The soft tissue response to osseointegrated dental implants

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The use of dental implants in the treatment of fully edentulous patients has become an important addition in oral/dental rehabilitation. The fact that these implants penetrate the oral mucosa can lead to the assumption that peri-implant tissues, similar to the periodontal tissues, are fulfilling an important function as a barrier to protect the bony anchorage underneath. It has been shown that insufficient plaque removal may lead to peri-implant tissue disease with bone loss similar to teeth. However, it is unclear how important this cause is as a source of implant failure compared with other factors, such as inadequate bone healing, unfavorable quantity and quality of bone, or (bio)mechanical and functional problems. It is also not understood if peri-implant epithelium and connective tissue are equally needed and/or qualified to slow down or prevent tissue breakdown as their periodontal counterparts. The scientific work focusing on peri-implant soft tissues has dramatically increased in the past few years. Most studies to date have examined and described their structure but little data exist on their true biologic function. This review analyzes the current understanding of morphologic and clinical features of the peri-implant soft tissues. Furthermore, evidence shall be provided that peri-implant soft tissues do not interfere with the current favorable results obtained when treating the edentulous patient with osseointegrated implants. (J Prosthet Dent 1998;79:79-89.)

The predominant biologic considerations in endosseous implant dentistry have focused on the bone-to-implant interface, because long-term stability of dental implants primarily depends on their anchorage in bone. However, because implants like teeth are transmucosal "devices" and as such penetrate the oral mucosa, the periodontal or peri-implant tissues are expected to exercise a protective barrier function. For the dentogingival tissues, the importance and the various functional components of the barrier properties are fairly well-understood. Although a microbial cause for peri-implant tissue disease has been documented indicating that some sort of protective barrier exists between the oral cavity and the internal aspects of the body, it is still not clear whether the peri-implant mucosa is able to provide barrier properties.[1,2]

The amount of scientific work focusing on peri-implant soft tissues has increased dramatically in the past few years. Most studies to date have examined the morphologic features of epithelium and connective tissue around implants, and only limited data exist on their function(s) and structural requirements as barrier tissues. It has been shown that insufficient plaque removal may lead to peri-implant tissue disease with bone loss similar to teeth.[1-3] However, it is unclear if this etiologic factor per se represents a predominant source of implant failure compared with other factors, such as inadequate bone healing, unfavorable quantity and quality of bone anchorage, or biomechanical and functional problems.

The purpose of this article is to analyze the current literature on peri-implant soft tissues and to put it in perspective with the treatment of the edentulous patient with osseointegrated implants.

PERI-IMPLANT TISSUES AND LONG-TERM IMPLANT SUCCESS

Various types of implants made of biologically compatible materials of various designs have been documented to maintain a healthy biologic relationship to their surrounding tissues over extended periods.[3] Longitudinal data show that implants that were placed with proper surgical techniques and restored (loaded) with adequately designed superstructures successfully support various types of prostheses in the completely or partially edentulous patient.[4-29] For the edentulous jaw, two prosthodontic treatment options are available: fixed prostheses (not soft tissue bearing) in various designs, as well as removable overdentures, either stabilized by implant-supported bar devices or individual anchors.[2]

The gold standard for evidence that one treatment modality is better than another is a randomized, controlled clinical trial of prospective nature.[30] There are no data of randomized trials available that directly compare various prosthodontic and preprosthetic modali-

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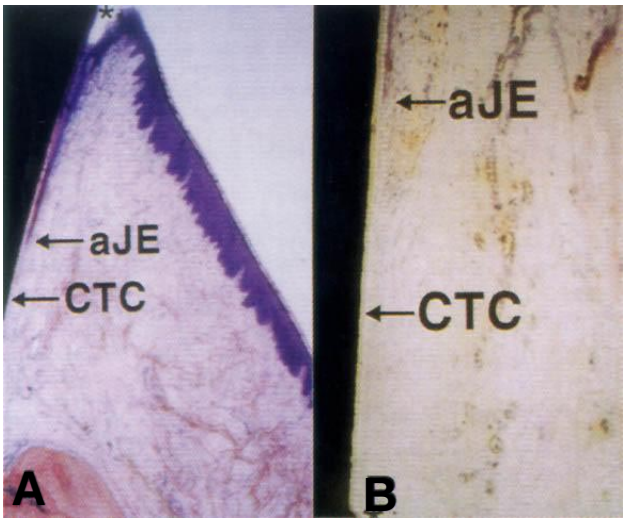

Fig. 1. A. Bucco-lingual section shows epithelial and connective portion of peri-implant mucosa, and most coronal part of alveolar bone. Rete peg formation is present only in area of keratinized oral gingival epithelium. Oral sulcular epithelium without keratinization. **B**. Same aspect as A in higher magnification. Difference between scar-like connective tissue contact adjacent to machined titanium surface and connective tissue supporting oral epithelium. *aJE* = Apical point of junctional epithelium; *CTC* = connective tissue contact. (Basic fuchsin stain, original magnification ×12.5 and ×25). With permission from Cochran DL, Hermann JS, Schenk RK et al. J Periodontol 1997;68:186-98.

ties, nonsurgical and surgical, in the management of the edentulous patient. Nevertheless, a number of prospective long-term studies regarding implant-supported prosthodontic treatment exists adequately documenting that both implant-supported fixed prostheses and overdentures represent predictable treatment modalities for the edentulous patient, with excellent patient satisfaction.[5] On the basis of a recent comprehensive meta analysis, including all available long-term studies on osseointegrated implants, the author[2] concluded that there is enough evidence to show that dental implants are a viable treatment modality.

Despite the excellent overall outcome of this treatment modality, failures and complications do occur.[2,12,31,32] The cause of implant failure appears to be multifactorial.[2] For soft tissue involvement, it has been shown that plaque accumulation on implants can lead to marginal tissue inflammation and, possibly, to periimplantitis with bone loss, which may ultimately cause the loss of an implant.[1,2] The importance of the soft tissues in the prevention, initiation, progression, and possible arrest of peri-implant tissue disease is not well-understood. It has been shown that with an adequate plaque removal discipline, no different than that for natural teeth, this etiologic factor as related to implant failure appears to be the one easiest to prevent.[2-33]

STRUCTURE OF THE PERI-IMPLANT MUCOSA

The soft tissues forming the implantogingival junction have to be examined both in terms of structure and physiologic function. Only minimal scientific information is available on the latter, whereas structure has been studied extensively.

Unlike teeth that have developed simultaneously with the periodontium and remain structurally continuous with these tissues, endosteal implants are placed into an osseous receptor site. The surrounding tissues are expected to adapt to the inserted posts, allowing them to function as tooth substitutes. It is important to recognize that, regardless of their various shapes and structural compositions, implants are not designed to interact with the surrounding tissues in the same manner as a tooth with its periodontium.[3] The absence of cementum on endosseous implants is not due to the inability of cementum to grow on an implant surface, as has been shown to occur in primates,[34-36] but rather is much more likely due to the absence of progenitor cells at sites prepared for implant insertion. The structure of soft tissues surrounding endosseous implants is in many ways analogous to the natural tooth.[3,37-39] A normal, gingiva-like tissue is frequently present around the transmucosal implant or abutment portion. This tissue consists of a dense, collagenous lamina propria, covered with a stratified, squamous, keratinizing oral epithelium. The main difference from the natural dentition is the manner in which the peri-implant connective tissues interface with the implant post.[3]

Peri-implant epithelium

Of the various tissues in contact with the implant, the epithelium most closely resembles that of the natural dentition.[3,37,39] The oral epithelium is continuous, with a sulcular epithelium lining the lateral surface of the gingival sulcus immediately apical to the gingival margin. As in the natural dentition, the sulcular epithelium resembles a nonkeratinized extension of the oral epithelium. The apical part of the sulcular epithelium is lined with the coronal cells of the junctional epithelium, forming a continuum between both tissues. The junctional epithelium provides an epithelial union between the implant and the surrounding gingiva (Fig. 1, *A* and *B*).

The junctional epithelium adheres to the implant surface through a basal lamina and hemidesmosomes.[3] The ultrastructure of this epithelial interface was first demonstrated by James and Schultz[40] using freeze-fractured preparations of vitallium implants. An intact epithelium-to-implant interface was later shown on epoxy resin replicas of extracted teeth replanted immediately into the extraction sockets of monkeys.[41] A similar relationship of the epithelium to titanium or titanium alloys was found using either evaporated layers of metal over plastic im-

plants[42] or freeze-fractured specimens from aluminum oxide ceramic implants in dogs.[43] The toothlike configuration of the peri-implant epithelium was also confirmed in the light microscopic analysis of healed peri-implant tissue sections from nonsubmerged implants placed in beagle dogs and fox hounds.[39,44]

Another study of beagle dogs compared the vascular topography of soft tissues around teeth and implants.[45] The vascular supply around teeth was derived from supraperiosteal vessels lateral to the alveolar process and from vessels within the periodontal ligament. The implant soft tissue blood supply originated from terminal branches of larger vessels from the bone periosteum at the implant site. Blood vessels adjacent to the junctional epithelium around both teeth and implants revealed a characteristic "crevicular plexus." Furthermore, although peri-implant soft tissues lateral to the implant had sparse blood vessels, the soft tissue lateral to root cementum was highly vascularized. A connective tissue zone lacking blood vessels directly adjacent to the implant surface (Fig. 2) was also described by Buser et al.[44]

Ultrastructurally, the mucosa around implants has great similarities to the mucosa around teeth.[46] In a beagle dog study of crystal sapphire implants that were placed in a nonsubmerged or one-stage procedure, the marginal soft tissues exhibited a collagenous stroma covered with an oral keratinized epithelium. The outer epithelial cells were joined by desmosomal contact. A junctional epithelium arranged as a collar was comprised of flattened, nonkeratinized squamous cells. Apical epithelial migration was not observed. At the electron microscopic (EM) level, mitochondria, rough endoplasmic reticula, Golgi complexes, and tonofilaments were observed. Hemidesmosomes were found at the basal lamina, which was smooth at the inner implant epithelium. An undulating basal lamina was found at the outermost epithelium, with connective tissue papillae interdigitated with the epithelial ridges. Collagen fibers ran in distinctively different directions and were embedded in ground substance. Biopsies from healthy implants contained few inflammatory cells.

The distribution of interstitial collagenous and noncollagenous glycoproteins of keratinized tissue was also studied.[47] Biopsies from healthy tissues around successful IMZ implants in five partially edentulous patients were compared with similar biopsies from healthy human teeth. Immunofluorescence techniques were used to identify types I, III, and IV collagen, laminin, and fibronectin. A stratified squamous keratinized oral epithelium, continuous with a nonkeratinized sulcular epithelium, was observed. The sulcular epithelium overlapped the outer coronal surface of the junctional epithelium and was separated from the underlying connective tissue by a basement membrane rich in type IV collagen and laminin. Dense connective tissue was observed under the oral epithelium. No significant difference was found in the distribution of collagenous components compared with the gingiva from teeth; however, the fibers in the connective tissue ran parallel to the long axis of the implant. Fibronectin, the major structural noncollagenous gingival glycoprotein, had the same distribution in tissues from both implants and teeth. Approximately half the biopsies contained inflammatory cells and a decreased collagen content below the junctional epithelium. In these areas, increased staining of type III collagen and fibronectin was seen. The authors[47] concluded that the distribution of interstitial collagenous and noncollagenous glycoproteins of keratinized tissue around successful implants was similar to that of normal gingiva around teeth.

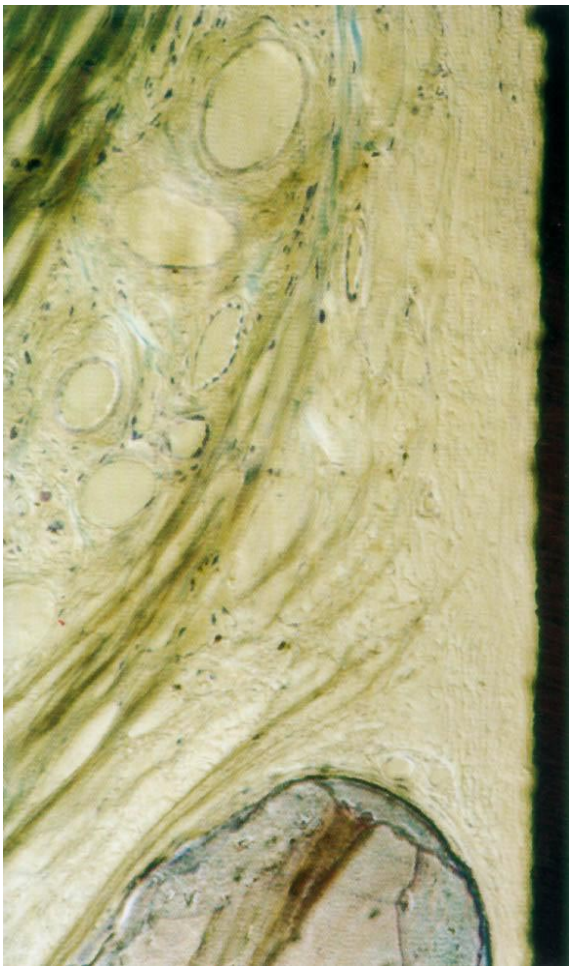

Fig. 2. Supracrestal area of peri-implant mucosa demonstrating fibers of gingival connective tissue and periosteum in zone distant to implant. Blood vessels are present only in this zone and not directly adjacent to implant. (Original magnification ×25.)

Monoclonal antibodies to specific cytokeratins and intercellular cell adhesion molecule 1 (ICAM-1) have been used to compare staining in gingival biopsies around teeth to gingival biopsies around implants.[48] These staining patterns allowed an evaluation of the differentiation

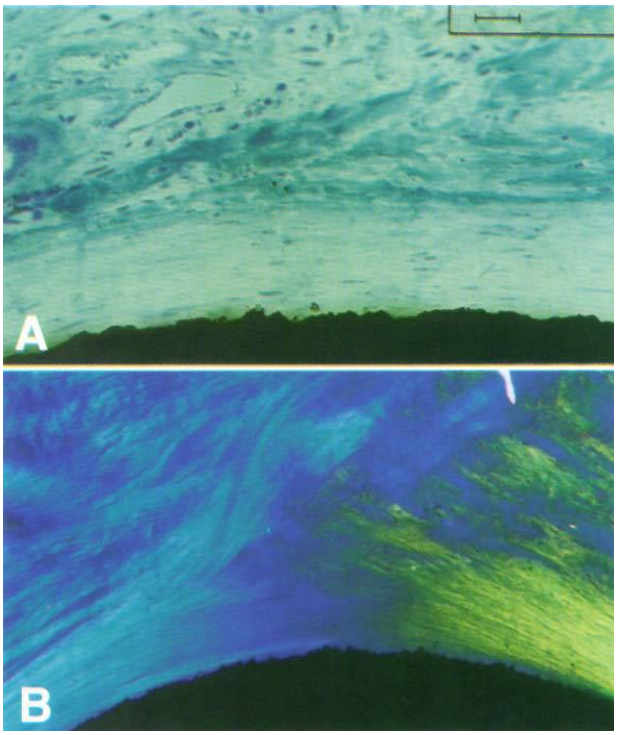

Fig. 3. A. Transverse section of implant demonstrates two different zones in connective tissue. Dense circular fiber arrangement without blood vessels is found in contact with implant post. Further distant to implant, thicker and somewhat looser arranged collagen fibers are found surrounding numerous blood vessels. **B**. Another view of dense circular fibers directly adjacent to implant post in cross-sectional view. (Toluidine blue stain and toluidine blue stain viewed with polarized light, original magnification ×50 and ×20.)

pattern of the epithelium. The results indicated that the formation of oral, oral sulcular, and junctional epithelium were phenotypically similar between teeth and implant gingiva. The implications of this work are interesting in the following respects: (1) Similar to the formation of a junctional epithelium after various periodontal surgical procedures, the presence of junctional epithelium around implants indicates that its formation is not dependent on odontogenic epithelium. This is in contrast to the junctional epithelium formation around erupting teeth where reduced enamel epithelial cells likely play a role. (2) Junctional epithelium and oral sulcular epithelium are not necessarily "dental" structures, as they are found around implants, implying that neither tooth structure nor periodontal ligaments are required for their formation; and (3) the formation of epithelial structures around implants involves normal patterns of epithelial macromolecular synthesis, indicating a basic host response to oral transmucosal structures. This report confirmed earlier immunohistochemical studies in which staining of keratins and desmoplakins in a total of six biopsies was used to show that gingiva and implant mucosa had similar staining patterns.[49]

A more recent study that uses immunohistochemistry techniques demonstrated structural differences in healthy human periodontal and keratinized peri-implant tissues.[50] Collagen types I, III, IV, and VII and fibronectin had similar distribution patterns between teeth and implants. Collagen types V and VI showed differing distributions between teeth and implants. The authors[49] suggested that the greater amounts of type V collagen around implants was significant due to its greater collagenase resistance.

Peri-implant connective tissue

A direct contact of connective tissue with the implant surface in the supracrestal area of nonsubmerged implants in monkeys was described by Schroeder et al.[37] This study showed that the implant surface characteristics influenced the orientation of the collagen fibers. In addition, the orientation of the fibers may have been affected by the relative mobility of the tissues surrounding the implant. The attached mucosa showed fibers arranged in both parallel and perpendicular directions, whereas the implants surrounded by alveolar mucosa had only parallel fibers. Implants with rough surfaces used in this study demonstrated connective tissue attachments with better developed, perpendicularly oriented dense fibers compared with the smoother surfaces. Nevertheless, dental implants currently in use exclusively have a smooth, machined surface in their suprabony, transmucosal portion. This is due to increased plaque retention being shown on rougher implant or abutment surfaces.[51-54] This may represent a stronger negative factor on the long-term stability and health of the peri-implant soft tissue collar than the possible positive effect on the type of connective tissue attachment if a rough surface is present in the area of the mucosa border.[51-54]

Buser et al.[44] evaluated the soft tissue healing around implants with different surface characteristics in their transmucosal portion, ranging from a smooth machined to a rough sandblasted surface. Healed tissues around one-piece nonsubmerged implants in beagle dogs were analyzed by light microscopy 3 months after placement (Figs. 2 and 3, *A* and *B*). Besides the previously described similarity between peri-implant and periodontal epithelial structures, a direct connective tissue contact was observed that was 50 to 100 μm wide and contained dense circular fibers without blood vessels (Figs. 2 and 3, *A* and *B*). Outside this area was a less dense connective tissue with horizontal and vertical collagen fibers and numerous blood vessels. No differences were found in the connective tissues between the rough sandblasted, fine sandblasted, and machined surfaces. There was, however, more coronal bone for the rougher surfaces than for the smoother surfaces. The authors[44] concluded that the different surface textures did not affect soft tissue healing, but did influence the location of the most coronal bone-to-implant contact. The finding of a par-

allel fiber orientation to a machined titanium surface was confirmed by Berglundh et al.[55] Similarly, Ruggeri et al.[56,57] demonstrated the presence of a "circular ligament" of densely packed collagen fibers free of inflammatory cells running parallel around nonsubmerged titanium screws in maxillary sites of monkeys. Although implant surface characteristics were not specified, the study could demonstrate that the smooth neck of the implant was surrounded by a narrow sulcus with junctional-like epithelium and few inflammatory cells.[56] The collagen fibers originated from the bone crest, adjacent teeth, and epithelial papillae and converged on the implant to form the circular fibers around the implant. Histochemical analysis revealed the presence of highly sulfated proteoglycans around the connective tissue fibrils.

CLINICAL CONSIDERATIONS FOR PERI-IMPLANT SOFT TISSUES

Mucosa or gingiva for long-term implant success?

Although it is considered preferable to locate implants within the masticatory mucosa, reports indicate that the placement of implants within the lining (alveolar) mucosa does not compromise long-term success (Fig. 4, *A* and *B*). The lining mucosa differs from the gingiva in several aspects. In addition to its lack of a keratinizing oral epithelium, the lining mucosa is less rigid, in part because of its lower content of collagen fibers and the presence of elastic fibers. Therefore the implant is surrounded by a moveable mucosa that, as some clinicians believe, favors the disruption of the implant-epithelial junction and the development of inflammatory changes.[37] This opens to discussion whether the presence of keratinized mucosa is required to maintain long-term tissue health.[6] The issue is important because it has considerable impact on treatment, especially for the resorbed edentulous mandible with often no or minimal remaining masticatory mucosa. Several clinical and experimental studies have addressed this question. In a study by Mericske et al.,[69] one-stage ITI implants placed in 33 elderly patients were followed for 5 years.[8] Each patient received two implants in the mandible to support overdentures. Approximately half the implants were located in nonkeratinized alveolar mucosa. The tissues around all implants were maintained in a healthy condition over the 5-year period with minimal or no attachment loss and with an average probing depth of approximately 3 mm. There was a tendency (which was statistically significant for certain areas) for the width of the keratinized mucosa to increase over time. Interestingly, Mericske et al. divided their patients between those who had been edentulous for a shorter period (implants placed within 2 years after the last tooth was lost) versus those who had been edentulous for longer periods (at least 5 years since last tooth loss). Patients who had been edentulous for longer periods had a significantly smaller zone of keratinized mucosa. This work in a population of elderly patients wearing implant-supported overdentures gave affirmation to the results of other studies in edentulous patients who had implants placed in nonkeratinized mucosa.[11,12]

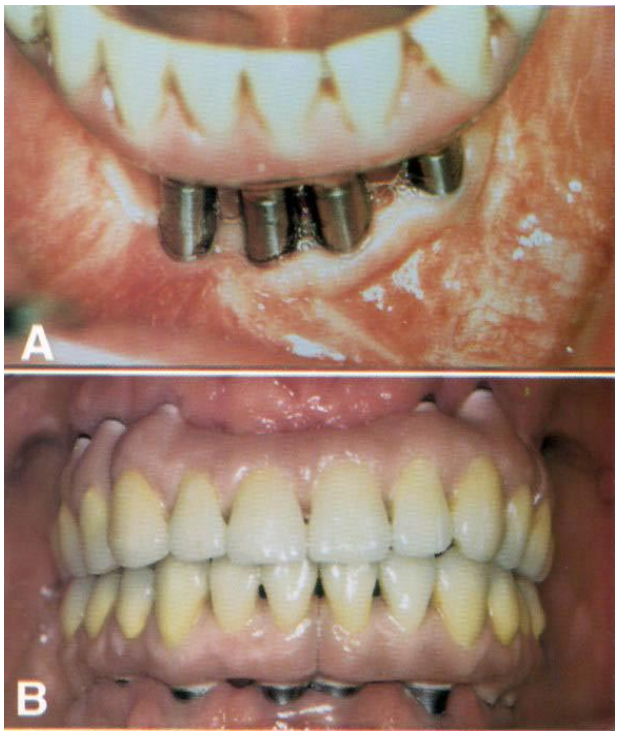

Fig. 4. A. Healthy peri-implant mucosa of different characteristics around four two-stage implants with transmucosal abutments supporting fixed detachable cantilever prosthesis in mandible for more than 5 years. Absence of keratinized attached mucosa around last abutment on right side of patient's mandible. **B**. Healthy peri-implant mucosa of different characteristics around maxillary and mandibular one-stage implants supporting fused-to-metal fixed full-arch prostheses for > 3 years. Different width of keratinized mucosa around various implants.

Wennstrom et al.[58] compared implants placed in varying amounts of masticatory mucosa. One hundred seventy-one Brånemark-type implants were examined; 24% of the implants were lacking a masticatory mucosa, 13% were in keratinized mucosa of < 2 mm width and mobility of the tissue (nonattached mucosa), and 61% of the implants were surrounded by mobile soft tissues. Multiple regression analysis indicated that neither the width of the masticatory mucosa nor the mobility of the mucosa had a significant effect on the plaque control level or the health of the soft tissues as determined by bleeding on probing. The authors[58] concluded that the lack of attached masticatory mucosa around an implant did not jeopardize the maintenance of soft tissue health.

Results from these clinical studies seem to offer sufficient evidence that the long-term prognosis of implants is not jeopardized by the absence of keratinized attached mucosa, provided that an adequate level of oral hygiene is maintained. In addition, experimental studies in canines and primates have been undertaken to assess the importance of a keratinized-type tissue to maintain long-term health of the implant bed.[59,60] However, extrapolation of these findings to clinical use is questionable because of the sample size and other study design features.

Altered tissue responses for implants and abutments in intraoral skin grafts have been reported. For example, persistent proliferation of epithelial tissue was discovered in two of five patients who received split-thickness skin grafts for mandibular vestibuloplasty.[61] Traditional surgical and oral hygiene techniques did not prevent further proliferation of the tissues. After removal of titanium abutments and replacement with custom gold abutments, the epithelial proliferation diminished. These findings suggested that these patients may have had an allergy to titanium.

Submerged or nonsubmerged implant placement?

On the basis of his early research in dogs, Brånemark[33,62] recommended the so-called submerged approach, namely, placing implants under the protective cover of the oral mucosa for a healing period of 3 to 6 months. After that time, a second surgical procedure is performed, during which a transmucosal component is placed on the implants.

Schroeder et al.[37] demonstrated in the mid to late 1970s that a nonsubmerged or one-stage surgical technique with a transmucosal implant placement at the first surgery allows equally successful tissue integration. The one-stage surgical concept has demonstrated sufficient experimental and clinical evidence to make it an accepted and attractive alternative to the two-stage approach.[8,14,16,63] Earlier fears that nonsubmerged implant placement would compromise successful healing due to soft tissue ingrowth in the coronal aspects of the bony implant bed have been dispelled. Nonsubmerged healing will lead to a soft tissue structure described in detail previously, provided a minimal level of oral hygiene is maintained.[64] The fact that only one surgery is necessary allows for soft tissue healing to the transmucosal portion of the inserted implant by primary intention from the time of implant placement. The healed peri-implant mucosa is not further disturbed with a second stage surgery for abutment placement or with later abutment exchanges. Various interesting questions regarding the biologic, for example morphologic and functional, significance of the placement technique and implant and component designs are currently being addressed in ongoing research.[39,64] Specifically, the gap location of the connection between implant and abutment in relation to the crestal bone and its influence on the hard and soft tissue structure, for example, the establishment of the so-called biologic width, are being studied intensively.

Microbiologic aspects, peri-implant soft tissue disease

Investigators have used periodontal criteria to describe the health and disease of soft tissues around implants. Only a few studies have attempted to assess cause and progression of peri-implant soft-tissue disease.[1,2,33,65]

Considering the oral microflora as a possible risk factor for peri-implant tissue disease, evidence suggests that no significant differences exist in the distribution of bacterial morphotypes around implants and teeth.[66] In contrast, significant differences were found in plaque composition between specimens taken from completely edentulous patients and plaque from teeth or implants in partially edentulous patients. This suggests that teeth may serve as a reservoir for the bacterial colonization of titanium implants placed in the same oral cavity. These findings confirmed an earlier study that examined crevicular fluid and plaque composition obtained from implants in human subjects. Similarities between teeth and implants in partially edentulous patients were observed; however, differences occurred in the plaque from implants in completely edentulous patients.[67]

Microbial differences between successful and failing implants and between the microbial flora in partially edentulous and fully edentulous patients were explored in two studies.[68,69] Facultative anaerobic cocci predominated, whereas low bacterial counts were measured from healthy implant specimens. Spirochetes and gram-negative anaerobic rods were present in high numbers in specimens from failing implants. Completely edentulous implant patients had fewer periodontium-associated pathogens than did implant specimens from partially edentulous patients. According to the authors,[68,69] this confirmed "that spirochetes are not commonly associated with successful implants."

When accepting a microbial cause for peri-implant tissue disease, it appears that the completely edentulous patient is considerably better off than the partially dentate in terms of absence of periodontal pathogens.

Histologic and immunohistologic analysis was performed in a study involving 18 gingival biopsies from clinically healthy implants and 9 similar specimens from implants with overt clinical signs of inflammation.[70] Histologically, all the specimens had some amount of inflammation, but the clinically inflamed sites had a much greater inflammatory infiltrate. Immunohistologic analysis included the proportion of T (50% to 60%) and B (40% to 50%) lymphocytes, the T-helper to T-suppressor ratio or CD4:CD8 ratio (1.6:1 and 2 for healthy and inflamed sites, respectively), the number of Langerhans cells (no significant differences between groups), and the human leucocyte antigen (HLA) class II positive cells

(healthy and inflamed specimens were significantly different). Biopsies were also taken from tissues between the implants. Again, no differences were found in the infiltrates from clinically healthy implants compared with inflamed implants. The authors[70] concluded from this immunohistologic analysis that the gingival lesion around clinically healthy and inflamed implants was stable and well-controlled. The inflammatory lesion consisted of a lymphocyte and macrophage infiltrate with only a few plasma cells. It was speculated that as the lesions became clinically significant in size, the proportion of T and B cells and T cell subsets remained relatively constant. Although T cells dominated the inflamed lesions, the substantial number of B cells with a lack of plasma cells suggested that activation of the B cell population was controlled. This was in addition to the finding of consistent CD4:CD8 ratios of between 1.5 and 2.0:1, a ratio found in delayed-type hypersensitivity reactions in the skin, peripheral blood and regional lymphatic tissue, putative stable periodontal lesions, gingivitis in children, and experimental gingivitis lesions. On the basis of the results of this study, the authors[70] suggested that gingivitis associated with osseointegrated implants is well-controlled immunologically and represents a stable condition.

The influence of plaque accumulation on the peri-implant tissue condition was also studied in animal experiments.[65,71] One study in beagle dogs compared the soft tissue reaction with plaque formation around implants with abutments versus teeth. Teeth and implants were cleaned for 4 months and a biopsy was taken. After 3 additional weeks with no oral hygiene procedures performed, specimens were again taken, including block biopsies. Specimens of healthy tissues around implants with abutments and around teeth revealed common features, including a keratinized oral epithelium, an approximately 2 mm long junctional epithelium, and an underlying connective tissue devoid of inflammatory cells. The differences between implants/abutments and teeth were predominantly in the direction and density of the collagen bundles. Specimens from implant/abutments and teeth that had no oral hygiene procedures performed for 3 weeks both revealed subgingival plaque (similar amounts) and an inflammatory infiltrate. Both lesions were characterized by decreased collagen content and a dense accumulation of inflammatory cells. The density of fibroblasts was the main difference between the inflamed dentogingival and implantogingival tissues, with the density around the implants the same as that found around healthy implant tissues, whereas the density of the dentogingival tissues in inflamed conditions was significantly decreased. Despite these differences, the authors[65,71] concluded that both implantogingival and dentogingival tissues had a similar reaction to plaque formation, both qualitatively and quantitatively, suggesting that the barrier function of both types of tissues was the same. These findings were confirmed to a certain extent in a later study in monkeys in which peri-implant infections were induced by ligature placement.[71]

Soft tissue complications around dental implants have been reported in several of the follow-up studies on fixed or removable prostheses in edentulous patients. These complications mostly represent adverse effects that are, in general, easy to treat and do not affect the long-term success of an implant. Nevertheless, they are a nuisance in daily practice, depending on how frequently they occur in a given patient and how involved the treatment of the respective problem is.

Proliferative gingivitis (gingival hyperplasia) and fistulae have been reported as the two main types of peri-implant soft tissue complications.[4,13,15] Proliferative gingivitis appears mostly associated with unfavorable conditions for local oral hygiene[4] and is significantly more frequent under overdentures.[72] Treatment approaches include longer abutments, gingivectomy, or flap procedures after establishing adequate plaque control.

Fistular tract lesions have been found mostly at the level of the implant/abutment connection.[4] The cause of these lesions may be in a loose or fractured abutment screw or plaque formation that occurs along the central abutment screw, again correlated to areas with unfavorable conditions for oral hygiene.[73] Several studies have now documented the microbial contamination of the microgap between implant and abutment in two-stage implant designs.[74,75] Recommended treatment steps include cleaning and sterilization of the abutment and abutment screw, application of sealing agents between the abutments and prosthesis, improvement of oral hygiene, and, if necessary, surgical soft tissue corrections. Obviously, loose components and subgingival gaps between components have an effect on the host response and the soft tissue health around the implant. This problem has not been reported for transmucosally placed one-stage implants.

Clinical monitoring of the peri-implant tissue condition

The same clinical techniques and parameters, including radiographs, are used to monitor peri-implant tissue health or disease. Monitoring as a preventive measure is certainly an important part of successful medicine and dentistry. However, the correlations between plaque accumulation, marginal tissue inflammation, probing depth, and bone loss are not well-understood for dental implants. The only significant correlation has been found between probing level and marginal bone level. A greater probing level is, however, not necessarily correlated to disease. Hence, the value of using periodontal parameters to determine peri-implant health is not clearly evident.[73,76-81] A prospective study that involved 174 fixed partial dentures on 460 Brånemark implants placed in partially edentulous patients at several centers evaluated

periodontal indices on teeth and implants over time.[76] After 3 years, the cumulative implant success rate was 93.9%, and failures appeared to be concentrated in patients who had a high plaque index. Only slight bone loss was found in the second and third years of the study, and the plaque and gingivitis indices were similar between implants and abutments and teeth. Probing depths (accessible in only one third of the sites) were found to decrease over time, which was attributed to shrinkage of the peri-implant soft tissues. According to the authors,[76] this study confirmed earlier data in fully edentulous patients.

In a prospective long-term follow-up of fully edentulous patients, Chaytor et al.[79] found no statistically significant correlations between marginal alveolar bone level change and plaque index and amount of keratinized mucosa. The authors[79] concluded that the weak correlations between bone level changes and the various periodontal indices suggest that these traditional measures of periodontal health should not be relied on to infer the state of bone-supporting implants.

The value of periodontal probing around endosseous dental implants remains an area of controversy in the literature. Whereas a study mentioned previously found a decrease of probing depth over time,[76] another report of 98 implants in 24 patients showed that significantly greater probing depths were found with increasing time ($r = 0.5$) after implant placement.[80] In an early review article comparing teeth with implants,[81] the consulted literature demonstrated that probing depths were greater around implants in the maxilla than in the mandible, and that deeper probing depths were significantly correlated with gingivitis. Interestingly, the deeper pockets associated with gingivitis were not associated with histologic changes of continuing destruction. This article pointed out that the probe tip was generally located millimeters coronal to the radiographic bone level.

Experimental data in animals exhibited somewhat different and more uniform results than results drawn from human data. A study in five beagle dogs evaluated probing (0.5 N force) of soft tissues around teeth and submerged implants with abutments connected after 3 months.[82] After 2 weeks of healing, radiographs and clinical measures were made for 1 year. The teeth were cleaned three times per week except for one tooth that had a ligature tied around it for 4 months. Resistance to probing was greater around teeth than implants, so probing depths were significantly deeper around the implants than around the teeth. Histologically, the probe tip (0.5 mm in diameter) was coronal to the apical extension of the junctional epithelium around teeth, whereas around implants the probe tip was always apical to the junctional epithelium and close to the alveolar bone crest. Quirynen et al.[77] pointed out that the data from this animal experiment conflicted with clinical data, which indicated that probing attachments and bone levels around the examined implants were comparable to those around teeth when moderately healthy soft tissues were present.

In another dog study, histologic assessment of probing around 30 one-stage nonsubmerged implants was evaluated.[83] After implant placement and healing with frequent plaque removal, the dogs were divided into three groups, including a group with healthy gingiva, a group in which plaque was allowed to accumulate naturally, and a third group in which ligatures were placed around the implants and plaque was allowed to accumulate. Probes were placed after 4 to 6 months with a standardized force (0.2 N) and fixed to the mesial and distal of each implant. Probe depth was located at the coronal aspect of the connective tissue in healthy tissues, but increased with the degree of inflammation. Probe penetration exceeded the connective tissue level in the ligature-induced group. Ericsson and Lindhe[82] concluded that probing around nonsubmerged implants was "a good technique for assessing the status of peri-implant mucosal health or disease." If this conclusion holds true for the human clinical situation, it could be useful only if a standardized force is applied and if a baseline value has been established at a given site around an implant to which future measurements are compared.

In summarizing the current interpretation on the use of periodontal parameters to monitor peri-implant tissue health, the consensus report of the 1996 Workshop in Periodontics concludes that there is no clear evidence for their value.[84]

SUMMARY

The supracrestal soft tissues around endosseous dental implants exhibit structures and features of noninflamed soft tissues analogous to noninflamed gingiva around teeth. These include the following:

1. Structures: Oral stratified squamous epithelium; sulcular nonkeratinized epithelium; nonkeratinized junctional epithelium; soft connective tissue contact; and vascular components.

2. Features: Basement membrane; rete pegs; connective tissue papillae; collagenous stroma; collagen and noncollagen glycoproteins; desmosomes and hemidesmosomes; structural and nonstructural proteins; immune cells.

Titanium, or more properly titanium oxide, does not appear to significantly affect epithelial cell structures or the formation of epithelial structures around transmucosal materials. This suggests that the location of the epithelium (in this case, oral gingival epithelium) is more influential in determining the structure of the epithelial components than is the substrate (implant versus tooth).

Evidence also exists that around titanium abutments, or nonsubmerged one-stage implants, the major connective tissue fibers run parallel to the long axis of the

implant. The connective tissue forms a nonvascularized, circular, scar-type structure surrounded by a less dense, vascularized connective tissue. Thus the epithelial components around implants appear to be consistent with epithelial components around teeth, whereas the connective tissue, although having a similar composition, has a dramatically different spatial orientation.

The factors that influence maintenance of dental implants are similar to those of the natural dentition. However, the value of periodontal parameters for monitoring peri-implant tissue health is not clearly evident, and differences in probing levels around implants are not strongly correlated to peri-implant tissue disease.

Peri-implant tissue inflammation and marginal bone loss are associated with the presence of periodontal pathogens in both partially and fully edentulous patients. Whereas, a high correlation between composition of the microbiota in the pockets around teeth and implants exists, periodontal pathogens are generally found in much lower levels in the fully edentulous case. Increased abutment roughness may lead to increased plaque accumulation and colonization with a pathogenic flora in the supracrestal tissues. The design of any prosthetic superstructure must offer sufficient access for plaque removal by the patient.

Clinical studies demonstrate evidence that keratinized, attached mucosa is not a prerequisite for long-term implant maintenance. However, in certain situations, patients may benefit from its presence. The physiologic and functional role of peri-implant soft tissues is not well understood. There is also no current documentation thus far about the role of host resistance in long-term implant maintenance.

FUTURE RESEARCH

Areas of future research on soft tissues around implants will need to address the physiologic and functional characteristics of these tissues.

Questions include:

1. Do the implantogingival tissues resist bacterial challenges in a manner similar to dentogingival tissues?

2. Is this true for both submerged and nonsubmerged implants?

3. Does the surface of the abutment or implant change the structure or function of the tissues and/or the relationships of the soft and hard tissue components?

4. Is the soft tissue contact around abutments, both structurally and functionally, the same as that around implants? How is it influenced by implant and abutment design, type, and location of implant and abutment connection?

5. What are the ideal surface characteristics and composition for optimal soft tissue contact and function?

6. Can the surface characteristics act to guide tissue integration around implants?

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Noteworthy Abstracts of the Current Literature

Repairs in complete denture: Results of a survey
Zissis AJ, Polyzois GL, Yannikakis SA. *Quintessence Dent Technol 1997;20:149-55.*

Purpose. This article presented the results of a survey of dental laboratories with regard to incidence of fracture and the type of repair needed and related the fracture type to the characteristics of complete dentures, including denture type, diastemas, deep frenal notches, previous reinforcement, and relining.

Material and Methods. A questionnaire submitted to three large dental labs in Athens, Greece, was used to collect data on the causes of denture fracture and the repairs needed of complete dentures submitted to these laboratories over a 6-month period. One certified dental laboratory technician examined the dentures and filled out the questionnaire. A total of 489 questionnaires were collected. There were 303 maxillary complete dentures and 186 mandibular dentures needing repair during the time of the survey. Acrylic resin was the denture base material of a majority of dentures.

Results. Deep frenal notches and diastemas between the central incisors, either singularly or in combination, accounted for more than 50% of the dentures needing repair. Of the dentures examined, nearly one third had been previously repaired. One fifth has been reinforced with a metal wire or metal mesh, and 5.3% had been previously relined. A midline fracture was the most common finding, tooth debonding was second, and other types of fractures, such as tooth fractures, were seen infrequently.

Conclusions. According to this survey, the majority of complete dentures requiring repair were maxillary, and the material most often used for their fabrication was acrylic resin. Midline fracture, associated with deep frenal notches and diastemas, accounted for more than 50% of the repairs. One third of the dentures had been previously repaired, and one fifth had been reinforced with wire or metal. Tooth debonding was a significant problem, with its occurrence higher in the maxillary than mandibular complete dentures. The material of choice for repair was acrylic resin and metal reinforcement rather than light-cured or microwave processing of the repair.
17 References.—*RP RENNER*