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REVIEW ARTICLE

WOUND HEALING IN PERIODONTAL REGENERATION

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ABSTRACT:
Healing following periodontal reconstructive therapy may result in formation of a long junctional epithelium at the tooth-mucogingival flap interface. In the context of periodontal regeneration, this should be considered an expression of wound failure, where maturation of a root surface-adhering fibrin clot has been disturbed by disruptive forces and/or properties of the root surface1. Periodontal regeneration is defined as the reproduction or reconstitution of a lost or injured part so that form and function of lost structures are restored2. Inconsequence, periodontal regeneration includes regeneration of alveolar bone, cementum, periodontal ligament and gingiva; however, the tissue structure may not be anatomically or functionally perfect and may include some evidence of scarring.
Keywords: Healing, regeneration.

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INTRODUCTION:

Healing following periodontal reconstructive therapy may result in formation of a long junctional epithelium at the tooth-mucogingival flap interface. In the context of periodontal regeneration, this should be considered an expression of wound failure, where maturation of a root surface-adhering fibrin clot has been disturbed by disruptive forces and/or properties of the root surface[1]. Periodontal regeneration is defined as the reproduction or reconstitution of a lost or injured part so that form and function of lost structures are restored[2]. Inconsequence, periodontal regeneration includes regeneration of alveolar bone, cementum, periodontal ligament and gingiva; however, the tissue structure may not be anatomically or functionally perfect and may include some evidence of scarring.

Clinical trials aimed at regeneration of tooth supporting structures have been based on varying biological rationales. Reconstructive modalities that appear to have merit and have demonstrated significant gain of clinical attachment and at least partial resolution of an associated bony defect include, separately or in combination: surgical debridement with adjunctive root surface or wound conditioning, implantation of bone, bone derivatives and substitutes, and placement of barrier membranes for guided tissue regeneration.[3-,5] The true histological nature of the clinical improvement, however, often remains obscure. Clinically, it may not be clear whether observed improvement has resulted from a functional collagenous scar or formation of a long junctional epithelium or whether periodontal regeneration actually has occurred. In addition, improved gingival tissue tonus or presence of an implant material invested in dense connective tissue may contribute to clinical improvement. Therefore, clinical healing may to a high degree reflect factors related to the particular procedure; while, on the other hand, periodontal regeneration more significantly is a consequence of biological factors that are active regardless of protocol.

Animal models offer the opportunity for histological observations, which may contribute to an improved understanding of the particular biology of healing following different reconstructive procedures. Such observations have demonstrated that formation of a connective tissue attachment rather than a long junctional epithelium is dependent on the unimpeded conclusion of a series of interactions between the root surface, plasma and tissue factors, and the connective tissue of the mucogingival flap and periodontal ligament. Eventually, wound maturation will result in a functional fibrous attachment, including formation of alveolar bone and cementum.

Dynamics of early healing events

The healing of an incisional or excisional wound has been studied extensively in non oral sites; however, the basic biological events apply to the tooth-mucogingival flap interface as well. Wound healing is commonly divided into three sequential phases: inflammation (early and late), granulation tissue formation, and matrix formation and remodeling.[6] Although a temporal representation leaves an impression of a highly organized and predictable system, considerable overlap exists and time needed for completion of each phase may vary considerably depending on wound morphology, condition of adjacent tissues, and other local and systematic factors.

When a mucogingival flap has been raised in a healthy periodontium and repositioned against the underlying periosteum and the severed supracrestalfibres, healing follows the same scheme because the wound is, in essence, a soft tissue wound. It has been noted, however, that supracrestal root-attached connective tissue is considerably less reactive and contributes less to the early stage of wound repair than does the soft tissue of the flap. This may in part, be explained by paucity of anastomoses connecting the periodontal ligament and the gingival microvasculature.[7]

These observations re-emphasize the importance of preserving root-attached connective tissue fibres when an intrasulcular incision is made.[8-10]

Conceptually, a more complex situation exists when a mucogingival flap is repositioned against a root that has been denuded of all organic material by mechanical instrumentation.

The undisturbed healing between an instrumented root surface and a mucogingival flap may be appreciated as follows. At wound closure, clotting blood fills the space between the transgingivally positioned tooth and the flap. Within seconds, plasma proteins, primarily fibrinogen, precipitate onto the wound surfaces and provide an initial basis for adherence of a fibrin clot.[11]Within one hour, the early inflammatory phase of healing is initiated by neutrophils infiltrating the clot from the mucogingival flap. Within six hours, the root surface becomes lined by neutrophils, which decontaminate the wound by phagocytosing injured and necrotic tissue. Within three days, the inflammatory reaction moves into its late phase as the neutrophil infiltrate gradually decreases while the influx of macrophages increases. The macrophage contributes to wound debridement by removing effete red blood cells, neutrophils, and residual tissue debris and, in addition, has a conspicuous role including release of growth factors which support fibroblast proliferation and matrix production, smooth muscle cell proliferation, and endothelial cell proliferation and angiogenesis. The macrophage, therefore, plays a key role in the transition from inflammation to granulation tissue formation.[12,13]Within seven days, the phase of granulation tissue formation gradually enters into the third phase of wound healing in which the newly formed cell-rich tissue undergoes maturation and remodeling to meet functional demands.[14]The histological observations suggest that healing at the tooth-mucogingival flap interface does not progress much differently form healing in incisional or excisional skin wounds, despite the fact that the tooth represents a transgingivally positioned, avascular and rigid wound margin. The observation also suggests that the first requirement for a new connective tissue attachment or periodontal regeneration to occur rest with adsorption of plasma proteins to the root surface.

Observation of wound healing following periodontal reconstructive surgery have nurtured the hypothesis that the gingival epithelium needs to be restrained access to the root surface for new connective attachment or periodontal regeneration to occur[15,16]. Early experimental observations by Linghorne and O'Connell, however, suggest that formation of a long junctional epithelium may only occur under certain circumstances.[17] In a periodontal dehiscence defect model, they observed that maxillary defects healed with a new connective tissue attachment following reconstructive surgery, whereas the mandibular defects formed a long junctional epithelium. Two decades later Hiatt et al reported temporal observations of wound healing in similar dehiscence defects.[14] Yhry observed those two to three days postsurgery "blood elements and debris prevented a reattachment of the epithelium to the tooth". From the seven-day observation interval they reported that "the epithelial attachment appeared normal and no evidence of down growth or increase of epithelial cell proliferation could be seen" apparently, the unimpeded adsorption, adhesion, and maturation of the fibrin clot at the tooth-mucogingival flap interface is critical for formation of new connective tissue attachment.

The frequently considered parameter of wound healing is the mechanical strength of the wound. Comparisons between temporal maturation of the

tooth mucogingival flap interface and incisional skin wounds reveal only minimal differences. Both periodontal and epidermal wound tensile strength increase significantly from approximately 200g within days or would closure to exceed 1700g within 15 days post surgery, the latter being a tensile strength that may resist most would rupturing challenges[14] these data suggest that wound integrity during the early healing phase rests primarily with that offered by suturing and that the tooth-mucogingival flap interfaces is vulnerable to disruption by mechanical forces for a considerable period of time post surgery. This knowledge also underscores the importance of securing and positioning the flap sheltered from tensile forces moreover, traumatic effect from post surgery procedures such as early suture removal, application of a surgical dressing and mechanical hygiene routines need to be recognized and considered.

Wound Maturation and Remodeling

While early would healing at the tooth mucogingival flap interface occurs by biological processes analogous to healing of a soft tissue wound, further maturation and functional adaptation require a mechanism by which collagen fibers become attached to the instrumented cementum or root dentin. This process may exhibit a number of variations, all of which may be present in a single wound.

First the healing connective tissue may recognize the instrumented root as an inert foreign body. Similar to an encapsulation process, collagen fibers form bundles parallel to the root surface usually in a vertical orientation. This type of healing has often been referred to as **"collagen adhesion"**[18] More recent ultrastructual data suggest, however, that although fibers are not visibly embedded in new cementum, close approximation of young collagen fibrils in the soft tissue to the collagen of the tooth matrix results in a physicochemically valid attachment resistant to mechanical disruptive forces.[19]Second the presence of a denuded root may stimulate the differentiation of cementoblasts, which will deposit a hard tissue into which new collagen fibers may be anchored. This seems to be slow process. Many studies have shown that cementum does not appear until after the third week following periodontal wounding in humans, and in canine and nonhuman primate models[19,20], although isolated deposits of new cementum have been reported at two weeks of healing. The interface between new cementum and underlying hard tissue may consists of a granular, fiber-free layer, possibly consisting of fibronectin.[21-23]

Third, resorptive activity may be initiated. This appears generally of a superficial and transient nature and is usually followed within a few weeks by deposition of cementum.[23]Osteoclastic or odontoclastic resorption implies, first, demineralization of the mineral component by acids produced by the clast cell and, second, degradation and removal of the organic matrix. The resorption process creates a surface in which collagen fibrils of the resorbed areas, thus completing the new attachment.[24-26] It should be noted. However, that aggressive, inflammatory resorption commonly originating in the cervical region has been reported as frequent, undesirable sequel in experimental regenerative studies.[26-30]

Finally, ankylosis may develop, primarily in the cervical region. The occurrence of ankylosis appears to be directly related to the vertical attachment achieved by the regenerative procedure.[31]Possibly the process of periodontal ligament regeneration is not as rapid as that of bone formation,[32] Hence progenitor attachment are "overrun" by bone-forming cells as the distance from the base of the wound increases.[30,31]This relationship may explain why, with few exceptions, ankylosis does not seen to have been a problem in human regenerative studies, where the amount of connective tissue attachment gain is `limited.

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