After exposing a submerged implant to the oral environment, bone loss occurs downwards along the implant body and stops at some predefined position. The implant abutment interface (IAI) is the joint between the implant and the abutment that is a feature common to two-piece implants (Fig. 1). A current area of hot debate is the significance of the IAI and its influence on crestal bone level in relation to the first thread. Several theories have been proposed based on a number of studies which attempt to explain this phenomenon. These range from trauma during surgery to load shielding resulting in crestal bone loss to the first thread or the inherent need for a minimum Biologic Width as seen in the natural dentition.

**Criteria for Implant Success**

Part of the early generally accepted criteria for implant success, is that less than 0.2 mm of alveolar bone loss occurs per year after the first year in function. However what is overlooked is that the success of implant therapy is determined after the first year of service because most of the bone loss occurs during the first 12 months following abutment connection. Therefore the loss of 2.0 mm of crestal bone over the first year has been considered a normal characteristic of a healthy functioning implant and this change in bone height is merely due to remodeling in responses to loading. In another words the bone is adapting to changes in load following prosthetic restoration. The question that needs to be redressed is, does this small amount of bone loss have any clinical significance and can it be considered acceptable. Dental implants unlike implants employed in other areas of medicine have two roles to fulfill, aesthetic and functional. The loss of seemingly small amounts of bone and soft tissue can have important implications on aesthetics of implant-borne restorations, which are reliant on healthy and vertically constant bony supported soft tissue dimensions over time.

**The Microgap**

In recent years the microgap which exists at the connection between the implant body and the restorative abutment-That is the IAI, has been implicated. This gap permits micro-leakage of fluids containing small molecules in the range of disaccharides and short peptides that contain bacterial by products or nutrients required for bacterial growth or abutment inflammatory cell infiltrate (a-ICT).

The sustained activation of inflammatory cells has been shown to promote osteoclast formation and activation, which can result in alveolar bone loss. It has been proposed that this in turn may initiate the one to two millimeters apical bone loss that occurs around submerged implants. This however remains to be clarified and distinguished from the phenomenon of bone resorption adjacent to the smooth implant neck. These infiltrates are invariably separated from the alveolar bone by a zone of non-inflammatory connective tissue (CT) showing clinically stable conditions (Fig. 2).

**Soft Tissue Interface**

Although the gingiva at teeth and the keratinized mucosa (peri-implant mucosa, PM) at titanium implants have features in common such as...
In the natural dentition, the junctional epithelium provides a seal at the base of the sulcus against bacterial penetration. The other line of defense present in the natural dentition and absent in implants is the periodontal ligament. Since no cementum or fibers are present on the surface of an implant, infection has the potential to spread directly into the osseous structures resulting in bone loss and ultimately implant failure. Thus, the maintenance of osseointegration and long term success of implants depends on the presence of a leak-proof peri-implant soft tissue cuff. This requires the formation of a biological seal dependent on the tight contact between the epithelium and adjacent connective tissue with the implant surface.

**Biologic Width**

The majority of research has been directed towards hard tissue integration with less emphasis on soft tissue integration involving epithelium and connective tissue. It is important to understand the mechanisms which permit the implant or abutment to transverse the soft tissues while maintaining a seal against bacterial ingress. The biologic width, is a soft tissue barrier composed of an epithelial component continuous with a zone of connective tissue. For natural teeth and implants alike, the biologic width is a physiologically formed and stable dimension. It is suggested that a certain width of peri-implant mucosa is essential in order to enable proper epithelial-connective tissue attachment and, if this absolute soft tissue dimension is not satisfied, bone resorption will occur to ensure the reestablishment of an attachment of the appropriate biologic width.

Therefore, the "Biologic Seal" is composed of junctional epithelium (JE) attachment to either the abutment of a 2-stage implant or neck section of a 1-stage implant and a tight band of connective tissue contact (CTC).

**Platform Switching**

Recently it has been suggested that the presence and position of the microgap may not be the principal cause of bone resorption phenomenon, but instead that it is due to the relative diameters of the abutment and the implant platform (Fig. 5). Radiographically the anticipated change in crestal bone level does not occur if the implant is restored with an abutment having a smaller diameter than the corresponding implant (Figs. 6 & 7). This observation has given birth to the concept of "Platform Switching".16,17,18

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(Figure 2)
IAI = Implant Abutment Interface, aICT = Abutment Inflammatory Cell Infiltrate (1.5mm = 0.75mm above IAI, 0.75mm below the IAI), CT = Zone of healthy connective tissue between the base of the aICT and crestal bone (1mm thick).

(Figure 3)
Histomorphometric Landmarks: GM = gingival margin, aS = apical extension of sulcus, aJE = apical extension of junctional epithelium, IAI = implant abutment interface, BC = bone contact. SD = sulcus depth, JE = junctional epithelium, CTC = connective tissue contact. BW = biologic width = SD + JE + CTC. From the cementum collagen fibers project laterally into the gingiva.

(Figure 4)
Histomorphometric Landmarks: PM = margin of peri-implant mucosa, aS = apical extension of sulcus, aJE = apical extension of junctional epithelium, IAI = implant abutment interface, BIC = Bone implant contact. BW = biologic width = SD + JE + CTC. There is no cementum or attached lateral collagen fibers.

(Figure 5)
Platform Switching. Abutment diameter is within implant platform.
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It is proposed that horizontal positioning of the implant abutment interface (IAI) has two effects.

1. The increased surface area created by the exposed implant seating surface, reduces the amount of crestal bone resorption necessary to expose a minimum vertical amount of implant surface for soft tissue attachment. In effect, the biologic width has been repositioned horizontally (Fig. 8).

2. Repositioning the IAI away from the outer edge of the implant and adjacent bone, confines the abutment inflammatory cell infiltrate (aICT) and reduces the overall effect on the surrounding tissues thus decreasing the resorptive effect on the crestal bone (Fig. 9).

**Conclusion**

It appears desirable to incorporate features into implant design which reduce microleakage or limit its effects. A tapered IAI creates a microseal and the hermetic seal produced eliminates the microgap preventing the passage of bacteria or aICT. Platform switching may limit or confine the effects of the aICT (Fig. 10).

**References**

9. Quirynen M, Bollen MC, Eyssen H, van Steenberghe D. Microbial penetration along the implant components of the Brånemark


