Current Concepts of the Dentogingival Junction: The Epithelial and Connective Tissue Attachments to the Tooth

Irving B. Stern

Pocket formation and recession are manifestations of an altered relationship of gingiva to tooth. Both occur in periodontal disease. To understand these disruptions, one must know the morphology and biology of the dentogingival junction. For periodontal disease to occur, the junction must be breached and its cytology altered. All theories of the etiology and pathogenesis of periodontal disease at some point describe the pathologic changes in the tissues of the gingiva. These tissues include the gingival epithelium, the junctional epithelium and the subjacent connective tissue.

Less than a century ago these structures were very poorly described. It was believed that the fibers of the ligament lay parallel to the tooth surface and that the gingiva contacted the tooth at the cemento-enamel junction. The 19th century was a period of explosive growth in the development of the biological sciences in general, and of histology in particular. Dental publications were concerned with anatomy (gross and comparative), anthropology, archaeology, and the growth and development of teeth. Clinical texts were equally scholarly and make fascinating reading, despite the relatively primitive state of the art and science of dentistry at that time. It was inevitable that the clinical and basic sciences would become increasingly integrated.

The Junctional Epithelium and the Epithelial Attachment

At that time the generally accepted concept of the gingival relationship to the tooth was based on clinical and histologic observation. The gingiva could be easily displaced during extraction, cavity preparation, restorative treatment, and scaling procedures. In addition, histologic examination required the use of decalcifying solutions to remove tooth mineral. Enamel is soluble in these solutions and leaves a space between the dentin and the gingiva in tissue sections. Based on these clinical and histologic observations, it was believed that the subgingival space extended to the cementoenamel junction under a loose fitting gingiva.

Histologic studies led Gottlieb to conclude in 1921 that there is no such space. Rather, the gingiva (via the enamel organ) forms an organic union with the enamel and is firmly bound to it (epithelansatz or epithelial attachment). He proposed that this epithelial attachment and its gingiva gradually stripped from the tooth, like the peel from an orange, to provide for eruption, pocket formation and recession. This concept met with increasingly general acceptance but some skepticism remained.

Waerhaug in 1952 reported clinical, histologic and experimental studies which led to the opposite conclusion. He described the gingiva as being separated from the tooth by a capillary space, forming an epithelial cuff. This cuff was believed to be weakly adherent to the tooth, and could be displaced from the tooth surface and then replaced against it without diminishing the strength of the adhesion. These observations divided dentists into two groups, those who believed in the epithelial attachment and those who believed in the adherent epithelial cuff.

In 1962 Stern first demonstrated that the ultrastructure of the epithelium of the ameloblast-enamel junction (the dentogingival junction) of rat incisor consists of a basal lamina and hemidesmosomes, and that the basal lamina has as components a lamina lucida and a lamina densa. (Fig. 1). This observation of a basal lamina and hemidesmosomes, plus those of other workers has led to a resolution of the controversy. Neither concept, Waerhaug’s weakly adherent cuff nor Gottlieb’s firm organic union of the last formed product of the reduced enamel organ bonding the gingiva to the tooth, is correct.

These concepts have been replaced by the concept of the junctional epithelium, a tissue capable of forming and renewing itself continuously throughout life. It forms at its base, migrates coronally and desquamates at its surface, all the while maintaining a biologic attachment to the tooth. Thus, the former relatively static concepts have been replaced by a dynamic concept.

The junctional epithelium forms in two ways. At first, it is derived from the ameloblasts and is referred to as
the primary junctional epithelium. In the course of ameloblast histodifferentiation the cells pass through two phases: forming enamel in the first, and primary junctional epithelium in the second. In the ameloblast life cycle, after the enamel matrix secreting and mineralizing stages of amelogenesis, the ameloblasts become smaller, terminate the enamel-forming function, and begin to form the epithelial attachment. When Gottlieb and his associates used the term "epithelial attachment," they referred to the epithelium in contact with the tooth, the cells as well as the junctional material. The epithelial cells are now referred to as the junctional epithelium, while epithelial attachment refers to the zone of attachment, its structure (i.e., basal lamina and hemidesmosomes) and its biochemical constituents.  

When the ameloblasts become depleted and are lost, their attachment role is taken over by the secondary junctional epithelium, a derivative of the gingival epithelium. The basal cells of oral epithelium are pluripotential for at appropriate times in their life cycle they may specialize to form odontogenic epithelium, glandular tissue, keratinocytes, and junctional epithelium. A differentiated cell cannot divide and does not ordinarily dedifferentiate. The secondary junctional epithelium is derived from undifferentiated basal cells. One must consider the ameloblasts to have been programmed for their attachment-forming role at some time during formation of the enamel organ.

The replacement of primary by secondary junctional epithelium occurs only in human or similar teeth—that is, in teeth having roots coated with radicular cementum and having closed apical foramina. These teeth have coronal enamel which is formed by an enamel organ situated in a coronal position. They are referred to as rooted teeth, or teeth of limited eruption. Continuously erupting or rootless teeth, on the other hand, maintain a
persistent dental organ at their base and have quite a different morphology. The rodent incisor, for example, has enamel coating on its labial surface only, and cementum on its lingual and lateral surfaces. There are two lateral cementotromatial junctions running from the base of the tooth, where enamel and cementum formation occur continuously, to the incisal surface (Fig. 2). After differentiation, the ameloblasts pass through enamel secretary and mineralizing functions, then enter a pigmentation stage, finally becoming the junctional epithelium over the enamel (Fig. 3). The ameloblasts form continuously at the base of the tooth. They move incisally with the ever-erupting tooth as if on a conveyor belt. When they ultimately desquamate from the tip of the junctional epithelium, they are replaced by the ameloblasts below them. The gingiva does not contribute to the ameloblast junctional epithelium. On the other hand, the odontogenic epithelium of the lingually situated portion of the dental organ breaks down and is lost during cementogenesis. After this, there is no epithelium in contact with the cementum until the tooth erupts into the oral cavity. Then the adjacent gingival epithelial basal cells divide and migrate over to the tooth and form the cemental junctional epithelium. There is no replacement of "primary" junctional epithelium by "secondary." For the rat incisor the word "secondary" is not quite appropriate. Both junctional epithelia are present when the tooth erupts, and they persist by means of continuous cell renewal through the life of the animal. The comparative anatomy and modes of formation of junctional epithelium in the rat incisor may be instructive when considering the source of the secondary junctional epithelium of rooted teeth.

The hemidesmosomes of the ameloblasts of the rat incisor become evident after mineralization (in the early maturation—late pigmentation stage). The ameloblasts of rat molars, which are rooted teeth, form hemidesmosomes at a similar stage prior to eruption. There is less information concerning timing of appearance of hemidesmosomes in human ameloblasts, but one would presume that they would appear at an equivalent stage.

The common ultrastructural morphology of the junctional epithelium and epithelial attachment in the rat incisor,44-47,54-58 rat molar,46,47,54-58 man,45,46,52-55 dog,53 monkey,48,49,55,56 cattle,57 and cat58 has led to acceptance of the concept of the junctional epithelium.

Various issues remain to be resolved, such as the biology and significance of the basal lamina in health and disease. Another is the site of pocket formation. Still another is the role of the fibroblasts subjacent to the junctional and sulcular epithelium.

The Basal Lamina

The width of the basal lamina is reported to be in the vicinity of 800 Å to 1200 Å.45,46,47,48,53,54 Its components, the lamina densa and lamina lucida, are not always evident,41,46,50-53 particularly in some decalcified preparations. At times in fully calcified specimens the lamina densa is not resolved, and may be calcified.45,48 Stern45,46,50-52 defined the "lamina lucida," ~400 Å, as the structure between the outer leaflet of the epithelial cell membrane and the lamina densa. Kobayashi et al.50 described a third lamina, the "sublamina lucida (120 ± 20 Å)," between the tooth and lamina densa. They defined the lamina lucida (140 ± 30 Å) as extending from the peripheral density of the hemidesmosome to the lamina densa (Fig. 4). The significance of the altered definition is that the contents of the lamina lucida may differ inside of and beyond the peripheral density. If one views the sub-lamina lucida or the lamina lucida as fluid-filled rather than as structural in nature, then for them to contribute to adhesion would require the presence of electrostatic repulsive and London-van der Waals attractive forces.54 These forces act over short distances: 100 to 200 Å would be the maximum.54 Within small dimensions, a "space" occupied by a molecular population of restrained mobility would be stabilized by temporary linkage of the molecules.54 Kobayashi et al.54 viewed the sub-lamina lucida as such a "space" created by an interplay of forces between the lamina densa and the tooth, thus having an important role in adhesion. Frank and Cimisoni54 and Stern45-52 implied that the lamina lucida plays the important role.

The lamina densa of various epithelial-connective tissue interfaces has a proteinaceous composition incorporating collagenous elements. It appears to function as a fibrillar attachment. Fine fibrils extend to it from the basal cell plasma membrane. Special fibrils extend to it from the connective tissue.

![Figure 2. Rat maxillary incisor. Longitudinal section showing dental organ at base. The tooth has erupted through the oral mucosa. The position of the junctional epithelium over the enamel space (on the labial) and cementum surface (on the palatal) is evident.](image-url)
Kobayashi et al. described similar filaments extending to the lamina densa of the junctional epithelium. These filaments appear to extend from pyramidal structures on the internal surface of the peripheral density of the hemidesmosome (Fig. 4). However, filaments can extend from the plasma membrane as well. There are no special fibrils, since the lamina densa abuts on tooth structure.

Movement of labeled proline from the junctional epithelium to the dentogingival junction indicates the presence of protein, possibly collagen. Ultrastructural histochemical stains, phosphotungstic acid (PTA), periodic acid silver methenamine (PA Silver), and periodic acid-thiosemicarbazide-silver methenamine (PA-TSC-Silver) demonstrate that the basal lamina contains glycoproteins (neutral mucopolysaccharides), supporting earlier light microscopic findings. The cuticle does not stain and thus doubt is cast on its possible derivation from the basal lamina.

Kobayashi and Rose reported that colloidal thorium which demonstrates acid-mucopolysaccharides is negative for the basal lamina, the hemidesmosomes and the cuticle. Ruthenium red, which also demonstrates acid mucopolysaccharides, is reactive with the lamina densa and also with the region between the peripheral density of the hemidesmosome and the plasma membrane. The authors interpreted this as signifying the presence of protein rather than acid mucopolysaccharides, since the basal lamina of other tissues is unreactive. Schroeder interpreted the positive reaction of ruthenium red as indicative of cell coating material (glycocalyx). The cuticle is non-reactive.

CHLORAMINE T SILVER methenamine demonstrates free α-amino acids (or highly condensed protein) in the
cuticle and outer border of acellular cementum. The protein is digestible with proteolytic enzymes (trypsin and chymotrypsin). The basal lamina is nonreactive.

These studies point out that there is a compositional difference between the lamina densa and the cuticle. On the other hand, the reactivity of the outer border of acellular cementum may demonstrate the presence of a coat of cuticle.

In 1980, Sankhyan used immunohistochemical fluorescent techniques to localize the collagen peptides of the dentogingival junction and presumably of the basal lamina. He found C-chain but neither A- nor B-chain, nor type I, II, III collagens.

Isolating the junctional material on the tooth surface by using sodium deoxycholate to remove the epithelial cells is a promising tool. Initial ultrastructural studies reveal that the residual material is granular and varies in thickness between 90 to 370Å.

The epithelial attachment is capable of being formed on enamel, cementum, afibrillar cementum and cuticle. A fibrillar cementum is probably mineralized and is usually found over enamel, but may be found on fibrous cementum in the vicinity of the dentogingival junction. Its outer coat is reactive with choline T-silver methenamine which stains cuticle and not basal lamina. Cuticle A has been reclassified as a form of fibrillar cementum. The suggestion has been made that the fibrillar cementum may carry a fine coat of cuticle.
The cuticle may form on the crown or root in the vicinity of the junction, and is probably protein, since it tests positively for $\alpha$-amino acids. It does not appear to be derived from basal lamina, because its histochemical reactions are different. It is thicker in younger and thinner in older beagle dogs, which implies that it is capable of being altered. The epithelial attachment has been observed to form over calculus covered by cuticle and not plaque. Although it is not considered part of the epithelial attachment, its role in providing a milieu for attachment, its derivation and its ultimate fate are intriguing problems which require further study.

The lamina densa and fine filaments extending to it require further study as well. The chemical composition of the lamina densa and of the fine filaments extending to it from the epithelial cells should be established if possible. The biochemical and biophysical properties of the clear structures (the sublamina lucida and lamina lucida) must also be examined. The adhering and attaching qualities of each should be studied.

Wound Healing

Surgical removal or detachment of the gingiva from the tooth is followed by formation of a new junctional epithelium and epithelial attachment. During healing after gingivectomy, hemidesmosomes appear before the lamina densa forms. In incisal wounds of the tongue, there is a progression of cell thickenings in 8 hours, definitive desmosomes by 18 hours, and bridging of the wound and the first indication of a lamina densa by 24 hours. Lamina densa continuity is established by 48 hours. In 72 to 96 hours an intact structure is present with tonofilaments inserting into the attachment plaques of the hemidesmosomes and anchoring fibrils inserting into the connective tissue side of the lamina densa. The sequence of events requires the presence of fibrin; the hemidesmosomes do not form in its absence. A similar situation must prevail for the junctional epithelium, although it should be noted that there are no anchoring fibrils at the basal lamina abutting the tooth. Tonofilaments do not form clearly evident plaque insertions in the junctional epithelium.

Wounds produced by the insertion of steel blades displacing the junctional epithelium from the teeth of marmosets heal in the following fashion. In 10 minutes only bacteria and cell debris are found in the site of the wound. In 1 day the cell debris is diminished and the bacteria are gone. At this time hemidesmosomes are absent from the superficial surface of the junctional epithelium which becomes infiltrated with leukocytes. In 2 days the separation gap becomes filled with leukocytes except at its greatest depth. There the junctional epithelial cells are found 200 $\mu$A from the enamel. They have rudimentary hemidesmosome formation in the form of condensation at the cell membrane. In 3 days attachment is evident in the lower two-thirds of the wound and mature hemidesmosomes are present. Complete restoration of the epithelial attachment is evident in 5 days.

Observations made in wound healing studies at longer intervals show normal junctional structure. While new attachment may occur in as little as 5 days in some cases, total restoration may take longer (10-20 days) in others. In all instances the presumptive evidence is that the regenerating tissues are derived from adjacent gingival epithelial basal cells.

These findings correspond with autoradiographic reports of cell migration in the junctional epithelium. Such reports indicate a repopulation of the junctional epithelium in ~72 hours. Total transit time (in and out of the junctional epithelium) may be 144 hours. One must assume that the attachment-forming capability is encoded in all epithelial cells but expressed only by undifferentiated basal cells or by postsecretory ameloblasts, given the appropriate conditions, and not by cells differentiated to form keratin. Thus, when the dentogingival junction is injured or displaced during therapy, or accidentally injured by function or some traumatic incident, it can completely regenerate.

The Epithelial Compartment of the Dentogingival Junction and Periodontal Disease

Details of the formation and progression of periodontal disease are summarized in various texts and reviews. The immunologic and microbiologic details, along with the role and progression of the cells of the inflammatory exudate, are outside the scope of this review, but clearly are involved in the events that influence the epithelial and connective tissue attachments to the tooth.

Morphologic changes of the epithelium in the presence of inflammation include intercellular edema, evident through widened intercellular spaces, decreased numbers of desmosomes, disruption of the outer (connective-tissue) facing basal lamina and cellular disruptions. All of these changes are related to pocket formation. The associated connective tissue changes will be discussed in the next section.

The intercellular edema, which may range from mild to severe, is attributable to the increased vascular permeability. Therefore, the intercellular "space" is occupied by fluid as well as cells. As the edema increases the numbers of tight junctions and of desmosomes decrease. These events are more evident in the junctional epithelium than in the gingival epithelium. Some disruption of the basal lamina at the epithelio-connective tissue junction occurs. The basal lamina may exhibit decreased or increased thickness, detachment, multilayer formation, etc. The basal lamina is considered to be a scaffold which defines spatial relationships and permits replenishment of cells in a wide variety of tissues. Multilayer formation, or reduplication, is believed to occur when the plasma mem-
branes of new cells are not opposed to the old basal lamina. When the new cells are separated by some distance from the old basal lamina, they develop a new basal lamina. This may result in the formation of two or more layers of the basal lamina. In human inflamed gingiva the area of basal lamina breakdown is always subjacent to an edematous intercellular space. Thickening of the basal lamina occurs before epithelial-connective tissue separation. Perforations in the basal lamina which are produced in inflammatory cells migrate through close afterward.

Anchoring (special) fibrils are diminished or absent in the presence of inflammation but are frequently related to reduplicated basal lamina. Microfibrils, resembling elastic fibrils, also penetrate the gingival epithelium basal lamina. These two types of fibrils, which may serve in a stabilizing function, are not found in the epithelial attachment to the tooth.

More recently there has been a classification of stages in the pathogenesis of pockets. These stages (initial, early, established and advanced) are related to the degree of inflammatory infiltrate. The progression of a contained initial lesion into a destructive lesion is accompanied by a widening of the intercellular space of junctional epithelium. The widened intercellular spaces, indicative of increased permeability, permit further ingress of plaque antigens, chemospecific substances, toxins, etc. and increased activity of the host response mechanism. The activity of the antigens and toxins and the inflammatory response both produce continuous cycles of reciprocal consequences. Eventually the junctional epithelium is disrupted and the periodontal pocket forms. The epithelial cells are damaged and the continuity of the tissue is interrupted. Inflammatory cells are found in increasing numbers in the junctional epithelium. Junctional epithelium and in addition to accentuating the opened intercellular space, they may displace the junctional epithelium from the tooth surface. On occasion, the widened intercellular spaces and tissue discontinuity may permit the entrance of bacteria. The provocation of the inflammatory reaction by plaque is now a proven fact, yet a cellular infiltration may be found in germ-free dogs, suggesting that other factors may also be operative.

The border between junctional epithelium and connective tissue is straight in health and contains projections (epithelial pegs or ridges) in disease. The apparent border between junctional epithelium and the adjacent gingival or sulcular epithelium becomes more marked as the cellular infiltrate in the junctional epithelium increases from 50% to 60%. Then the most coronal cells of the junctional epithelium disintegrate and are lost and the sulcus bottom/junctional epithelium top shifts apically. The resultant pocket has as its soft tissue wall the remaining gingival epithelium, now constituting pocket or sulcular epithelium. The superficial cells of the sulcular epithelium do not form an epithelial attachment to the tooth.

The Connective Tissue Compartment of the Dentogingival Junction and Periodontal Disease

The connective tissues of the dentogingival junction are of major importance in health and disease. The gingival fibers provide structural support for the gingival and junctional epithelia. The fibers of the periodontal ligament provide for tooth support. The vessels are contained within the connective tissue and permit transport to and from the epithelium and the connective tissue, as well as providing for the mobilization of inflammatory cells. The connective tissue is the primary site of the inflammatory response of periodontal disease. The connective tissue also contributes to epithelio-mesenchymal inductive interactions and to those events based upon such phenomena.

The structure of the periodontal and gingival ligaments, the role of the fibroblast in collagen synthesis, and the structure of collagen are not discussed in this paper and may be found elsewhere.

In periodontal disease, the collagen fibrils of the ligament diminish in diameter and the interfibrillar space widens. The gingival fibers are destroyed subjacent to the junctional epithelium, but are first degraded and become less numerous. The endoplasmic reticulum and ribosomal arrangement are more random than organized, indicating a disturbance in collagen synthesis. Lympocytes may be found in close apposition to the fibroblasts, although they are less numerous. The fibroblast has been shown to be active in collagenolytic and via intracellular phagocytosis. The fibroblasts do not appear to be stationary. They are capable of movement. They contain contractile filaments and can form gap junctions with other fibroblasts, suggestive of mobility. They enter into close proximity with lymphocytes and with monocytes, which may have a capacity for collagen phagocytosis.

Wound-healing studies indicate that the macrophage is essential for wound debridement. When macrophages are absent, the proliferation of fibroblasts and fibrosis are markedly delayed. Cell contact and cell proximity between fibroblasts and some lymphocytes seems to indicate collaboration. In addition, the gap junctions evident between some fibroblasts of the periodontal ligament may be related to the deposition and resorption of collagen. Collagenolysis in periodontal disease may be a fibroblastic function; however, other pathways (epithelial cells, leukocytes, bacteria) are possible.

The uptake of labeled proline is an indication of
collagen synthesis but can signify uptake into glycoproteins as well. It is reported to be higher in the periodontal ligament than in the gingiva.136-139 Twenty-four hours after administration, it is incorporated into noncollagenous protein, not into collagen.140

Pocket Formation

Histologic studies have delineated a sequence of events that occur in gingival inflammation and in destructive periodontal disease without necessarily demonstrating the progression of one to the other. Historical synopses of the earlier histologic work may be found in Waterhag140 and in Page and Schroeder.141

Page and Schroeder142 indicate that in the initial lesion, the connective tissue is rarely more than 5 to 10% involved. In the early lesion collagen loss can reach 60 to 70% and most of the fibroblasts appear to be altered. In the established lesion the junctional and subepithelial structure proliferate into the infiltrated connective tissues and along the root surface. The gingival sulcus deepens and the coronal portion of the junctional epithelium is converted into pocket epithelium. In the advanced lesion the fiber bundles of the marginal gingiva lose their characteristic orientation completely, while the transseptal fiber bundles appear to be continuously regenerated farther apically as the lesion extends. Fingerlike projections of pocket epithelium extend deeply into the connective tissue and the junctional epithelium migrates apically. The collagen-poor zone adjacent to the pocket may remain constant in size indefinitely, and the lesion stable. Other lesions become more aggressive. The formation of a pocket appears to be necessary for a lesion to become aggressive. Morphometric analyses of ultrastructural photomicrographs have indicated that infiltrated gingival connective tissue in man contains 70% less collagen than noninfiltrated. One feature of the infiltrated connective tissue is the increased size of the fibroblasts (reported to be threefold). They have close cell-to-cell contact with lymphocytes, and this may be related to cytoplasmic alterations found in the fibroblasts. It has been suggested that the altered fibroblasts have a diminished capacity to maintain collagen.143

Another ultrastructural study of established periodontitis in the rat showed that fiber loss takes place below the junctional epithelium. It can be divided into three zones: a) complete loss immediately apical to the junctional epithelium, b) partial loss apical to the zone of complete loss, and c) a normal-appearing zone apical to that. Many of these studies are of naturally occurring periodontal disease, whose course may be slow and intermittent.

To overcome the difficulties inherent in studies of slowly progressing disease, which must be carried out over a long period, animal models are being sought in which the disease and the dentition resemble those of man, and where the progress of the disease is quick. Periodontal disease in the rice rat,144-148 ligation-induced destructive periodontal disease in the dog,149 and dietary sucrose-spontaneous periodontal disease in conventional rats149-150 are promising because the onset and progress of the disease are rapid.

In the progression of disease in rice rats the inflammatory exudate is present in the gingival epithelium and lamina propria 2 weeks after weaning. In the next 6 weeks the gingival epithelium narrows, the number of polymorphonuclear leukocytes infiltrating the epithelium increases and there is a downgrowth of junctional epithelium. From the 9th to 14th week the thickness of the junctional epithelium is reduced in many areas to one to three cells. Breaks occur in the gingival epithelium. The transseptal area becomes infiltrated at this time, although it has not been infiltrated earlier.150 Degenerating fibroblasts are found in this area combined with "clumping" with inflammatory cells.151

In ligation-induced rapidly destructive periodontitis in dogs, the destruction of supra-alveolar connective tissue in the initial phase is not accompanied by complete destruction of the connective tissue attachment (fibers remain attached to cementum) and is not immediately followed by apical migration of the junctional epithelium.152

The periodontal disease in conventional rats which occurs as a consequence of a high sucrose diet indicates that an alteration of connective tissue precedes the apical migration of the junctional epithelium. The cemental surface is denuded. There is a loss of collagen associated with the presence of fibroblasts containing a dilated endoplasmic reticulum. The fibroblasts in the infiltrated connective tissue are small but round. There are areas of focal basal lamina breakdown. The fibroblasts appear to be damaged near the epithelium and the collagen breakdown is also subepithelial.

Pocket formation has been attributed to a loss of cellular continuity in the coronal portion of the junctional epithelium,153-156 recalling concepts of Wessely157 and Eulber158 who attributed pocket formation to an intraepithelial split. While loss of cell continuity certainly plays a contributory role, the loss of subepithelial collagen, the lysis of collagen fibers inserting into the cementum, and the breakdown of the basal lamina are the main factors. The pocket is a result of the subsequent apical reconstitution of the basal lamina (Fig. 5).159-160 Below the junctional epithelial cells that have migrated apically, the basal cells attempt to reconstitute a basal lamina over the pathologically altered connective tissue. The cells migrate as they do in wound healing. In the rat, the junctional epithelium is renewed from adjacent proliferative centers in the gingival epithelium.159 The data given for primates can be interpreted similarly161-164 except that some portion of the junctional epithelium seems to have proliferative capability.

The basal lamina is necessary for maintenance of tissue architecture, for diffusion, and for the performance of inductive interactions165 (see discussion of basal lamina in the section on Epithelial Compartment).
The dynamic nature of the junctional epithelium and its basal lamina is made more evident by age comparison studies of germ-free rats where the base of the junctional epithelium of the molars appears to retract, that is, migrate coronally for a short distance uncovering enamel near the cementoenamel junction.  

CONCLUDING REMARKS

This review leads to a concept in which the tissues of the dentogingival junction are dynamic rather than static. Even when they are pathologic, they can be reconstituted by repair. Both their cellular and extracellular components exhibit a high rate of turnover. Some of the cells are specialized for specific functions, such as attachment formation, and do not generate additional cells, but generative pools are always nearby. The cells are capable of movement and of positional change. The junctional epithelium can advance and retract. The cuticle width is alterable. The entire tissue is capable of regeneration after wounding. This dynamic group of tissues is well adapted for the healing of direct injuries produced during mastication. The tissues do remarkably well, over long periods, in their response to periodontal disease, whether due to direct bacterial or toxic damage, or to indirect damage via the migration of inflammatory cells into the lesion. The tissues show a capacity for repair and regeneration following the elimination of plaque formation and the resultant resolution of the inflammatory infiltrate.

The complete story is not yet developed. The past 60 years are replete with fine contributions by distinguished workers. Additional contributions continue to be made. The inheritance from our predecessors has been used well and our expanded knowledge in this area now serves as the conceptual framework for further study.
REFERENCES


Send reprint requests to: Dr. Irving B. Stern, 1148 Medical-Dental Building, Seattle, WA 98101.