

# The maintenance of **crestal bone** around dental implants

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## \_Introduction

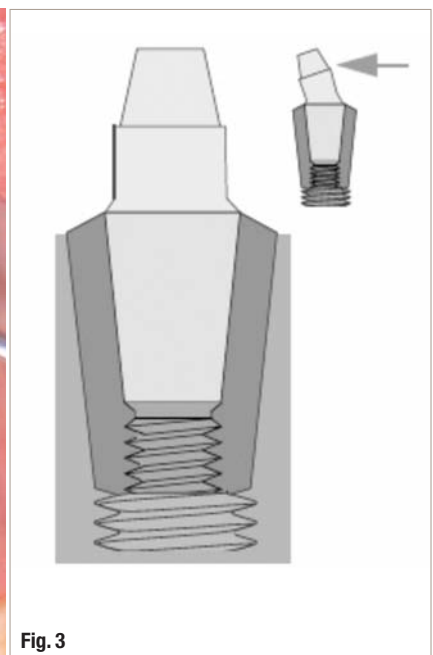
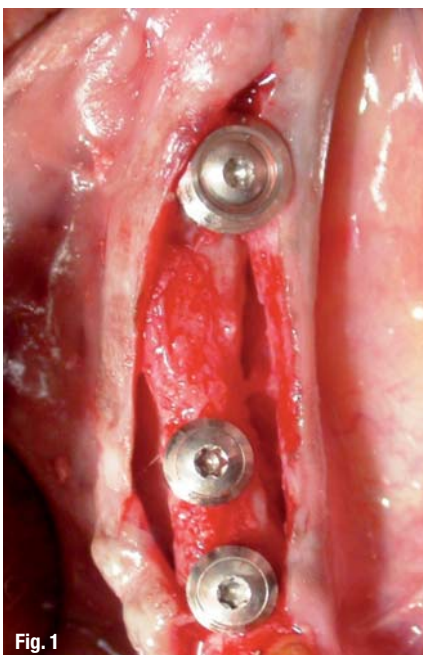
The longevity of dental implants is highly dependent on integration between implant components and oral tissues, including hard and soft tissues. Studies have shown that submerged titanium implants had 0.9 to 1.6 mm marginal bone loss from the first thread by the end of the first year in function, while only 0.05 to 0.13 mm bone loss occurred after the first year.<sup>1-3</sup>

The first report in the literature to quantify early crestal bone loss was a 15-year retrospective study that evaluated implants placed in edentulous jaws.<sup>1</sup> In this study, Adell *et al.* reported an average of 1.2 mm marginal bone loss from the first thread

during healing and the first year after loading. In contrast with the bone loss during the first year, there was an average of only 0.1 mm bone lost annually thereafter.

Based on the findings on submerged implants, Albrektsson *et al.* and Smith and Zarb proposed criteria for implant success, including a vertical bone loss of less than 0.2 mm annually following the implant's first year of function.<sup>4,5</sup>

Non-submerged implants have also demonstrated early crestal bone loss, with greater bone loss in the maxilla than in the mandible, ranging from 0.6 to 1.1 mm, at the first year of function.<sup>6-8</sup>



**\_Surgical trauma**

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.

*Heat generation and excessive pressure*

Eriksson and Albrektsson reported that the critical temperature for implant site preparation was 47°C for one minute or 40°C for seven minutes.<sup>9</sup> Matthews and Hirsch demonstrated that temperature elevation was influenced more by the force applied than drill speed.<sup>10</sup> When both drill speed and applied force were increased, no significant increase in temperature was observed owing to efficient cutting.<sup>10, 11</sup>

Sharawy *et al.* compared the heat generated by the drills of four different implant systems run at speeds of 1,225, 1,667 and 2,500 rpm.<sup>12</sup> All of the drill systems were able to prepare an 8 mm site without the temperature rising by more than 4°C (to 41°C). For all drill systems, the 1,225 rpm drill speed required a 30 to 40% longer drilling time when compared with 2,500 rpm and a 20 to 40% reduction in the time required for bone temperature to normalise. With greater depth of preparation and insufficient time between drill changes, a

detrimental temperature rise to 47°C or greater may be reached. The authors recommend that surgeons interrupt the drilling cycle every five to ten seconds to allow irrigant time to cool the osteotomy.

*Periosteal flap*

The periosteal elevation has been suggested as one of the possible contributing factors to 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) underneath the cortical bone.<sup>13</sup> The bone loss at stage II implant surgery in successfully osseointegrated implants is generally vertical and noted only around the implant characterised by saucerisation, not the surrounding bone even though during surgery all the bone was exposed. Therefore, this hypothesis is not generally supported.

**\_Occlusal overload**

Research has indicated that occlusal overload often resulted in marginal bone loss or de-osseointegration of successfully osseointegrated implants.<sup>1, 3, 14-20</sup> The crestal bone around dental im-

**Table I** Comparison between tooth and implant.

	<b>Tooth</b>	<b>Implant</b>
Connection	Periodontal ligament (PDL)	Osseointegration (Brånemark <i>et al.</i> , 1977), functional ankylosis (Schroeder <i>et al.</i> , 1976)
Proprioception	Peridontal mechanoreceptors	Osseoperception
Tactile sensitivity (Mericske-Stern <i>et al.</i> , 1995)	High	Low
Axial mobility (Sekine <i>et al.</i> , 1986; Schulte, 1995)	25–100 µm	3–5 µm
Movement phases (Sekine <i>et al.</i> , 1986)	Two phases Primary: non-linear and complex Secondary: linear and elastic	One phase Linear and elastic
Movement patterns (Schulte, 1995)	Primary: immediate movement Secondary: gradual movement	Gradual movement
Fulcrum to lateral force	Apical third of root (Parfitt, 1960)	Crestal bone (Sekine <i>et al.</i> , 1986)
Load-bearing characteristics	Shock absorbing function Stress distribution	Stress concentration at crestal bone (Sekine <i>et al.</i> , 1986)
Signs of overloading	PDL thickening, mobility, wear facets, fremitus, pain	Screw loosening or fracture, abutment or prosthesis fracture, bone loss, implant fracture (Zarb & Schmitt, 1990)

**Table I**

plants could be a fulcrum for lever action when a bending moment is applied, suggesting that implants could be more susceptible to crestal bone loss by mechanical force.

Factors associated with increased bending overload in dental implants:

- \_ Prostheses supported by one or two implants in the posterior region (Rangert *et al.* 1995);
- \_ Straight alignment of implants;
- \_ Significant deviation of the implant axis from the line of action;
- \_ High crown/implant ratio;
- \_ Excessive cantilever length (>15 mm in the mandible, Shackleton *et al.* 1994; >10–12 mm in the maxilla, Rangert *et al.* 1989; Taylor 1991);
- \_ Discrepancy in dimensions between the occlusal table and implant head;
- \_ Para-functional habits, heavy bite force and excessive premature contacts (>180 µm in monkey studies, Miyata *et al.* 2000; >100 µm in human studies, Falk *et al.* 1990);
- \_ Steep cusp inclination;
- \_ Poor bone density/quality; and
- \_ Inadequate number of implants.

The cortical bone is known to be least resistant to shear force, which is significantly increased by bending overload. The greatest bone loss was seen on the tension side.<sup>29</sup> According to Von Recum, when two materials of different moduli of elasticity are placed together with no intervening material and one is loaded, a stress contour increase is observed where the two materials first come into contact.<sup>30</sup> Photoelastic and 3-D finite element analysis studies demonstrated V- or U-shaped stress patterns with greater magnitude near the point of the first contact between implant and the photoelastic block, which is similar to the early crestal bone loss phenomenon.<sup>31</sup>

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 mineralisation after one year alters the stress-strain relationship and reduces the risk of microfracture during the following years.<sup>32</sup> Wiskott and Belser described a lack of osseointegration attributed to increased pressure on the osseous bed during implant placement, establishment of a physiological biological width, stress shielding and lack of adequate biomechanical integration between the load-bearing implant surface and the surrounding bone.<sup>33</sup> They focused on the significance of the relationship between stress and bone homeostasis.

Based on a study by Frost,<sup>34</sup> five types of strain levels interrelated with different load levels in the bone were described:

- 1) Disuse, bone resorption;
- 2) Physiological load, bone homeostasis;
- 3) Mild overload, bone mass increase;
- 4) Pathological overload, irreversible bone damage; and
- 5) Fracture.

The concept of "microfracture" was proposed by Roberts *et al.*, who concluded that crestal regions around dental implants are high-stress-bearing areas.<sup>35</sup> They explained that if the crestal region is overloaded during bone remodelling, "cervical cratering" is created around dental implants. The study recommended axially directed occlusion and progressive loading to prevent microfracture during the bone-remodelling 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

**Table II** \_ Studies regarding the biologic width around natural teeth or dental implants.

	Dental Implants				
	Natural teeth		Non-submerged	Submerged	
	Gargiulo <i>et al.</i> <sup>57</sup> 30 human skulls	Vacek <i>et al.</i> <sup>58</sup> 10 human skulls	Cochran <i>et al.</i> <sup>68</sup>	Berglundh <i>et al.</i> <sup>53</sup>	Abrahamsson <i>et al.</i> <sup>71</sup>
Sulcus depth (SD)	0.69 mm	1.34 mm	0.16 mm	2.14 mm	2.14 mm
Junctional epithelium (JE)	0.97 mm	1.14 mm	1.88 mm		
Connective tissue attachment (CT)	1.07 mm	0.77 mm	1.05 mm	1.66 mm	1.28 mm
Biologic width	2.04 mm	1.91 mm	3.08 mm	3.80 mm	3.42 mm
	(JE + CT)	(JE + CT)	(SD + JE + CT)	(SD + JE + CT)	(SD + JE + CT)



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.<sup>36</sup> Appleton *et al.* reported a decrease in crestal bone loss in progressively loaded implants, compared with 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.<sup>37</sup> Greater crestal bone loss observed at the first year of function compared with following years can be explained by a reduced occlusal overload or increased resistance to occlusal overload after the first year of function including a functional adaptation of the oral musculature, wear of the prosthesis material, and/or an increase in bone density after a certain time period.

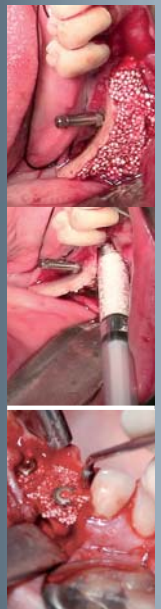
## Peri-implantitis

Peri-implantitis is one of the two main causative factors of implant failure in later stages. A correlation between plaque accumulation and progressive bone loss around implants has been reported in experimental studies and clinical studies. Tonetti and Schmid reported that peri-implant mucositis is a reversible inflammatory lesion confined to peri-implant mucosal tissues without bone loss. Peri-implantitis however begins with bone loss around dental implants.<sup>18</sup>

Clinical features of peri-implantitis were described by Mombelli as including radiographic evidence of vertical destruction of the crestal bone, formation of a peri-implant pocket in association with radiographic bone loss, bleeding after gentle probing, possibly with suppuration, mucosal swelling, redness and no pain typically.<sup>38</sup> In an experimental study evaluating the pattern of ligature-induced breakdown of peri-implant and periodontal tissues in beagle dogs, significantly greater tissue destruction was demonstrated clinically, radiographically, and histomorphometrically at implant areas than at tooth sites. It was also found that significantly fewer vascular structures existed at implant sites compared with periodontal tissues.

The difference in collagen fibre direction (parallel to the implant surface and perpendicular to tooth surface) and amount of vascular structure may explain the faster pattern of tissue destruction in peri-implant tissues than periodontal tissues. Literature has shown that peri-implantitis is similar in nature to periodontitis in that the microbiota of peri-implantitis resemble the microbiota of periodontitis; however, there has been no evidence that peri-implantitis induces crestal bone loss during healing and in the first year of function at a faster rate than following years.

Early crestal bone loss may result in an environment favourable for anaerobic bacterial growth, thus possibly contributing to more bone destruction in following years. In the majority of implants however the bone loss is dramatically reduced after the first year of prosthesis loading. Therefore, peri-implantitis as the main causative factor for early implant bone loss may not be justified.



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## \_Micro-gap and the platform-switching concept

Many implant systems have an abutments used with conventional implant types that are flush with the implant shoulder in the contact zone. This results in the formation of microcracks between the implant and the abutment. Numerous studies have shown that bacterial contamination of the gap between the implant and the abutment adversely affects the stability of the peri-implant tissue. If above-average axial forces are exerted on the implant, a pumping effect may ensue (depending on the positive internal/external connection at the interface), which may then result in a flow of bacteria from the gap, causing the formation of inflammatory connective tissue in the region of the implant neck.<sup>39-41</sup>

Berglundh and Lindhe evaluated the micro-gap of the Brånemark two-stage implant and found that inflamed connective tissue existed 0.5 mm above and below the abutment-implant connection, which resulted in 0.5 mm bone loss within two weeks after the abutment had been connected to the implant.<sup>42</sup> Ericsson et al. coined the term distance-sleeve-associated infiltrated connective tissue to describe this phenomenon. They interpreted this to be a biological protective mechanism against the bacteria residing in the microcrack, explaining the plaque-independent bone loss of approximately 1 mm during the first year. This bone loss may result in a reduction of the marginal bone level in both the vertical and the horizontal dimensions.<sup>43</sup>

If the microcrack is located close to the bone, the creation of the biological width will occur at the expense of the bone. The platform-switching effect was first observed in the mid-1980s. At the time, larger-diameter implants were often restored with narrower abutments (Ankylos, DENTSPLY Friadent; AstraZeneca; Bicon), as congruent abutments were often still unavailable. As it later turned out, this was a remarkable coincidence.<sup>44</sup> The platform-switching concept requires that this micro-gap be placed away from the implant shoulder and closer toward the axis in order to increase the distance of this micro-gap from the bone as a protective measure.

## \_Biological width

The clinical term biological width denotes the dimensions of periodontal and peri-implant soft-tissue structures such as the gingival sulcus, the junctional epithelium, and the supra-crestal connective tissues.<sup>45</sup> According to measurements conducted by Gargiulo *et al.*, the average biological width (from the base of the sulcus to the alveolar bone margin) is 2.04 mm, of which 0.97 mm is epithelial attachment

and 1.07 mm is connective tissue attachment.<sup>46</sup> These dimensions, however, are in no way static but subject to interindividual variation (from tooth to tooth and from patient to patient) and will also vary according to gingival type and implant concepts.

Numerous studies have shown that bone resorption around the implant neck does not start until the implant is uncovered and exposed to the oral cavity. This invariably leads to bacterial contamination of the gap between the implant and the superstructure.<sup>47-50</sup> Bone remodelling will progress until the biological width has been created and stabilised. This width progresses not only apically along the vertical axis (Fig. 1), but also 1 to 1.5 mm horizontally, according to studies conducted by Tarnow *et al.* This is the reason for maintaining a minimum distance of 3 mm between two implants and platform switching in the aesthetic reconstruction zone in order to obtain intact papillae and stable inter-implant bone.<sup>51-53</sup>

## \_Summary

Maintenance of crestal bone around dental implants is one of the critical factors that affect its longevity and aesthetic soft-tissue architecture. Preservation of such bone is a multifactorial process; as summarised in this article some other factors related to crestal bone loss have been investigated. These includes bone volume, bone quality, soft-tissue biotype, condition of the adjacent teeth, implant design, implant dimensions, abutment design, augmentation procedures, implant insertion depth, time of loading, time of restoration, frequency of prosthetic secondary-component replacement, suturing techniques and patient compliance.

Proper tissue maintenance and care, regular hygienic evaluations and patient education on proper methods for home care are vital. Continued evaluation via probing, radiographic assessment and oral examination will allow the clinician to ensure long-term maintenance and overall treatment success.

*Editorial note: A list of references is available from the publisher.*

## \_contact

## implants

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