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Review Article

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PERIMPLANT HEALTH

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ABSTRACT

Soft tissue relationship to implant surface is one of the most challenging area for implant manufacturers as it is evident by different kind of connections, implant shoulders and platforms. The soft tissue compartment is denoted "peri-implant mucosa" and is formed during the wound healing process that follows implant/abutment placement.[1] The hard tissue compartment forms a contact relationship to the implant surface to secure implant stability.[2] Due to their histologic and anatomic features, peri-implant tissues carry out two basic functions: the mucosa protects the underlining bone, while the bone supports the implant. Indeed, the destruction of peri-implant tissues can jeopardize the implant success and survival,[3] and the understanding of the characteristics of healthy peri-implant tissues allows the recognition of disease. Thus, the aim of the present review was to define clinical and histologic characteristics of periimplant tissues in health and describe the mucosa–implant interface.

KEYWORDS: Connective tissue biology, diagnosis, implantology, osseointegration, Periimplant tissues.

INTRODUCTION

Teeth are anatomically unique because they are the only structures of the body that penetrate a lining or covering epithelium. Thus, teeth and dental implants are two isolated examples of structures that pierce the integument. While proper anchorage of an implant in the bone (osseointegration) is a prerequisite for its stability, long-term retention of an implant seems to depend on the epithelial and connective tissue attachment to the titanium sur face, ie, a complete soft tissue seal protecting the bone from the oral environ ment (eg, Branemark 1985, Gould 1985, Ten Gate 1985, McKinney et al 1988, Carmichael et al 1989).

Peri-implant tissues are those that occur around osseointegrated dental implants. They are divided into soft and hard tissue compartments. The soft tissue compartment is denoted "peri-implant mucosa" and is formed during the wound healing process that follows implant/abutment placement.[1] The hard tissue compartment forms a contact relationship to the implant surface to secure implant stability.[2] Due to their histologic and anatomic features, peri-implant tissues carry out two basic functions: the mucosa protects the underlining bone, while the bone supports the implant. Indeed, the destruction of peri-implant tissues can jeopardize the implant success and survival,[3] and the understanding of the characteristics of healthy peri-implant tissues allows the recognition of disease.

Peri-Implant Mucosa

Most information regarding the structural features of the peri-implant mucosa is derived from animal studies using dog models.4–15 In such studies implants were placed in the edentulous ridge (alternatively, the fresh extraction socket), the outer osseous part of which was covered with masticatory mucosa. It was also shown that the healed peri-implant mucosa on the buccal aspect averaged about 3 to 4 mm high when measured from the mucosal margin to the crest of the peri-implant bone. In addition, this mucosa contains a core of connective tissue, mainly comprised of collagen fibers and matrix elements (85%), comparatively few fibroblasts (3%), and vascular units (5%). The outer (oral) surface of the connective tissue is covered by an often orthokeratinized epithelium.

The portion of the peri-implant mucosa that is facing the implant (abutment) contains two distinct parts, a "coronal" portion that is lined by a thin barrier epithelium (similar to the junctional epithelium of the gingiva) and sulcular epithelium, and a more "apical" segment in which the connective tissue appears to be in direct contact with the implant surface. This apical portion of the peri-implant mucosa is designated zone of connective tissue adhesion.

In the connective tissue immediately lateral to the barrier and sulcular epithelium, a delicate plexus of vascular structures, similar to the dentogingival vascular plexus,[4]

is consistently present,5 while the connective tissue adhesion zone appears to harbor only limited amounts of vascular structures.

At implants placed into masticatory mucosa, the main collagen fiber bundles are anchored in the crestal bone and extend in a marginal direction parallel to the surface of the metal device. It is assumed that circular fibers may also be present in this type of peri-implant mucosa.

Moon et al.6 analyzed under electron scanning microscope the zone of connective tissue adhesion confined to a 200-μm wide zone of the connective tissue facing the implant. The findings demonstrated that the adhesion includes two distinct layers: one inner layer, about 40 μmwide, which harbors large amounts of fibroblasts (32% of volume) that appear to be in intimate contact with the surface of the implant; and one outer layer, about 160 μmwide, that is dominated by collagen fibers (83%), smaller amounts of fibroblasts (11%), and larger volumes of vascular structures (3%).[6]

Valid histologic information is not currently available regarding the peri-implant mucosa when implants are placed in non-keratinized lining or alveolar mucosa.

Berglundh et al (1994)7 studied the vascular topography of the periodontium and the peri-implant soft and hard tissues using the beagle-dog model. The authors observed that the gingiva and the supracrestal connective tissue at teeth are supplied by (1) supraperiosteal vessels lateral to the alveolar process and (2) vessels from the periodontal ligament. The periimplant mucosa, on the other hand, was found to be supplied by terminal branches of larger vessels originating from the periosteum of the bone at the implant site.

In both situations, the blood vessels built a characteristic "crevicular plexus" lateral to the junctional epithelium (Egelberg 1966). At teeth, the supracrestal connective tissue portion demonstrated a rich vascularization, while at the corresponding implant sites very few, if any, vessels were observed (Fig 1-2). These observations support the suggestion made by Buser et al (1992) that the peri-implant soft tissue may have an impaired defense capacity against exogenous irritation.

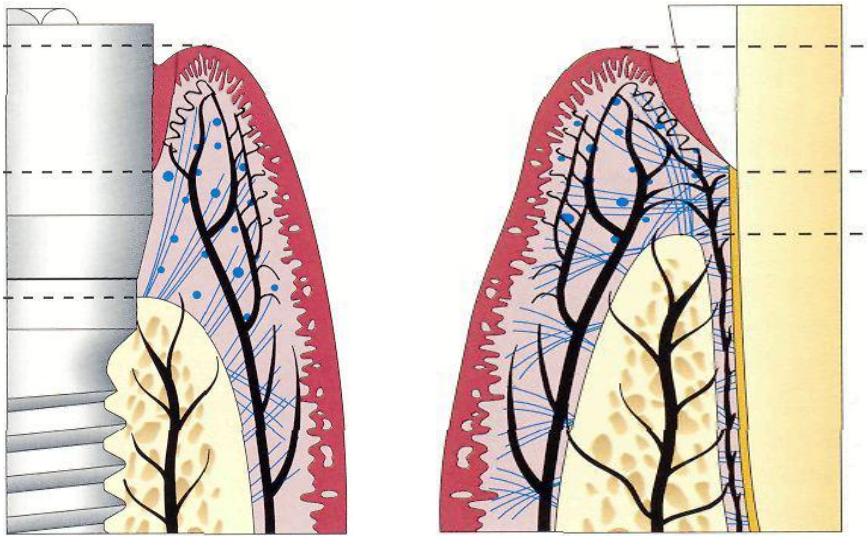


Fig. 1.1: Vascular topography of the peri-implant soft and hard tissues *(left)* and of the penodontium *(right)*. PM - peri-implant soft tissue margin; aJE - apical termination of the junctional epithelium; AFJ = abutment-fixture junction; BC = marginal bone crest; GM - gingival margin; CEJ = cementoenamel junction.

Ericsson and Lindhe (1993),[8] using the beagle-dog model, examined the resistance to mechanical probing offered by clinically healthy gingival tissues and periimplant mucosa at titanium dental implants. The authors reported that the probe penetration was more advanced at implants than at teeth {- 2.0 mm and ~ 0.7 mm, respectively). Thus, at the implant sites the probe tip displaced the junctional epithelium as well as the connective tissue portion facing the abutment surface in the lateral direction and stopped close to the bone crest (Fig 1-6). The tip of the probe thus stopped within the supracrestal connective tissue portion, and occasional rupture of some blood vessels resulted in bleeding. At the tooth sites, however, the tip of the probe consistently terminated coronally to the apical portion of the junctional epithelium, thus roughly identifying the bottom of the gingival pocket (Fig 1-2).

Bleeding on probing is an important tool to properly diagnose the condition of the apical portion of the periodontal soft tissues. In this study, however, bleeding on probing was sometimes observed at implants, but rarely at teeth. Based on current knowledge, the importance of such an observation at implants is doubtful.

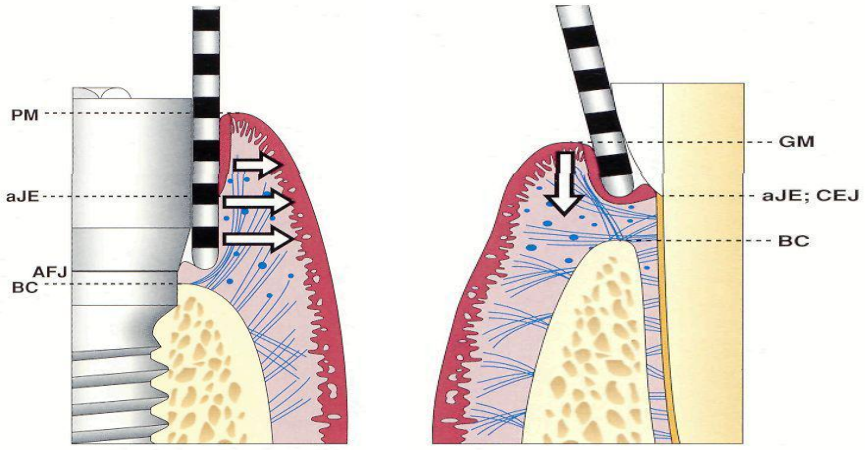


Fig. 1-2: Results of probe penetration at Branemark System® implants *(left)* and teeth *(right)*. PM – periimplant soft tissue margin; aJE = apical termination of the junctional epithelium; AFJ - abutment-fixture junction; BC = marginal bone crest; GM = gingival margin; CEJ = cementoenamel junction

Probing Peri-Implant Tissues

For many years it was incorrectly assumed that the tip of the periodontal probe in a probing depth (PD) measurement identified the apical base of the dento-gingival epithelium.[9] Later research documented, however, that this was not the case. At healthy sites the tip of the probe failed to reach the apical portion of the epithelial barrier, while at diseased sites the probe found the apical base of the inflammatory cell infiltrate.

Hence, PD measurements assess the depth of probe penetration or the resistance offered by the soft tissue.[9,10]

The influence of the condition (health, disease) of the periimplant mucosa on the outcome of the probing measurement was studied in animal models. Lang et al.[11] reported that at sites with healthy mucosa or mucositis, the tip of the probe identified the apical border of the barrier epithelium with an error of approximately 0.2 mm, while at sites with peri-implantitis, the measurement error was much greater at 1.5 mm. Abrahamsson and Soldini,[12] in a subsequent study, stated that the probe penetration into the healthy soft tissues at the buccal surface of teeth and implants in dogs was alike and similar to the length of the junctional/barrier epithelium.

It was assumed that probing the implant–mucosa interface would sever the soft tissue seal and jeopardize the integrity of the adhesion. This issue was examined in a dog study13 that documented that already after 5 to 7 days following clinical probing, the soft tissue seal had regenerated to its full extent.

The authors reported,[14] that the prolonged period of plaque accumulation resulted in a development of an inflammatory- cell infiltrate in the gingiva and the peri-implant mucosa. The two infiltrates had many features in common, but the apical extension was more pronounced in the peri-implant mucosa than in the corresponding lesion in the gingiva (Fig 1-3).

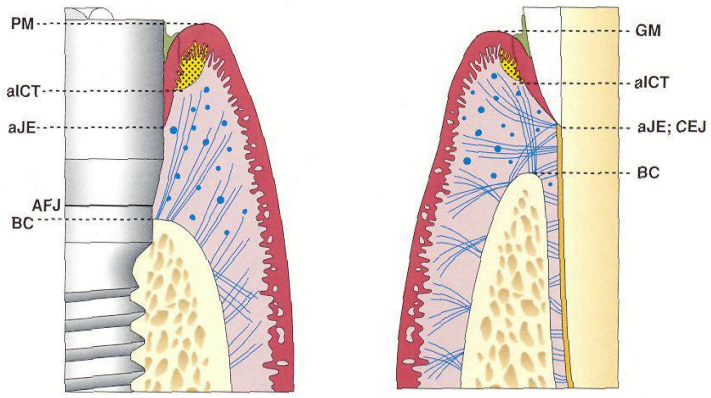
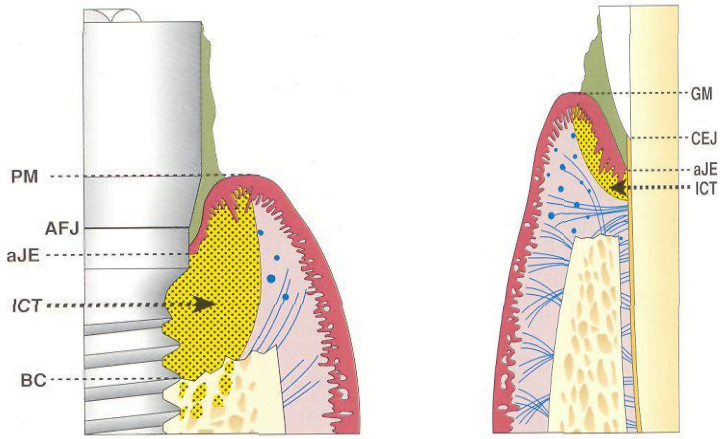


Fig. 1.3: Anatomical landmarks of the peri-implant soft and hard tissues *(left)* and the periodontium *(right)* following long-standing plaque formation. PM = peri-implant soft tissue margin; alCT = apical termination of the infiltrated connective tissue; aJE = apical termination of the junctional epithelium; AFJ = abutment-fixture junction; BC - marginal bone crest; GM - gingival margin; CEJ = cementoenamel junction.

The data reported above indicated the following: (1) for teeth, 3 weeks to 3 months of undisturbed plaque accumulation resulted in no further extension of the inflammatory lesion, but (2) at implants, under identical experimental conditions, a further spread in apical direction of the inflammatory- cell infiltrate was consistently observed.

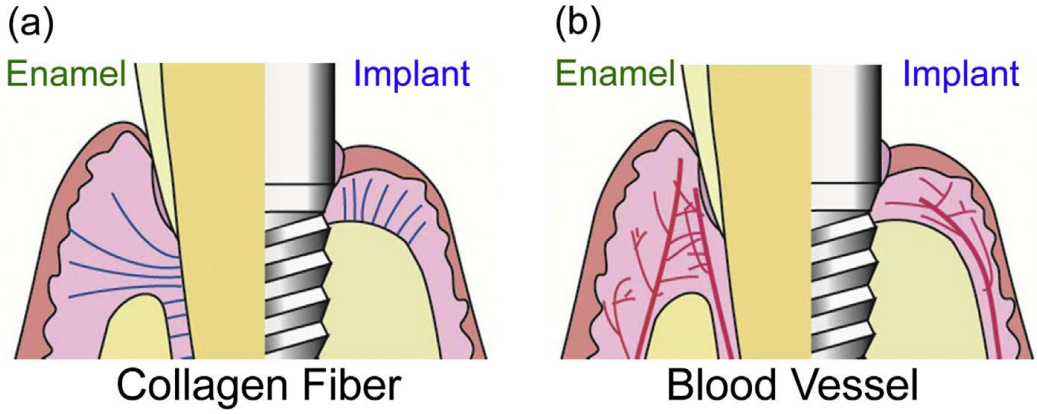
This implies that the defense mechanism of the gingiva may be more effective than that of the peri-implant mucosa in preventing further apical propagation of the pocket microbiota. This hypothesis is further supported by Lindhe15 et al (1992) and Marinello et al (in press).16 Lindhe and coworkers (1992) induced experimental breakdown of peri-implant and periodontal tissues in dogs by placing cotton-floss ligatures submarginally and reported that 1 month following ligature removal, (1} "the resulting tissue destruction was more pronounced at implants than at teeth, (2) the size of the soft tissue lesion was larger at implants than at teeth, and (3) the lesion at implants but not at teeth frequently extended into the bone marrow"



(Fig 1-4): Anatomical landmarks of the peri-implant *(left)* and the periodontal *(right)* tissues following experimental breakdown. PM = peri-implant soft tissue margin; AFJ = abutment-fixture junction; ICT = infiltrated connective tissue; aJE = apical termination of the junctional epithelium; BC = marginal bone crest; GM - gingival margin; CEJ - cementoenamel junction

Structure of the interface between the implant and connective tissue

In case of natural teeth, the connective tissue attachment is apical to the JE and resists the physical invasion of bacteria by providing strong adhesion between the special fibers as periodontal ligament and cementum, and through compact type III collagenous fibers. However, around an implant there are many type V collagenous fibers with resistance to collage- nase, so peri-implant connective tissue is generally a chronic inflammatory condition rather than intercept or defence structure. In addition, the fiber orientation and attachment patterns of the epithelium to the implant and tooth are fundamentally different because of the absence of cementum and periodontal ligament around the implant.[16] In short, while the fiber orientation in the connective tissue around natural teeth is perpendicular to the root surface, it runs parallel to the surface around dental implants.[13]



(Fig. 1.5). Locus of collagen fibers and blood vessels in gingivae. (a) Natural tooth has collagen fibers perpendicular to the cementum surface, whereas around implants, these fibers extend from the bone and run parallel to the implant surface. (b) Normal periodontal soft tissue is supplied by blood from vessels running both outside the alveolar bone and through the periodontal ligament; in contrast, the peri-implant tissue has a reduced blood supply as the periodontal ligament source is not present.

This weak, poorly-sealing connective tissue around the implant may accelerate horizontal recession. The role of connective tissue around both implant and tooth is not only for the protection from the extra stimulation as oral bacteria, but also for the supply of nutrients from the blood vessel. However, the PIE is also disadvantaged in comparison with the JE by its limited supply of nutrients. While the periodontal tissue has ample blood flow from the periodontal ligament, periosteum, and connective tissue, the blood supply to peri-implant soft tissue is mainly from the connective tissue.[17] (Fig. 1.5). In addition, the soft tissue around the implant is dependent upon the alveolar bone for its blood supply in the absence of other supporting periodontal tissues

Structure of soft-tissue sealing around implant

The goal of management for peri-implant tissue depends on many clinical factors, including the location of implant placement, the form of the implant abutment, and the clinical type of gingiva. For example, in a posterior tooth, the acquisition of attached gingiva is important because effective cleaning is a high priority and the peri-implant soft tissue must be able to withstand it. In anterior teeth, esthetics is a higher priority, so natural gingival form and a healthy color are the prerequisite properties.18 Physiologically, the attached gingiva is a firmly anchored with oral mucosa that has less mobility, being tethered to the underlying periosteum by epithelial and connective tissue attachments (JE and periosto-gingival fibers, respectively) (Fig1.6).

The width of this attached gingiva varies, even in the same oral cavity; upper and anterior sites have wider attached gingivae than lower and posterior sites.

The biologic significance of peri-implant soft tissue

The creation of a soft tissue barrier around a dental implant at the point where it emerges into the oral cavity is an important stage in the process of rendering the implant functional and ensuring the esthetic integration of the implantsupported prosthetic restoration (Fig 1-7). Maintaining this seal in a condition of health is critical to the function and long-term prognosis of the implant.[19] The ultimate purpose of the peri-implant soft tissue seal is to protect the underlying bond between the implant and bone tissue created through the osseointegration process.

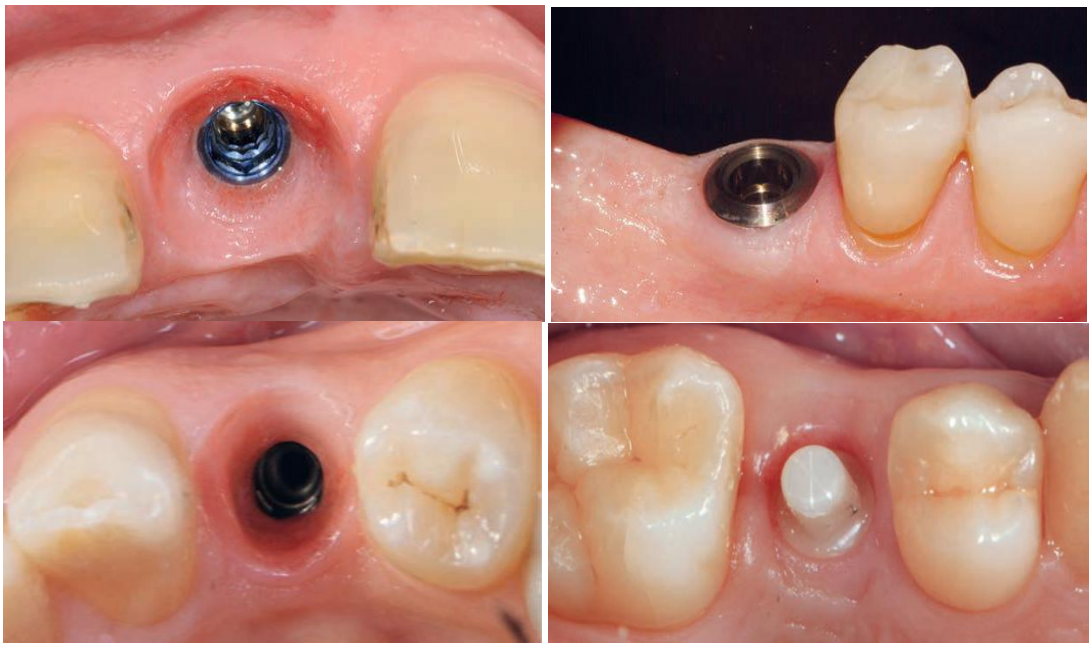


Fig. 1.7: Physiologic appearance of peri-implant soft tissues around different implant types. *(a and b)* Bone-level titanium implants. In these cases the depth of the peri-implant mucosal tunnel, ie, the distance from the gingival margin to the implant connection, is greater. *(c)* Tissue-level titanium implant. In these cases the peri-implant mucosal tunnel is more shallow. *(d)* Single-component zirconia implant. With tissue-level implants, the implant prosthetic platform is located closer to the surface in a juxtagingival or slightly subgingival position

Healing of Soft Tissue around an implants

Formation of a transmucosal or peri-implant attachment begins with the implant placement for single-component implants (Fig 1-8). The epithelial cells at the margin of the surgical flap adapted to the implant or abutment neck proliferate and migrate to cover the underlying connective tissue and adhere to the implant or abutment surface, forming a junctional epithelium. Apical migration of the epithelial cells ends at a band characterized by dense connective tissue and located immediately above the bone ridge, which also comes into contact with the implant surface (Fig 1-9). For two-piece implants, formation of the peri-implant attachment begins at surgical reopening and abutment attachment.[20]

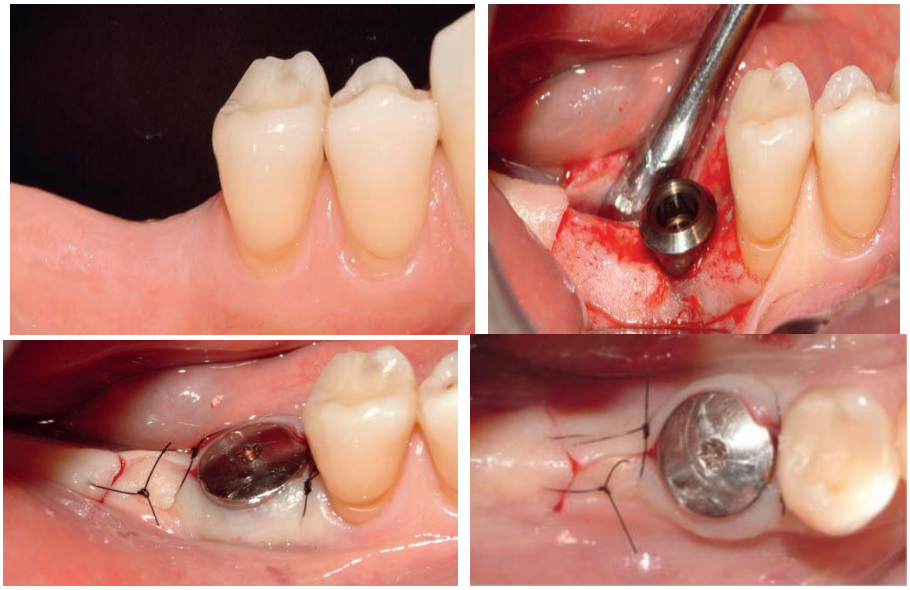


Fig. 1.8: ***(a to d)*** **The peri-implant mucosal seal around transgingival or single-component implants begins to form immediately after implant insertion, when the soft tissues are adjusted to fit the smooth implant neck by means of sutures.**

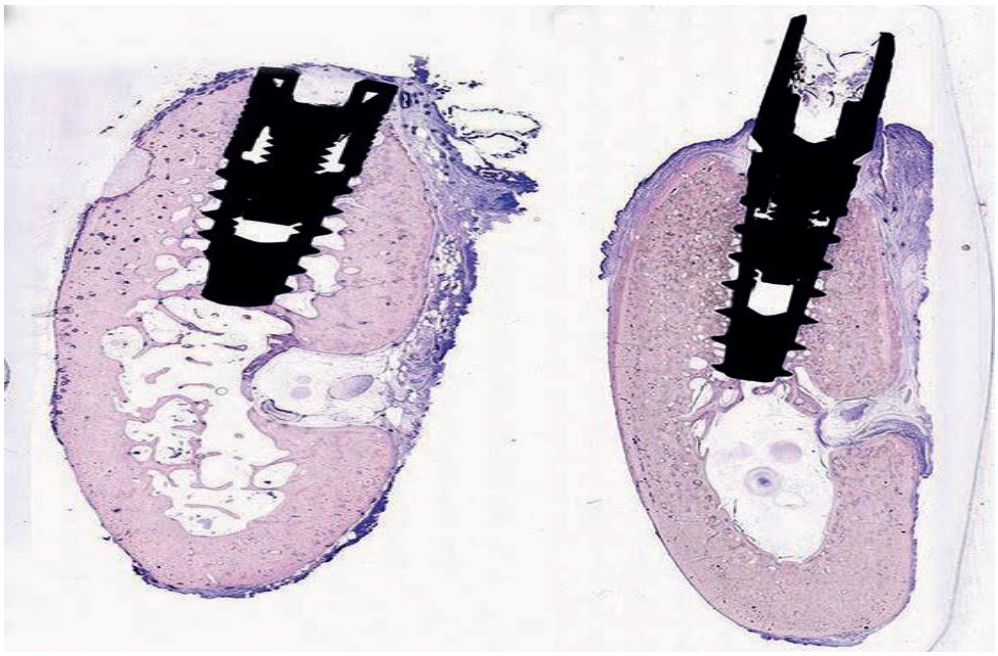


Fig. 1.9: Histologic evaluation of peri-implant hard and soft tissue healing in a dog model 12 weeks after insertion. ***(a)*** **With submerged healing, the implant achieves secondary stability through the osseointegration process. The soft tissues above the implant, consisting of epithelium and connective tissue, completely cover the head of the fixture.** ***(b)*** **After reopening and abutment connection, the soft tissues adapt around the abutment and heal by creating a mucosal tunnel consisting of an epithelial attachment and connective tissue attachment up to the first contact between bone and implant. (Courtesy of Prof J. L. Calvo Guirado, Murcia, Spain.)**

CONCLUSIONS

The healthy peri-implant mucosa is comprised of a core of connective tissue covered by either a keratinized or nonkeratinized epithelium. Most of the intrabony part of the implant is in contact with mineralized bone, while the remaining portion faces bone marrow, vascular structures, or fibrous tissue. The characteristics of peri-implant tissues in health are properly identified in the literature. According to the available definitions of peri-implant mucositis and peri-implantitis, the absence of signs of clinical inflammation is necessary for concluding that a site has peri-implant health

The influence of five different factors on peri-implant biologic width dimensions has been evaluated reviewing the available literature, these are: surgical technique, loading time, titanium surfaces and abutment materials, implant structure and position, immediate post-extractive

insertion. Microgap between implant and abutment when present can modify the dimension of biologic width, the longer epithelial component described may be determined by bacterial colonization or abutments micro movements.

REFERENCES

1. Adell, R., Lekholm. U., Rockier, B. Branemark, R-l. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *international Journal of Oral Surgery*, 1981; 6: 387-416.
2. Berglundh T, Abrahamsson I, Welander M, Lang NP, Lindhe J. Morphogenesis of the peri-implantmucosa: an experimental study in dogs. *Clin Oral Implants Res*., 2007; 18: 1–8.
3. Albrektsson T, Sennerby L. State of the art in oral implants. *J Clin Periodontol*, 1991; 18: 474–481.
4. Egelberg J. The blood vessels of the dento-gingival junction. J Periodontal Res., 1966; 1: 163–179.
5. Berglundh T, Lindhe J, Jonsson K, Ericsson I. The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. J Clin Periodontol, 1994; 21: 189–193.
6. Moon IS, Berglundh T, Abrahamsson I, Linder E, Lindhe J. The barrier between the keratinized mucosa and the dental implant. An experimental study in the dog. J Clin Periodontol, 1999; 26: 658–663.
7. Berglundh, T., Lindhe, J., Jonsson, K., Ericsson, I. The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. *Journal of Clinical Periodontology*, 1994.
8. Ericsson, I., Lindhe, J. Probing at implants and teeth. An experimental study in the dog. Journal of Clinical Periodontology, 1993; 20.
9. Listgarten MA, Mao R, Robinson PJ. Periodontal probing and the relationship of the probe tip to periodontal tissues. *J Periodontol*, 1976; 47: 511–513.
10. Schou S, Holmstrup P, Stoltze K, Hjorting-Hansen E, Fiehn NE SL. Probing around implants and teeth with healthy or inflamed peri-implant mucosa / gingiva: a histologic comparison in cynomolgus monkeys (Macaca fascicularis). *Clin Oral Implants Res*., 2002; 13: 113–126.
11. Lang NP,Wetzel AC, Stich H, Caffesse RG. Histologic probe penetration in healthy and inflamed peri-implant tissues. *Clin Oral Implants Res*., 1994; 5: 191–201.
12. Abrahamsson I, Soldini C. Probe penetration in periodontal and peri-implant tissues.An experimental study in the beagle dog. *Clin Oral Implants Res*., 2006; 17: 601–605.
13. Etter TH, Hakanson I, Lang NP, Trejo PM, Caffesse RG. Healing after standardized clinical probing of the perlimplant soft tissue seal: a histomorphometric study in dogs. *Clin Oral Implants Res*., 2002; 13: 571–580.
14. Ericsson, L, Berglundh, T., Marinello, C. P., Liljenberg. B., Lindhe, J. (plaque and gingivitis at implants and teeth in the dog. *Clinical Oral Implants Research.*
15. Lindhe, J., Berglundh, T., Ericsson, I., Liljenberg, B., Marinello, C. P. Experimental breakdown of peri-implant and periodontal tissues. A study in the beagle dog. Clinical Oral Implants Research, 1992; 3: 9-16.
16. Marinello, C. P., Berglundh, T. Ericsson, I., Klinge, B., Glantz, R-O., Lindhe, J. Resolution of ligature induced periimplantitis lesions in the dog. Journal of Clinical Periodontology, in press.
17. Buser D, Bragger U. Two-part ITI-hollow cylinder and hollow screw implant. Phillip J., 1989; 6: 263–74.
18. Moon IS, Berglundh T, Abrahamsson I, Linder E, Lindhe J. The barrier between the keratinized mucosa and the dental implant. An experimental study in the dog. J Clin Periodontol, 1999; 26: 658–63.
19. Ramfjord SP. Periodontology and restorative dentistry. 2. Phillip J Restaur Zahnmed, 1984; 1: 163–70.
20. Schroeder HE. Healing and regeneration following periodontal treatment. Dtsch Zahnarztl Z, 1986; 41: 536–8.
21. Tarnow DP, Wallace SS, Froum SJ, Rohrer MD, Cho SC. Histologic and clinical comparison of bilateral sinus floor elevations with and without barrier membrane placement in 12 patients: Part 3 of an ongoing prospective study. Int J Periodontics Restor Dent, 2000; 20: 117–25.
22. Grunder U. Stability of the mucosal topography around single-tooth implants and adjacent teeth: 1-year results. Int J Periodontics Restor Dent, 2000; 20: 11–7.