.8 mm[21], other studies suggested a buccal bone envelope ≥2.0 mm[22-24] or even ≥2.5 mm[25]. Opposite results come from a prospective study by Mehreb and co-workers, reporting stability of marginal bone levels also for initially thin (<1 mm) buccal plates[26]. However, the majority of these studies are narrative reviews, expert opinions or investigations conducted with questionable methodology and/or small sample size. Recently, a multi-centre prospective study investigated the influence of buccal and palatal bone thickness at the time of implant placement on horizontal and vertical IBR during the submerged healing period, with a strict control of the possible confounding factors[27]. Results suggest that bone envelope >2 mm on the buccal side and >1 mm on the palatal side may effectively prevent peri-implant vertical bone resorption following surgical trauma, avoiding early implant surface exposure. These findings are clinically relevant, as a previous study demonstrated that the presence of a buccal bone dehiscence at second stage surgery is significantly correlated with more apically located buccal bone level at 10 years[28].

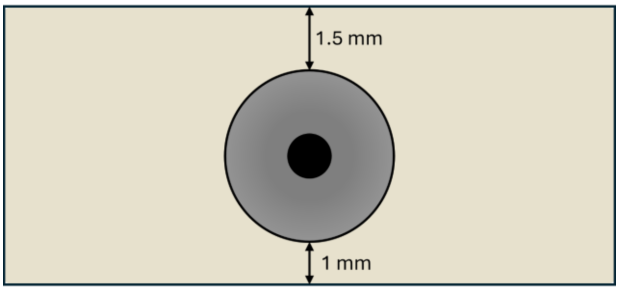

Figure 2. Schematic representation of minimum crestal bone width requirements for long-term peri-implant stability.

A buccal bone thickness >1.5 mm and a palatal/ lingual bone thickness >1.0 mm are associated with reduced risk of IBR and implant surface exposure.
Even if available data still do not support sufficient evidence to define precise thresholds, a rational choice of implant diameter eventually coupled with regenerative procedures should be planned to obtain an adequate peri-implant bone envelope helping to maintain marginal bone stability and obtain optimal aesthetic outcomes[29] (Fig. 2).

1.3 Supracrestal Tissue Height

Biological width is a histologic concept which has been described around natural teeth and may be defined as the physiological and stable vertical space extended from the alveolar crest to the gingival margin, including sulcular epithelium, junctional epithelium and connective tissue attachment[30]. The traditional term “biological width” has recently been replaced by the term “supracrestal tissue attachment”[31]. Similarly, the biological width around dental implants has been redefined as “supracrestal tissue height” (STH)[32], while the process of its establishment around the implant is more recently referred to as “supracrestal tissue adhesion” (STAd)[33]. Peri-implant supracrestal tissue height is the vertical dimension of the soft tissue surrounding an implant from the crestal bone to the mucosal margin. Specifically, in corono-apical direction, STH includes: 1- the sulcular epithelium between the peri-implant mucosal margin and the most coronal point of junctional epithelium; 2- the junctional epithelium; 3- the supracrestal connective tissue between the most apical point of the junctional epithelium and the first bone-to-implant contact[34]. Histologic studies in humans indicated that vertical dimension of STH around two-piece implants ranged between 1.8 and 3.6 mm, being stable and mature after an 8-week healing period[35,36]. Tissue-level implants presented a smaller mean STH (about 2.5 mm): this difference was determined by a variation in the mean vertical dimension of supracrestal connective tissue (1.24 mm for tissue-level versus 1.87 mm for bone-level implants), with the epithelial portion presenting the same dimension[35].

Based upon these premises, initial mucosal dimensions should be carefully evaluated as they could significantly influence IBR during STAd. Berglundh and Lindhe (1996) demonstrated in an animal model that, in presence of thin mucosa, STAd consistently included marginal bone resorption to allow the establishment of a stable soft tissue attachment[37]. Linkevicius and co-workers confirmed these preliminary findings with numerous clinical studies on bone-level implants, showing that the presence of thin mucosa (≤2 mm vertical thickness) at the time of implant placement is significantly correlated with the development of crestal bone loss during STAd[38-40].

Further supporting this concept, recent studies have shown that during STAd, tissue-level implants also exhibit greater IBR at sites with thin mucosa (≤ 2.5 mm vertical thickness) compared with sites with medium or thick mucosa, prior to crown delivery and prosthetic loading[41,42]. Therefore, adequate clinical surgical and prosthetic strategies are required to limit and, possibly, overcome this problem.

2. IMPLANT CHARACTERISTICS

2.1 Implant-Abutment Connection

In two-piece implants, IBR is influenced by the presence of a micro-gap at the implant-abutment interface[43]. This internal space of variable dimensions is rapidly colonized by bacteria after abutment connection[44], determining a local inflammatory reaction characterized by connective tissue infiltration by neutrophils, lymphocytes, macrophages and plasma cells (Fig. 3). This infiltrated connective tissue (ICT) surrounds three-dimensionally the implant-abutment junction and, in external hex implants, extends about 1.5 mm high and 0.5 mm wide from the micro-gap[45]. ICT leads to marginal bone resorption within four weeks from the exposure of the implant-abutment junction to the oral cavity[46].

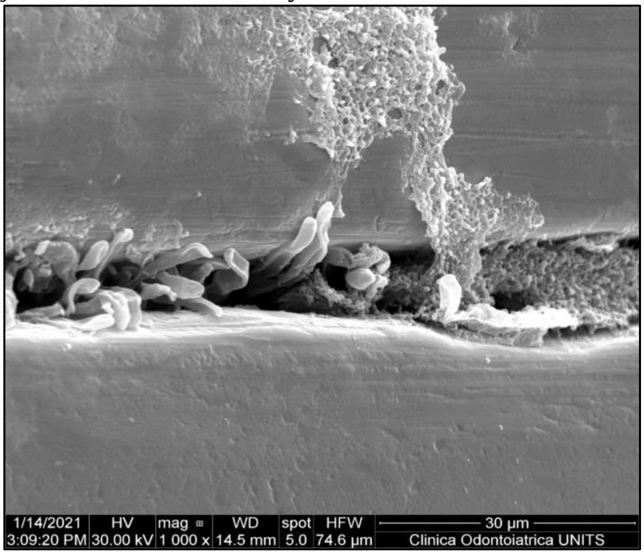

Figure 3 Microscopic image of the implant–abutment interface showing the microgap and the presence of bacterial infiltrate.

Mean micro-gap dimension varies among the different connection types, being significantly greater in external hex connections (10-50 μm) than in internal connections (<10 μm)[47]. The consequence is that crestal bone levels are better maintained in the short to medium term when internal connections are adopted. Furthermore, marginal bone loss around implants with internal, conical connections, is significantly lower than around implants with internal, non-conical connections[48]. An additional strategy to preserve peri-implant bone level is to increase the distance from the micro-gap (with the surrounding ICT) to the crestal bone, with the aim to reduce marginal bone resorption. This concept, named platform switching, was proposed by Gardner and Lazzara in the early 2000's[49,50], and implies the use of an abutment narrower than the implant platform, with the resulting mismatch determining a greater horizontal distance between the bacterial leakage originating from the micro-gap and the crestal bone. The clinical effectiveness of platform-switching design in the limitation of crestal bone loss around two-piece implants was subsequently demonstrated by numerous studies both for equicrestal and subcrestal placement[51-53]. However, a meta-analysis by Atieh and co-workers highlighted that the amount of marginal bone resorption is inversely correlated to the extent of implant-abutment mismatch and, particularly, that a difference ≥0.4 mm between implant and abutment diameters is needed to minimize marginal bone resorption[54].

Implant-abutment connection mechanical stability is another important factor influencing IBR. The application of functional loading may lead to elastic deformation of implant-abutment interface, enlarging micro-gap and allowing fluid infiltration into the inner part of the implant. This fluid, containing high concentrations of bacteria, endotoxins and acids, is pumped by cyclical opening and closing of the microgap, contaminating the surrounding peri-implant tissue[55,56]. Moreover, it was demonstrated that micro-gap dimension increases after the application of cycling loading (> 200.000 cycles) due to wear of metal surfaces[57]. As a result of this degradation, titanium particles are a common finding in soft and hard peri-implant tissue biopsies and their role in the pathogenesis of peri-implant diseases is currently under investigation[58,59]. Numerous comparative studies demonstrated that conical connections showed no or reduced micro-gap enlargement during dynamic loading, when compared with internal flat connections, with external hex connections showing the worst biomechanical performance[60-62].

A completely different option to eliminate the detrimental effects of micro-gap and poor connection stability on crestal bone is represented by the use of tissue-level implants. The absence of a micro-gap at crestal bone level can limit peri-implant bone resorption and guarantee a better peri-implant connective tissue organization compared with bone-level implants. The substantial bone stability around tissue-level implants is confirmed in literature by numerous studies: a retrospective analysis conducted on 1692 tissue-level implants reports mean marginal bone loss of 0.14 ± 0.41 mm, and 0.17 ± 0.45 mm after 7 and 9 years of loading, respectively[9].

2.2 Implant Crest Module

The crest module of an implant, defined as the most coronal portion of the fixture, represents the transition between the implant body and the prosthetic component (abutment or crown). Its location varies according to implant positioning: completely endosseous in subcrestal implants, transosteal in equicrestal implants, and transmucosal in tissue-level implants. Variations in the geometry and surface characteristics of the crest module influence how occlusal forces and related stresses are transferred to the surrounding bone. Since cortical bone—where most of these forces are dissipated—is more resistant to compression than to tension (critical thresholds of 170 and 100 MPa, respectively), optimizing load transfer is crucial to preserving marginal bone[63]. Numerous finite element method (FEM) analyses, particularly on equicrestal implants, have examined the relationship between crest module design and stress distribution[64]. There is general agreement that a parallel-walled crest module is the least favorable configuration for cortical load transfer. Evidence is more controversial when comparing divergent and convergent crest modules. Some FEM analyses indicate that a divergent geometry can reduce the transmission of detrimental tensile and shear forces to the cortical bone[10,65,66]. In contrast, Bozkaya and co-workers, using a bone overload criterion, found that at moderate loads (100–300 N) both designs maintained stress levels within the bone's physiological limits, whereas at high loads (1000–1200 N) only convergent necks avoided compression overload in the crestal bone[67] (Fig. 4). Based on these findings, convergent crest modules may offer a biomechanical advantage under heavy occlusal forces, whereas divergent designs—despite their potential benefits in reducing tensile and shear stresses—present additional drawbacks, including the need for a wider alveolar crest and highly precise cortical preparation to avoid excessive compression during implant insertion. The surface texture of the crest module is also a key factor in limiting IBR. There is broad consensus that a moderately rough titanium surface (1 µm < Ra < 2 µm)[68], by increasing bone-to-implant contact, can reduce stress magnitude and promote the transfer of beneficial compressive rather than detrimental shear forces to the cortical bone when compared with smoother surfaces[10,69].

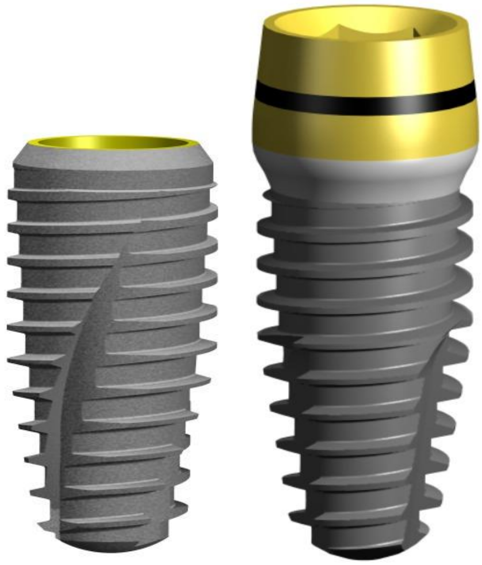

Figure 4 Two dental implants—bone-level (left) and tissue-level (right)—both featuring a convergent crestal module.

However, greater roughness may also facilitate bacterial biofilm adhesion if the collar becomes exposed in the oral cavity. Achieving an optimal balance between osseointegration quality and load distribution on one side, and minimizing plaque accumulation on the other, is essential for long-term success. To address this, minimally rough crest modules (0.5 µm < Ra < 1 µm)[68] have been incorporated into hybrid implants[70].

Tissue-level implants, in which the crest module is located transmucosally, display different biomechanical behavior under load[71,72]. FEM studies have shown that, compared with bone-level designs, tissue-level implants distribute lower compressive and tensile stresses to the peri-implant cortical bone under both vertical and oblique forces[72]. Furthermore, a concave or convergent transmucosal profile appears to be associated with reduced IBR compared with parallel or divergent profiles[73,74].

3. SURGICAL STRATEGIES

3.1 Avoid Bone Overheating During Site Preparation

Thermal injury to cortical bone is a well-documented cause of osteocyte apoptosis, osteoclastic activation, and subsequent marginal bone resorption. Bone heating above the critical threshold of 47°C for more than one minute can irreversibly compromise cell viability and osseointegration[75]. The risk is greater in dense cortical bone due to its low vascularity and reduced thermal conductivity. Therefore, osteotomy preparation should be performed with sharp drills, copious irrigation, intermittent drilling with minimal pressure, and controlled rotational speed. Special attention is needed when preparing dense bone: in such cases, the use of well-irrigated piezoelectric devices or low-speed drilling can help further reduce temperature rise and minimize the risk of thermal injury[76].

3.2 Prevent Excessive Cortical Compression at Implant Insertion

Overcompression of cortical bone during implant placement can generate microfractures, disrupt vascular supply, and trigger inflammatory-mediated bone resorption[77]. This risk increases in high-density bone and in cases with a thin buccal or palatal plate, where stress concentration is higher[78]. To avoid excessive compression, final osteotomy diameter should be carefully matched to implant dimensions according to bone density, and insertion torque should be monitored—maintaining values generally below 50 Ncm to achieve primary stability without overloading the cortical layer. In dense bone, the use of cortical taps or countersinks may reduce insertion stress, while in softer bone, underpreparation can still be applied strategically to enhance stability without inducing excessive cortical compression.

3.3 Position the Implant at the Optimal Apico-Coronal Level

A fundamental surgical objective during STAd is to maintain complete bone coverage of the treated implant surface, thereby preventing its exposure to the oral environment — a condition that promotes bacterial biofilm formation and increases the risk of peri-implant disease[6]. This can be predictably achieved by calibrating the apico-coronal position of the implant according to the preoperative measurement of STH with a tissue probe (Fig. 5):

- thick mucosa (> 3.5 mm): position the implant platform at crestal level or slightly subcrestal; the available soft tissue height allows STAd to be completed without marginal bone resorption, ensuring the treated surface remains fully covered;
- medium mucosa (2.5–3.5 mm): position the platform approximately 1 mm below the bone crest; this depth accommodates the limited marginal bone resorption expected in this soft tissue condition during STAd, which will occur entirely above the platform;
- thin mucosa (< 2.5 mm): position the platform approximately 2 mm subcrestal; this ensures that the marginal bone resorption needed for STAd takes place completely above the implant platform, maintaining bone coverage of the treated surface.

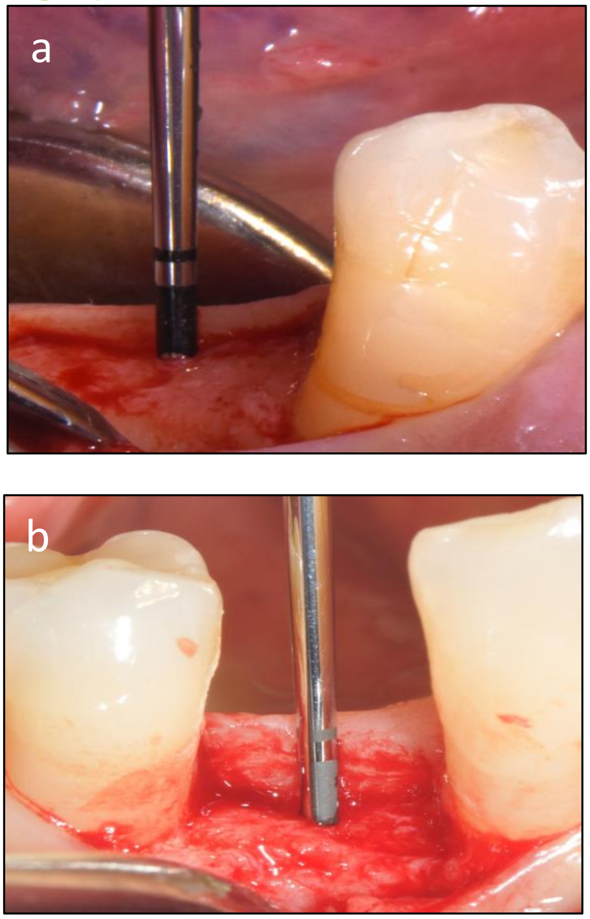

Figure 5 Soft tissue thickness at the implant site preparation. a) Thin tissues (< 2.0 mm). b) Thick tissues (≥3.5 mm).

By adapting implant depth to soft tissue thickness, bone resorption during STAd is confined to the peri-implant bone coronal to the platform (bone remodeling), avoiding progression to the peri-implant bone apical to the platform (bone loss)[79]. This approach is effective only when using implant–abutment connections with minimal microgap-induced resorption. External hex designs are unsuitable, as the inflammatory infiltrate at the microgap predictably causes bone loss down to the platform[80]. A platform-switched internal connection — preferably conical — provides optimal conditions for preserving crestal bone stability and maintaining long-term bone coverage of the treated implant surface.

4. PROSTHETIC PROCEDURES

Even when surgical procedures are performed in full compliance with biological principles aimed at preserving peri-implant bone stability, the prosthetic phase still represents a critical period for marginal bone preservation. Several technical and biological factors inherent to prosthetic procedures — if not properly managed — can trigger inflammation through different biological mechanisms, potentially leading to marginal bone loss. Careful attention to these aspects is therefore essential to ensure that the stability achieved during surgery is maintained.

4.1 Abutment Disconnections/Reconnections

In bone-level implants placed at or below the crestal level, repeated connection and disconnection of healing or provisional abutments should be strictly limited. Multiple abutment manipulations—commonly performed for impression taking, prosthesis try-in, or adjustments—have been shown to induce measurable peri-implant marginal bone resorption[10,81,82]. This may result from both the disruption and repeated re-establishment of the peri-implant soft tissue seal and from potential microbial contamination of the implant cavity (e.g., via saliva) during clinical procedures[83]. The deeper the implant is positioned, the more these adverse effects are amplified.

To mitigate this problem, current prosthetic protocols should be reconsidered. Possible strategies include selecting configurations that relocate the implant–abutment interface coronally, within the soft tissue zone and far from the crestal bone. This is the case for tissue-level implants, where the connection is inherently positioned transmucosally, well above the bone crest; in such designs, repeated connection and disconnection of prosthetic components can be performed without affecting the peri-implant bone interface or triggering marginal bone remodeling. Alternatively, for bone-level implants, adopting restorative approaches such as the "one abutment–one time" concept—where the definitive abutment is connected at the time of surgery and left undisturbed thereafter—can help maintain the integrity of the peri-implant soft tissue seal and reduce the risk of early bone resorption[84,85].

4.2 Prosthetic Abutment Height

Prosthetic abutment height — defined as the distance from the implant platform to the apical edge of the crown — has a decisive influence on marginal bone stability in bone-level implants placed at or below the crestal level. Short abutments (< 2 mm) position the prosthetic emergence profile too close to the peri-implant bone crest, encroaching upon the supracrestal tissue height (STH) that has recently formed during supracrestal tissue adhesion (STAd). This invasion of the biologic width can force a re-adaptation of the soft tissue seal, a process frequently accompanied by coronal bone remodeling and subsequent marginal bone resorption to recreate a stable STH.
Clinical evidence strongly supports this mechanism. Galindo-Moreno and colleagues first reported that multi-unit screw-retained implants restored with abutments longer than 2 mm exhibited better preservation of IBR compared with shorter abutments[86]. Subsequent studies extended this finding to both cement- and screw-retained single and multi-unit prostheses, consistently showing an inverse relationship between abutment height and the magnitude of IBR[87,88]. A different scenario applies to tissue-level implants, where the influence of abutment height on marginal bone stability is markedly reduced compared with bone-level designs[89]. In these implants, the traditional concept of abutment height is less applicable; the parameter of interest is the transmucosal collar height, defined as the distance from the bone crest to the most apical point of the crown margin. From an anatomical perspective, the mean supracrestal tissue height (STH) is significantly lower in tissue-level implants than in bone-level designs (2.55 ± 0.16 mm vs. 3.26 ± 0.15 mm)[35] and may be further reduced when a convergent transmucosal profile is used instead of a straight one[90,91]. Clinically, in bone-level implants the choice of abutment height—and consequently the vertical position of the crown margin—is made without precise knowledge of individual connective tissue height and sulcus depth, which increases the risk of encroaching upon the soft tissue seal. In contrast, with tissue-level implants the impression directly records the transmucosal portion of the fixture, allowing accurate reproduction of the emergence profile and precise crown margin placement within the gingival sulcus (Fig. 6). This workflow preserves the established connective tissue attachment and virtually eliminates the risk — common in bone-level designs — that an inappropriate abutment height could disrupt the seal, necessitate its re-adaptation, and cause marginal bone remodeling.

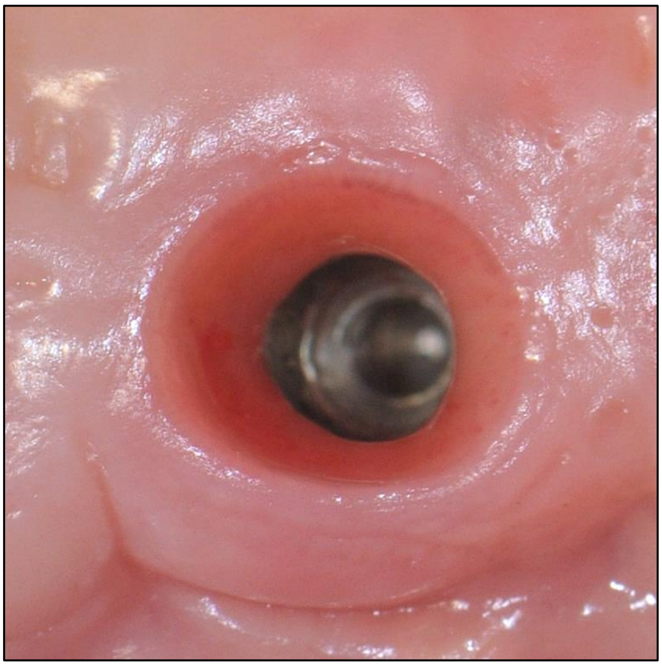

Figure 6 Bone-level implant (previous page) versus tissue-level implant (above). In bone-level implants, abutment height and crown margin are chosen without exact reference to connective tissue and sulcus depth, increasing the risk of soft tissue seal violation. Tissue-level implants, instead, allow direct recording of the transmucosal portion, ensuring accurate emergence profile and crown margin placement.

4.3 Prosthetic Retention

The type of prosthetic retention—cement-retained, screw-retained, or conometric—can exert a considerable influence on peri-implant tissue health and, consequently, on the extent of initial bone remodeling (IBR). Cement-retained restorations are associated with a significantly higher risk of peri-implant inflammation, largely due to undetected residual cement at the restoration margins[92,93]. This residual material can elicit an inflammatory response via two distinct mechanisms: (i) as a foreign-body stimulus, inducing a local immune reaction, and (ii) by providing an ideal substrate to bacterial adhesion and biofilm maturation. Both pathways may synergistically promote marginal bone resorption. A prospective clinical investigation on cemented restorations reported that 81% of peri-implantitis cases were attributable to cement remnants, and 74% of these lesions resolved following their mechanical removal[94]. Screw-retained prostheses effectively eliminate the risk of cement-associated inflammation, making them the preferred option in implant-supported rehabilitations. The primary limitation—compromised aesthetics when the screw access channel emerges on the buccal aspect—can be mitigated through careful three-dimensional implant positioning and, when available, the use of angulated screw channels. More recently, conometric retention systems have been introduced, providing a screw-free and cement-free alternative based on a precision friction-fit interface between the abutment and the restoration[95]. This configuration maintains retrievability while avoiding cement-associated risks and eliminating the prosthetic access channel, potentially offering biological and technical advantages. However, the current lack of robust long-term clinical evidence suggests caution until further studies confirm their long-term efficacy and stability.

4.4 Crown Emergence Profile

Although the evidence is still limited, current studies suggest that also crown emergence profile may play a role in influencing IBR around dental implants[96,97]. In particular, emergence profiles with a marked convexity—especially when the emergence angle exceeds 30°—may reduce the space available for soft tissue accommodation, create mechanical compression of the peri-implant mucosa, and hinder plaque control (Fig. 7). These conditions could trigger peri-implant soft tissue inflammation and subsequent bone remodeling aimed at re-establishing an adequate STH. Conversely, more concave or flatter emergence profiles may enhance peri-implant soft tissue stability, favor the formation of a thicker connective tissue layer, and facilitate access for oral hygiene, thereby potentially contributing to the maintenance of marginal bone levels. Despite being supported by preliminary clinical studies, these concepts require further research to establish definitive guidelines for optimal emergence profile design in implant-supported restorations.

Figure 7 Example of a definitive crown showing a convex emergence profile with an emergence angle greater than 30°.

CONCLUSIONS

Maintaining stable marginal bone levels is essential for the long-term success of implant therapy. Initial bone remodeling can be minimized through a combination of careful surgical execution, appropriate implant selection, and precise prosthetic management (Table 1).

Table 1. Main clinical determinants of initial bone remodeling (IBR) around dental implants, categorized into site-specific characteristics, implant-related features, surgical strategies, and prosthetic procedures.

Site-Specific Characteristics
• Bone Density • Bone Crest Width • Supracrestal Tissue Height
Implant Characteristics
• Implant-Abutment Connection • Implant Crest Module
Surgical Strategies
• Avoid Bone Overheating • Prevent Excessive Cortical Compression • Apico-Coronal Position
Prosthetic Procedures
• Abutment Disconnections/Reconnections • Prosthetic Abutment Height • Prosthetic Retention • Crown Emergence Profile

Key factors include preserving cortical bone during site preparation, ensuring an adequate bone envelope, and adapting implant depth to soft tissue thickness. Implant features such as a stable internal conical connection, favorable crest module design, and appropriate surface texture further support peri-implant bone preservation.

During the prosthetic phase, minimizing abutment manipulations, using adequate abutment height, avoiding residual cement, and designing restorations with favorable emergence profiles are all critical.

Rather than accepting marginal bone loss as inevitable, modern protocols integrating these principles can significantly reduce its incidence, enhancing the predictability and longevity of implant-supported rehabilitations.

DECLARATIONS

Ethics approval and consent to participate
Not applicable.

Conflict of interest
The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding
This research received no external funding.

REFERENCES

1. Albrektsson T, Zarb G, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: a review and proposed criteria of success. Int J Oral Maxillofac Implants. 1986;1(1):11-25.
2. Smith DE, Zarb GA. Criteria for success of osseointegrated endosseous implants. J Prosthet Dent. 1989;62(5):567-572.
3. Misch CE, Perel ML, Wang HL, Sammartino G, Galindo-Moreno P, Trisi P, Steigmann M, Rebaudi A, Palti A, Pikos MA, Schwartz-Arad D, Choukroun J, Gutierrez-Perez JL, Marenzi G, Valavanis DK. Implant success, survival, and failure: The International Congress of Oral Implantologists (ICOI) Pisa Consensus Conference. Implant Dent. 2008;17(1):5–15.
4. Papaspyridakos P, Chen CJ, Singh M, Weber HP, Gallucci GO. Success criteria in implant dentistry: a systematic review. J Dent Res. 2012;91(3):242-248.
5. Galindo-Moreno P, León-Cano A, Ortega-Oller I, Monje A, O'Valle F, Catena A. Marginal bone loss as success criterion in implant dentistry: beyond 2 mm. Clin Oral Implants Res. 2015; 26(4):e28-e34.
6. Windael S, Collaert B, De Buyser S, De Bruyn H, Vervaeke S. Early peri-implant bone loss as a predictor for peri-implantitis: A 10-year prospective cohort study. Clin Implant Dent Relat Res. 2021;23(3):298-308.
7. Galindo-Moreno P, Catena A, Pérez-Sayáns M, Fernández-Barbero JE, O'Valle F, Padial-Molina M. Early marginal bone loss around dental implants to define success in implant dentistry: A retrospective study. Clin Implant Dent Relat Res. 2022;24(5):630-642.
8. Cassetta M, Pranno N, Calasso S, Di Mambro A, Giansanti M. Early peri-implant bone loss: a prospective cohort study. Int J Oral Maxillofac Surg. 2015;44(9):1138-1145.
9. Kang MH, Jung UW, Cho KS, Lee JS. Retrospective radiographic observational study of 1692 Straumann tissue-level dental implants over 10 years. II. Marginal bone stability. Clin Implant Dent Relat Res. 2018;20(5):875-881.

10. Oh TJ, Yoon J, Misch CE, Wang HL. The causes of early implant bone loss: myth or science? J Periodontol. 2002;73(3):322–333.
11. Spinato S, Stacchi C, Lombardi T, Bernardello F, Messina M, Zaffe D. Biological width establishment around dental implants is influenced by abutment height irrespective of vertical mucosal thickness: A cluster randomized controlled trial. Clin Oral Implants Res. 2019;30(7):649-659.
12. Berglundh T, Armitage G, Araujo MG, Avila-Ortiz G, Blanco J, Camargo PM, Chen S, Cochran D, Derks J, Figuero E, Hämmerle CHF, Heitz-Mayfield LJA, Huynh-Ba G, Iacono V, Koo KT, Lambert F, McCauley L, Quirynen M, Renvert S, Salvi GE, Schwarz F, Tarnow D, Tomasi C, Wang HL, Zitzmann N. Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol. 2018;89(Suppl.1):S313-S318.
13. Qian J, Wennerberg A, Albrektsson T. Reasons for marginal bone loss around oral implants. Clin Implant Dent Relat Res. 2012;14(6):792-807.
14. Fetner M, Fetner A, Koutouzis T, Clozza E, Tovar N, Sarendranath A, Coelho PG, Neiva K, Janal MN, Neiva R. The effects of subcrestal implant placement on crestal bone levels and bone-to-abutment contact: A microcomputed tomographic and histologic study in dogs. Int J Oral Maxillofac Implants. 2015;30(5):1068-1075.
15. Heitz-Mayfield LJ, Aaboe M, Araujo M, Carrión JB, Cavalcanti R, Cionca N, Cochran D, Darby I, Funakoshi E, Gierthmuehlen PC, Hashim D, Jahangiri L, Kwon Y, Lambert F, Layton DM, Lorenzana ER, McKenna G, Mombelli A, Müller F, Roccuzzo M, Salvi GE, Schimmel M, Srinivasan M, Tomasi C, Yeo A. Group 4 ITI Consensus Report: Risks and biologic complications associated with implant dentistry. Clin Oral Implants Res. 2018;29(l 16):351-358.
16. Rho JY, Ashman RB, Turner CH. Young's modulus of trabecular and cortical bone material: ultrasonic and microtensile measurements. J Biomech. 1993;26(2):111-119.
17. Ru JY, Wang YF. Osteocyte apoptosis: the roles and key molecular mechanisms in resorption-related bone diseases. Cell Death Dis. 2020;11(10):846.
18. Insua A, Monje A, Wang HL, Miron RJ. Basis of bone metabolism around dental implants during osseointegration and peri-implant bone loss. J Biomed Mater Res A. 2017;105(7):2075-2089.
19. Kristensen HB, Andersen TL, Marcussen N, Rolighed L, Delaisse JM. Osteoblast recruitment routes in human cancellous bone remodeling. Am J Pathol 2014;184(3):778-789.
20. Belser UC, Bernard JP, Buser D. Implants in the esthetic zone. In: Lindhe J, Lang NP, Karring T, eds. Clinical Periodontology and Implant Dentistry.1146–1174. Oxford: Blackwell Publishing Ltd. 2008.
21. Spray JR, Black CG, Morris HF, Ochi S. The influence of bone thickness on facial marginal bone response: stage 1 placement through stage 2 uncovering. Ann Periodontol. 2000;5(1):119-128.
22. Buser D, Martin W, Belser UC. Optimizing esthetics for implant restorations in the anterior maxilla: anatomic and surgical considerations. Int J Oral Maxillofac Implants. 2004;19(l):43-61.
23. Chen ST, Darby IB, Reynolds EC. A prospective clinical study of non-submerged immediate implants: clinical outcomes and esthetic results. Clin Oral Implants Res. 2007;18(5):552-562.
24. Nohra J, Dagher M, Matni G, Mokbel N, Jobaili E, Naaman N. Effect of primary stability and soft- and hard-tissue thickness on marginal bone loss: A prospective pilot study. Implant Dent. 2018;27(5):542-546.
25. Grunder U, Gracis S, Capelli M. Influence of the 3-D bone-to-implant relationship on esthetics. Int J Periodontics Restorative Dent. 2005;25(2):113-119.
26. Merheb J, Vercruyssen M, Coucke W, Beckers L, Teughels W, Quirynen M. The fate of buccal bone around dental implants. A 12-month postloading follow-up study. Clin Oral Implants Res. 2017;28(1):103-108.
27. Cicciù M, Pratella U, Fiorillo L, Bernardello F, Perillo F, Rapani A, Stacchi C, Lombardi T. Influence of buccal and palatal bone thickness on post-surgical marginal bone changes around implants placed in posterior maxilla: A multi-centre prospective study. BMC Oral Health. 2023;23(1):309.
28. Schropp L, Wenzel A, Spin-Neto R, Stavropoulos A. Fate of the buccal bone at implants placed early, delayed, or late after tooth extraction analyzed by cone beam CT: 10-year results from a randomized, controlled, clinical study. Clin Oral Implants Res. 2015;26(5):492-500.
29. Tatarakis N, Bashutski J, Wang HL, Oh TJ. Early implant bone loss: preventable or inevitable? Implant Dent. 2012;21(5):379-386.
30. Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. J Periodontol. 1961;32(3):261-267.
31. Jepsen S, Caton JG, Albandar JM, Bissada NF, Bouchard P, Cortellini P, Demirel K, de Sanctis M, Ercoli C, Fan J, Geurs NC, Hughes FJ, Jin L, Kantarci A, Lalla E, Madianos PN, Matthews D, McGuire MK, Mills MP, Preshaw PM, Reynolds MA, Sculean A, Susin C, West NX, Yamazaki K. Periodontal manifestations of systemic diseases and developmental and acquired conditions: consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol. 2018;89(1):237-S248.

32. Avila-Ortiz G, Gonzalez-Martin O, Couso-Queiruga E, Wang HL. The peri-implant phenotype. J Periodontol. 2020;91(3):283–288.
33. Liu W, Misch J, Wang HL. The influence of supracrestal tissue adhesion on implant marginal bone level. Int J Oral Implantol 2023; 16(1):3-6.
34. Araujo MG, Lindhe J. Peri-implant health. J Periodontol. 2018;89(Suppl 1):S249-S256.
35. Judgar R, Giro G, Zenobio E, Coelho PG, Feres M, Rodrigues JA, Mangano C, Iezzi G, Piattelli A, Shibli JA. Biological width around one- and two-piece implants retrieved from human jaws. Biomed Res Int. 2014;2014:850120.
36. Tomasi C, Tessarolo F, Caola I, Wennström J, Nollo G, Berglundh T. Morphogenesis of peri-implant mucosa revisited: An experimental study in humans. Clin Oral Implants Res. 2014;25(9):997–1003.
37. Berglundh T, Lindhe J. Dimension of the peri-implant mucosa. Biological width revisited. J Clin Periodontol. 1996;23(10):971–973.
38. Linkevicius T, Apse P, Grybauskas S, Puisys A. Reaction of crestal bone around implants depending on mucosal tissue thickness. A 1-year prospective clinical study. Stomatologija. 2009;11(3):83-91.
39. Linkevicius T, Apse P, Grybauskas S, Puisys A. Influence of thin mucosal tissues on crestal bone stability around implants with platform switching: A 1-year pilot study. J Oral Maxillofac Surg. 2010;68(9):2272-2277.
40. Linkevicius T, Puisys A, Steigmann M, Vindasiute E, Linkeviciene L. Influence of vertical soft tissue thickness on crestal bone changes around implants with platform switching: A comparative clinical study. Clin Implant Dent Relat Res. 2015;17(6):1228-1236.
41. Spinato S, Bernardello F, Lombardi T, Soardi CM, Messina M, Zaffe D, Stacchi C. Influence of apico-coronal positioning of tissue-level implants on marginal bone stability during supracrestal tissue height establishment: a multi-centre prospective study. Clin Implant Dent Relat Res. 2022; 24(5):611-620.
42. Garaicoa-Pazmino C, Mendonça G, Ou A, Chan HL, Mailoa J, Suárez-López Del Amo F, Wang HL. Impact of mucosal phenotype on marginal bone levels around tissue level implants: A prospective controlled trial. J Periodontol. 2021;92(6):771-783.
43. Quirynen M, van Steenberghe D. Bacterial colonization of the internal part of two-stage implants. An in vivo study. Clin Oral Implants Res. 1993;4(3):158-161.
44. Jansen VK, Conrads G, Richter EJ. Microbial leakage and marginal fit of the implant-abutment interface. Int J Oral Maxillofac Implants. 1997;12(4):527-540.
45. Ericson I, Persson LG, Berglundh T, Marinello CP, Lindhe J, Klinge B. Different types of inflammatory reactions in peri-implant soft tissues. J Clin Periodontol. 1995;22(3):255-261.
46. Berglundh T, Lindhe J, Ericsson I, Marinello CP, Liljenberg B, Thomsen P. The soft tissue barrier at implants and teeth. Clin Oral Implants Res. 1991;2(2) 81-90.
47. Caricasulo R, Malchiodi L, Ghensi P, Fantozzi G, Cucchi A. The influence of implant-abutment connection to peri-implant bone loss: A systematic review and meta-analysis. Clin Implant Dent Relat Res. 2018;20(4):653-664.
48. Yu X, Han Y, Wang J. Is an internal tapered connection more efficient than an internal non-tapered connection? A systematic review and meta-analysis. J Prosthet Dent. 2020;124(4):431-438.
49. Gardner DM. Platform switching as a means to achieving implant esthetics. NY State Dent J. 2005;71(3):34-37.
50. Lazzara RJ, Porter SS. Platform switching: a new concept in implant dentistry for controlling postrestorative crestal bone levels. Int J Periodontics Restorative Dent. 2006;26(1):9-17.
51. Spinato S, Galindo-Moreno P, Bernardello F, Zaffe D. Minimum abutment height to eliminate bone loss: influence of implant neck design and platform switching. Int J Oral Maxillofac Implants. 2018;33(2):405–411.
52. Santiago JF Jr, Batista VE, Verri FR, Honório HM, de Mello CC, Almeida DA, Pellizzer EP. Platform-switching implants and bone preservation: A systematic review and meta-analysis. Int J Oral Maxillofac Surg. 2016;45(3):332–345.
53. Hsu YT, Lin GH, Wang HL. Effects of platform-switching on peri-implant soft and hard tissue outcomes: A systematic review and meta-analysis. Int J Oral Maxillofac Implants. 2017;32(1):e9–e24.
54. Atieh MA, Ibrahim HM, Atieh AH. Platform switching for marginal bone preservation around dental implants: A systematic review and meta-analysis. J Periodontol. 2010;81(10):1350-1366.
55. do Nascimento C, Miani PK, Pedrazzi V, Gonçalves RB, Ribeiro RF, Faria AC, Macedo AP, de Albuquerque RF Jr. Leakage of saliva through the implant-abutment interface: in vitro evaluation of three different implant connections under unloaded and loaded conditions. Int J Oral Maxillofac Implants. 2012;27(3):551-560.
56. Assenza B, Tripodi D, Scarano A, Perrotti V, Piattelli A, Iezzi G, D'Ercole S. Bacterial leakage in implants with different implant-abutment connections: an in vitro study. J Periodontol. 2012;83(4):491-497.
57. Blum K, Wiest W, Fella C, Balles A, Dittmann J, Rack A, Maier D, Thomann R, Spies BC, Kohal RJ, Zabler S, Nelson K. Fatigue induced changes in conical

implant-abutment connections. Dent Mater. 2015;31(11):1415-1426.
58. Fretwurst T, Buzanich G, Nahles S, Woelber JP, Riesemeier H, Nelson K. Metal elements in tissue with dental peri-implantitis: a pilot study. Clin Oral Implants Res. 2016;27(9):1178-1186.
59. Ivanovski S, Bartold PM, Huang YS. The role of foreign body response in peri-implantitis: What is the evidence? Periodontol 2000. 2022;90(1):176-185.
60. Sailer I, Sailer T, Stawarczyk B, Jung RE, Hämmerle CHF. In vitro study of the influence of the type of connection on the fracture load of zirconia abutments with internal and external implant-abutment connections. Int J Oral Maxillofac Implants. 2009;24(5):850-858.
61. Zipprich H, Weigl P, Ratka C, Lange B, Lauer HC. The micromechanical behavior of implant-abutment connections under a dynamic load protocol. Clin Implant Dent Relat Res. 2018;20(5):814-823.
62. Vinhas AS, Aroso C, Salazar F, López-Jarana P, Ríos-Santos JV, Herrero-Climent M. Review of the mechanical behavior of different implant-abutment connections. Int J Environ Res Public Health. 2020;17(22):8685.
63. Martin RB, Burr DB, Sharkey NA. Skelatal tissue mechanics. 1st ed. New York: Springer; 1998. p. 127-178.
64. Cervino G, Fiorillo L, Arzukanyan AV, Spagnuolo G, Campagna P, Cicciù M. Application of bioengineering devices for stress evaluation in dentistry: the last 10 years FEM parametric analysis of outcomes and current trends. Minerva Stomatol. 2020;69(1):55-62.
65. Shen WL, Chen CS, Hsu ML. Influence of implant collar design on stress and strain distribution in the crestal compact bone: a three-dimensional finite element analysis. Int J Oral Maxillofac Implants. 2010;25(5):901-910.
66. Costa C, Peixinho N, Silva JP, Carvalho S. Study and characterization of the crest module design: A 3D finite element analysis. J Prosthet Dent. 2015;113(6):541-547.
67. Bozkaya D, Muftu S, Muftu A. Evaluation of load transfer characteristics of five different implants in compact bone at different load levels by finite elements analysis. J Prosthet Dent. 2004;92(6):523-530.
68. Albrektsson T, Wennerberg A. Oral implant surfaces: Part 1: Review focusing on topographic and chemical properties of different surfaces and in vivo responses to them. Int J Prosthodont. 2004;17(5):536–543.
69. Cheng HY, Chu KT, Shen FC, Pan YN, Chou HH, Ou KL. Stress effect on bone remodeling and osseointegration on dental implant with novel nano/microporous surface functionalization. J Biomed Mater Res A. 2013;101(4):1158-1164.
70. Spinato S, Bernardello F, Sassatelli P, Zaffe D. Hybrid and fully-etched surface implants in periodontally healthy patients: A comparative retrospective study on marginal bone loss. Clin Implant Dent Relat Res. 2017;19(4):663–670.
71. Araki H, Nakano T, Ono S, Yatani H. Three-dimensional finite element analysis of extra short implants focusing on implant designs and materials. Int J Implant Dent. 2020;6(1):5.
72. Avağ C, Akkocaoğlu M. The evaluation of stress on bone level and tissue level short implants: A Finite Element Analysis (FEA) study. J Stomatol Oral Maxillofac Surg. 2023; 124(1S):101298.
73. Valente NA, Wu M, Toti P, Derchi G, Barone A. Impact of concave/convergent vs parallel/divergent implant transmucosal profiles on hard and soft peri-implant tissues: A systematic review with meta-analyses. Int J Prosthodont. 2020;33(5):553-564.
74. Agustín-Panadero R, Bermúdez-Mulet I, Fernández-Estevan L, Solá-Ruíz MF, Marco-Pitarch R, García-Selva M, Zubizarreta-Macho A, León-Martínez R. Peri-implant behavior of tissue level dental implants with a convergent neck. Int J Environ Res Public Health. 2021;18(10):5232.
75. Eriksson R, Albrektsson T. Temperature thresholds for heat induced bone tissue injury: A vital-microscopic study in the rabbit. J Prosthet Dent. 1983;50(1):101-107.
76. Lamazza L, Lollobrigida M, Vozza I, Palmieri L, Stacchi C, Lombardi T, De Biase A. Piezoelectric implant site preparation: Influence of handpiece movements on temperature elevation. Materials (Basel). 2020;13(18):4072.
77. Cha JY, Pereira MD, Smith AA, Houschyar KS, Yin X, Mouraret S, Brunski JB, Helms JA. Multiscale analyses of the bone-implant interface. J Dent Res. 2015;94(3):482–490.
78. Barone A, Alfonsi F, Derchi G, Tonelli P, Toti P, Marchionni S, Covani U. The effect of insertion torque on the clinical outcome of single implants: A randomized clinical trial. Clin Implant Dent Relat Res. 2016;18(3):588-600.
79. Spinato S, Lombardi T, Stacchi C. Bone remodeling and bone loss around subcrestal implants: Two distinct entities. Int J Periodontics Restorative Dent. 2023;43(4):410-411.
80. Weng D, Nagata MJ, Bosco AF, de Melo LG. Influence of microgap location and configuration on radiographic bone loss around submerged implants: An experimental study in dogs. Int J Oral Maxillofac Implants. 2011;26(5):941-946.
81. Koutouzis T, Gholami F, Reynolds J, Lundgren T, Kotsakis GA. Abutment disconnection/reconnection affects peri-implant marginal bone levels: A meta-analysis. Int J Oral Maxillofac Implants.

2017;32(3):575–581.
82. Vaténas I, Linkevičius T. One abutment one time vs. repeatable abutment disconnections in implants, restored with cemented / screw retained fixed partial dentures: Marginal bone level changes. A systematic review and meta-analysis. Stomatologija. 2021;23(2):35-40.
83. Becker K, Mihatovic I, Golubovic V, Schwarz F. Impact of abutment materials and dis-/re-connection on soft and hard tissue changes at implants with platform-switching. J Clin Periodontol. 2012;39(8):774-780.
84. Degidi M, Nardi D, Piattelli A. One abutment at one time: Non-removal of an immediate abutment and its effect on bone healing around subcrestal tapered implants. Clin Oral Implants Res. 2011;22(11):1303-1307.
85. Atieh MA, Tawse-Smith A, Alsabeeha NHM, Ma S, Duncan WJ. The One Abutment-One Time protocol: A systematic review and meta-analysis. J Periodontol. 2017;88(11):1173-1185.
86. Galindo-Moreno P, León-Cano A, Ortega-Oller I, Monje A, Suárez F, ÓValle F, Spinato S, Catena A. Prosthetic abutment height is a key factor in peri-implant marginal bone loss. J Dent Res. 2014;93(7 Suppl.):80S–85S.
87. Spinato S, Stacchi C, Lombardi T, Bernardello F, Messina M, Dovigo S, Zaffe D. Influence of abutment height and vertical mucosal thickness on early marginal bone loss around implants. A randomized clinical trial with an 18-month post-loading clinical and radiographic evaluation. Int J Oral Impl (Berl) 2020;13(3):279-290.
88. Blanco J, Pico A, Caneiro L, Nóvoa L, Batalla P, Martín- Lancharro P. Effect of abutment height on interproximal implant bone level in the early healing: A randomized clinical trial. Clin Oral Implants Res 2018;29(1):108–117.
89. Spinato S, Bernardello F, Stacchi C, Soardi CM, Messina M, Rapani A, Lombardi T. Marginal bone changes around tissue-level implants after prosthesis delivery: A multicenter prospective study. Clin Implant Dent Relat Res. 2025;27(3):e70071.
90. Huh JB, Rheu GB, Kim YS, Jeong CM, Lee JY, Shin SW. Influence of implant transmucosal design on early peri-implant tissue responses in beagle dogs. Clin Oral Implants Res. 2014;25(8):962-968.
91. Bolle C, Gustin MP, Fau D, Exbrayat P, Boivin G, Grosgogeat B. Early periimplant tissue healing on 1-piece implants with a concave transmucosal design: A histomorphometric study in dogs. Implant Dent. 2015;24(5):598-606.
92. Staubli N, Walter C, Schmidt JC, Weiger R, Zitzmann NU. Excess cement and the risk of peri-implant disease: A systematic review. Clin Oral Implants Res. 2017;28(10):1278-1290.
93. Dalago HR, Schuldt Filho G, Rodrigues MA, Renvert S, Bianchini MA. Risk indicators for peri-implantitis. A cross-sectional study with 916 implants. Clin Oral Implants Res. 2017;28(2):144-150.
94. Wilson TG Jr. The positive relationship between excess cement and peri-implant disease: A prospective clinical endoscopic study. J Periodontol. 2009;80(9):1388-1392.
95. Degidi M, Nardi D, Sighinolfi G, Piattelli A. The conometric concept: Definitive fixed lithium disilicate restorations supported by conical abutments. J Prosthodont. 2018;27(7):605-610.
96. Katafuchi M, Weinstein BF, Leroux BG, Chen YW, Daubert DM. Restoration contour is a risk indicator for peri-implantitis: A cross-sectional radiographic analysis. J Clin Periodontol. 2018;45(2):225-232.
97. Soulami S, Slot DE, van der Weijden F. Implant-abutment emergence angle and profile in relation to peri-implantitis: A systematic review. Clin Exp Dent Res. 2022;8(4):795-806.