Histological Evaluation of Human Intraosseous Healing Responses to the Placement of Tricalcium Phosphate Ceramic Implants*

I. Three to Eight Months

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EIGHT INTRABONY LESIONS IN FOUR PATIENTS were removed *en bloc* 3 to 8 months after periodontal flap debridement. At the time of debridement, the position of the gingival margin and the most apically located calculus were notched to serve as reference points. All lesions received "Synthograft†" implants and lesions healed uneventfully. Even though these were severely involved periodontal sites, clinical measurements at time of block removal demonstrated gingival recession (average = 2.9 mm) and a gain in clinical closure (average = 2.6 mm).

Histologically, graft particles were present in each specimen. They were walled off by collagen and did not appear to enhance new attachment nor did they induce an inflammatory infiltrate. Thus, they seemed to act as nonirritating fillers. Microscopically, closure of the lesions demonstrated repair with limited evidence of new connective tissue attachment. Histologic expression of the clinical gain in closure was the result of closure by long epithelial adhesion (long junctional epithelium) and possible linkage of dentinal collagen with gingival fibers at areas of dentinal resorption. These variations in closure were often seen within the same clinical site.

A current review of histologic evidence for new attachment in human intrabony pockets indicated that the presence of bone grafts seemed to enhance new attachment potential. Based on findings reported in 123 human specimens, the authors concluded that (1) new attachment is possible on root surfaces denuded by periodontal disease and (2) new attachment is more likely to occur when various grafted materials are used than in nongrafted sites, particularly if root planing is performed. There is presumptive evidence that bone grafted materials enhance osteogenesis and cementogenesis.[1] The recent introduction of ceramic implants mandates similar histologic study of human periodontal repair sequences in the presence of such implants. Clinically, a comparison between healing responses of intrabony lesions treated by implantation of Durapatite* ceramic and debridement versus debridement alone showed Durapatite*-treated sites to be stable over a 3-year period, while areas treated with debridement alone regressed.[2] On the other hand, histologic evaluation of ceramic implants, thus far, did not indicate enhanced osteogenesis, cementogenesis and new attachment in the presence of these implants.[3-5] This may be due, in part, to the lack of stimulating proteins found in mineralized tissues which are not present in ceramic implants.[6,7]

In view of the limited number of human block sections depicting repair of intraosseous lesions with tricalcium phosphate implants, the periodontal healing responses of eight lesions will be described both clinically and histologically over an 8-month period.

MATERIALS AND METHODS

Eight periodontal sites in four patients, who volunteered for this evaluation, were selected for histologic study of the effects of a tricalcium phosphate ceramic† implant in the repair of intraosseous lesions (Table 1).

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† The graft material is marketed as "Synthograft" by Johnson and Johnson Dental Products Co. East Windsor, NJ 08520.

Table 1
Presurgical and Postsurgical Clinical Findings at Implant Sites

Patient	Age (yrs)	Site	Initial pocket depth (mm)	Mobility	Initial osseous depth (mm)	Observation period (months)	Postoperative pocket depth (mm)	Postoperative gingival recession (mm)	Gain in closure (mm)
P	58	#4 d	8.5	III	3.5	8	2.9	3.1	2.5
		#4 L	9.5		3.0		4.5	2.0	3.0
		#3 m	10.2		3.9		4.6	2.5	3.1
B	45	#7 m	9.5	II	3.2	8	3.0	4.5	2.0
		#8 d	10.4		3.3		2.8	4.3	3.3
M	55	#21 m	7.1	II	5.6	3	3.7	0.8	2.6
W	48	#13 d	9.9	II	3.7	8	4.5	3.0	2.4
		#14 m	9.8		3.6		4.4	3.1	2.2

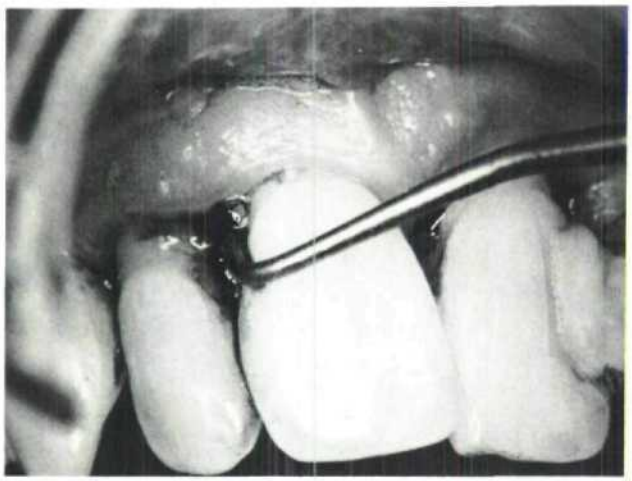

Figure 1. *Preoperative clinical photograph of site at Tooth #7 of Patient B.*

Figure 2. *Preoperative radiogram of site shown in Figure 1.*

Each patient had advanced periodontitis and no contributory medical history. The teeth to be removed *en bloc* were considered to have a hopeless prognosis by the dental treatment team. Teeth at the involved sites received a notch at the gingival margin which served as a reference point for pocket measurements to 0.1 mm. The notch was created using a half round bur. After obtaining local anesthesia, a full-thickness mucoperiosteal flap was elevated. Prior to debridement, and again using a half-round bur, a notch was created through the most apical extension of calculus. The lesion was then thoroughly debrided. Following intramarrow penetration, the site was overfilled with the ceramic graft material. The flap wall was then positioned incisally and complete closure was attempted. Interrupted sutures (4.0) were used and a periodontal dressing was applied over the sutured site. All appropriate presurgical and postsurgical measurements and photographs were taken during this time. Ten days after graft placement, dressings and sutures were removed and the site cleansed. Patients received weekly professional plaque removal of the surgical site for the first 6 weeks and then once every 2 to 4 weeks until the block was removed. At time of block removal, appropriate clinical records, photographs and radiograms were taken and pocket depth, recession and clinical closure gain recorded, again using the gingival marginal notch as the fixed point of reference. Blocks were removed 3 to 8 months after graft placement (Table 1). No adverse reactions were noted during our observation time.

Upon removal, all specimens were decalcified and prepared for histologic study. Step serial, mesiodistally cut sections were prepared and selectively stained with hemotoxylin-eosin, Mallory-trichrome, and Van Kossa stains.

Clinical Observations. **(Table 1).** Preoperative pocket depth at the eight sites varied from 7.1 mm to 10.2 mm and intraosseous depth varied from 3.0 mm to 5.6 mm. The osseous configurations were essentially 1-to-2 wall lesions. Following treatment, the sites showed pocket depths varying from 2.8 mm to 4.6 mm. Gingival recession was seen at all sites and varied from 0.8 mm

to 4.5 mm. Clinical gain in closure varied from 2.0 mm to 3.1 mm (Figs. 1–6).

Histological Observations. Since all sites showed similar histologic responses, the healing pattern will be reported as a composite.

At the time of block removal, gingival recession apical to the marginal notch had occurred in all specimens. Closure was seen via a long junctional epithelium (JE), parallel connective tissue fiber adhesion and limited new attachment, as evidenced by cementogenesis and functionally inserted fibers at this site. Cementogenesis was confined to small distances incisal to the adjacent crest and did not seem to relate spatially to the presence of the implant particles. Implant particles were present within the vertical lesions and supracrestally in all specimens. All implant particles were surrounded by a connective tissue capsule. No significant inflammation was seen at these sites, nor was there any evidence of osteogenesis surrounding the implant particles. The alveolar crest demonstrated remodeling with limited osteogenesis and osteoclasis taking place at the crestal bony surfaces (Figs. 7–9).

Figure 3. *Site shown in Figure 1 debrided.*

Figure 4. *Site shown in Figure 1 filled with graft particles.*

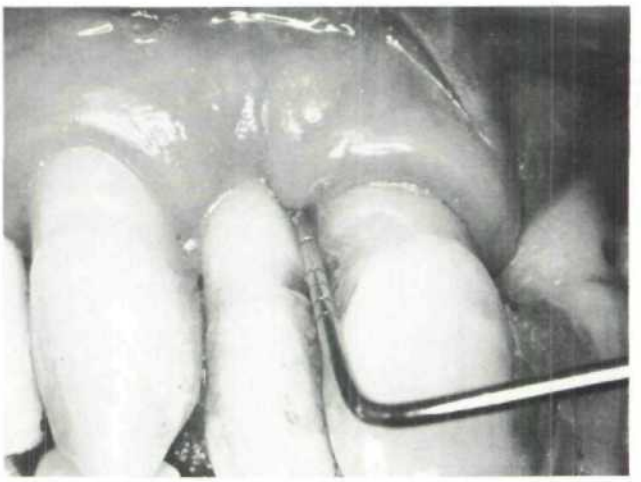

Figure 5. *Site shown in Figure 1, 8 months postsurgery.*

Figure 6. *Postoperative radiogram of site shown in Figure 1, 8 months postsurgery.*

Of particular interest were responses seen within the apical notch which identified the most apical deposition of root accretions within each site. The responses are particularly well seen in specimen mesial Tooth #7, Patient B and will be described in detail. In sections 34 and 35 of this block, cementogenesis is present at the most apical portion of the notch. Immediately incisal to that, right angle-oriented collagen fibers arise from the dentinal wall of the notch. Careful examination of the dentinal wall shows an undulent surface suggesting dentin resorption (Fig. 10). These fibers appear to link with fibers from the gingival connective tissue which had filled the space and stain positively for collagen with a Mallory trichrome stain (Fig. 11). Figures 12 to 14 show a serial section from the same site in which

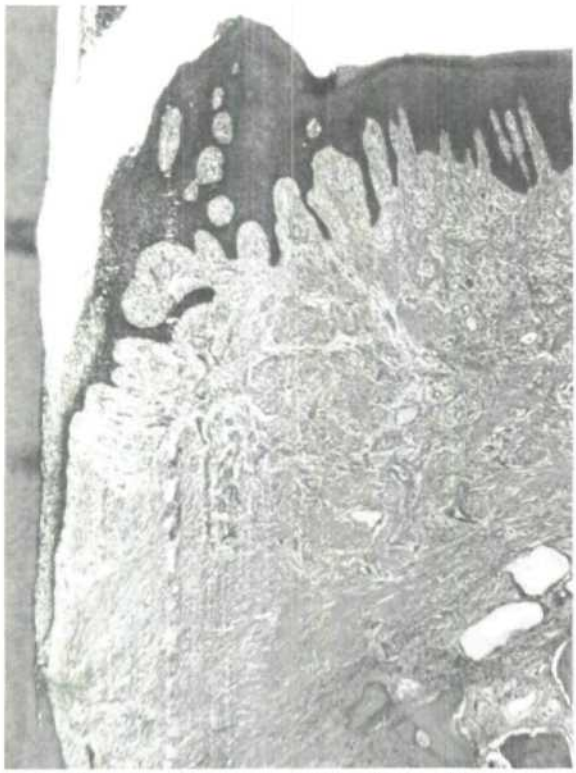

Figure 7. *Overview of site at tooth #4 in Patient P 8 months after graft placement (magnification ×10).*

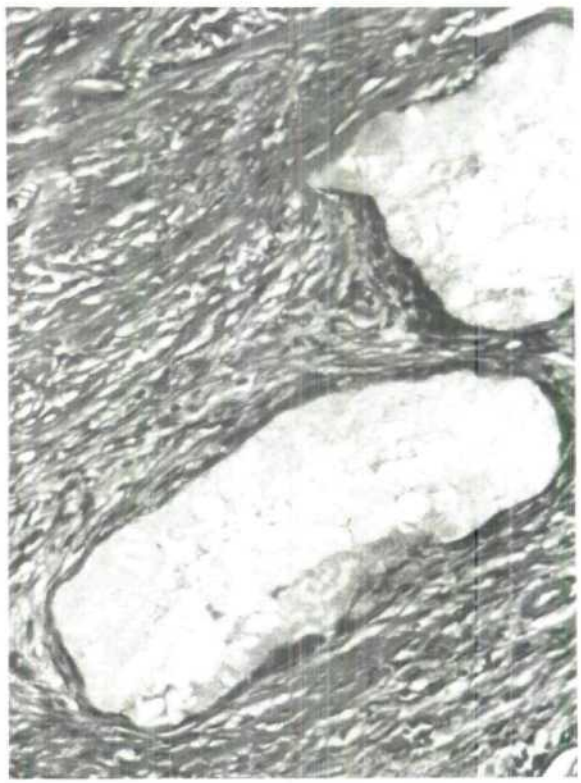

Figure 9. *Higher magnification of graft particles shown in Figure 7. Note collagen wall surrounding particles (resorbed due to histologic processing) and lack of inflammation. No evidence of osteogenesis is seen.*

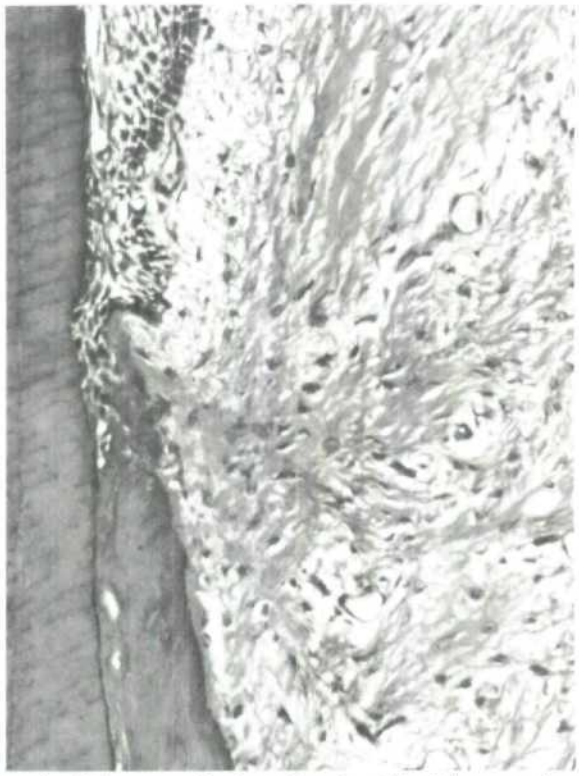

Figure 8. *Higher magnification of interface (Fig. 7) between the most apical portion of the JE and evidence of cementogenesis at the root surface (magnification ×64).*

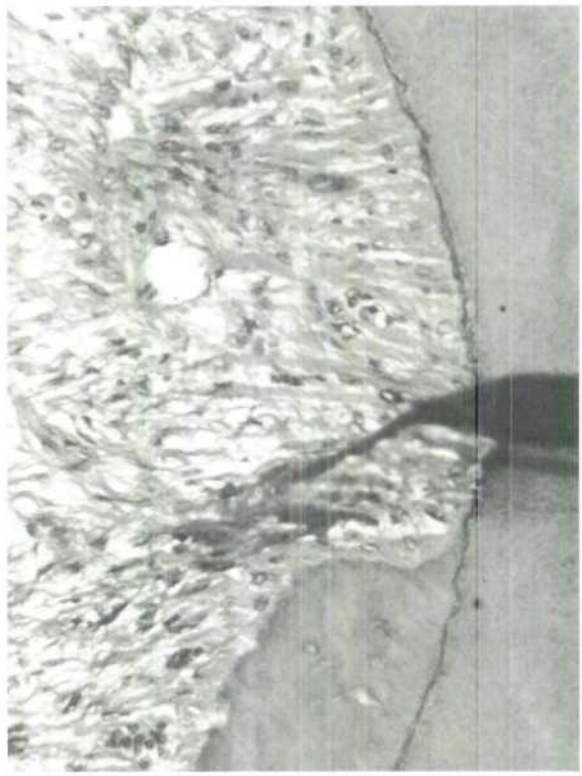

Figure 10. *Area of apical notch, (apical extent of calculus) at Tooth #7, Patient B 8 months after graft insertion. Note cementogenesis at apical portion of notch, incisal to which right angle oriented collagen fibrils appear to arise from resorbing dentinal wall. Note also evidence of dentinal surface resorption (magnification ×64).*

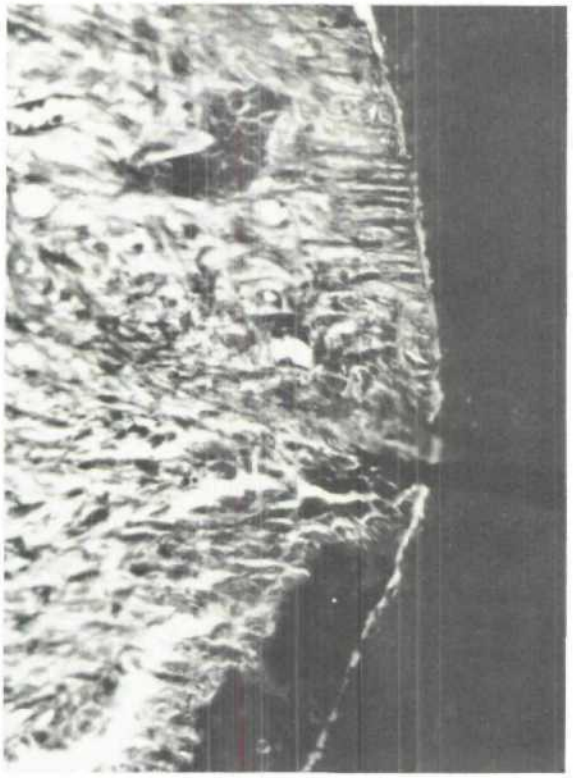

Figure 11. *Step serial section of specimen shown in Figure 10. Mallory trichrome stain. Fibrils arising from dentin stain positive for collagen (magnification ×64).*

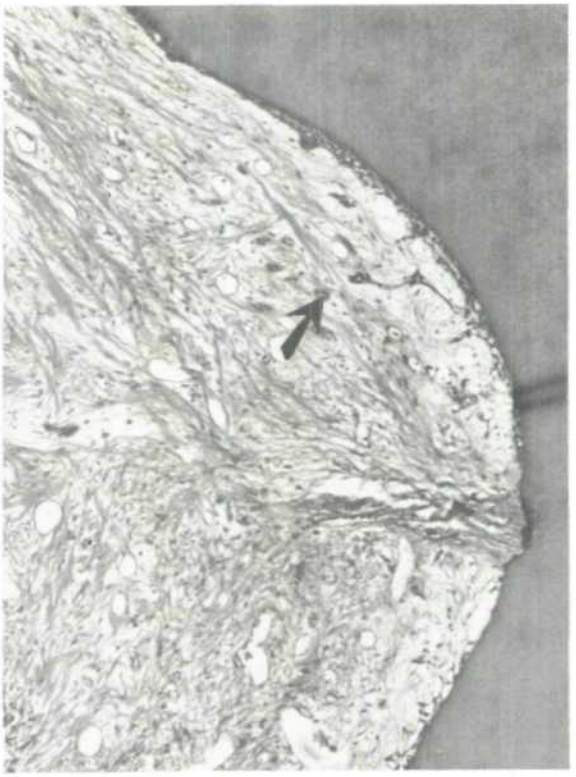

Figure 13. *Higher magnification of notch area shown in Figure 12. Note incisal portion of notch covered with epithelium (arrow). Epithelial migration ends at interface with right angle oriented collagen fibrils arising from cut dentin (magnification ×25).*

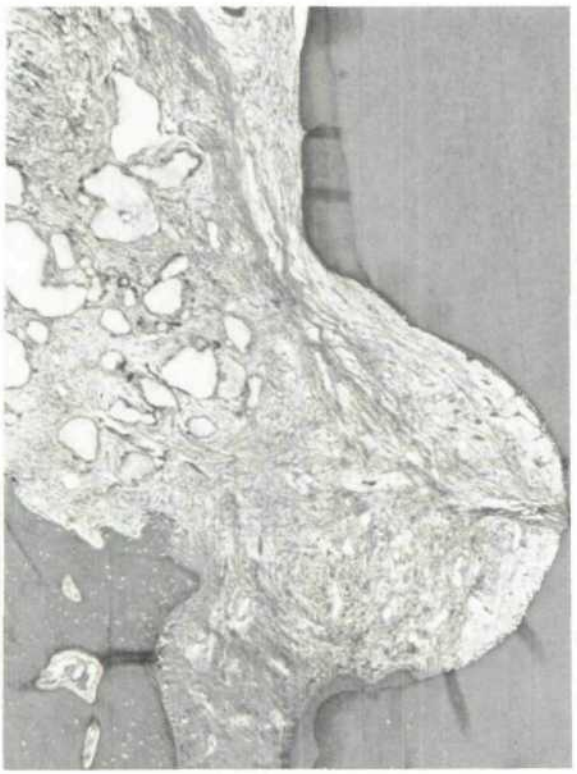

Figure 12. *Serial section from specimen shown in Figure 10. Overview section. Note apical portion of notch shows cementogenesis. Graft particles are also present (magnification ×10).*

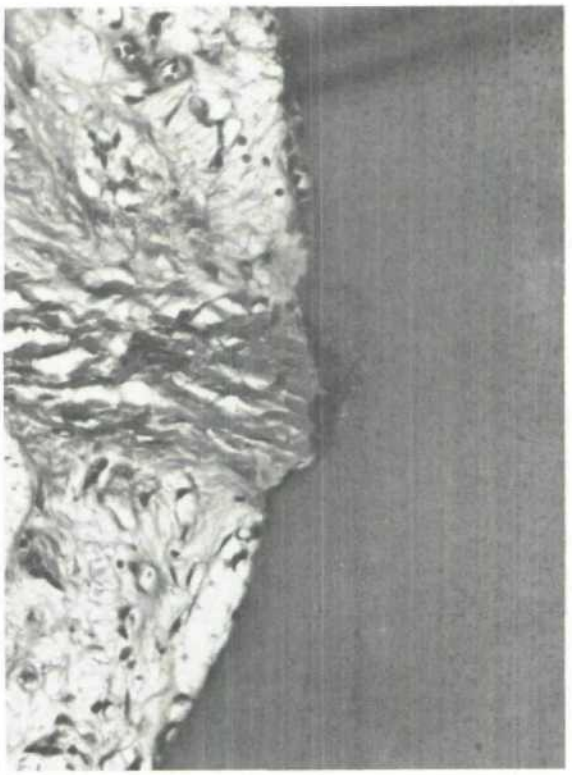

Figure 14. *Higher magnification of cut dentin surface in notch shown in Figure 13. Note resorption of dentin and presence of collagen fibrils arising from dentinal surface (magnification ×64).*

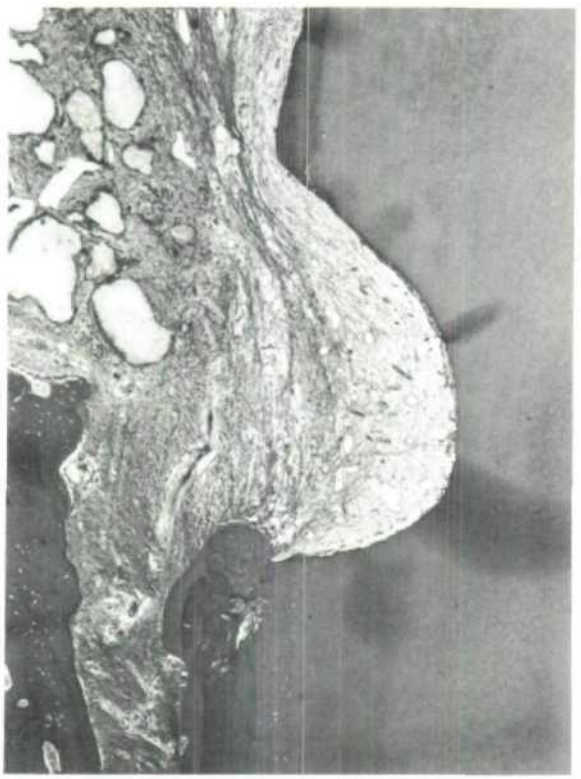

Figure 15. *Serial section from site shown in Figure 10. Note notch is lined with epithelium. (long JE) (magnification ×10).*

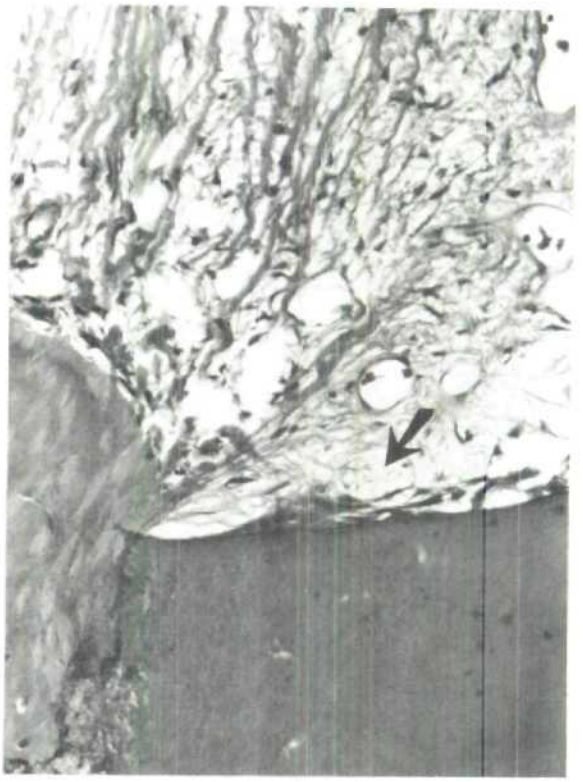

Figure 17. *Higher magnification of epithelial-cementum interface at apical portion of notch shown in Figure 15. Arrow points to epithelial cells (long JE) (magnification ×64).*

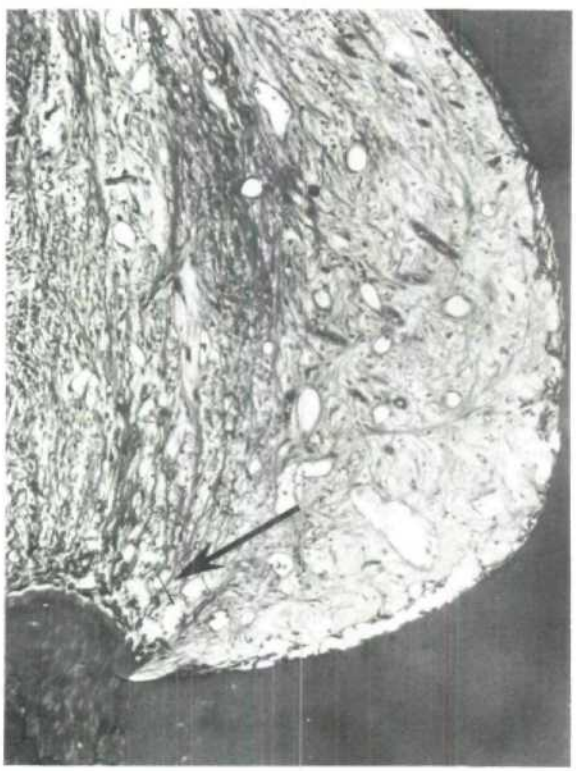

Figure 16. *Higher magnification of notch site shown in Figure 15. Note epithelial lining of entire notch surface except at the apical portion of the notch where cementogenesis is evident. (arrow) (magnification ×25).*

the incisal portion of the notch is covered by junctional epithelium. At about midpoint within the notch, fibrils arise from the resorbing dentinal surface which appear to link with repairing gingival collagen fibrils. Apical to this area, loose connective tissue abuts the dentinal wall except at the very apical edge where cementogenesis is again evident. Figures 15 to 17 show further serial sections of this site. Here the entire notch, except for its most apical edge, is covered by a very thin epithelial lining, a direct extension of the junctional epithelium into the notch. Graft particles are seen in this area but do not appear to be involved in the repair process.

The preceding series of histologic observations, at a notched site previously covered with calculus, demonstrate varying repair processes occurring within the same site after debridement of a human intrabony lesion. They vary from (1) epithelial adhesion of the previously exposed site to (2) partial blockage of epithelial migration by exposure of dentinal collagen which appears to link with gingival collagen to (3) limited new attachment as evidenced by cementogenesis and functionally inserted fibers into newly formed cementum. Furthermore, it should be noted that such repair sequences were seen as late as 8 months postsurgery and appeared to be part of an overall repair process. The implantation of ceramic particles was not connected with the above described repair process.

COMMENT

The healing processes of the intraosseous lesion described above are similar to those previously reported in human intrabony healing.[1] Even though clinical observations indicated that ceramic implants improve clinical closure,[2,8-10] the histologic evidence does not demonstrate that these implants enhance new attachment. Thus, the findings presented here are in concert with those reported by Baldock et al,[5] namely, the particles do not resorb quickly, do not appear to induce inflammation, but rather seem to represent an inert fill material which becomes well encapsulated by gingival connective tissue.

The present material is of further interest because it indicates a variety of closure mechanisms within the same site. Such responses were reported previously and consist of (1) closure by a long junctional epithelium, (2) possible linkage of gingival and dentinal collagen at sites of dentine demineralization and (3) new attachment.[11-15] Of particular interest were the findings of a thin junctional epithelium which had migrated laterally and apically to wall off the entire notch-surface from the gingival connective tissue in at least one section. No significant inflammation was seen in this site. These findings support the concept that the long junctional epithelium is able to act as an effective closure mechanism following the treatment of intrabony lesions[16-19] and is maintainable over many months.

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