Implant-Abutment Connections: A Review of Biologic Consequences and Peri-implantitis Implications

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Clinicians very often have seen marginal bone loss around dental implants at the crest level early on after implant placement and uncovering. Early clinical publications had suggested that this bone loss occurred during the first year of loading. Thus, numerous attempts have been made to minimize or eliminate such bone loss. However, the timing and reason for this bone loss are not always apparent. The objective of this study was to review the evidence regarding marginal bone loss around dental implants from the standpoint of biologic consequences to help understand marginal bone changes around dental implants. One hypothesis for the bone loss around these implants was related to the presence of bacteria in the interfaces between the implant and abutment connections. The literature was reviewed regarding the three major types of implant-abutment crestal connections, including butt-joint, platform-switched, and no interface (tissue-level or one-body). This review article revealed that 1.5 to 2.0 mm of bone loss occurred around bone-level, butt-joint connections when the interface was created because the microgap was wide enough for penetration and colonization of bacteria, and that this bone loss was not observed around implants with no interface because they did not have a contaminated interface at the bone crest. Many studies have shown an advantage in the amount of marginal bone resorption for implants with a platform-switched connection, and there appears to be a significantly different biologic reaction. Recent publications indicate that such contaminated implant-abutment connections might have an effect on peri-implantitis and failure over time. Int J Oral Maxillofac Implants 2017;32: 1296–1307. doi: 10.11607/jomi.5732

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Historically, marginal or crestal bone levels around dental implants have been used as a sign of health and to help indicate to the clinician whether further diagnosis or treatment may be indicated for that implant. Bone loss at the crest of the bone, however, occurs around many implants, and the timing and reason for this bone loss are not always apparent. Typically, so-called "bone-level implants," which historically were referred to as "submerged" or "two-stage" implants, are placed in the osteotomy such that the top of the implant is level with the crestal bone. For "tissue-level implants," historically referred to as "nonsubmerged" or "one-stage" implants, the border between the rough implant surface and the smoother transmucosal surface was placed at the crestal bone level, with the top of the implant either within or coronal to the soft tissues (Figs 1a and 1b). Generally, after some period of healing, the bone-level or submerged implants were uncovered at a second surgery and a healing abutment placed so that soft tissue healing could occur. Finally, a provisional and then a definitive crown were fabricated on the implant. During these processes, the soft tissues were manipulated by raising a mucosal flap or using a tissue punch, and the soft tissues were "shaped" using a provisional restoration. Radiographs can be taken at the time of implant placement and/or at the time of provisional restoration and/or at the time of definitive restoration. In the literature, when evaluating marginal bone levels, the "baseline" radiograph could be any one of these three radiographs: the one taken at implant placement, the one taken upon provisional restoration, or the one taken at definitive restoration. For this reason, evaluation of bone-level changes and

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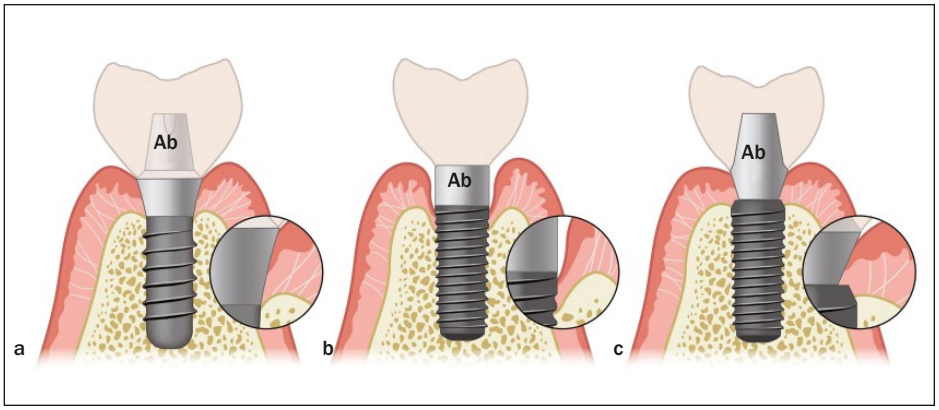

Fig 1 Three major types of implant-abutment crestal connections: *(a)* no interface (tissue-level or one-body implants), *(b)* butt-joint, and *(c)* platform-switched. Ab = abutment. Reprinted with permission from The University of Texas Health Science Center at San Antonio (UTHSCSA).

bone loss around the implant can be confusing. Furthermore, success criteria are often used based upon the amount of bone loss that occurs during the "first year" and during the time after the first year. Without an agreed-upon baseline time point, the literature varies greatly in the amount of bone loss that is reported around implants. This is further compounded by the fact that the greatest amount of bone loss that occurs around implants generally takes place soon after the implant receives a healing abutment or restoration. For these reasons, it is important to understand when and why marginal bone loss occurs around dental implants. Furthermore, marginal bone level changes are historically critical in determining whether or not an implant is experiencing so-called "peri-implantitis."

Historically, placement of dental implants as recommended by Brånemark et al was to be performed only by oral surgeons, placed in an operating room environment; implants were placed in the edentulous mandible to incorporate bicortical stabilization; and the taking of a radiograph was prohibited due to fear of damaging the bone cells responsible for osseointegrating the implant.[1] As this was the predominant type of implant used in the early days of endosseous cylindrical screw-type implants, the baseline radiograph was taken when the definitive restoration was placed on the implant and not at the time of implant placement. Any marginal bone loss that occurred prior to that point was not detected in these cases. Later on, it became evident that radiographs could be taken at the time of implant placement without interfering with osseointegration, and when these radiographs were used as the baseline, all marginal bone changes associated with the implant could be evaluated. Using radiographs taken at implant placement as the baseline and evaluating marginal bone changes that occurred following placement, it became obvious that predictable initial bone loss occurs early on after implant placement and uncovering. The reasons for such bone loss have been speculated on and include a "natural" remodeling after implant placement, surgical trauma, loss of blood supply, establishment of biologic width, a reaction to stress, occlusal loading, influence of disconnection/reconnection of the abutment, or a response to bacteria from contaminated implant components and peri-implant infections.[2,3] A response to bacteria and peri-implant infection are especially thought to be main factors for bone loss. Adding to the complexity of understanding marginal bone changes is the fact that several different types of implant-abutment (or crown) connections and resulting interfaces at the bone crest are utilized, including butt-joints, platform-switched connections, or no interfaces with tissue-level or one-body implants (Figs 1 and 2). Recently, many implant manufacturers have produced various implants with these types of connections (Table 1). In addition, changes have also occurred in the attachment mechanism of the abutment (and/or crown) to the implant that include different types of both external connections and internal connections, as well as connections held together by screws and/or Morse taper–like designs. The objective of the present study was to review the evidence regarding predictable initial marginal bone loss around dental implants from the standpoint

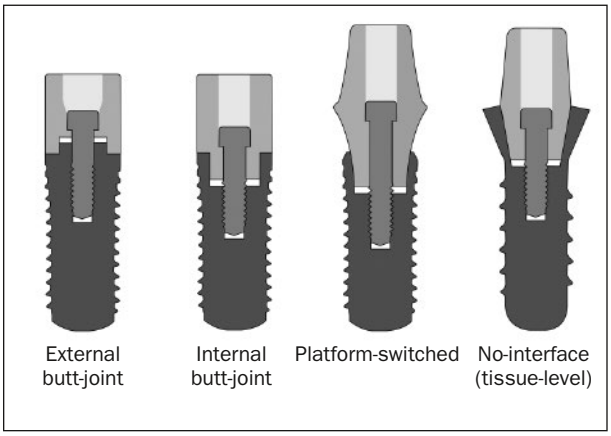

Fig 2 Different types of implant-abutment (or crown) connections at crestal bone: external butt-joints, internal butt-joints, platform-switched connections, and no interfaces (tissue-level or one-body). Reprinted with permission from UTHSCSA.

Table 1 Representative Manufacturers of Dental Implants and Their Availability of Products with Different Implant-Abutment Connections

	Connection		
Manufacturer	No interface	Butt-joint	Platform-switched
Biohorizons	×	×	×
Biomet 3i		×	×
Camlog		×	×
Dentsply	×	×	×
Nobel Biocare	×	×	×
Straumann	×		×
Zimmer Implant	×	×	×

of biologic consequences to help understand marginal bone changes around dental implants; to do this, it is best to examine different implant types and what bone changes occur, and why, around that type of implant. A working hypothesis for understanding marginal bone changes is that implant interfaces at the bone crest, contaminated with bacteria, such as the butt-joint interface, have localized infections that exist 360 degrees around the implant and that such localized infections, after initial consequences (typically 1.5 mm of bone loss), exist in equilibrium ("lying in wait") with the host (similar to gingivitis lesions) until a complication occurs that disrupts the equilibrium and causes more marginal bone loss due to an inflammatory front. This hypothesis should be interpreted in light of the fact that if bone is healthy, it does not remodel and lose volume (resorb). Rather, it exists in a steady state of equilibrium where bone resorption is balanced by bone formation and resorption only occurs when bone loss is not replaced by bone formation.

BUTT-JOINT CONNECTIONS

During the 1980s and 1990s, the predominantly placed dental implants were two-stage implants with an external hexagon (Brånemark implants) that were surgically submerged below the soft tissues and were uncovered at a stage-two surgery, where an abutment was placed. Binon[4] discussed these abutments: "The external hexagonal design, ad modum Brånemark, originally intended as a coupling and rotational torque transfer mechanism, consequently evolved by necessity into a prosthetic indexing and antirotational mechanism. The expanded utilization of the hexagonal resulted in a number of significant clinical complications." Another design feature at the time was to allow flexibility between implant components since it was felt that, like natural teeth that move due to the periodontal ligament, the implant needed to have a certain degree of mobility built into the design of the implant components. An example of compensating for tooth mobility was the "intramobile element" incorporated by one implant manufacturer. Khraisat et al[5] discussed external hexagons and their role as an antirotational element. These authors concluded, "The results indicated a direct correlation between the implant-abutment rotational misfit and the screw loosening." After a number of years, it became obvious that compensating for mobility of teeth is not important for implant restorations. After the submerged implant with an external hexagon had an abutment placed, a butt-joint connection was created at the interface between the implant and abutment. Many experimental studies and clinical case cohort studies documented that marginal bone loss occurred around these implants.[6–10] In fact, marginal bone loss occurred so routinely that Albrektsson et al[11] suggested that the success criteria for these implants included up to a mean 1.5 mm of marginal bone loss in the first year of restoration (because the baseline radiograph could only be taken at the time of restoration) and less than 0.2 mm of bone loss annually thereafter. Also noteworthy were many studies that documented that such interfaces become contaminated with bacteria (Table 2)[12–30] after the abutment is placed on the implant, which can cause inflammation.[31,32] Because the implant with the external hexagon and butt-joint connection was the predominant implant being placed, most assumed that the 1.5 mm of marginal bone loss that occurred in the first year of restoration (as a success criteria) applied to all dental implants. In addition, it was not known what, if any, bone changes occurred after implant placement, because the protocol at the time for these implants prohibited radiographs from being taken during, or immediately after, osteotomy preparation due to a fear that the radiograph may interfere with osseointegration.[1]

The important questions then became, did all implants lose 1.5 mm of marginal bone in the first year of restoration, and did bone loss occur prior to implant restoration? A study by Hermann et al[6] investigated if the 1.5 mm of marginal bone loss could be replicated in an experimental model. Also, because the bone loss occurred after stage-two surgery and crown placement, one suggestion was that the 1.5 mm of bone loss was associated with the creation of an interface (microgap) at the implant-abutment interface. To test this hypothesis, an experimental condition needed to be established that could demonstrate such bone loss. Also, a control was needed where there was no interface at the bone crest. In the study by Hermann

Table 2 Connection Type and Microbial Penetration

Author (year)	External hexagon	Internal hexagon	Platform-switched	Loading
Quirynen et al (1994)[12]	C			NL
Jansen et al (1997)[13]	C	C	C	NL
Gross et al (1999)[14]	C			NL
Piattelli et al (2001)[15]	C			NL
Steinebrunner et al (2005)[16]	C	C		L
Dibart et al (2005)[17]			NC	NL
Coelho et al (2008)[18]		C		NL
Tesmer et al (2009)[19]		C	C	NL
Harder et al (2010)[20]			C	NL
Aloise et al (2010)[21]			C	NL
do Nascimento et al (2011)[22]	C			NL
Teixeira et al (2011)[23]		C	C	NL
Assenza et al (2012)[24]		C	C	NL
do Nascimento et al (2012)[25]	C	C	C	L/NL
Jaworski et al (2012)[26]	C		C	NL
D'Ercole et al (2014)[27]		C	C	NL
Koutouzis et al (2014)[28]			C	L/NL
Koutouzis et al (2015)[29]			C	L
Ranieri et al (2015)[30]		C	C	NL

C = that type of connection was examined and was found to be contaminated with bacteria; NC = that type of connection was examined and was found not to be contaminated with bacteria; L = loading; NL = no loading.

et al, a tissue-level (one-stage or nonsubmerged implant) was used as a control since no interface was present at the bone crest. This study was able to demonstrate that 1.5 to 2.0 mm of bone loss did occur around bone-level, external-hexagon butt-joint implants when the interface was created (at stage-two surgery) or immediately, if the abutment was placed on the implant (interface created) at the time of first surgery (implant placement) and that when no interface existed (the tissue-level or one-body implants), no or minimal bone loss was observed. Thus, the 1.5 to 2.0 mm of bone loss was associated with the butt-joint interface being placed at the bone crest and was not observed around tissue-level implants (that did not have an interface at the bone crest). Furthermore, interestingly, if the interface was moved apically, more bone loss occurred, and if the interface was moved coronally, less bone loss was observed. In every case, however, this interface to first bone-to-implant contact was approximately 1.5 to 2.0 mm from the interface. Additionally, it was observed that the bone loss occurred relatively quickly and then became stable, which was similar to what had been observed in patients. While the experimental study conducted by Hermann et al was performed in animals, the findings reinforced what was observed clinically.

Buser et al[33] demonstrated in a long-term study of 65 patients with 97 tissue-level implants over 8 years that minimal to virtually no bone changes occurred in patients treated with tissue-level implants. This was further documented in a human clinical study by Hartman and Cochran,[34] who showed that the amount of bone loss was related to the location of the interface, relative to the crest of bone. The closer the implant-abutment interface to the original bone level, the more bone loss was observed. This appears to be a universal finding around butt-joint connections and has been shown by others as well.[7,35] Another important finding from the study by Hermann et al[6] was that the 1.5 to 2.0 mm observed bone loss occurred immediately when the implant-abutment interface was created, therefore demonstrating that occlusion did not play a role in the observed bone loss (also, the implants were never loaded in this experimental setup). Furthermore, because this experimental design evaluated bone changes from the time of implant placement, the 1.5 to 2.0 mm of bone loss was shown to occur when the interface was created. Early clinical publications had suggested that this bone loss occurred during the "first year of loading,"[11] because the baseline radiographs used were taken at the time of restoration and not at the time of implant placement. Therefore,

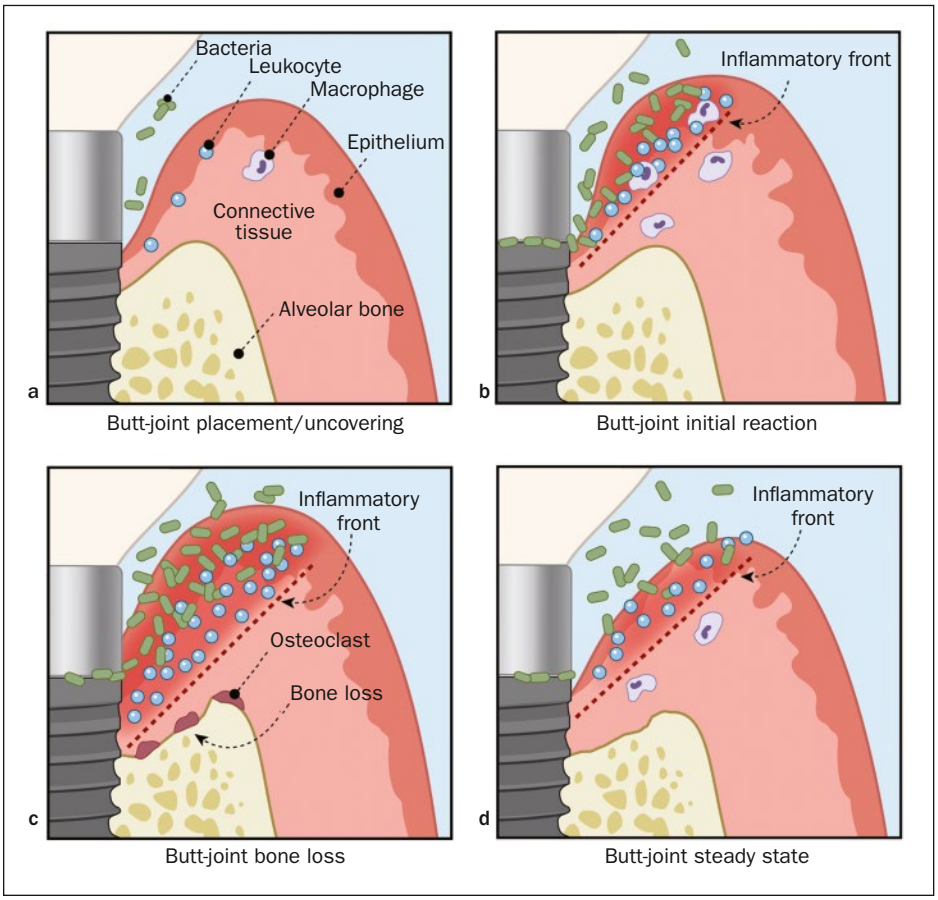

Fig 3 The spatial relationship between inflamed connective tissue (inflammatory front) and alveolar bone resorption around butt-joint interfaces. *(a)* A butt-joint connection is created at the interface between the implant and abutment. *(b)* As a butt-joint initial reaction, leukocytes in connective tissue are primarily found close to epithelium, and the interface becomes contaminated. *(c)* When the bacteria or their products penetrate further into connective tissue, the inflammatory front moves closer to peri-implant bone, where mediators produced by inflammatory cells stimulate osteoclastogenesis and bone resorption. *(d)* Once a "safe" distance (approximately 2 mm) has been created by the resorbing bone, an equilibrium can be established, but the bacteria remain in the interface. Reprinted with permission from UTHSCSA.

these earliest bone-level changes were not specifically evaluated in those studies. Importantly, therefore, the experimental design used in the Hermann et al study not only replicated what was observed clinically but provided, for the first time, new information as to when the bone changes actually occurred. Thus, the Hermann et al study demonstrated that the 1.5 to 2.0 mm of bone loss observed around bone-level, external-hexagon butt-joint connections (and documented as a success criteria by Albrektsson et al) should only be applied to this type of implant-abutment connection. Furthermore, the reason for the bone loss and then the stabilization of the bone crest was unknown.

One hypothesis for the bone loss around these implants was related to the presence of bacteria in the interface between the implant and abutment connection. Many publications have documented the presence of a reservoir of bacteria in the interface (Table 2), and as such, the host resists such an infection in a very predictable way. Graves and Cochran[36] described the presence of an inflammatory front that exists under such conditions and that the inflammatory front is responsible for the bone loss until a "safe" distance (approximately 2 mm) is created from the bone. Once such a distance has been created, an equilibrium can be established between the bacteria and their components and the host response, similar to the equilibrium that exists around teeth in gingivitis (Fig 3). This spatial relationship between inflamed connective tissue and alveolar bone resorption has been documented over many years.[37–39] This relationship between inflammation and bone also exists around teeth, and an interventional study demonstrating that inflammation causes some of the bone loss around teeth in an experimental periodontitis study reinforced this relationship.[40] In fact, an area of science referred to as "osteoimmunology" is dedicated to this relationship between inflammation and the immune system and bone and involves the science related to osteoclast development.[41,42]

Since it has been well documented that a contaminated microgap exists in the interface between the implant and abutment connection in the widely used external-hexagon butt-joint implant systems, as mentioned earlier, prevention of microbial leakage at the interface has been a challenge for the restoration of two-stage implants in order to minimize inflammatory reactions and to maximize bone stability around the implant. Thus, attempts have been made to minimize or eliminate the inflammation, which can cause the bone loss around the implant. One approach regarding this phenomenon is to decrease the size of the interface or microgap. Since the microgap is wide

enough for penetration and colonization of bacteria and it influences the level of crestal bone, it is possible that the size of the microgap may have an effect on the crestal bone level. This has formed the basis for research into implant system designs that provide an intimate seal at the implant-abutment interface trying to exclude the bacteria and reduce the amount of inflammation and bone loss. A study by Hermann et al[43] and King et al[44] investigated whether the size of the microgap (10, 50, or 100 μm) between the implant and abutment could influence the rate and amount of marginal bone loss in unloaded two-piece experimental butt-joint implants at the histologic and radiographic levels in the canine mandible. These papers concluded that the size of the butt-joint did not significantly influence the amount of bone loss observed around the interface, even with the most precise fit between implant components (10 μm); marginal bone loss could not be prevented. From a clinical point of view, these findings were reinforced in the study by Harder et al.[20] They investigated whether endotoxin could penetrate the interface between an implant and abutment in a platform-switched configuration instead of bacteria itself because it is well-known that endotoxin, a smaller molecular complex of lipopolysaccharides and proteins, is one of the most important toxins of gram-negative bacteria and plays a major role in bone destruction processes. It was reported that higher rates of leakage and a faster increase in endotoxin contamination occurred. Because the size of the endotoxin molecules can be smaller than 1 to 2 μm, the authors speculated that endotoxin could easily penetrate the interface rather than bacteria. It was also shown by Hermann et al[43] and King et al[44] that crestal bone changes were significantly influenced by possible movements between implants and abutments. These studies suggested that manufacturing small interfaces between butt-joint components would have a limited effect on the biologic consequences that result from the presence of the bacteria in the interface. However, these studies also suggested that making the implant-abutment interface more stable might be a promising goal to achieve.

PLATFORM-SWITCHED CONNECTIONS

Another potential way to prevent marginal bone loss is to physically move the implant-abutment interface horizontally. Thus, many manufacturers have changed the configuration from a butt-joint to an internal cone connection combined with a nonmatching implant diameter and a smaller-diameter abutment. These types of connections result in a horizontal offset at the implant-abutment interface and have been termed "platform-switched connections" (Fig 2). Lazzara and Porter[45] found better long-term preservation of marginal bone around wide-diameter dental implants connected with standard-diameter restorative components, due to a temporary coincidental commercial unavailability of a matching-diameter abutment. These radiographic observations led to the development of the platform-switched technique. This type of connection was reported radiographically to result in less marginal bone loss than the bone loss around butt-joint connections, where 1.5 to 2 mm of bone loss routinely occurs. Cochran et al[46] histologically and systematically evaluated the bone reaction to loaded bone-level implants that incorporated a platform-switched configuration (nonmatching implant-abutment diameter). The major finding of that study was that only minor bone loss was observed for platform-switched implants placed both submerged (below the soft tissues) and nonsubmerged (with a transmucosal abutment at the time of implant placement). The observed bone loss (0.34 and 0.38 mm for the submerged implants and nonsubmerged implants, respectively) for the implants placed at the bone crest level was much less than the 1.5 to 2.0 mm of bone loss that occurs around bone-level implants that have butt-joint connections.

Other studies[47–49] have confirmed that marginal bone loss around such diameter-mismatched components is significantly lower than around butt-joint components; however, the reason for this reduced bone loss is not clearly known. It can be speculated that since platform-switched implants displace the implant-abutment interface further from the alveolar bone, in theory, even if bacterial penetration were to occur in the interface, the bacteria would be at a greater distance from peri-implant bone. On the other hand, other possible reasons may exist for this phenomenon. Cochran et al[46,50] evaluated platform-switched implants histologically. The most significant consequence of their study was the fact that the connective tissue covered the implant-abutment interface (microgap) in some of these mismatched implant-abutment–diameter implants (Fig 4). This represented a paradigm shift in the host biologic reaction compared with implants with butt-joint connections, where the apical extension of the junctional epithelium is always located below the interface (microgap). They speculated that one reason for this major change in biologic reaction to mismatched implant-abutment configurations might be related to a reduction of bacteria and/or the stability provided by internal cone (Morse taper–like) connections rather than just the distance from the interface that is essential for this major change in tissue response.

Another possibility for this very different biologic reaction might be that an internal cone connection may be much more stable than an external connection,

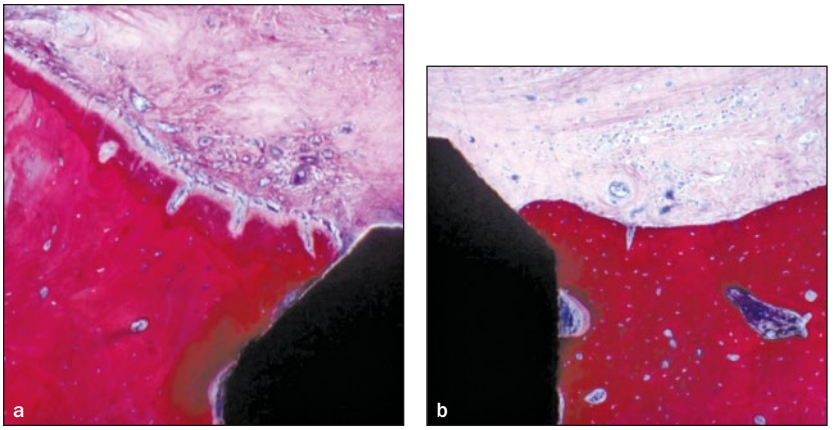

Fig 4 Two examples illustrating an implant-abutment interface covered by connective tissue around platform-switched implants. The horizontal (flat) aspect of the implant is only 0.2 mm; thus, the bone is extremely close to the interface between the implant and abutment. This figure is taken from Ref 46, reprinted with permission from the American Academy of Periodontology.

confirming the earlier studies by the same group (Hermann et al[43] and King et al[44]) that demonstrated that the stability of the components is important for maintenance of the crestal bone level. Cochran et al[50] concluded that the results from their study suggest that in clinical indications where bone quantity in a vertical dimension is critical (such as above the mandibular canal or below a sinus), a one-piece implant or an implant with a platform-switched connection would be favored over implants with butt-joint connections where a 1.5- to 2-mm loss of supporting bone will occur.

Recently, many implant manufacturers have produced implants with an internal cone for the abutment connection. The most stable cone connections utilize Morse taper–like connections. Such Morse taper systems in dental implantology use a concept developed originally by Stephen A. Morse in 1864 for tools and machinery.[51] The basic principle of the system, "a cone within a cone," results in not only improved sealing capacity of the interface between the abutment and internal walls of the body of the implant, but also an increased stability between the two components. A Morse taper is defined by the angle that the taper surfaces make relative to the longitudinal axis of the component and by the mismatch angle between the male and female part. The original Morse taper angle defined by Stephen Morse for tools was a relatively small angle of 2° 50, with the mathematical relation that tang 2° 50 = 5%. Most implants with internal connections use a higher percentage of taper, and thus, are not true Morse tapers. One huge advantage of a Morse taper–like connection can be that it takes more force to dislodge the components than it took to connect the components. Additionally, the screw threads of the abutment are not used for retention of the two components; rather, the screw threads serve only to locate the components so that the Morse taper–like connection occurs.

The capacity of preventing microbial penetration by Morse taper–like connections is somewhat controversial. Several studies have evaluated the potential microbial leakage of different implant–abutment connections, utilizing either nonloading or loading conditions, and all but one of them have reported some contamination with Morse taper–like connections (Table 2). Although Morse taper–like implants provided a better bacterial seal than external-hexagon implants, they seem to be unable to completely prevent bacterial leakage itself. Irrespective of the lack of a bacterial seal, implants with Morse taper–like connections have been proposed as an alternative to external-hexagon implants because it is clear that this type of connection increases the stability of the implant-abutment interface and decreases the occurrence of micromovements observed with the external connections.

From a clinical standpoint, many studies have shown an advantage in the amount of marginal bone resorption with implants having platform-switched connections. A systematic review of 10 studies with 1,239 implants was conducted by Atieh et al.[52] These 10 studies included only papers that directly compared platform-switched implants to implants without platform switching. The results of the analysis revealed a significant advantage for the platform-switched implants, and the authors concluded that platform-switched implants could be considered a desirable morphologic feature that may prevent horizontal saucerization and preserve vertical crestal bone levels. Al-Nsour et al[53] conducted a systematic review of nine articles to investigate the effect of platform switching on marginal bone loss. Although a meta-analysis was not conducted because of the heterogeneity of study designs and implant characteristics, they concluded that the use of abutments with a smaller diameter than their corresponding implant platforms seemed to exert beneficial effects on the peri-implant marginal bone. These authors mentioned that various factors, for example, the depth of implant placement, implant microstructure, the amount of platform-switch, and the reliability of examination methods, might influence the interpretation of the research data.

The coronal/apical level of implant placement in relation to the alveolar crest appears to be an important factor influencing marginal bone loss. Cochran et al[46] histologically evaluated the bone reaction to bone-level implants loaded for 6 months that incorporated a platform-switched configuration. They placed dental implants at three levels: even with the alveolar crest, 1 mm above the alveolar crest, and 1 mm below the alveolar crest in the canine mandible. The implants were submerged on one side of the mandible. On the other side, healing abutments were placed and exposed the implant to the oral cavity (nonsubmerged). These authors demonstrated that significantly less bone loss occurred when the dental implants were placed at the bone crest (–0.34 and –0.38 mm of bone change for the submerged and nonsubmerged implants, respectively) compared with the implants placed 1 mm below the alveolar crest (–1.29 and –1.13 mm of bone change for the submerged and nonsubmerged implants, respectively). Furthermore, there was bone gain around implants placed 1 mm above the bone crest (+0.04 and +0.29 mm of bone change for the submerged and nonsubmerged implants, respectively); relative to implants placed at the bone crest, this change was significantly different. Thus, the apical/vertical position of the implant is important in regard to marginal bone change.

Another potentially important factor influencing peri-implant marginal bone around platform-switched implants is the amount of the horizontal offset between the side of the abutment and the outer edge of the implant. However, this horizontal offset effect of platform switching on marginal bone level appears controversial. Canullo et al,[47] for example, conducted a randomized controlled trial aimed at evaluating marginal bone level change around platform-switched implants, using different implant-abutment mismatching. They showed that the greatest platform-abutment mismatch resulted in the least marginal bone loss and concluded that the degree of platform switching might significantly influence peri-implant marginal bone remodeling. On the other hand, however, a retrospective study including 228 platform-switched implants by Galindo-Moreno et al[54] demonstrated that greater mismatching did not minimize the marginal bone loss. Interestingly, they investigated not only horizontal mismatching but also vertical mismatching. They found that much less crestal bone loss occurred when the height of the abutment was 2 mm or greater. Therefore, it is not known if there is an indirect relationship between the amount of horizontal offset in a platform-switched implant and the amount of marginal bone loss. However, the importance of the apical/vertical positioning was reinforced.

Another factor, mucosal tissue thickness, has been shown to take part in the etiology of crestal bone loss as well. A study by Vandeweghe and De Bruyn[55] showed that the creation of a biologic width affects peri-implant bone loss to a significant extent and that platform switching is only effective when the mucosal thickness allows the establishment of a biologic width. Another study by Vervaeke et al[56] has also shown anticipation on the bone remodeling that occurs after implant placement in healed sites by adapting the vertical position of the implant to the thickness of the gingiva. A recent comparative clinical study by Linkevicius et al[57] concluded that in healed ridges, soft tissue thickness around platform-switched implants appears to influence marginal bone loss with thin-tissue patients (< 2 mm), losing more bone at 1 year compared with thick-tissue patients (> 2 mm). They reported that the thin group had marginal bone loss of 1.18 mm (minimum: 0.1 mm, maximum: 2.1 mm), while the thick group had a mean bone loss of 0.22 mm (minimum: 0.0 mm, maximum: 1.1 mm). Thirty-eight of 40 implants in the thin group had at least 0.5 mm of bone loss, while 6 of 40 implants in the thick group had more than 0.5 mm of bone loss at 1 year.

It is also important to note that not all Morse taper–like connections are the same in dental implants. The angle used varies, as does the length of contact of the cone, and these factors are determined by each manufacturer (Fig 5). From a mechanical standpoint, the smaller the Morse taper–like angle is, the higher the stability is (Fig 5a). Ranieri et al[30] investigated the ability of four commercially available Morse taper–like connections to impede bacterial penetration through their implant-abutment interfaces in vitro. Although there were microgaps of sufficient size for the penetration of bacteria commonly found in the oral cavity in all four systems tested, and bacteria were found in all cases, there was a difference with one specific connection compared with the other connections regarding the extent of microbial penetration. Whereas in the latter, the presence of bacteria was entirely observed inside of the implant, in the former, bacteria were not evidenced on the screw threads. The authors concluded that although the reasons for the findings were unknown, one possible explanation might be the differences in the degree of taper of the abutment posts. The degree of taper in the least-contaminated connection was the smallest (5.6 degrees) compared with the other groups (11.5 and 13 degrees). The authors suggested that the smaller degree of abutment taper might lead to greater attrition between the body of the implant and the abutment, resulting in a more intimate contact between the two parts. Importantly, however, all the Morse taper connections were contaminated with bacteria.

Likewise, the length of the contact between the implant and abutment wall appears to vary as well and

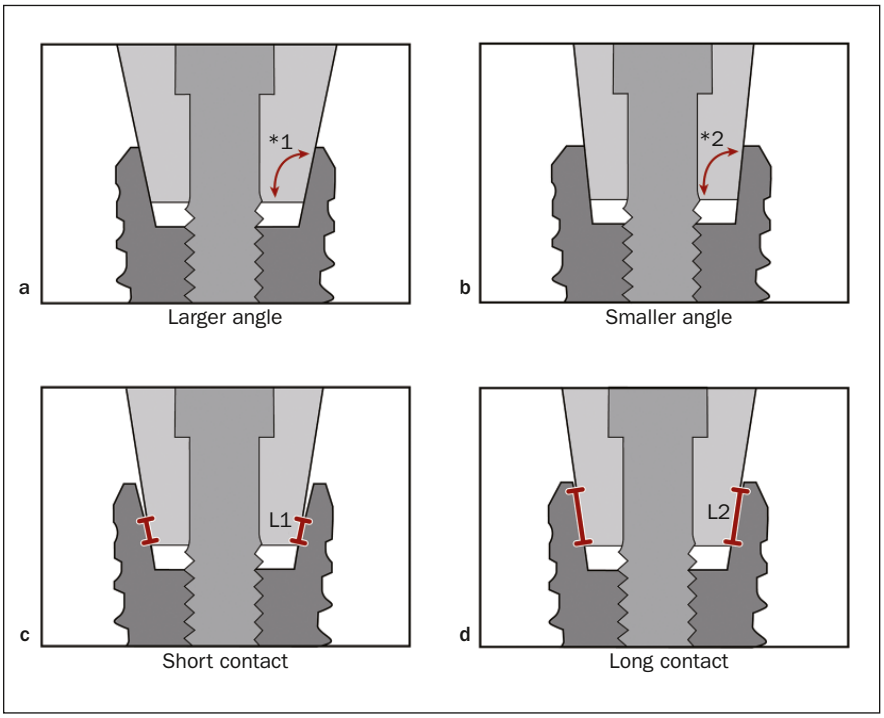

Fig 5 The difference of *(a, b)* taper angle and *(c, d)* length of contact in Morse taper–like connections. A connection with a *(b)* smaller angle can influence the stability of the connection compared with a *(a)* larger angle. Likewise, a connection with a *(d)* long contact can influence the stability of the connection compared with a *(c)* short contact length. Reprinted with permission from UTHSCSA.

could also be an important factor influencing stability, contamination, and peri-implant marginal bone levels (Fig 5b). Both taper angle and length of contact can influence the stability of the connection between the components (implant and abutment). Studies based upon microbial penetration indicate that under load (less stability), implant components result in contamination; however, with more static conditions of testing (no loading), bacteria can sometimes (at least in one paper) be excluded (Table 2). These findings suggest that the stability of the implant-abutment connection may be important in regard to maintenance of crestal (marginal) bone loss. This was confirmed by Hermann et al,[43] in that when the abutment was welded to the implant, even with a microgap (three sizes were examined), less marginal bone loss occurred around the implants compared with nonwelded implants with similar microgap sizes.

ONE-PIECE IMPLANTS (TISSUE-LEVEL OR ONE-BODY)

Experience with butt-joint (and external hexagon) connections between the implant and abutment have revealed that microbiologic contamination of that interface results in host inflammation and predictable marginal bone loss of approximately 1.5 to 2.0 mm. Platform-switched implants with internal connections have more stability between components, and bacterial contamination occurs, but apparently to a lesser degree, and hence, inflammation-induced bone resorption of the marginal bone is reduced. Thus, one critical factor in regard to marginal bone resorption around implants is the amount of inflammation present, since a direct spatial relationship exists between inflammation and bone.[36–39] Therefore, the optimal condition would be not to have any inflammation at the alveolar crest, and with a dental implant and abutment, this means that no opportunity for bacterial contamination at this point would be ideal. This is exactly the situation when no microgap or interface is present at the marginal bone, and as such, the driving force for initial bone resorption (the inflammation) is eliminated.

One-piece implants (currently referred to as tissue-level or one-body implants) provide exactly this scenario, since no opportunity for initial bacterial contamination at the bone crest exists. The implant is typically made with two implant surfaces, one on the part of the implant that is placed in the bone tissue (usually some type of osteoconductive surface), while a different surface is placed on the implant in contact with the soft tissues (connective tissue and epithelium), which is typically some type of smoother surface. Many experimental studies reinforce the concept that without a microgap or interface at the marginal bone level, minimal if any initial bone loss occurs, and in careful evaluations, most implants actually gain some bone (have bone formation) around the implant. This has been repeatedly demonstrated in both preclinical[6] and in clinical trials.[33,34] Such bone stability can be a clinical advantage. For example, in the posterior mandible,

there is typically a limited amount of bone above the lingual nerve to place an implant. The clinician typically is concerned about this amount and measures carefully to determine the length of the implant that can be placed above the nerve to maximize the amount of support (osseointegration) for the implant restoration, but does not, however, always account for the bone loss that might occur at the marginal level simply due to his/her selection of implant and abutment connection. This same effect can be important for implants placed below the sinus and especially for implants placed in the esthetic zone, where recession of tissue is to be avoided. For these biologic considerations, it certainly makes more clinical sense to use an implant-abutment configuration that minimizes initial marginal bone resorption, even it is "only" 2 mm of bone loss. Two millimeters with an 8-mm-long implant is 25% of its length, 20% of a 10-mm implant. That is a lot of support to sacrifice when one is trying to support a restoration, and thus, a one-piece (tissue-level or one-body) implant would be a first choice and a platform-switched implant would be a second choice if one is truly concerned with optimizing the osseous support for the implant-retained restoration. These considerations are beneficial for the implant survival rate as well. For example, French et al[58] conducted a retrospective cohort study of 4,591 Straumann implants, including tissue-level implants and platform-switched implants in a private practice setting, with up to 10-year follow-up. Generally, implants had a very high survival rate. At the implant level, implant survival at 1 year was 99%, and at 10 years, it had only dropped to 98%.

PERI-IMPLANTITIS

Another important consideration when choosing the type of implant-abutment connection is to consider what the implication is for the long-term success of the implant. Peri-implantitis has been reported to be a prevalent problem for implants over time.[59–61] In virtually all of these cases, bacterial contamination and inflammation is either a cause of or a part of the pathogenic mechanism of this condition. Where do the bacteria originate for such infections? One potential source, in addition to the peri-implant sulcus and tissues, is the external hexagon butt-joint contaminated interface (Fig 3d). Such implants in a steady state have approximately 2 mm of bone loss initiated by the host reaction to the initial infection since the bacteria are located within the interface. Many clinicians who use such implants acknowledge such bone loss but quickly point out that it does not jeopardize the success of the implant. What they do not consider, however, is that under these conditions, there is an infection completely around the implant that could be "lying in wait," much like a patient with gingivitis. Gingivitis is also a localized infection that is "lying in wait" until something either stimulates the infection to proliferate or the host becomes compromised, and under either condition, the lesions progress to a periodontitis lesion, and more bone loss occurs. It is generally accepted that peri-implant mucositis is the precursor of peri-implantitis, as it is accepted that gingivitis is the precursor of periodontitis. Likewise, it is also generally accepted that peri-implantitis, like periodontitis, occurs primarily as a result of an overwhelming bacterial insult and subsequent host immune response.[62] Broggini et al[31] have shown that the interfaces around connections become contaminated with bacteria after the abutment is placed on the implant, which can cause "persistent acute inflammation" at the implant-abutment interface. Their study provided histomorphometric evidence that a unique pattern of inflammatory cell infiltrate develops adjacent to implants and varies on the basis of implant design. For one-piece implants, there was no peak of inflammatory cells at the alveolar crest. However, two-piece implants resulted in a peak of inflammatory cells approximately 0.50 mm coronal to the connection and consisted primarily of neutrophilic polymorphonuclear leukocytes.

Having a 360-degree localized infection around contaminated butt-joint interfaces places such implants at risk for either a stimulation of the infection to proliferate or for the host to become compromised, and the lesions then progress to peri-implantitis. Recent evidence exists that suggests this risk is real. Derks et al[63,64] have demonstrated in a prevalence study of randomly selected patients and implants that tissue-level implants have significantly fewer early failures and fewer late failures and less marginal bone loss than implants with interfaces at the alveolar crest. These data support the notion that contaminated interfaces have three very significant consequences for the implant: *(1)* the initial infection results in approximately 2 mm of marginal bone loss that is so predictable that such bone loss is considered a success criteria for such implants[11]; *(2)* they place the implant at greater risk for failure over time[63]; and *(3)* the implant experiences more marginal bone loss ("peri-implantitis") over time.[64]

CONCLUSIONS

In this review, the authors examined different implant-abutment connections and what initial marginal bone changes occur, when, and why, around each type of connection. Based on the current available evidence, 1.5 to 2.0 mm of bone loss occurs predictably around bone-level, external-hexagon

butt-joint implants where the interface is contaminated with bacteria. This bone loss is not observed around one-piece implants because there is no interface at the bone crest, and hence, no opportunity for infection from contaminated implant interfaces and a host inflammatory reaction. Many studies have shown an advantage in the amount of marginal bone resorption with implants having platform-switched connections compared with external-hexagon butt-joint connections. Further investigations are needed to understand the influence of specific factors used in internal cone connections, including the angle and length of contact, as well as the influence in the amount of horizontal offset on marginal bone loss. In addition, well-designed, large, and long-term studies are also needed to investigate the association between the types of implant-abutment connections and prevalence of peri-implantitis.

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