Implant Cervical Collars: Preserving Crestal Bone

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ABSTRACT
Roughened implant surfaces have been instrumental in bolstering long-term implant survival rates, but can also promote microbial attachment if exposed to the gingival crevice or oral cavity. The likelihood that some bone loss will occur from surgical trauma or other contributing factors over time is considerable. Consequently, maintaining a machined surface on the implant collar can help to prevent or minimize the percentage of roughened surface exposure after implant placement.

INTRODUCTION
Implant restoration of missing teeth has become increasingly predictable since 2000, when long-term implant survival rates began to exceed 95%. Although a host of internal and external variables can still significantly affect the osseointegration process (Table 1), this significant enhancement in implant predictability over the preceding two decades can be attributed to a number of technological improvements in component designs and surgical techniques. Among these, dental implant surfaces are a crucial aspect of the osseointegration process because they directly influence bone and soft tissue attachment to the implant. Numerous studies have reported a positive correlation between implant surface roughness and the overall percentage of bone-to-implant attachment that develops during osseointegration. For this reason, most contemporary manufacturers further roughen their dental implant surfaces after machining. However, there is currently no clinical consensus as to what degree of surface roughness is clinically optimal, or even how surface roughness should be measured. For example, while one surface may appear rougher than another surface when average roughness is measured in a two-dimensional (2D) roughness profile (Ra value), the reverse can be true when measuring the same two surfaces using a three-dimensional (3D) roughness profile (Sa value). Although some researchers have strongly asserted that 3D Sa values are more accurate and preferable to measuring implant surface roughness than 2D Ra values, there is still no clinical consensus and both Ra and Sa roughness measurements are used in the industry today.

Despite the recent advancements in implant predictability, crestal bone loss continues to be a matter of concern. This can be especially problematic following immediate implant placement into fresh extraction sockets, because bone will typically resorb 0.5-1.0 mm in both the vertical and horizontal dimensions during the first year after implant placement due to the post-extraction healing process. Such bone loss not only harbors a risk of exposing the roughened implant surface to the gingival crevice, but may also directly expose the roughened surface to the oral cavity if there is corresponding gingival recession during the early stage of healing. Any degree of roughened surface exposure may also enhance attachment by the 100-200 varieties of intraoral microbiota that inhabit neighboring dentition at any given time. While clinically harmless below a certain count, unchecked bacterial reproduction results in biofilm formation than can trigger gingival and periodontal diseases in the gingival sulcus regions of both implants and teeth. While infection is usually manifested as localized gingival inflammation, continued release of inflammatory cytokines, chemokines, and other intracellular mediators from the biofilms can progressively lead to chronic gingivitis, periodontal attachment loss, bone resorption, and eventual tooth loss or implant failure.
paper will review issues related to roughened implant surfaces and crestal bone loss as the basis for advocating maintenance of a 1.0 mm machined collar below the prosthetic platform of dental implants.

**PERIIMPLANT BONE LOSS**

For implants placed into healed extraction sites, approximately 0.5-1.0 mm of periimplant marginal bone loss — often described as *saucerization* — has been observed around implants at their second-stage surgical uncovering. Because surgical insult to the bone tissue during routine implantation procedures is widely assumed to be the cause, such preliminary bone loss is generally accepted as an unavoidable consequence of implant surgery. Disruption of the vascular network during elevation of the mucoperiosteum has also been cited as an additional theoretical cause of this bone loss. The latter theory is not universally accepted, however, because similar saucerization does not appear around natural teeth after soft tissue elevation for osseous surgery, and some bone loss still occurs on implants placed via a transmucosal or flapless technique. Consequently, many implant studies have either failed to report any marginal bone changes, or only calculated marginal bone loss after definitive implant loading (i.e. at least 3-6 months after implant placement). As technology improved, early researchers gradually applied new definitions of acceptable periimplant bone loss over time (Table 2).

In addition to the effect of surgical trauma on short-term periimplant bone loss, long-term bone loss and eventual implant failure have also been linked to many other variables, such as bacteria-related infections, chronic inflammation, and occlusal overloading. In a prospective, multicenter study conducted by the U.S. Department of Veterans Affairs, researchers measured the residual facial plate thickness of approximately 3,000 implant osteotomies and followed patients for three years. The study found that, as residual facial plates approached 2.0 mm in thickness, there was a corresponding decrease in facial bone loss, bone gain in some patients, and an increase in implant survival rates. Conversely, as facial plate thickness decreased below the 2.0 mm threshold, there was a corresponding increase in facial bone loss and implant failure. Implant placement in low-density bone, tobacco use and excessive alcohol consumption have also been associated with increased peri-implant bone loss in the dental literature.

In the same prospective multicenter study conducted by the U.S. Department of Veterans Affairs, other researchers evaluated clinician subjective estimates of bone density during osteotomy preparations for 2,839 implants. Results indicated that the greatest bone density was located in the anterior mandible, with diminishing bone density in the posterior mandible, anterior maxilla, and posterior maxilla, respectively. In an unrelated study, researchers used finite element analysis (FEA) to evaluate bone strains relative to bone properties and simulated periimplant loading conditions. In cancellous bone models with a lower range of Young's modulus values, FEA results showed that 50% of patients experienced hyperphysiologic periimplant strains in the crestal bone region where short-term periimplant crestal bone loss (saucerization) is commonly reported. In denser bone with higher ranges of Young's modulus values, only 25% of the cancellous bone models exhibited excessive periimplant bone strains. These findings suggest that implants placed in low-density bone with a lower range of Young's modulus values characteristic of the anterior and posterior maxillary jaw may be at greater risk for bone loss than implants placed in other jaw locations with higher Young's modulus values. It is important to note, however, that low-density bone can also be found in other jaw locations, such as the posterior mandible. Maintaining 1.0 mm of machined surface on the implant collar can help to prevent exposure of the roughened surface in the likely event of some crestal bone loss over time.

**IMPLANT CERVICAL MICROARCHITECTURE**

Traditional implant dentistry strictly advocated a two-stage, delayed loading technique to prevent implant micromovements that might result in fibrous tissue encapsulation and clinical failure of the implant. More recently, cellular, animal and human studies have suggested that implant cervical regions with microgrooves,
microthreads, microtextured surfaces or a combination of these features might help to foster bone attachment to the cervical region of the implant surface and bone formation inside the microgrooves or between the microthreads to impede epithelial tissue down-growth. The shapes and dimensions of the microgrooves themselves have also been reported to influence the behavior of epithelial and connective-tissue cells differently in vivo. Although adequate, prospective, comparative research data on microgrooves and microthreads are still generally lacking, short-term results have been variable in both animal and human models, while longer term results have suggested that any possible benefit of cervical microthreads may disappear after 5 years in function. It is currently unknown whether the perceived benefits of cervical microgrooves or microthreads will outweigh the potential hygiene risks of thread exposure should bone recession occur.

Extending roughened implant surfaces to the tops of implant necks has been reported to reduce the amount of periimplant crestal bone loss without adversely affecting soft tissue health. Implants with roughened, acid-etched (test group) and machined (control group) surfaces were placed in a canine model and allowed to accumulate plaque for 6 months. There were no significant differences in plaque formation or establishment of inflammatory cell lesions in the periimplant mucosa between the test and control groups. It should be noted, however, that the acid-etched implants are much smoother than many of the roughened surfaces on the market today. Nonetheless, concern about the possibility of increased bacterial attachment to rougher implant surfaces in humans has been expressed in the dental literature.

**RISKS OF ROUGHENED SURFACE EXPOSURE**

Concern about possible bacterial colonization of the implant-abutment microgap within the biologic width may be significantly heightened if the implant neck has a roughened surface in close proximity. Although some clinicians have advocated moving the microgap away from the outer circumference of the implant with the use of an abutment that is smaller in diameter than the implant itself (platform switching), the effect of a roughed surface at the microgap remains uncertain. One study used FEA to evaluate whether platform switching provided any biomechanical advantages. Results showed that, although platform switching helped to shift the stress concentration area away from the cervical bone-implant interface, it increased stress concentrations in the abutment screw and the abutment body. Excessive occlusal stresses directed at the abutment fixation screw have been cited as a leading cause of screw loosening. If left untreated, loose abutment screws can lead to crestal bone loss that would further exposure of the roughed surface to the biologic width.

In an animal model, another study evaluated the influence of the implant-abutment microgap on periimplant tissues in the mandibles of 4 canines. The researchers placed implants and abutments in matching diameters 1 mm above, 1 mm below (countersunk), and level with the crestal bone. After a 3-month nonsubmerged healing period, abutments were placed on the implants and the animals were allowed to function for 3 months before sacrifice and histologic evaluation. Results showed that not only did placing the implant-abutment microgap deeper in the bone not result in additional bone loss, but that implants with countersunk microgaps had the least bone loss of the 3 study groups, which challenged the microgap theory. Results of comparative short-term clinical studies have been inconclusive regarding the clinical efficacy of platform switching. Additional long-term comparative data are still needed before definitive conclusions can be drawn to adequately support evidence-based treatment planning. Thus, while researchers have proposed platform shifting and surgical positioning of the implant collar at or above the bony crest, there is little data to suggest that these strategies will prevent all crestal bone loss around the implant. Therefore, maintaining 1.0 mm machined surface on the implant’s cervical collar can help to resist the risk of bacterial attachment in the event of exposure to the gingival crevice or the oral cavity itself.
PERIIMPLANTITIS RISKS

Bacteria are significantly involved in the etiology of periimplant diseases, which can range from implant peri-mucositis to full periimplantitis that affects the supporting tissues around a dental implant and abutment. The periimplantitis process typically begins as mucositis after 10-14 days of plaque retention, and triggers the same pathogen-induced inflammatory progression as periodontitis. Consequently, patients with a history of periodontitis remain periodontitis-susceptible and have a higher risk of developing periimplantitis and implant failure than patients with no history of the disease. An estimated 16-28% of all implant patients will develop peri-implantitis lesions with marginal bone loss of at least 2 mm after 5-10 years. This same disease process will also occur at a higher prevalence among patients with multiple implants than with an implant-supported, single tooth restoration. For long-term implant health, and especially in periodontitis-susceptible patients, maintaining a 1.0 mm machined collar on dental implants may thus help to avoid roughened surface exposure and help to mitigate bacterial plaque adhesion to the dental implants, especially in periodontitis-susceptible patients.

REFERENCES


## Table 1. Variables that May Adversely Affect Osseointegration or Implant Survival

<table>
<thead>
<tr>
<th>Source</th>
<th>Type</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manufacturer</td>
<td>Manufacturing errors</td>
<td>Metal burs from machining, Surface contamination (e.g., manufacturing oil, particulate debris), Wrong labeling</td>
</tr>
<tr>
<td></td>
<td>Design errors</td>
<td>Interfacial gaps between machined components, Abutment rotational instability, Inadequate or poor engagement of screw threads</td>
</tr>
<tr>
<td>Patient</td>
<td>Uncontrolled diseases</td>
<td>Periodontitis, Diabetes mellitus, Osteoporosis</td>
</tr>
<tr>
<td></td>
<td>Compromising medications</td>
<td>Intravenous bisphosphonates, Long-term corticosteroid use</td>
</tr>
<tr>
<td></td>
<td>Parafunctional habits</td>
<td>Bruxing, Fingernail biting / pencil chewing</td>
</tr>
<tr>
<td></td>
<td>Compromising personal habits</td>
<td>Smoking, Alcoholism, Poor oral hygiene</td>
</tr>
<tr>
<td>Clinician</td>
<td>Latrogenic errors</td>
<td>Excessive drilling heat, Improper implant angulation, Ineffective occlusal scheme</td>
</tr>
</tbody>
</table>

*Examples are not an exhaustive list; other examples also apply

## Table 2. Evolution of Acceptable Bone Loss Definitions by Early Researchers

<table>
<thead>
<tr>
<th>Time Interval</th>
<th>1979†</th>
<th>1985‡</th>
<th>1986‡</th>
<th>1993‡</th>
<th>1998‡</th>
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<tbody>
<tr>
<td>General</td>
<td>1/3 of implant height66</td>
<td>Not Reported</td>
<td>Not Reported</td>
<td>Not Reported</td>
<td>Not Reported</td>
</tr>
<tr>
<td>After submerged healing</td>
<td>Not Reported</td>
<td>Not Reported</td>
<td>Not Reported</td>
<td>Not Reported</td>
<td>Not Reported</td>
</tr>
<tr>
<td>First year of loading</td>
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<td>1.0-1.5 mm22</td>
<td>&lt;1.0 mm42</td>
<td>&lt;1.0 mm47</td>
<td>Not Reported</td>
</tr>
<tr>
<td>Follow-up years</td>
<td>Not Reported</td>
<td>Not Reported</td>
<td>0.05-0.0142</td>
<td>&lt;0.2 mm47</td>
<td>&lt;0.2 mm48</td>
</tr>
</tbody>
</table>

†Blade implants only  
‡Root-form implants only