The success of dental implants is highly dependent on integration between the implant and intraoral hard/soft tissue. Initial breakdown of the implant-tissue interface generally begins at the crestal region in successfully osseointegrated endosteal implants, regardless of surgical approaches (submerged or non-submerged). Early crestal bone loss is often observed after the first year of function, followed by minimal bone loss (≤0.2 mm) annually thereafter. Six plausible etiologic factors are hypothesized, including surgical trauma, occlusal overload, peri-implantitis, microgap, biologic width, and implant crest module. It is the purpose of this article to review and discuss each factor. Based upon currently available literature, the reformation of biologic width around dental implants, microgap if placed at or below the bone crest, occlusal overload, and implant crest module may be the most likely causes of early implant bone loss. Furthermore, it is important to note that other contributing factors, such as surgical trauma and peri-implantitis, may also play a role in the process of early implant bone loss. Future randomized, well-controlled clinical trials comparing the effect of each plausible factor are needed to clarify the causes of early implant bone loss. J Peridontol 2002;73:322-333.

KEY WORDS
Biologic width; bone loss/etiology; dental implants/complications; dental implants failure; dental occlusion, traumatic; peri-implant diseases.
surgical trauma are often surrounded by fibrous connective tissues or have an apical extension of the junctional epithelium. Heat generated at the time of drilling, elevation of the periosteal flap, and excessive pressure at the crestal region during implant placement may contribute to implant bone loss during the healing period.

In 1984, Eriksson and Albrektsson reported that the critical temperature for implant site preparation was 47°C for 1 minute or 40°C for 7 minutes. When the bone is overheated, risk of implant failure is significantly increased. Overheating may be generated by excessive pressure at the crestal region during implant surgery. Matthews and Hirsch demonstrated that temperature elevation was influenced more by the force applied than drill speed. However, it was found that when both drill speed and applied force were increased, no significant increase in temperature was observed due to efficient cutting.

The periosteal elevation has been speculated as one of the possible contributing factors for crestal implant bone loss. Wilderman et al. reported that the mean horizontal bone loss after osseous surgery with periosteal elevation is approximately 0.8 mm, and the reparative potential is highly dependent upon the amount of cancellous bone (not cortical bone) existing underneath the cortical bone. The bone loss, if observed, at stage II implant surgery (implant uncovering surgery in submerged implants) in successfully osseointegrated implants is generally vertical and has been measured to be between 0.2 mm and 1.3 mm. However, the bone loss noted was only around the implant, not the surrounding bone even though during the surgery all the bone was exposed, not just the implant region. Additionally, the pattern of bone loss differs between early implant bone loss and the bone loss after osseous surgery in natural teeth; the early implant bone loss is characterized by “saucerization” rather than horizontal resorption noted after osseous surgery in natural teeth. Therefore, this hypothesis is not generally supported.

In summary, the signs of bone loss from surgical trauma and periosteal reflection are not commonly observed at implant stage II surgery in successfully osseointegrated implants; furthermore, the pattern of bone loss in implants is more likely to be vertical than horizontal. Hence, the hypothesis of the surgical causes of early implant bone loss remains to be determined.

OCCLUSAL OVERLOAD

Occlusal overload is considered a major cause of implant failure. Research has indicated that occlusal overload often resulted in marginal bone loss or de-osseointegration of successfully osseointegrated implants. Unlike natural teeth, osseointegrated implants are ankylosed to surrounding bone without the periodontal ligament which has mechanoreceptors and shock-absorbing function. In addition, the crestal bone around dental implants could be a fulcrum point for lever action when a bending moment is applied, suggesting that implants could be more susceptible to crestal bone loss by mechanical force. Rangert et al., in a retrospective clinical analysis, described contributing factors associated with increased bending overload in dental implants. These included: prostheses supported by 1 or 2 implants in the posterior region, straight alignment of implants, significant deviation of the implant axis from the line of action, high crown/implant ratio, excessive cantilever length, discrepancy in dimensions between the occlusal table and implant head, and parafunctional habits. The cortical bone is known to be least resistant to shear force, which is significantly increased by bending overload. Therefore, the above-mentioned factors may result in progressive marginal bone loss or even deosseointegration if the bending overload increases beyond the threshold of bone homeostasis.

Loss of osseointegration by occlusal overload was observed in monkeys by Isidor. Among 5 implants
Early Implant Bone Loss

placed in each of 4 monkeys, 2 implants received occlusal overload 6 months after implant placement by prostheses causing lateral displacement of the mandible during occlusion. Oral hygiene was employed to the overloaded implants. The remaining 3 implants were not loaded but plaque accumulation was encouraged (ligature placement without oral hygiene). The results demonstrated that 5 out of 8 implants with overload lost osseointegration 4.5 months to 15.5 months after initiation of the occlusal overload, whereas all implants with plaque accumulation remained osseointegrated. Of the 3 implants which did not fail among the implants with occlusal overload, 1 lost approximately one-half of the crestal bone, and the other 2 in the same animal showed the highest bone-to-implant contact and bone density. Isidor24,25 stated that the increased bone density in the 2 implants would prevent the implant failure and provide limited amount of crestal bone loss. During 18 months after initiation of overload or plaque accumulation, there was progressive marginal bone loss observed in both groups: 5.5 mm for the overload group versus 1.8 mm for the plaque group at 18 months. Even though the bone loss in the overload group was 3 times greater than the bone loss in the plaque group, there was no statistically significant difference between the 2 groups, probably due to the small sample size. The study concluded that occlusal overload can be a causative factor for implant failure, and both occlusal overload and peri-implant infection can result in progressive marginal bone loss. Contradictory to Isidor,24,25 Hürzeler et al.28 showed histologically that a repetitive mechanical trauma did not influence peri-implant bone loss in healthy or in diseased implant sites up to 16 weeks in monkeys. The difference of the results in these studies may be attributed to the different experimental periods and different levels of forces used to induce occlusal overload.

In a series of the experimental studies in monkeys by Miyata et al.,29-31 the influence of occlusal overload on peri-implant tissue was histologically investigated. In the first part, they found that peri-implant bone loss was not observed when occlusal overload was applied by a superstructure with an excess occlusal height of 100 μm.29 On the other hand, in the second report, peri-implant bone destruction was clearly demonstrated by a combination of occlusal overload (provided by an excessive occlusal height of 100 μm on a superstructure) and experimental inflammation.30 In the last part,31 occlusal overload was employed with 3 different excess occlusal heights (100 μm, 180 μm, 250 μm) on implant prostheses for 4 weeks, and oral hygiene was performed. Bone destruction was observed in 180 μm and 250 μm excess occlusal height groups, indicating existence of a critical point of excessive occlusal height on the prostheses for crestal bone loss. From these studies, it can be concluded that peri-implant bone resorption may occur under severe occlusal overload or under co-existence of inflammation and occlusal overload.

The modulus of elasticity is a measure of material stiffness whereas a stress value is expressed as force divided by area. The modulus of elasticity of titanium is approximately 5 times greater than the cortical bone.32 According to VonRecum,33 when 2 materials of different moduli of elasticity are placed together without intervening material and one is loaded, a stress contour increase is observed where the two materials first come into contact. Photoelastic and 3-dimensional finite element analysis (FEA) studies demonstrated V- or U-shaped stress patterns with greater magnitude near the point of the first contact between implant and photoelastic block, which is similar to the early crestal bone loss phenomenon.34,35 Misch claimed that the stresses at the crestal bone may cause microfracture or overload, resulting in early crestal bone loss during the first year of function, and the change in bone strength from loading and mineralization after 1 year alters the stress-strain relationship and reduces the risk of microfracture during the following years.36 In addition, he stated that the etiology of early crestal bone loss and early implant failure after loading is primarily from excessive stress transmitted to the immature implant-bone interface.36 This suggests that stress reduction might be needed in early stages of bone healing and in poor quality bone. Stress reduction can be achieved by increasing surface area and decreasing forces because stress is force divided by area.

Wiskott and Belser37 described a lack of osseointegration attributed to an increased pressure on the osseous bed during implant placement, establishment of a physiologic "biologic width," stress shielding and lack of adequate biomechanical coupling between the load-bearing implant surface and the surrounding bone. Among these causes, they focused on the significance of the relationship between stress and bone homeostasis. Based on the previous study by Frost,38 5 types of strain levels interrelated with different load levels in the bone were described: 1) disuse, bone resorption; 2) physiologic load, bone homeostasis; 3) mild overload, bone mass increase; 4) pathologic overload, irreversible bone damage; and 5) fracture. The study concluded that adequate strain
levels, such as bone homeostasis and bone mass increase, are the only requirement for successful integration of load bearing surfaces.

Several authors speculated that “local overload” might contribute to the formation of “saucerization” of crestal bone adjacent to implants. The concept of “microfracture” proposed by Roberts et al.39 is often used. The article described that crestal regions around dental implants are high stress bearing areas, and further explained that if the crestal region is overloaded during bone remodeling, “cervical cratering” is created around dental implants. The study also suggests that axially directed occlusion as well as progressive loading are recommended to prevent “microfracture” during the bone remodeling periods.

Progressive loading on dental implants during healing stages was first described by Misch in the 1980s to decrease early implant bone loss and early implant failure. Based on the concept, progressive loading needs to be employed to allow the bone to form, remodel, and mature to resist stress without detrimental bone loss by staging application of diet, occlusal contacts, prosthesis design, and occlusal materials.40 Misch et al.41 evaluated 364 consecutively placed implants in 104 patients where the progressive protocol had been employed. It was reported that a 98.9% success rate at stage II uncovering surgery was observed, followed by no early loading failures during the first year of function. However, no control group without progressive loading was used in the study, and it was not possible to objectively determine the influence of progressive loading on early crestal bone loss. In another study, a decrease in crestal bone loss was observed in progressively-loaded implants, compared to implants without progressive loading, within a similar healing and loading period; in addition, digital radiographs indicated an increase in bone density in the crestal 40% of the implant in the progressive loaded crowns.42 The study suggests that controlling occlusal load with progressive loading in accordance with bone density may be beneficial to reduce early implant bone loss in healing periods.

Occlusal overload can result in progressive marginal bone loss or even complete loss of osseointegration, and when traumatic occlusion is combined with inflammation, the progression of bone destruction is accelerated.24,25,30 However, considerably greater crestal bone loss observed at the first year of function compared to following years may not be clearly explained only by occlusal overload because bone loss resulting from occlusal overload is considered to be progressive rather than a phenomenon limited to the first year after loading. A possibility of the cause for reduced occlusal overload or increased resistance to occlusal overload after the first year of function includes a functional adaptation of the oral musculature, wear of the prosthetic material, and/or an increase in bone density after a certain time period.

In summary, implant bone loss may occur if the stress is excessive (i.e., pathologic overload). Accordingly, the early implant bone loss may be induced by occlusal overload or some types of excessive stress on immature bone-implant interface in the early stage of implants in function. Also, it could explain how the “saucerization” forms up to the first year of function.

**PERI-IMPLANTITIS**

Together with occlusal overload, peri-implantitis is one of the two main causative factors for implant failure in later stages. A correlation between plaque accumulation and progressive bone loss around implants has been reported in experimental studies43-45 and clinical studies.1,19,46 Tonetti and Schmid reported that peri-implant mucositis is a reversible inflammatory lesion confined to peri-implant mucosal tissues without bone loss; on the other hand, peri-implantitis begins with bone loss around dental implants.23

As in the case of the natural teeth, peri-implant mucositis and peri-implantitis occur as a result of breaking down host-parasite equilibrium. Clinical features of peri-implantitis were described by Mombelli47 as including: 1) radiographic evidence of vertical destruction of the crestal bone; 2) formation of a peri-implant pocket in association with radiographic bone loss; 3) bleeding after gentle probing, possibly with suppuration; 4) mucosal swelling and redness; and 5) no pain typically. Mombelli et al.48 evaluated the microbiota associated with successful or failing implants and suggested that “peri-implantitis” is regarded as a site-specific infection and has microbial features similar to chronic periodontitis. The healthy sites harbored small amounts of bacteria, mainly coccoid cells. On the other hand, microbiota obtained from failing implants consisted of a large proportion of Gram negative anaerobic rods, with black-pigmented *Bacteroides* and *Fusobacterium* spp. as well as spirochetes. The microbial features were site-specific rather than host-dependent. Lee et al.49 investigated microbiota of successfully osseointegrated dental implants in 43 partially edentulous patients. The results suggested that a history of periodontitis had a greater impact on the peri-implant microbiota than implant loading time. It was also demonstrated that the microbiota on remaining teeth
Early Implant Bone Loss

significanty influenced the composition of peri-
implant microbiota.

In an experimental study evaluating the pattern of
ligature-induced breakdown of peri-implant and peri-
odontal tissues in beagle dogs, significantly greater
tissue destruction was demonstrated clinically, radi-
ographically, and histomorphometrically at implant
areas than at tooth sites. It was also found that sig-
nificantly fewer vascular structures existed at implant
sites compared to periodontal tissues. The difference
in collagen fiber direction (parallel to the implant sur-
face and perpendicular to tooth surface) and amount
of vascular structure may explain the faster pattern
of tissue destruction in peri-implant tissues than peri-
odontal tissues.

Literature has shown that peri-implantitis is simi-
lar in nature to periodontitis in that the microbiota of
peri-implantitis resemble the microbiota of peri-
odontitis; however, there has been no evidence that
peri-implantitis induces crestal bone loss during heal-
ing and the first year of function at a faster rate than
following years. In fact, the early crestal bone loss
may result in an environment that is favorable for
anaerobic bacterial growth, thus possibly contribut-
ing to more bone destruction in following years.

Nonetheless, in the majority of implants the bone loss
is dramatically reduced after the first year of pros-
thesis loading. Therefore, it may not be justified that
peri-implantitis is the main causative factor for early
implant bone loss.

MICROGAP

In implant dentistry, there are 2 basic approaches to
place endosseous implants, including submerged (2-
stage) and non-submerged (1-stage) implants. In
most 2-stage implant systems, after the abutment is
connected, a microgap exists between the implant and
abutment at or below the alveolar crest. In non-
submerged implant designs, the implant itself extends
above the alveolar crest level; therefore, such a micro-
gap does not exist at the level of the bone.

Implant countersinking below the bone crest was
recommended in the Brånemark surgical protocol to
minimize the risk of implant interface movement
during bone remodeling, and to prevent implant ex-
posure during healing. Implant countersinking is also
used to accommodate the wider implant platform in
Brånemark implants or its clones and to enhance
emergence profile for implant prostheses at the
expense of the crestal bone. The countersinking per-
formed for the above purposes places the abutment-
implant microgap below the crestal bone.

Quirynen and van Steenberghe and Persson et
al. found microbial species cultivated from internal
surfaces of submerged implants or their restorative
component parts. The Quirynen and van Steenberghe study demonstrated the presence of microorganisms
in the inner thread of submerged implant fixtures in
9 subjects. The apical part of 2 abutment screws that
had been in place for 3 months were examined by
means of differential phase-contrast microscopy. The
results showed that all screws harbored a significant
quantity of microorganisms, mainly coccoid cells
(86.2%) and nonmotile rods (12.3%). Motile organ-
isms (1.3%) or spirochetes (0.1%) were only spor-
adically registered. The study implied that a micro-
bial leakage from the microgap between the abutment/fixture interface in submerged implants is
the most probable origin for this contamination. How-
ever, the possibility of microbial contamination through microgap between the abutment and fixture in sub-
merged implants is related to development of peri-
implantitis, and its consequence is not limited to the
first year after loading.

Berglundh et al. and Lindhe et al. also evalu-
ated the microgap of the Brånemark 2-stage implant
and found inflamed connective tissue existed 0.5 mm
above and below the abutment-implant connection,
which resulted in 0.5 mm bone loss within 2 weeks
after the abutment was connected to the implant.

The influence of the microgap on the peri-implant
tissue formation during healing was studied radi-
ographically in dogs by Hermann et al. Six differ-
ent types of implant design were used, 2 types of 1-
part implants and 4 types of 2-part implants. The
1-stage approach (non-submerged) was used for the
1-part implant types and 1 of the 2-part implant
types; a 2-stage approach (submerged) was employed
on the remaining implant types. Abutment connec-
tion was carried out 3 months after implant place-
ment on the implants previously submerged. After 3
months of additional healing, all animals were sacri-
ficed for histologic analysis. Radiographic evaluation
included the distance between the top of the implant/abutment and the most coronal bone-to-
implant contact using standardized radiographs taken
monthly and bone density changes using computer-
assisted densitometric image analysis (CADI). The
results indicated that in 1-part, non-submerged
implants, the crestal bone levels followed at all time
points the rough/smooth implant interface; on the
other hand, for all 2-part implants, the crestal bone
levels appeared dependent on the location of the
microgap, approximately 2 mm below the microgap.
In addition, CADIA values for all submerged implants were decreased in the most coronal areas, but increased at the new bone level after abutment connection. This study first demonstrated that the microgap between implant/abutment has a direct effect on crestal bone loss independent of surgery approaches, submerged or non-submerged. The study also suggested that epithelial proliferation to establish a biologic width could be responsible for the crestal bone loss found about 2 mm below the microgap. Later, the radiographic findings were further supported by a histometric analysis performed by the same group.55

Even though a microgap does not exist in non-submerged implants, crestal bone loss during the first year of function in non-submerged implants has been reported, being equivalent or slightly less than submerged implants.2,8 However, stable alveolar bone crest levels from 1 year up to 8 years after implant placement were reported in non-submerged implants.56 From the literature review, it can be speculated that although microgap may not be considered as the only cause of early implant bone loss, it might cause implant crestal bone loss during the healing phase if it is placed at or below the bony crest.

**BIOLOGIC WIDTH (BIOLOGIC SEAL)**

In natural teeth, the dentogingival junction consists of 3 components: the gingival sulcus, the epithelial attachment, and the connective tissue attachment. The dimensions of the dentogingival junction were studied in human skulls by Gargiulo et al.57 and Vacek et al.58 Gargiulo et al.57 reported that the average value of sulcus depth was 0.69 mm, and the average values for the epithelial attachment and connective tissue attachment were 0.97 mm and 1.07 mm, respectively. The biologic width included the latter 2, the epithelial attachment and connective tissue attachment, which was 2.04 mm. The values found in Vacek et al.58 corresponded to Gargiulo et al.’s findings,57 which were 1.14 mm for epithelial attachment and 0.77 mm for connective tissue attachment. Both studies concluded that the most consistent value between individuals was the dimension of the connective tissue attachment.

Likewise, around dental implants, the epithelial attachment (or zone) and connective tissue attachment (or zone) exist (Fig. 2), comprising the biologic seal around dental implants that acts as a barrier against bacterial invasion and food debris ingress into the implant-tissue interface.59 The epithelial attachment in both implant and natural tooth is composed of hemidesmosome and basal lamina, whereas collagen fiber direction in the connective tissue attachment is different, being parallel to implant surfaces and perpendicular to natural teeth.60-62 Table 1 describes differences noted between implant and tooth.

Cochran et al.68 performed a study on loaded and unloaded non-submerged titanium implants and found that the dimensions of the implant/gingival junction remained constant over time up to 12 months after loading. The dimensions were comparable to the dentogingival tissues as described by Gargiulo et al.57 After 12 months of loading, the values were 0.16 mm for the sulcus depth, 1.88 mm for the junctional epithelium, and 1.05 mm for the connective tissue attachment. The biologic width reported in the study was 3.08 mm, including the sulcus depth, epithelial
attachment, and connective tissue attachment. Later, Hermann et al.\textsuperscript{69} histometrically evaluated the dimensional change of the biologic width around non-submerged implants. They observed that each dimension of the sulcus depth, epithelial attachment, and connective tissue attachment changed over time, but within the overall biologic width dimension. The dimensions of the biologic width around submerged implants have also been reported.\textsuperscript{53,70,71} Table 2 lists biologic width studies associated with the natural tooth and dental implant.

Berglundh and Lindhe\textsuperscript{70} studied the effect of subcrestal placement of the polished surface of non-submerged implants on marginal soft and hard tissues in 11 patients. At test sites, the apical border of the polished surface was placed about 1 mm below the alveolar crest, while the junction between rough and polished surface was located at the crest in control sites. After 1 year of function, the average crestal bone loss was 2.26 mm in the test group and 1.02 mm in the control group. The study suggested that during the first year of function, the biologic seal was established 1 mm apical of the rough implant portion at the expense of the crestal bone independent of an initially increased countersink depth.

In a study comparing healed tissues in submerged and non-submerged unloaded dental implants in dogs, it was found that apical extension of epithelial attachment in submerged implants was located below the microgap and significantly greater than that in non-submerged implants.\textsuperscript{73} It was speculated that the greater apical extension of epithelial attachment in submerged implants might have been due to microbial leakage from the microgap after abutment connection at stage II surgery. However, there was no significant difference between the 2 groups in the distance between implant top and first bone-implant contact (2.92 mm in submerged versus 2.95 mm in non-submerged implants).\textsuperscript{73} The study hypothesized that the extent of epithelial downgrowth was not related to the amount of bone resorption occurring after surgery, and that connective tissue appeared to fill that space. Wallace\textsuperscript{74} emphasized the significance of biologic width in dental implants and stated “The
The fact that the ultimate location of the epithelial attachment, following phase two surgery, will be on the implant body is of clinical significance to the implant surgeon since it will in part determine the amount of early post-surgical bone loss.

Based upon these findings, it is apparent that early implant bone loss, in part, is from the processes of establishing the biologic width. The amount of bone loss and location of the biologic width may be associated with thickness of soft tissue around implants, location of the junction between rough and polished surfaces in non-submerged implants, and location of the microgap in submerged implants. However, the reformation of the biologic width may not solely satisfy the causes of early crestal bone loss. As demonstrated in the Weber et al. study, the maxillary arch had more bone loss than the mandibular arch, which might have been attributed to lower bone density in the maxilla compared to the mandible. Yet, the biologic width should be similar in both arches.

**CREST MODULE CONSIDERATIONS**

The crest module of an implant body is defined as the transosteal region of the implant and serves as the region which receives the crestal stresses to the implant after loading. This region of the implant is often not designed for load bearing, instead is usually designed to minimize plaque accumulation, and acts as a transition zone to the load-bearing structure of the implant body in submerged implants. With regard to the concept of preventing plaque accumulation, two problems may be observed. First, since toothbrush bristles cannot enter a sulcus on a routine basis more than 1 mm, and the tissue height above the implant body in submerged implants is usually 2.5 mm or more, the implant crest module does not provide an environment favorable for hygiene to remove plaque. Second, a smooth crest module may actually contribute to the crestal bone loss. Cortical bone is strongest to compressive loads, 30% weaker to tensile forces, and 65% weaker to shear forces compared to compressive forces. Misch and Bidez claimed that a smooth, parallel-sided crest module may result in shear stresses in this region, and that an angled crest module of more than 20 degrees with a surface texture that increases bone contact might impose a slight beneficial compressive and tensile component to the contiguous bone and decrease the risk of bone loss (Fig. 3).

Significant loss of crestal bone has been reported for implants with 3-mm long, machined (smooth) coronal regions. It can be speculated that this bone loss may be attributed to the lack of effective mechanical loading between the machined coronal region of the implant and the surrounding bone.

It has been clinically observed that bone is often lost to or below the first thread in some types of submerged implants after loading. Bone grows above the threads during healing as often demonstrated at stage II surgery, but after prosthesis loading, the bone loss down to the first thread is often noted after first year of prosthesis loading. Yet, in many submerged implant systems, the distance between the implant platform and the first thread varies, ranging from 1 to 3 mm (e.g., 1.2 mm in the Bränemark system and 3 mm in many screw-vent implant systems). Therefore, the bone loss is probably not related to a specific anatomic length, but

**Table 2. Studies Regarding the Biologic Width Around Natural Teeth or Dental Implants**

<table>
<thead>
<tr>
<th></th>
<th>Natural Teeth</th>
<th>Dental Implants</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30 human skulls</td>
<td>10 human skulls</td>
</tr>
<tr>
<td>Sulcus depth (SD)</td>
<td>0.69 mm</td>
<td>1.34 mm</td>
</tr>
<tr>
<td>Junctional epithelium (JE)</td>
<td>0.97 mm</td>
<td>1.14 mm</td>
</tr>
<tr>
<td>Connective tissue attachment (CT)</td>
<td>1.07 mm</td>
<td>0.77 mm</td>
</tr>
<tr>
<td>Biologic width</td>
<td>2.04 mm (JE + CT)</td>
<td>1.91 mm (JE + CT)</td>
</tr>
</tbody>
</table>
Early implant bone loss may be in part related to crest module design. Also, it can be hypothesized that the bone loss may slow down at the first thread because the first thread changes the shear force of the crest module to a component of compressive force to which bone is the most resistant.

The beneficial effect of rough surfaces in the implant crest region in the reduction of crestal bone loss was also demonstrated by the previously presented report by Hermann et al., which compared 2 different 1-part implant bodies. The first implant had the rough/smooth region placed at the bone crest at surgery, while the other placed the rough/smooth region 1.5 mm below the bone. After 6 months the bone level remained at the original height of the first implant, while bone loss of 1.5 mm occurred on the second implant, which corresponded to the rough/smooth region. The study suggests that the bone loss found in the second implant might have been attributed to reformation of biologic width at the expense of crestal bone, which was related to the implant crest module design. Norton radiographically evaluated 33 single tooth implants for up to 4 years and reported considerably smaller amounts of crestal bone loss, 0.32 mm mesially and 0.34 mm distally. The study postulated that the significantly low degree of crestal bone loss resulted from a modification of the study postulated that the significantly low degree of crestal bone loss, 0.32 mm mesially and 0.34 mm distally. The study postulated that the significantly low degree of crestal bone loss resulted from a modification of the surface structure, both at the microscopic (micro-threaded crest module) and macroscopic level (rough surfaces: grit blasted with TiO₂ particles), as well as an altered implant-abutment interface design (internal conical interface). Further research in this area is needed to clarify the relationship between implant crest module designs and early implant bone loss.

CONCLUSION
Early implant crestal bone loss during healing and the first year of function, often greater than bone loss occurring at following years, is generally observed regardless of implant types. Possible causative factors for early implant bone loss include surgical trauma, occlusal overload, peri-implantitis, microgap, biologic width, implant crest module, and others. As demonstrated in photoelastic and finite element analysis studies, stress is concentrated around the crestal region when two materials with different moduli of elasticity are placed together. If some type of excessive stress (pathologic overload) is present at the crestal region after prosthesis loading, implant bone loss begins at this region. This may partly explain why saucerization patterns of marginal bone loss are noted in some types of implants after the first year of function. It has been observed that bone density may also affect the amount of early implant bone loss, and implants with progressive loading in accordance with bone density levels, although hypothetical, may provide less bone loss compared to implants with non-staged loading. In addition, the reformation of biologic width around dental implants can contribute to the early implant bone loss. This process starts immediately after stage II surgery in submerged implants and after implant placement in non-submerged implants. The dimension and position of the biologic width which are related to the degree of early implant bone loss during surgical healing phase may be determined by the location of the microgap if present, or implant crest module designs such as surface textures, implant-abutment interface designs in 2-part implants, and the location of a junction between rough and polished surfaces in non-submerged implants. The biologic width inevitably occurs following biomechanisms regardless of implant type, but may not be considered a sole factor associated with early implant bone loss since different levels of early implants bone loss have been reported in the literature depending on implant types. Therefore, it would appear that among all possible contributing factors, reformation of biologic width, occlusal overload, microgap and implant crest module are the most likely contributing causes for the early implant bone loss phenomenon. However, early implant bone loss may also result from or depend on surgical trauma, peri-implantitis, and others.

There has been little evidence on the mechanism of early implant bone loss, lacking studies comparing possible causative factors of early implant bone loss. Therefore, randomized well-controlled clinical trials are needed to determine the true mechanism of early implant bone loss.

ACKNOWLEDGMENT
This article was partially supported by the University of Michigan, Periodontal Graduate Student Research Fund.

REFERENCES

330 Early Implant Bone Loss
1097_IPC_AAP_553092 3/1/02 2:19 PM Page 331

J Periodontol • March 2002

41. Roberts WE, Garetto LP, De Castro RA. Remodeling of devitalized bone threatens periosteal margin integrity...


Send reprint requests to: Dr. Hom-Lay Wang, Department of Periodontics/Prevention/Geriatrics University of Michigan School of Dentistry 1011 North University Avenue, Ann Arbor, MI 48109-1078. Fax: 734/936-0374. e-mail address: homlay@umich.edu.

Accepted for publication August 20, 2001.