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## The Gingival Sulcus and the Periodontal Pocket Immediately Following Scaling of Teeth

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THE clinical and histopathologic features of gingival and periodontal pockets in relation to therapeutic procedures have been discussed in several recent publications.<sup>1,2,8,9</sup> The wide variety of opinions expressed on the nature and response of the epithelial attachment to therapeutic measures appears very confusing to the practitioner. Because of the apparent confusion regarding this problem, a clinical and histologic study of gingival and periodontal pockets was initiated using various intervals of time following common therapeutic procedures.

This report will deal with the histologic findings in specimens obtained immediately following scaling of teeth for removal of plaques and subgingival calculus.

### MATERIAL

The specimens were obtained from patients who needed partial resection of the alveolar process in association with insertion of prosthetic appliances. A technic was developed for removal of suitable specimens of the type shown in Fig. 1. Buccal and some interproximal soft tissues with 2 to 4 mm. depth of alveolar process were separated surgically from the surrounding tissues. A sharp knife was used and attempts made to leave the soft tissue attachment to the tooth undisturbed. Periosteal flaps were elevated from the specimen, and diamond discs or sharp burrs were used to cut through the bone into the tooth at the periphery of the soft tissue specimens without the disc or the burr touching the soft tissue. Then the tooth with the adherent soft tissue was carefully removed. Immediately before this procedure a routine scaling of the tooth was performed. A deliberate, but careful attempt, was made to nick the surface of the cementum for a reference mark by utilizing a very definite finger rest to prevent any slip of the instrument, and moving a curette in a coronal direction from the bottom of the pocket. (Bunting's curettes No. 5 and No. 6, made by S. S. White, were used for the scaling and the marking of the surface of the tooth.) Two teachers of periodontics each scaled about half of the teeth. No flushing or rinsing of the pocket was done following the scaling. As a matter of convenience, the mesiobuccal, buccal, and distobuccal areas of mandibular and maxillary anterior teeth were selected for the investigation.

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From each specimen numerous sections were prepared for microscopic study. Specimens that contained artifacts from accidental trauma during the surgical removal were discarded. Microscopic study showed that nine of fourteen specimens were free from soft tissue distortion and could be used. Clearly recognizable markings from the scaling could be located on these teeth. These nine specimens were obtained from patients varying in age from 18 to 58 years of age, and with clinical diagnoses varying from "normal gingiva" to "moderately severe periodontitis." Giemsa stains and an examination for micro-organisms were done on 2 sections from each case.

#### FINDINGS

Case No. 1. Female. Age 42. Tooth No. 9. Labial crevice. Ulceration of apical one half of the crevicular surface. Parakeratosis of gingival surface. Active chronic inflammation both in the free and the attached gingiva with ingrowth of rete pegs and proliferation of epithelial rests of Malassez. Free gingival groove situated apically to bottom of pocket. No residual epithelial attachment at bottom of pocket in the area which had been scaled. (Fig. 2). Accumulation of debris on crevicular surface of pocket. Evidence of slight superficial injury to periodontal fibers at the bottom of the pocket. New bone formation on tip of alveolar crest.

In two areas where the crevicular surface appeared torn, micro-organisms (cocci) and extravascular red blood cells were observed within the traumatized tissues immediately beneath the crevicular surface.

Case No. 2. Male. Age 58. Tooth No. 9. Labial crevice. Ulceration of crevicular lining. Parakeratosis of gingival surface. Mild chronic inflammation. No residual epithelial attachment at bottom of crevice. (Fig. 3). Some periodontal fibers have been severed and cementoblasts have been removed from the surface of the tooth at the bottom of the crevice. The traumatized area was filled with coagulated blood. Bac-

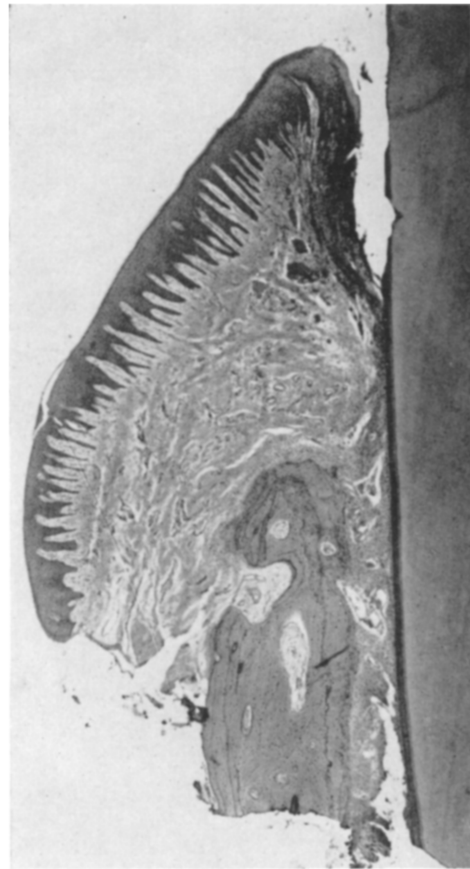


Fig. 1. (X20) Type of specimen usually obtained.

teria and debris on crevicular surface, but no invasion of bacteria. A few bacteria observed within the connective tissue at site of surgical crushing in the apical end of the specimen.

Case No. 3. Male. Age 40. Tooth No. 12. Distobuccal crevice. Erosion and ulceration of crevicular surface. Parakeratosis of gingival surface. Active chronic inflammation extending into the periodontal membrane. Pronounced mucoid degeneration associated with the inflammation. No evidence of residual epithelial attachment in the area marked by the scaling. (Fig. 4). Some periodontal fibers have been severed at the bottom of the pocket and coagulated blood has filled this site of injury. Active resorption of alveolar crest.

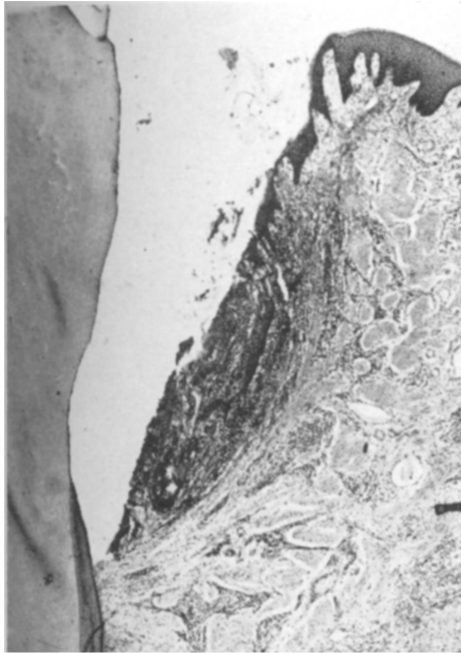


Fig. 2. (X45) No residual epithelial attachment. Debris and ulceration on crevicular surface. Demarkation for scaling not well shown in this picture.



Fig. 3. (X65) The defect produced in cementum by scaling is clearly shown. No residual epithelial attachment. Evidence of traumatic soft tissue injury from the scaling.



Fig. 4. (X231) The cementum has been fragmented during the scaling procedure. No residual epithelial attachment. Blood between the bottom of the crevicular lining and the surface of the tooth, indicating traumatic soft tissue injury from the scaling.

Colonies of filiform organisms were observed on the surface of the crevicular lining. Some cocci were found in an area of hemorrhage and inflammation,  $\frac{1}{2}$  mm. beneath the crevicular surface.

Case No. 4. Male. Age 50. Tooth No. 28. Distobuccal crevice. Extensive crevicular ulceration. Parakeratosis of gingival surface. Severe chronic inflammation extending into the periosteum and the adjacent marrow spaces. Marked osteoclastic activity at alveolar crest. No residual epithelial attachment present at the bottom of the pocket. (Fig. 5). Evidence of rather severe traumatic injury from the scaling with cutting and tearing of periodontal fibers was seen mostly in association with fibers that were partially destroyed during the preceding course of inflammation. This area of injury was filled with debris (calculus, bacterial colonies, and fragments of cementum). Large colonies of bacteria were observed on the crevicular surface as well as superficial invasion of micro-organisms associated with coagulating blood in cracks of the crevicular lining. No bacterial invasion into



Fig. 5. (X308) No residual epithelial attachment. Debris in an area of injury from the scaling. The zone of injury extends apically to the mark produced by scaling on the tooth surface (A).

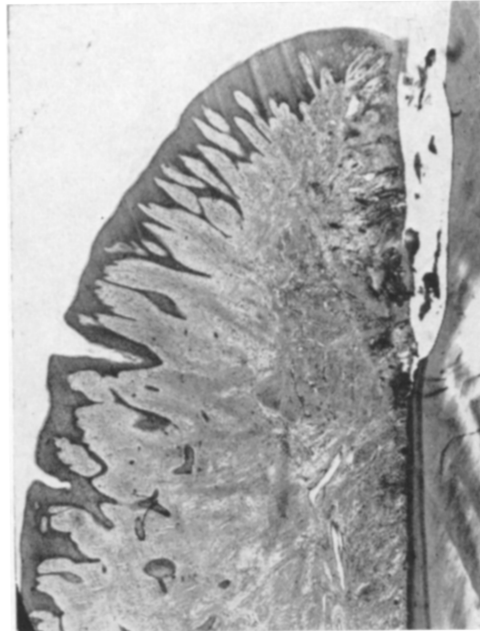


Fig. 6a. (X16) Defect in cementum and dentin demarcated the area of scaling on the surface of the tooth. Two gingival grooves.

living tissues was found.

Case No. 5. Female. Age 46. Tooth No. 23. Mesiolabial crevice. Small crevicular ulcerations. Parakeratosis and hyperkeratinization of gingival surface with evidence of marked stippling and two free gingival grooves. (Fig. 6a). Small gingival cyst. Marked squamous character of Malassez's epithelial rests (Fig. 6b). Chronic inflammation. No evidence of residual epithelial attachment in the scaled areas. Some specimens showed that periodontal fibers were cut at the bottom of pocket from the scaling (Fig. 6c). This area of injury was filled with coagulating blood and debris. New bone formation at alveolar crest.

Bacteria on crevicular surface. Some mononuclear cells contained phagocytized debris (possibly bacterial origin). No bacterial invasion found.

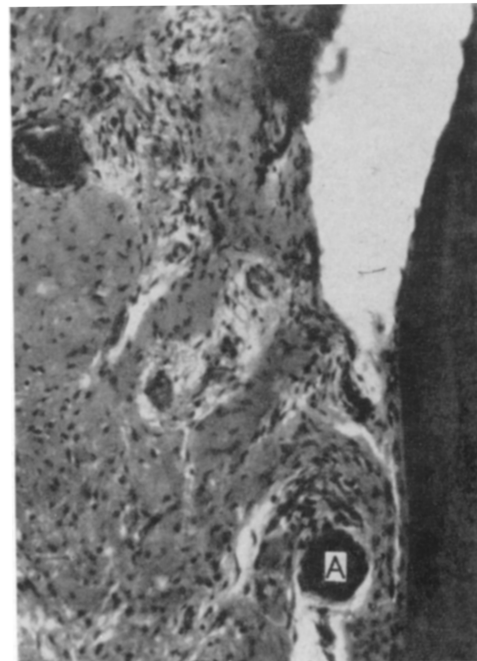


Fig. 6b. (X399) Large Malassez's epithelial rest (A). Tear in periodontal fibers at bottom of gingival sulcus. This area of injury appears partly filled with blood.



Fig. 6c. (X217) Severe injury at the bottom of the pocket. Debris and blood partially filling the crevice. Extravasation of blood in surrounding tissues.

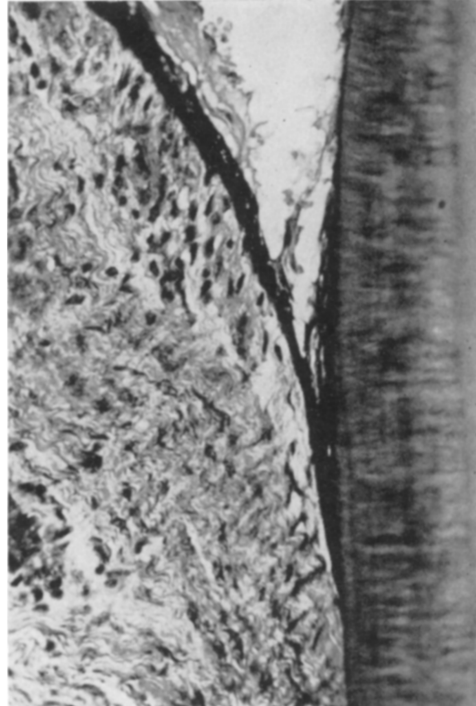


Fig. 7b. (X658) High magnification from 7a showing tear in the epithelial attachment with living epithelial cells attached to the cementum and blood within the split of the epithelium.

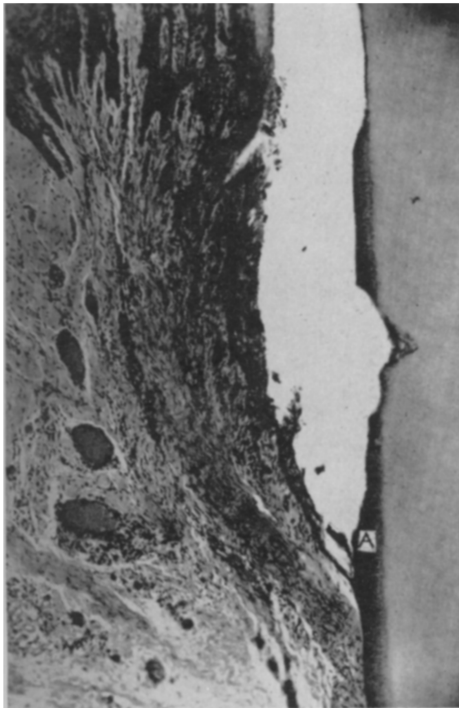


Fig. 7a. (X196) Thin residual epithelial attachment below marks produced by scaling (A).

Case No. 6. Female. Age 20. Tooth No. 20. Buccal crevice. Degenerative changes present in crevicular epithelium and partial split of rete pegs. Parakeratosis and hyperkeratinization of gingival surface. Acute passive congestion. Mild chronic inflammation. New bone formation at alveolar crest. In the area of deepest scaling (according to marks on the tooth) some trauma to periodontal fibers was observed and no residual epithelial attachment was present. However, in a closely adjacent area a slender projection of the epithelial attachment, 2 to 3 layers of cells in thickness, was seen apically to the reference mark for the scaling (Fig. 7a). This residual epithelial attachment showed evidence of a tear (Fig. 7b) and the presence of numerous red blood cells in the fissure of tear indicated that the tear had occurred before the fixation of the tissues.



Fig. 8. (X189) No residual epithelial attachment. Note cuticle (A) and blood between the cuticle and the crevicular epithelium indicating that this split was present before the fixation of the specimen.

An abundance of filiform and coccal organisms mixed with blood were observed in the gingival crevice. No invasion of organisms into living tissues.

Case No. 7. Male. Age 20. Tooth No. 27. Buccal crevice. Degeneration of crevicular epithelium. Split in rete peg of the crevicular lining (Fig. 8). Hyperkeratinization of gingival surface. Chronic inflammation. Resorption of alveolar crest and fibrosis of bone marrow. No evidence of residual epithelial attachment in area scaled, but a tear in residual epithelial attachment in adjacent areas. Small extravasation of blood into the tissues in immediate vicinity to marks from the scaling at the bottom of pocket. Abundant accumulations of coccal, filamented, and rod-shaped organisms mixed with desquamated epithelial debris and blood were observed in the gingival crevice. No bacterial invasion into living tissues.

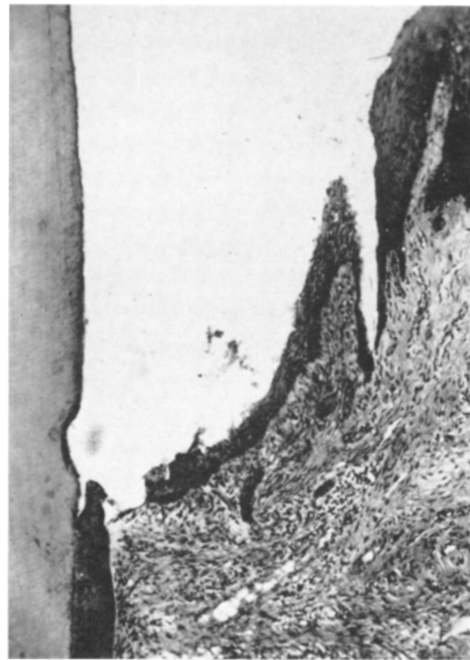


Fig. 9a. (X196) Split of crevicular rete peg and tear of epithelial attachment.

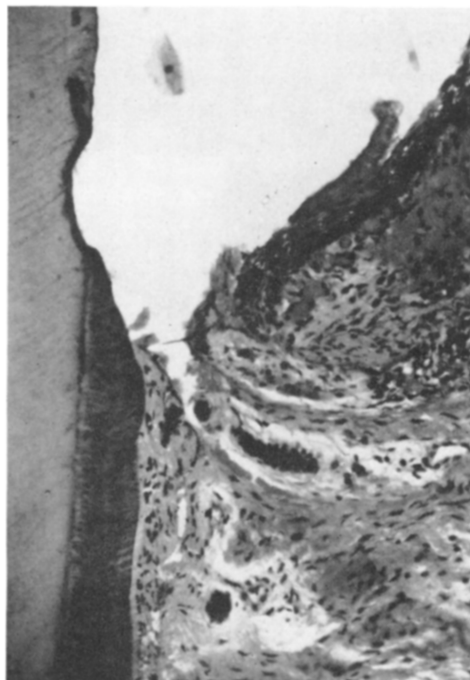


Fig. 9b. (X399) No residual epithelial attachment. Tear of a few connective tissue fibrils at the bottom of the crevice. The tear is partially filled with blood and extends into Malassez's epithelial rests.



Fig. 9c. (X826) Decalcified enamel (A). Primary and secondary enamel cuticle (B) with attached epithelial cells. Crevicular lining (C). Red blood cells present in the crevice indicating pre-fixation separation.

Case No. 8. Male. Age 20. Tooth No. 29. Mesio Buccal area. Clinically normal gingiva. Deep split of rete peg in otherwise unbroken crevicular epithelial lining (Fig. 9a). Hyperkeratinization of gingival surface. Mild chronic inflammation. New bone formation at alveolar crest. No residual epithelial attachment, but trauma from scaling has severed a few periodontal fibrils at the bottom of the pocket (Fig. 9b) and coagulating blood has partially filled this fissure of tear. Remains of enamel cuticle and a few attached epithelial cells were found on a small fragment of decalcified enamel (Fig. 9c).

Bacterial stains revealed mouth organisms only on the surface of the specimen.

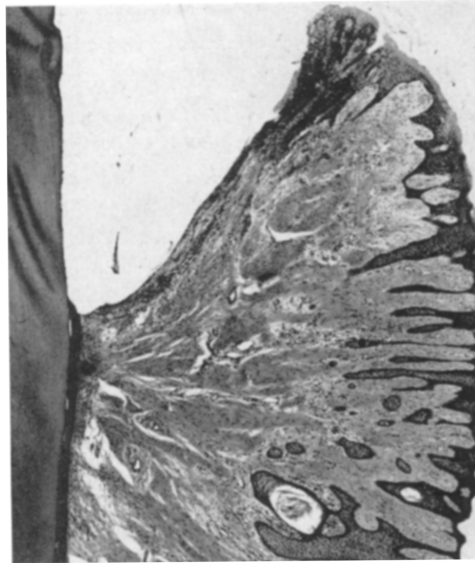


Fig. 10. (X30) No residual epithelial attachment. Gingival microcysts.

Case No. 9. Male. Age 18. Tooth No. 9. Distobuccal crevice. Crevicular epithelial lining thin and completely missing in some areas (Fig. 10). Parakeratosis and hyperkeratinization of gingival surface. Two gingival microcysts in association with hyperplastic rete pegs. Mild chronic inflammation. No evidence of residual epithelial attachment at the bottom of the pocket. No bacterial invasion.

#### EVALUATION AND CLINICAL SIGNIFICANCE OF FINDINGS

It appears from this material that thoroughly executed routine scaling of teeth with fine instruments will sever the epithelial attachment and the cut will extend into the adjacent connective tissue attachment. Only in one case was a slender epithelial projection (2 to 3 cells in thickness) observed that extended slightly apically to the scaling marks on the tooth. In the presence of severe inflammation and partial destruction of the collagen fibers, it was noted that the scaling tended to injure an area slightly apically to the bottom of

the epithelial attachment, and aggregations of surface granulation tissue and epithelial cells were loosened and sometimes removed from their normal site.

This observation is of great clinical significance since it will help to understand and explain the satisfactory results which have been obtained on the basis of seemingly different approaches to conservative periodontal therapy. It has been claimed,<sup>3</sup> but not wholly explained, that periodontal pockets could be eliminated and reattachment obtained without intentional removal of the epithelial attachment and the epithelial lining of the pocket. As brought out by Orban,<sup>4</sup> this seems theoretically impossible. However, it is evident from the present investigation that the epithelium in the bottom of periodontal pockets actually can be removed unintentionally by routine scaling thereby acting as subgingival curettage, and creating the biologic possibility for reattachment.

In recent publications it has been widely discussed as to how far below the bottom of the pocket one should remove soft tissues in order to assure elimination of the epithelial attachment.<sup>1, 2, 8</sup> A main argument against the use of subgingival curettage has been the often stressed possibility that one by this deliberate surgical or chemical extension of the pocket had to consider the risk of improper healing and loss of some periodontal attachment as a direct consequence of the therapy.<sup>8</sup>

To measure the depth of the epithelial attachment from sections is inadequate because it does not account for the often wavy surface of the bottom of the gingival crevice or pocket, but to assume that thorough scaling prior to subgingival curettage or gingivectomy will extend only to the degenerated cells on the surface of the epithelium at the epithelial attachment is a far greater error.

It is evident that the surface of the epithelial attachment, following oral prophylaxis, will not be at the same level as before the scaling, and that the measure-

ments of the epithelial attachment indicated by Ritchey and Orban<sup>8</sup> do not apply to a case that has been prepared for subgingival curettage or gingivectomy by previous scaling.

On the basis of the presented findings there does not seem to be any rationale for removal of soft tissues below the bottom of the pocket during subgingival curettage or gingivectomy provided that a thorough scaling has preceded these operations.

The epithelial lining of the wall of the pocket was partially removed by the scaling in areas of severe inflammation and mainly in the apical one third of the pocket, but it is obvious from the presented photomicrographs that the epithelial lining of the pocket has to be deliberately removed in order to obtain maximum extent of reattachment following subgingival curettage. Ritchey and Orban<sup>8</sup> state, "Curettement of the soft tissue side of the pocket in expectation of an epithelial attachment should include the removal of *the entire pocket epithelium* . . . . ." This statement should not be taken literally because it has been shown experimentally<sup>6</sup> that both connective tissue reattachment and new epithelial attachment may occur in the presence of numerous epithelial inclusions that remain of the previous lining of the pocket. An attempt to insure complete removal of very deep projections of rete pegs in the crevicular epithelium would lead to unwanted and unnecessarily extensive removal of soft tissues, a widened pocket, and delayed healing. Even the presence of a few remaining epithelial cells in small areas at the bottom of the epithelial attachment would not prevent reattachment from a blood clot in the pocket that was coronal to these remaining cells provided a fairly adequate removal of the epithelial lining of the crevice was accomplished.<sup>6</sup>

It was also interesting to note that the scaling tended to split deep rete pegs in the wall of the pocket as well as split the epithelial attachment. The extent of healing of these splits is not known, and it



will be investigated further. It is conceivable that a caustic drug such as phenol camphor would reach and more dependably destroy the epithelial cells within such fissures than would be possible by superficial surgical curettage.

There was a tendency for the scaling effects to extend apically to the epithelial attachment resulting in subepithelial injury. It is possible that this injury will deepen the pocket during the course of periodontal treatment especially in cases with severe active inflammation. The evidence of trauma is particularly significant because bacteria and debris commonly were observed in these areas mixed with the coagulating blood. The extension of scaling injury to Malassez's epithelial rests (Fig. 9b) may cause proliferation of these cells, and thereby be the source of a new epithelial attachment before connective tissue healing can take place, or before crevicular epithelium can reach the area. The practical implication of these findings should be to do a careful, but not necessarily a complete, scaling in severely inflamed pockets at the first sitting. The area should be rinsed thoroughly, and a blood clot allowed to form in the bottom of the pocket. Detailed scaling should be delayed for 7 to 10 days until the inflammation has subsided. During this period an epithelial lining of the crevice will form, and some organization and maturation of the subepithelial granulation tissue will take place so that definite resistance can be felt against instruments during the scaling procedure.

If subgingival curettage or gingivectomy has to be utilized for elimination of residual periodontal pockets, this should of course be done after the periodontal tissues have reached a maximal state of preoperative health in order to facilitate repair and regeneration.<sup>7</sup> Following the scaling, neither subgingival curettage nor gingivectomy should include tissues below the clinical bottom level of the pocket.

The significance of bacteria being present in the coagulating blood and in associa-

tion with traumatized tissues can not be ascertained from this study. It may be assumed that they delay the process of healing. As to whether or not these bacteria are apt to invade further into the traumatized or the adjacent tissues can not be stated on basis of the present findings. That bacteria were observed on the root surfaces following extraction was in accord with what should be expected from operating in a non-sterile field.

Five gingival microcysts were incidental findings in serial sections of 16 specimens of the before described type. The free gingival groove, if present, did not necessarily correspond to the level of the bottom of the gingival crevice or pocket,<sup>5</sup> and in one case two gingival grooves were observed.

#### CONCLUSIONS

1. Routine thorough scaling of teeth will tear and split the epithelial attachment, and commonly the scaling procedure will extend the tear into the connective tissue attachment.
2. Subgingival curettage or gingivectomy, if done following thorough scaling, should be extended only to the bottom of the clinical pocket at that time, because the epithelial attachment has already been severed by the previous scaling.
3. Scaling should be done with special care in areas of severe gingival inflammation to avoid deepening of the gingival crevice or the periodontal pocket.
4. The pocket should be thoroughly rinsed of debris, and a blood clot allowed to form following the scaling.

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