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Human Clinical and Histologic Repair Responses following the Use of Citric Acid in Periodontal Therapy

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RECENT PUBLICATIONS have reported on various attempts to accelerate repair and/or regeneration of periodontal attachment lost due to inflammatory periodontal disease.[1-5] However, all authors agree that results of these procedures are unpredictable at both the clinical and microscopic levels. Among the many items influencing the uncertain results of our "reattachment" procedures is the relative lack of knowledge concerning factors governing cementogenesis over pocket exposed cementum. Such cementogenesis, in turn, could lead to connective tissue attachment incisal to its position at time of therapy.[6] Recently, Register[7] and Register and Burdick[8] reported that accelerated reattachment with cementogenesis to dentin may take place when root surfaces are demineralized in situ. This approach led us to evaluate pocket closure both clinically and histologically in patients treated with the demineralization procedure.

MATERIAL AND METHODS

Patients selected for this study were under treatment for periodontitis at the periodontal clinic at New York University College of Dentistry. An informed consent form was obtained from each patient. Patients were selected from those who had at least one or more teeth with pocket depth greater than 4 mm remaining on the *facial aspect* following initial therapy. Only those patients who were able to maintain an oral hygiene index (OHI-S)[9] approaching zero were included in the study.

Presurgical preparation in all cases consisted of oral hygiene instruction, root planing and curettage, occlusal equilibration and dietary evaluations.

Two of the patients taking part in the clinical phase of the present study permitted surgical removal of seven treated teeth for histologic study. Extraction of these teeth was necessitated by prosthetic considerations.

Patient "A" was a 73-year-old Caucasian male with no significant medical history who required extraction of tooth No. 29. Patient "B" was a 53-year-old Black male with no significant medical history who required extraction of six remaining maxillary teeth in order to construct a maxillary full denture. These teeth were Nos. 2, 4, 5, 12, 13, and 14 (Figs. 1-3). Clinical pocket depth around these teeth ranged from 4.0 to 8.1 mm with an average depth of 5.3 mm.

METHODS OF MEASUREMENT

Following initial therapy, two sets of study models were obtained and an omnivac stent was fabricated. The method of measurement and the fabrication of individualized stents have been previously described.[10] For each of the teeth in the study the conditions at the time of surgery were recorded on a specially prepared data sheet. Immediately prior to surgery, the following measurements were recorded: (a) stent to gingival margin; (b) stent to depth of pocket; and (c) stent to cementoenamel junction (CEJ) to check for seating of the stent.

During flap surgery, the following measurements were recorded: (a) stent to the coronal insertion of the gingival fibers; and (b) stent to CEJ.

Following suturing, the following measurements were recorded: (a) stent to coronal height of the gingival margin; and (b) stent to CEJ.

Measurements identical to those performed immediately prior to surgery were recorded at 4, 8, 12, and 16 weeks after surgery.

SURGICAL PROCEDURES

The surgical procedure for all teeth was as follows: An inverse bevel incision to the alveolar crest with maximum retention of the gingival margin was performed and a periosteal flap reflected, using the predetermined pocket probing as a guide. A horizontal incision was then made at the base of the pocket and the contents of the pocket lining and connective tissue coronal to this incision were removed with a curette.

Prior to root preparation, a rubber dam was fitted and placed over the tooth being treated in order to

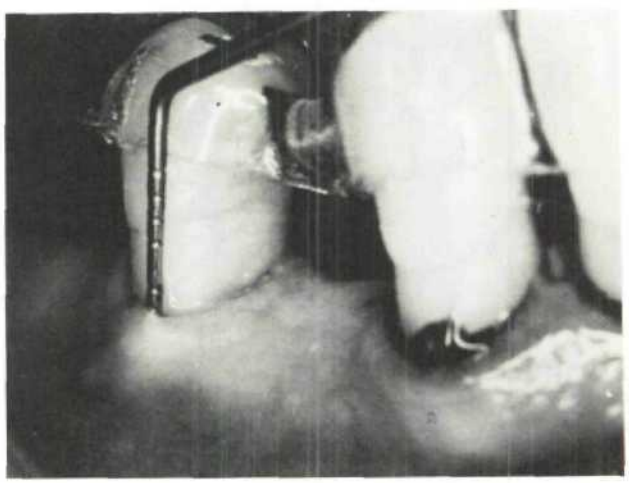

FIGURE 1. *Preoperative photograph of Patient "B".*

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isolate the tooth, and the tooth was randomly assigned to one of four different demineralization procedures, Groups A to D.

Group A. Following flap reflection and removal of both root accretions and pocket wall, the entire root surface to the crest of bone was treated with citric acid solution (pH = 1) for 2 minutes. The citric acid was continuously applied with four cotton pellets, each in contact with the tooth for 30 seconds. Two minutes later the root surface was dried and rinsed thoroughly with sterile saline (3 teeth).

Group B. Following flap reflection and removal of both root accretions and pocket wall, only the pocket-exposed cementum was treated with citric acid. The rubber dam was adjusted accordingly (2 teeth).

FIGURE 2. *Clinical lesions of patient "B" at time of surgery.*

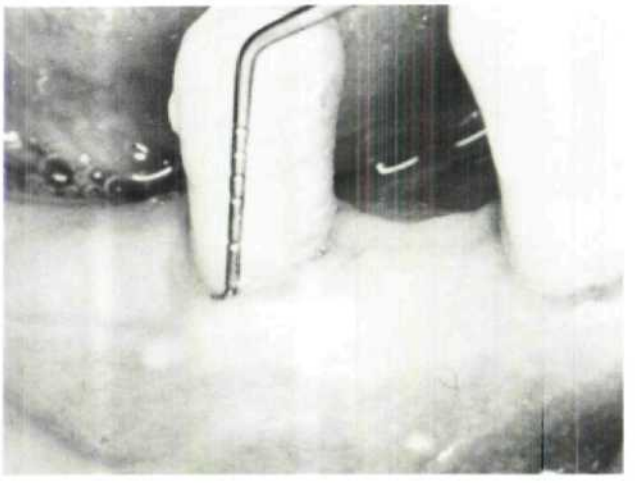

FIGURE 3. *Postoperative photograph of patient "B" 16 weeks after surgery.*

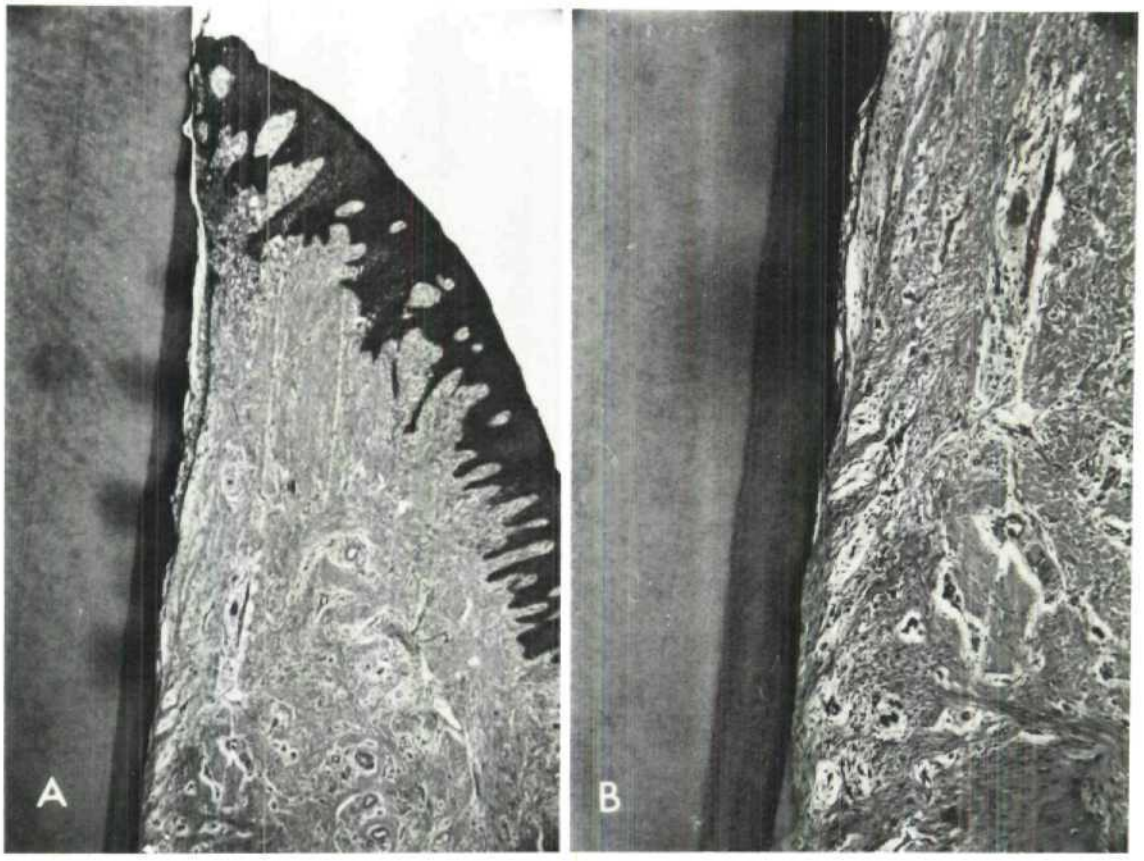

FIGURE 4A. *Human specimen from Group A removed 16 weeks after surgery. In these specimens citric acid was applied to the root planed surface and the crestal collagen mantle (H & E. Original magnification × 10). Note the lack of cementogenesis above the alveolar crest and the long junctional epithelium.* B. *Higher magnification of supracrestal area from specimen shown in Figure 4A (Original magnification × 25). Note parallel oriented fibers below the most apical position of the junctional epithelium. Apical to these fibers are functionally inserted fibers without evidence of cementogenesis at this site.*

Group C. Surgical procedures were identical to those described for Group B. However, in this group the pocket-exposed cementum was thoroughly curetted in an attempt to remove all of it. Citric acid was then applied to the exposed dentin (1 tooth).

Group D. Surgical procedures were identical to those described for Group B. In this group, however, removal of accretions was followed by treating the exposed root with sterile saline for 2 minutes. This group served as the control specimen (1 tooth).

In all cases, flaps were replaced at a position as close as possible to their presurgical level and were sutured. Measurements and photographic documentation were obtained immediately and at 4, 8, 12, and 16 weeks postoperatively.

Sixteen weeks after surgery, the seven teeth were removed using a "block section technique" and prepared for routine histologic study. All extraction sites healed uneventfully.

Clinical Observations

At 16 weeks after surgery, gingival recession had taken place on all teeth. Recession varied from 0.4 to 1.5 mm with an average recession of 0.7 mm. Pocket depth at 16 weeks post-surgery varied from 1.6 to 4.2 mm with an average depth of 2.7 mm. Since these findings are limited to only a few specimens, statistical relevance of healing responses will have to await clinical healing trends monitored in larger groups of patients. Such a study is currently in preparation.

Histologic Observations

For clarity, the histologic responses will be described for each of the four groups.

Group A (3 teeth—entire root (CEJ to crest) treated with citric acid). In the three specimens available, evidence of cemental removal was present. However, the degree of cemental removal varied within each specimen and within the group. The new gingival margins were usually apical to the exposed dentin. The histologic form of pocket closure against root planed cementum appeared to be a long junctional epithelium which adhered to the root surface. No evidence of cementogenesis was noted in these areas (Figs. 4A and B). The connective tissue apical to the junctional epithelium was oriented parallel to the root surface for a short distance. Apically to this collagen, fibers appeared to be functionally oriented. No evidence of cementogenesis was noted in these areas. Since crestal collagen fibers were left inserted into the root during the initial surgery and no evidence of cementogenesis was present at the supracrestal level, we must assume that the functionally oriented collagen fibers seen inserting into the cementum were in that position prior to surgery. Inflammation in the gingival margin was limited and crestal remodelling was noted. It was of interest that the application of citric acid to the supracrestal collagen mantle showed no ill effects on collagen at this level of investigation.

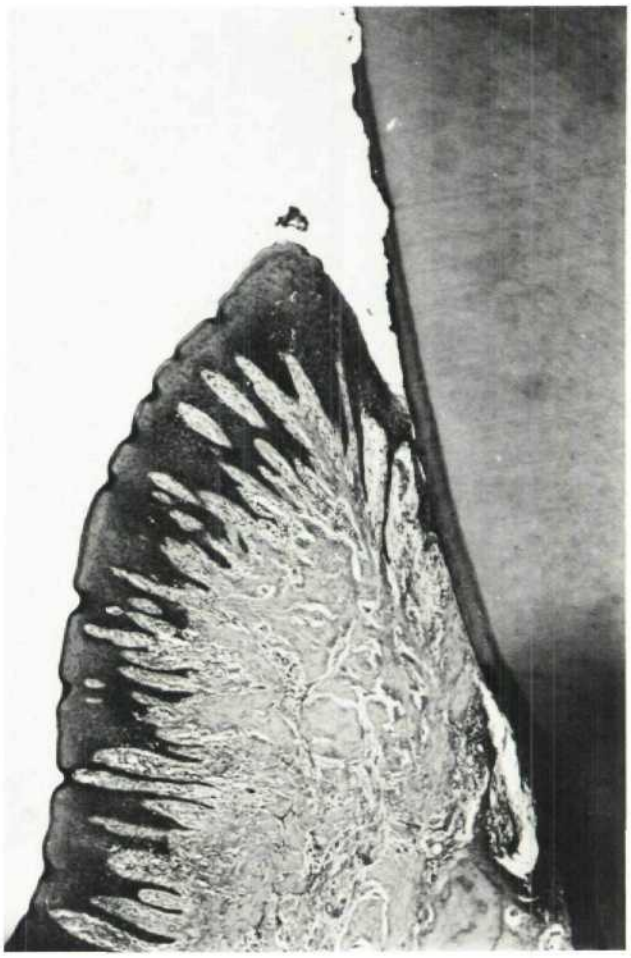

FIGURE 5. *Human specimen from Group B. In these specimens, citric acid was only applied to the planed root surface (H & E. Original magnification × 10). No evidence of cementogenesis is seen at the cemental surface.*

Group B (2 teeth—only pocket-exposed cementum treated with citric acid). These specimens responded very much like those described in Group A (Fig. 5), except for one area where apparently new cementum was present on the surface of acellular cementum. It appeared similar to the repair cementum seen in association with the placement of osseous autografts [11] (Figs. 6A and B). This repair cementum was found incisal to the crest with a small portion of it incisal to the base of the pocket. The greatest bulk of the "repair" cementum however was seen below the junctional epithelium. Collagen fibers appeared to be functionally inserted into this cementum. Obviously our procedures cannot, with certainty, identify the time of deposition of the "repair cementum." Therefore we are unable to state that this repair phenomenon can be associated directly with the application of citric acid. Furthermore, repair cementum was not present at the most incisal portions of the planed root surface in this specimen.

Group C (1 tooth—cementum planed to crest of bone and entire exposed root surface treated with citric acid).

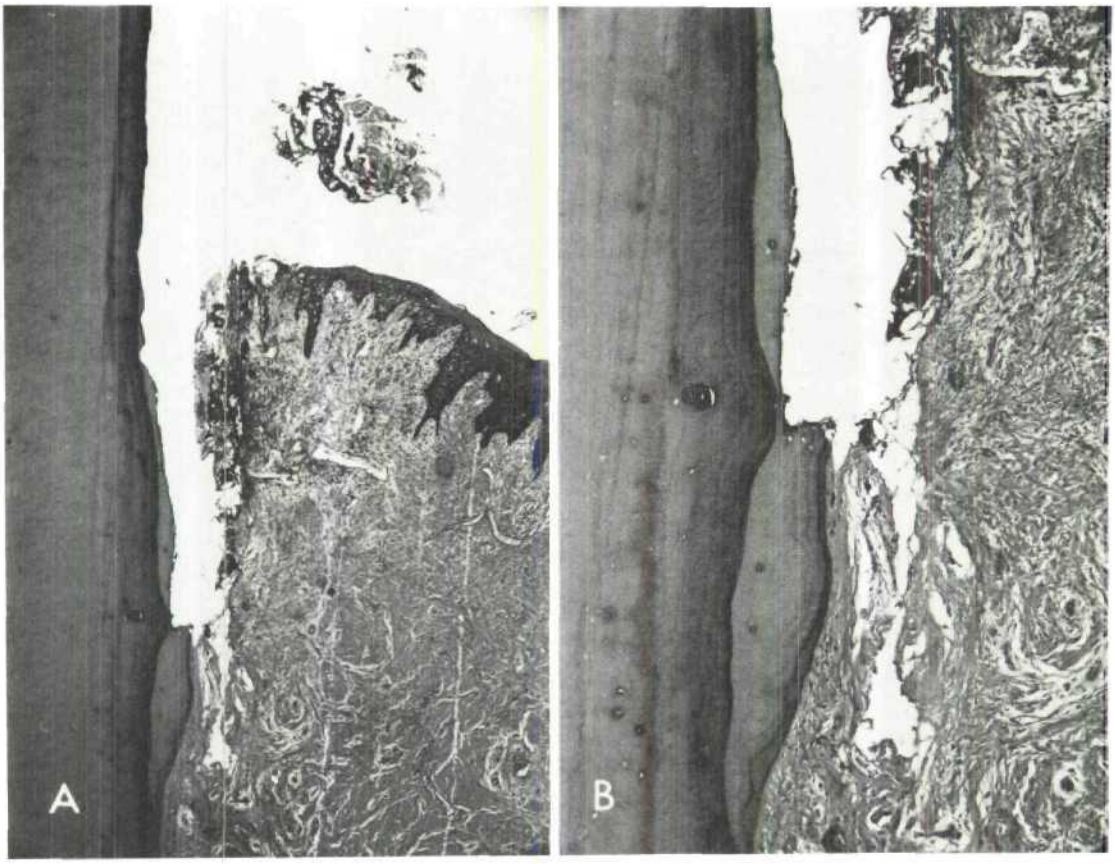

FIGURE 6A. *Human specimen from Group B (H & E. Original magnification × 10). Note evidence of "repair cementum" within the pocket as well as apical to the junctional epithelium.* B. *Higher magnification of "repair cementum" from specimen shown in Figure 6A (Original magnification × 64). Note the "repair cementum" adhering to acellular cementum. Fibers appear to be inserted into this structure. Also note the markedly thicker "repair cementum" below the junctional epithelium than that seen incisal to it.*

This specimen showed evidence of cementum remaining on the root surface especially near the crest of bone where collagen fibers may also have been left on the root surface. No evidence of cementogenesis was noted on the root surfaces treated with citric acid. The mode of pocket closure was essentially by epithelial adhesion (Fig. 7).

Group D (1 tooth—pocket-exposed cementum planed and saline applied to the root surface). This specimen showed acellular cementum remaining at the planed root surface. An epithelial-lined pocket and a somewhat elongated junctional epithelium were seen in association with significant local inflammation. Some crestal remodelling was also present (Fig. 8).

COMMENTS

As in many human histologic investigations, the present study demonstrates case reports rather than information which can be statistically evaluated. The difficulties in obtaining human dental block sections obviously necessitate such limitations. However, a repair trend can be observed with even limited specimens and compared with extensive data obtainable from human clinical trials and animal experimentations.

Within these limitations, our material does not indicate that citric acid applied to planed, pocket-exposed cementum induced or accelerated cementogenesis. Nor did it create an environment for new or accelerated attachment of functionally-oriented collagen fibers at root sites previously exposed to the oral environment in association with inflammatory periodontal disease.

However, the histologic responses provided some further clinical insight into the healing patterns following periodontal flap surgery. Our specimens demonstrated the difficulty a clinician faces when asked to remove all of the cementum. In our specimens, some cementum was present in all specimens in which the clinician attempted total removal.

The mode of pocket closure was essentially similar to that observed following root planing and retention of gingival fibers;[12] namely, (a) recurrence of some pocket depth; (b) an elongated junctional epithelium; (c) a small area of parallel oriented fibers immediately apical to the junctional epithelium; and (d) functionally inserted collagen fibers without evidence of cementogenesis at the site of their insertion.

A special comment must be made regarding the finding of repair cementum in one citric acid treated speci-

men. Since all of the citric acid treated specimens came from the same patient, this evidence of repair cementum represents only one of six potential sites. Nevertheless, its presence must be evaluated. As stated before, similar repair responses were frequently observed following the insertion of osseous autografts in humans.[11, 13] Our observations thus suggest a repair sequence which apparently may take place following varying modes of periodontal therapy. The present data is too limited to comment on the frequency of occurence of this type of repair when different therapeutic techniques are performed.

Since our study focuses on histologic observations, *clinical results* following demineralization procedures cannot be adequately discussed from this sample. More extensive clinical trials hopefully will provide further information regarding the usefulness of root demineralization in periodontal therapy.

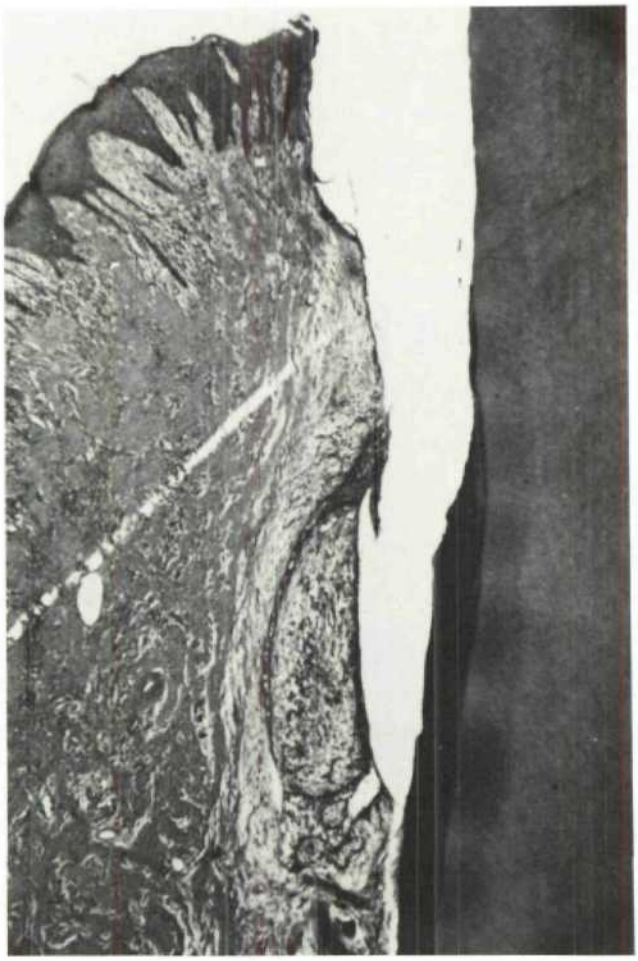

FIGURE 7. *Human specimen from Group C removed 16 weeks after surgery. In this specimen, attempts were made to completely remove the cementum. Subsequent to root planing, citric acid was applied to the root surface (H & E. Original magnification × 10). Complete cemental removal was accomplished in the incisal third of this root specimen. Here too, no evidence of cementogenesis is seen. However, a rather long epithelial-tooth interface is present.*

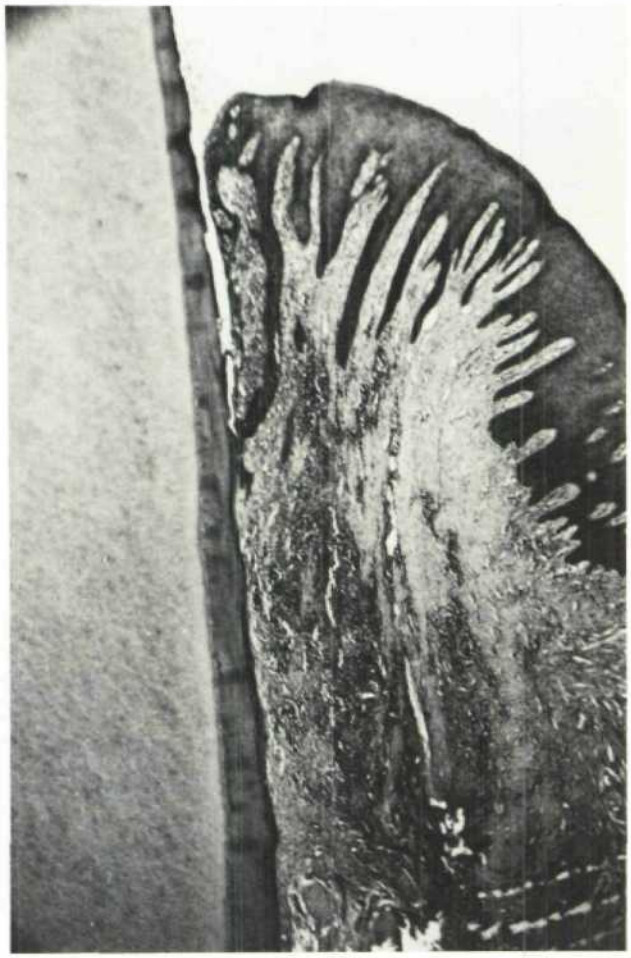

FIGURE 8. *Human specimen from Group D removed 16 weeks after surgery. In this specimen, saline was applied to the root surface after surgical preparation (H & E. Original magnification × 10). Note the reformation of a pocket. No evidence of cementogenesis was seen at this root surface.*

SUMMARY AND CONCLUSIONS

Seven blocks containing one tooth each were obtained from two patients undergoing treatment for periodontitis. Sixteen weeks prior to block removal, these sites were treated by periodontal flap surgery. In six teeth, a citric acid solution (pH = 1) was applied for 2 minutes to the planed root surfaces. The seventh tooth received identical periodontal therapy except that saline was applied to the planed root for 2 minutes.

At the end of the experimental period, the blocks were removed and prepared for histologic study. In five out of six citric acid treated specimens, no evidence was observed suggesting that citric acid applications either initiated or accelerated cementogenesis or functional connective tissue attachments at root surfaces previously exposed to periodontal pockets. In one specimen, repair cementum was seen crestally to the alveolar margin. This cementum was deposited on the surface of acellular cementum. Unfortunately, however, the specimen does not allow us to date the deposition of the repair cementum. It thus remains questionable whether

this cemental deposition is indeed a repair response to the specific treatment performed.

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Letter to the Editors:

In the November 1976 issue of the Journal I was impressed with the article by Lavine, Page and Padgett on the Chediak-Higashi Syndrome. Their Plate II, Figures G and H, is very similar to the clinical manifestations of periodontal disease found in patients with chronic neutropenia. In particular the cherry red cervical gingival lesion coupled with severe alveolar bone destruction, are the hallmarks of the periodontal aspects of chronic neutropenia. It is most interesting that humans with Chediak-Higashi Syndrome also suffer from a neutropenia. Therefore, it would be worthwhile if investigators would try to find out whether the neutrophils in patients with chronic neutropenia possess a similar defect as those in the Chediak-Higashi Syndrome. If this were so, we would finally have a mechanism explaining the unusual gingival appearance and the severe alveolar bone loss accompanying patients with chronic neutropenia. The same mechanism might be responsible for producing the periodontal destruction in both of these diseases.

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