HEALING SUBSEQUENT TO CIRCUMFERENTIAL SUPRACRESTAL FIBROTOMY IN HUMANS

J. Timothy Quinn, D.D.S.

This paper submitted in partial fulfillment of the requirements for a Certificate in Orthodontics, Oregon Health Sciences University.

June 1982

ACKNOWLEDGMENTS

I would like to thank Dr. Douglas Buck for his suggestions, editorial help and guidance; to Fran Miller of the Anatomy Department for her assistance and Diane Sullivan for typing and preparing the manuscript.

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INTRODUCTION

Orthodontic relapse following the correction of rotational tooth position is a commonly observed clinical phenomenon. Securing a stable tooth correction was focused on by several investigators, including Edwards.[1,2] His investigation elucidated the supracrestal fibers as responsible for rotational relapse and advocated severing these fibers subsequent to rotational tooth movement. Concern about this procedure is centered on whether in providing for a more stable tooth position, a concomitant result would be instigation of a process of compromised periodontal support. Therefore, evaluation of this procedure, circumferential supracrestal fibrotomy (C.S.F.), could be enhanced by microscopic scrutiny of the postsurgical sequelae. Although biologic consistency of the gingival repair phenomenon is well documented, a study of a simple incision of the PDL may detail some specific information on a postorthodontic procedure some may routinely perform. If supracrestal fibers are accountable for rotational relapse, then what is the histologic change of the fibers from the prefibromy state? Can we expect the connective tissue to regenerate its original architecture? And does the fibrotomy result in aggravating or instigating a pathological response of increased sulcular depth or loss or change of attachment.

This report will present an observation of the relatively short term gingival healing sequelae following C.S.F. and determine if a

typical histologic pattern is evident and if this pattern is consistent with gingival healing previously reported.[17,22,25,29,40] Also, the later stages of healing will be compared to the connective tissues' original structure to determine architectural alterations. Specifically, cell types and arrangement, fiber pattern and maturation and hard tissues response to C.S.F. will be explored.

REVIEW OF THE LITERATURE

Retention, after the active phase of orthodontic treatment, has long been recognized as a major problem. Hawley[3] in 1919, whether in hyperbole or not, stated that he would gladly give half his fee to anyone who would take his cases when finished, retain them and be responsible for them afterward.

Prevention of rotational relapse is a specific area of retention that orthodontists have addressed definitive procedures to overcome. Skogsburg[4] advocated fissuring the bone on both sides of the orthodontically corrected rotation to "neutralize the unfavorable forces within the tissue." He succeeded in preventing relapse by this method, whereas transection or even surgical removal of the supra-alveolar structures appeared to be insufficient. Hallet[38] saw benefit in immediate correction of single-rooted teeth. The procedure, immediate torsion, entailed twisting or luxating the tooth with forceps to correct irregularity of position. He reported relapse less than an orthodontically achieved correction but tooth vitality was an obvious problem. Reitan[5] reported that microscopically, fibers appeared "taut" after tooth rotation and gross gingiva was directionally deviated. He advanced the idea of overrotating the tooth and then some relaxation of the displaced supra-alveolar fibers would be permissable. He also depicted an age factor as something to take into account as well as

other local factors. This would explain his observation of a differential tendency to relapse in some persons more than others. It was also advocated by Reitan to correct rotations before the apical portion of the root is fully developed. Ramfjord and Ash[6] maintain that retention problems are considerably alleviated by properly equilibrated occlusion.

Edwards,[1,2] as well as others,[7,8,9] limited the causative factor of rotational relapse to the supracrestal fibers. Thompson and Boese,[9] in an animal study, stripped the attached tissue from the rotated teeth and in conjunction with a period of retention reported a reduction in relapse. The fibers of the periodontal ligament apical to the supra-alveolar fibers and adjacent to alveolar bone are discounted in the relapse phenomenon for the most part due to the plastic osseous tissue which remolds and allows the fibers to return to their original and stable relationship. The supracrestal fibers do not possess the plastic osseous tissue to eliminate their distortions after tooth movement. Using this concept Boese[7] points to two phases of orthodontic relapse. Prior to eight weeks after orthodontic rotation correction, relapse is caused by stretched principal fibers. This phase is terminated when alveolar bone is remodeled providing new attachments for the fibers. The second phase is due to the supra-alveolar fibers and will continue until the relapse is almost total since no cemental attachments of the fibers have been altered. Boese went on to elucidate the specific characteristics of the supracrestal tissue. This area of tissue showed an increase in quantity and definition of an elastic type of fiber, oxytalan.[7,11] These fibers would exert an elastic force by stretching

rather than lengthening on an orthodontically rotated tooth, resulting in relapse.

Brain[10] and Edwards[2] demonstrated greater tooth stability of orthodontically rotated teeth after experimental removal of the supracrestal fibers. Edwards'[1,2] procedure, circumferential supracrestal fibrotomy, attains a desired reduction or elimination of rotational relapse by the process of creating a gingival wound. The healing of that wound would be of considerable concern to those involved with such a procedure.

Ross[12] states that the process of wound repair in man differs little from one kind of tissue to another and is generally independent of the form of injury. Melcher[13] points out, however, that the periodontium presents a unique situation with its relationships between the epithelia and the hard and soft connective tissues. These relationships would result in a difference in some aspects of the organization and synchronization of wound healing compared to other areas of the body. These differences are in respect to detail only and basic principles of tissue repair remain constant. The literature is replete, since Arey[14] first described the basic tissue response (1936), with information directed mainly in the area of cutaneous lesion repair. The information concerning that aspect of the periodontium is more limited and a select group of investigators, including Melcher, Ramfjord, Listgarten, and Stahl are largely responsible. The investigations of healing of simple incised wounds of the gingiva are few. Mittleman[15] et al described the healing of a linear incised wound of marginal gingiva in the attached tissue of humans. He described the healing in three phases as has been described in cutaneous repair[12] but the timing

was of notable difference. The gingiva's healing rate was reported significantly more rapid. Winters[16] attributes this to healing taking place in a wet environment which results in a more superficial epithelial migration and thus more rapid process.

The phases and timing of tissue repair according to Mittleman[15] consists in the initial phase of bleeding and clotting (0 → 9th hr.), anchoring of the clot and inflammation (6 → 48th hr.) and then resolution of the inflammation (18 → 48 hr.). The second phase is made up of proliferation and bridging of the epithelia (27 → 32 hr.), capillary budding with perivascular proliferation (21 → 72 hr.) and then production of collagen (28 → 72 hr.). The final phase involves keratinization and maturation of epithium (42 → 72) and progressive proliferation and partial maturation of connective tissue (28 → 72 hr.).

The first phase is concerned chiefly with the cell population involved in inflammation. The fibrin clot achieves an emergency seal and also elicits invasion of polymorphonuclear leukocyte. Engler et al[17,19] noted the presence of acute inflammation, characterized by emigration of PMN's within two hours following gingivectomy in monkeys. Cutaneous lesions did not present this cell type until nearly the sixth hour. Neutrophils function as a local barrier to bacterial invasion and their phagocytic nature is well recognized.[18] The leukocytes migrate out of the vessels at the site of injury and form a band between the surface of the wound and the connective tissue to protect against invading organisms. Ramfjord[19] considers this "bacteria proof seal" the most critical phase of his healing experiment with monkey gingivectomies. Mittleman[15] reported the presence of mild inflammation up until the 18th

hour in his linear incision wound investigation and thereafter inflammatory cells decreased and resolution continued up till the 40th hour. The almost complete disappearance of PMN's was noted as soon as the wound surface is protected by epithelium.[19] Ramfjord reported the change from acute to chronic inflammatory cell types occurring after 48 hours. This next generation of inflammatory cells function in phagocytosis and digestion of bacteria and debris. The lymphocytes, with a much longer life span than the PMN's, were evidenced for several weeks after the healing of the wound.[19]

It was noted in the second phase of healing that the epithelial cells migrate by sliding over the surface of the clot from the cut edge.[15] In human gingiva with an incision-type wound, the epithelial cells had joined across the wound by 24 hours. At this time the basal layer was not well defined and not till the 36th hour was the epithelium reported to be eight layers thick.[15] Others[20,12] have reported epithium migrating deep or through the clot but one constant finding is that the continuity of the epithelia is established prior to that of the connective tissue.[19,15]

In linear incisive wounds mobilization and migration of adjacent preexisting epithelial cells take place without the necessity of new cells through mitosis.[21] Melcher[13] explained the mobilization of epithelial cells occurring when contact between like cells is broken, as when an incision is made. Then through the phenomenon of contact guidance the cells move across the surface of the substrate in the newly formed space. Fibrin strands within the clot may provide pathways for the cells to be directed.

It was explained[15] that during the process of epithelial bridging concomitant activity of capillary budding and perivascular cell proliferation is evidenced. The clot at this time is still unorganized but fibroblasts were observed projecting into the clot setting up for the final phase of tissue repair.

The third phase of healing deals principally with progressive proliferation and partial maturation of the connective tissue. This process in incised human gingiva was not observed until 48 hours and fibrogenesis reported well advanced, although not complete, at the 72-hour period.[15] Melcher[13] described fibroblasts moving from between the collagen bundles into the clot. However, the collagen fiber origin in wound repair is uncertain. The collagen molecular units which the fibroblasts secrete have been demonstrated to become mature collagen fibers.[23] It has also been postulated[22] that preexisting collagen may be depolymerized to molecular units or subunits which reaggragate to contribute to newly forming connective tissue. The injured fibers may only serve as a guiding framework for orientation of repairing fibers.

The fibers, as they heal, run in all directions at first.[15] Remodeling then entails reorientating the first deposited fibers and then increasing their number. Connective tissue maturation appears difficult to define. In experiments that were reported to mimic an incisive wound of the periodontal ligament, Loe and Waerhaug[39] note that the PDL fibers were still partly disorganized 80 days postoperatively. They also reported that the fibers were eventually functionally orientated but initially the fiber bundles were observed parallel to the root surface, for reasons unknown. Giblin et al[24] in incised dog gingiva observed

in the 60-day specimens that collagen fibers were dense and well oriented to the adjacent tissue and the cellular fiber ratio was indistinguishable from the adjacent tissue. Stahl[25] summarized a variety of gingival surgical procedures in which he noted a range of between 10 and 30 days postsurgery for connective tissue maturation to occur. Nasjleti[28] didn't note maturation and orientation of the PDL fibers until four months after his reimplantation study. Maturation at the electron microscopic level is characterized by cross-linking of the collagen fibril bundles.

Researchers have observed a relationship between the formation of collagen and deposition of new cementum. Surgical wounds created in the periodontium have shown connective tissue healing with the formation of new cementum.[13,17,28] Andreasen[26] found in humans that cementum formation seldomly would take place if the periodontal ligament fiber repair is prevented. He later found in rats that deposition of new cementum was always associated with insertion of mature collagen fibers.[27] Nasjleti's[28] study noted that cementoblasts were not observed along the root surface postsurgically and could not be demonstrated until the repair process was well advanced. Cementum formation thus appears to follow repair of PDL fibers and could possibly be dependent upon that process.

Staffileno[31] demonstrated in dogs that the alveolar crest, although not directly traumatized, resorbed in response to incising the overlying soft tissue. This occurred as early as two days postoperatively. Others reported bone resorption occurring within the first week.[30] Resorption then gives way to osteogenesis, and former height and contour

are achieved.[13] Cementum, likewise, demonstrates a localized resorptive response to soft tissue surgical wounding.[13,20]

The nature of this observed response to cementum and bone to wounding of the overlying soft tissue is obscure. Melcher[13] describes the cells that are responsible for the initial destruction and later repair as being derived from a progenitor pool of cells in the periodontal ligament. Humoral factors, not completely understood presently, may elicit differentiation into the appropriate cell.[32]

Of practical consideration is the healing of the epithelial attachment. Reattachment after wounding was confirmed among others by Edwards[2] but the critical question concerned whether the attachment, as a consequence of the healing, moves apically.

Several investigators[33,34,35,36] reported through ultrastructural evaluation that the mode of epithelial adherence to tooth surface is similar after wounding to that seen in untreated controls. Taylor and Campbell[37] described in marmosets after the attached gingival epithelium was separated from the enamel that reattachment had reached the level of the best controlled unoperated gingiva. Electron micrographs depicted the epithelium attached to the enamel cuticle by well formed hemidesmosomes. The attachment was near the cemento-enamel junction and appeared confined solely to the proliferating basal cell layer of the epithelium. Stahl et al[23] reported that the epithelial cuff often migrates apically since the tooth surface alone does not present sufficient contact inhibition. Sufficient resistance to migration is created by connective tissue fiber remnants left on the tooth surface. Staffileno[29] in dogs found a complete functional repair of the dento-gingival

junction, but the epithelial cuff was slightly apical to its preoperative position and the sulcus was also observed to be more shallow. At present no definite conclusion can be drawn on the effect of wounding on the epithelial attachment.

Much investigation has taken place on the various host factors and how they affect healing. Research on nutrition, aging, and endocrine secretions are among these. At present the studies are inconclusive. The affects of oral hygiene on healing is likewise currently undetermined. Engler et al[17] emphasized the need for meticulous oral hygiene if there was to be complete healing. In their study with monkeys, it was reported that if bacterial plaques were established on the tooth surfaces during healing an adequate epithelial attachment could not be established. Kohler and Ramfjord[40] in humans, however, found that poor oral hygiene and its associated areas of inflammation did not seem to influence the healing process. Mittleman[15] claimed a bacteriostatic action from the saliva that inhibits the production of inflammation due to its buffering action. It will be for future research to determine the affect of various factors involved in the repair process.

MATERIALS AND METHODS

Fifteen patients who required extractions of maxillary first bicuspids for orthodontic treatment agreed to participate in this study. Participants ranged in age from 11 to 15 years and health histories were nonremarkable. Details and possible consequences were explained. Specific oral hygiene instructions were intentionally not given.

The circumferential supracrestal fibrotomy procedure as described by Edwards[2] was performed on the maxillary first premolars. This procedure involves directing a #11 scapel into the gingival sulcus encircling the tooth. The apical extent of the incision penetrates the PDL to just below the alveolar crest.

In this study the procedure was limited to the labial surface of the bicuspids following injection of 1-100,000 lidocaine. The contralateral tooth's fibers were subsequently severed in like manner at a later predetermined date. After healing had taken place for a specified period, both maxillary premolars were extracted concurrently. The extraction procedure involved placing vertical incisions mesial and distal of the buccal surface. These incisions extended 5 mm apically from the gingival margin and were connected by a horizontal cut. A mallet, chisel and fissure burr were utilized to free the experimental specimen from adjoining tissue. The study teeth were then palatally

elevated and extracted, followed by suturing with 4-0 B.S.S. Little discomfort was reported during or postsurgery.

A problem became apparent of detachment of the soft tissue and bone from tooth during removal. This was most apparent in the shorter healing times. Therefore, a modification of the C.S.F. was introduced on the remaining five cases. The buccal area of fibrotomy was restricted to approximately two millimeters. A vertical notch was placed on the buccal surface of enamel just occlusal to the gingival margin of the surgical area. A modified Orban knife was inserted opposite the enamel marker. Restricting the surgical area resulted in amelioration of the detachment occurrence.

Once obtained, the specimens were fixed in 4% glutaraldehyde for approximately 24 hours. This fixative permits the same specimen to be used in either electron or light microscopy.

Subsequent to fixation the specimens were decalcified in EDTA for approximately six months. To expedite this process the teeth were sectioned in half mesiodistally and a large segment of root removed. Demineralization was monitored by radiographs. Light microscopic specimens were additionally placed in 10% formalin for 10 days. This aids visualization of certain stains (ex. H. & E).

Seven micron sections were removed buccolingually and slides prepared. The stains were incorporated using the standard procedures. These included hematoxylin and eosin, Verhoeff elastic fiber stain, Wilder's reticulin stain, Mallory's connective tissue stain, Aldehyde Fuschin oxytalin fiber stain, and Van Gieson connective tissue stain.

FINDINGS

Control Specimens

Periodontal tissues of control specimens showed normal arrangement in agreement with reports on young healthy human subjects (see Fig. 1). The bottom of the junctional epithelium was located very close to the cemento-enamel junction. A mild inflammatory infiltrate was observed in the connective tissues adjacent to the crevice. Small localized areas of osteoclastic activity at the alveolar crest were evident in some sections.

Day 1

At 24 hours histologic C.S.F. sections revealed incised periodontal fibers connected by a clot. The clot extended from the base of the gingival sulcus apically within the PDL to the level just beneath the alveolar crest. Pieces of clot and cell debris were evident in the crevice, however, the bulk of such tissue in that area was probably lost during tissue preparation. The clot contained disorganized and divided collagen fiber bundles and was infiltrated with polymorpholeukocytes and erythrocytes. The PMN distribution was not of the "polyband" arrangement described by Ramfjord but certain areas of circumscribed dense acute inflammatory cells were present (see Fig. 2). The cemental Sharpey's fibers were still intact and directed into the clot. Some specimens, however, failed to demonstrate these fibers at the CEJ level.

The epithelial cells bordering the incision were ragged and no crevicular attachment was present. No specimen presented epithelial remnants still attached to the tooth at this time interval.

Inflammatory cells, mainly composed of PMN's spread from the free gingiva apically to the alveolar crest. Acute inflammatory cells appeared concentrated in the CEJ region and capping the periodontal ligament side of the alveolar bone. PMN's were observed marginating capillary spaces and round cells were evident in larger vessel lumina.

Cementoblasts appeared to lose their normal arrangement along the cementoid of the incision but no resorption of cementum was evidenced.

Mallory's connective tissue stain depicted the extent of collagen fiber destruction. Disrupted fibers of the dentogingival, transeptal and horizontal dentogingival groups were obvious and interspersed with areas of extravasated blood cells. Fibrin appeared to be the other notable clot constituent, evidenced in the trichrome sections.

The alveolar bone was unremarkable from the control specimen with the exception that adjacent to the bone increased cellular activity was evident. Connective tissue on the labial side of alveolar bone demonstrated an arrangement characteristic of the control sections.

Day 4

At day 4 specimens demonstrated some reorganization of collagen fibers. Fiber bundles of the gingival and transeptal ligaments acquired patterns more typical of the control fiber patterns but were still loosely organized. Alveolar dental ligament reorganization was not

as evident; however, artifacts were conspicuous in the limited specimens in this area. Extravasated blood was still prominent but clot islands appeared more localized than the generalized destruction of day 1 specimens.

Endothelial cells appeared to be proliferating to form capillary budding, another indication of connective tissue organization. An impression of reduced perivascular activity was noted. The influx of PMN's in day 1 was not conspicuous at this time interval. Round cells were a typical observation, concentrated in areas approximating the clot islands and in the connective tissue adjoining the crevice. Other localized areas were noted where a high ratio of fibroblast to fiber content were evident.

The cementoblast arrangement viewed in the control was still absent. No cementum resorption was observed.

Epithelium was still separated from the tooth. In the apical region of what was previously the epithelial attachment small ridges of epithelium projected into the connective tissue, a finding not evident in the control sections.

Alveolar bone resembled control. Periosteal activity was increased over day 1.

Day 7

Adequate sections were not available.

Day 10

Day 10 specimens presented considerable indication of healing

progressing over earlier sections. The crevicular epithelium was now attached at or near the cemento-enamel junction (see Fig. 3). Epithelial cell layers resembled the control sections in appearance and number. No epithelial ridge projections in the attachment zone were evident. Due to decalcification, epithelium and its relationship to enamel could not be determined and study design was not established to evaluate attachment level. In a 10-day section as well as in one control specimen, cementum was observed overlapping what was previously enamel. This is a normal variation of the CEJ.[41]

Distinct gingival fiber patterns were now identified. Many young, dark staining fibroblasts were observed within collagen fiber bundles. Younger fiber bundles stained lighter and ran parallel to the tooth surface as compared to the older, darker fibers which appeared more perpendicular to the long axis.

Collagen bundle insertion into the alveolar bone was no longer observed and the immediate zone adjacent to the bone presented heavy cell concentrations. The tooth side of alveolar crest manifested a scalloped pattern, suggestive of resorption.

The cementum was not noteworthy. Cementoblast arrangement seen in control specimens was not observable at 10 days.

Day 14

Day 14 specimens presented a general refinement in the healing sequelae observed in the sections four days prior. The dentogingival junction appears to have a complete functional repair.

The connective tissue attachment to tooth also appeared normal. Fibers were darker staining and possessed a wavy characteristic similar to control fibers. Fiber direction was more perpendicular or oblique to the long axis of tooth versus the vertical pattern seen earlier. However, areas were evident where reorganization was not as complete. These were characterized by regions of which a high ratio of fibroblast to fiber content were present. Tissue appeared more cellular, less dense, less organized in these regions in the trichrome connective tissue sections. Round cell infiltrate was noteworthy adjacent to the crevice and capping the alveolar crest.

The first instance of cementum resorption was apparent at this time. One section demonstrated, coronal to the alveolar crest, extensive cementum loss. Other specimens in this time interval failed to exhibit this phenomenum.

Alveolar bone presented considerable resorption lacunae (see Fig. 4). Osteoclasts were generally inconspicuous with the exception of one section. Round cells appeared concentrated in the connective tissue near alveolar resorption regions.

Day 21

Collagen fiber bundles were now orientated typical of control sections. Wavy, dark staining fibers indicated further maturation from the earlier sections. However, unlike the controls, island were still present in which there existed a high ratio of fibroblasts to fibers.

The most dramatic finding at this time period was the considerable cemental resorption located between the CEJ and alveolar crest. Cementoclasts were observed within the resorptive lacunae. Circumscribed areas of chronic inflammatory cells were present, some opposite these areas of resorption (see Fig. 5).

Osteoclastic activity was evident on the periodontal ligament aspect of alveolar crest. Howship's lacunae were responsible for the scalloped appearance of bone. Osteoclasts were sparingly apparent.

Day 28

Day 28 specimens bore subtle differences from the 21-day sections. Fibroblasts appeared mature and their ratio to fibers appeared more in line with the control sections. Collagen bundles likewise were orientated similar to the unoperated specimens (see Fig. 6). Cemental resorption was evident in the PDL only in a couple specimens. Osteoclastic activity appeared consistent with the 21-day sections. The extent of any osteoblastic activity was difficult to determine.

Post 28 Day

Areas coronal to the alveolar crest appeared undistinguishable from the controls.

Cementum resorption was evident in some sections at the 90-day period. Thereafter, no cementoclastic activity was observed. Osteoclastic activity was still noteworthy at 90 days. At 120 days bone resorption was still present but at a much reduced level.

DISCUSSION

This study has presented the short-term healing sequalae following circumferential supracrestal fibrotomy. Subjects were representative of those who would be likely candidates for such a procedure. These participants would be expected to heal rather rapidly being young, healthy, normal and well nourished. Variables would likewise be expected given the limited number of specimens and factors such as female cyclic variations. This study was hoped to corroborate the findings of previous investigators who delineated the healing response.

Epithelial Tissues

Much of the concern directed at the CSF procedure centered on what deleterious effects it may have on the epithelial attachment. Is the increase in tooth stability worth the possible initiation of a periodontally harmful response, specifically, deepening of the gingival sulcus? This report can only give limited insight into this question. The apical extent of the epithelial attachment did not deviate from the control following incision and subsequent healing. The apical end of the epithelial cuff reattached at the cemento-enamel junction. Orban's[43] describes this state of the dentogingival junction as stage 1 in passive exposure. The findings of this study appear to indicate that the C.S.F. procedure did not accelerate the passive eruption to a later stage where the apical epithelial attachment is secured to the cementum (stage 2).

Once secured at the CEJ, the apical extent of epithelium is reported to migrate coronally.[37] Sharpey's fibers embedded along the cementum may act as a barrier to converse migration.

Listgarden[33] reported that the initial epithelial-dental union takes place at the apical edge of the epithelial cuff where basal cells were closest to the tooth. This union was first noted at the 10-day period in this study. Limited specimens in the early healing periods precluded this report from gaining a comprehensive grasp of the timing of this phenomenon. Table I depicts the number of specimens available for inspection in each time interval. What the table does not indicate is that problems in experimental management resulted in tissue tearing away from the tooth in some early time intervals which further decreased the specimens available for inspection.

Decalcification of enamel during specimen preparation precluded evaluation of the change in sulcular depth following the C.S.F. Investigators have reported that following surgical removal of the epithelial attachment the mode of reattachment remains unchanged from the original organic union.[33,35] Microscopic studies of ground sections of frozen specimens where enamel and soft tissues are retained in their normal relation would be useful in these areas.

Connective Tissues

The connective tissues responded to the C.S.F. procedure predictably with inflammation. It has been observed that the connective tissue routinely contains a cell population characteristic of a mild chronic

inflammatory state in the area adjoining the depth of the gingival sulcus. Bacterial plaque occurrence is generally universal to some degree in the sulcus and inflammatory cells are believed to be a response to its presence.

At 24 hours a large influx of acute inflammatory cells were observed. This early response had changed to a more chronic phase of inflammation by day 4. The exact division could not be determined between these phases even with additional time interval specimens owing to healing variation and the gradual process of change. Reports[19] have placed a round cell predominance after 24 to 48 hours.

Inflammatory cells demonstrated concentrated areas in the connective tissue adjoining the gingival crevice. After day 10 areas of concentrated inflammation also were noted capping the alveolar crest. The degree of inflammation as measured by the number of PMN's and round cells was variable. Round cells, as stated earlier, were present in all sections but the major inflammatory response comprised the first 21 days.

Proliferative activity in the connective tissue appeared to occur early in the healing process. Young, dark staining fibroblasts appeared most prominent in the 10-day specimen and thereafter were decreasingly evident.

The collagen fiber destruction began to regenerate subsequent to epithelialization. Adjacent to the incision, fibers were reorienting to definite patterns at 10 days. Factors involved in directing the various groups of fiber bundles are not presently known but functional tension is assumed to play a part. The genetic factor is also believed

to contribute to this phenomenon. When comparing the regeneration of the various fiber bundles, no difference could be ascertained. Vascularization is not significantly different to the various fiber regions which may account for the similarity in healing. By 28 days collagen fiber regeneration appeared considerable. This appeared to concur with other reports of fiber regeneration taking place within a 30-day period.

Maturation of fibers could not be reliably identified and chronicled. What could be observed are fibers assuming the orientation patterns identified in control sections. Fiber waviness and dark eosinic staining was taken as signals of fiber reorganization. Fiber stability and insolubility, characteristic of maturation, involves covalent bonding between neighboring protein chains. Some reports indicated maturation of fibers not being complete until six months postsurgically. Electron micrographs could provide some insights.

Bone

Bone although not directly traumatized, responded with resorption subsequent to the C.S.F. procedure. Although some minor osteoclastic activity appeared even in the control sections, the first evidence of significant resorption was noted at 10 days. Other reports[30,31] noted resorption taking place within the first week of healing. Adequate sections were not available to determine bone activity between the four-day and 10-day interval. Resorption appeared to give way to osteogenesis after 21 days, although osteoclastic activity was still noticeable at 120 days.

Cementum

Cementoblasts disappeared subsequent to the C.S.F. incision all along the root surface. Other periodontal surgical procedures had similar reports.[19,41,42] These cells did not reappear until healing was well advanced. However, very early in healing, fibers were evidenced attached to the root surface. Since collagen fibers in the PDL regenerated prior to cementum and cementoblast formation, cementoblasts appear to only play a secondary role in fiber attachment.

Cementum resorption was noted spanning from the 14-day section until 90 days. This was explained as part of the inflammatory response in the healing sequalae and not a direct traumatic result of the scapel since cementoclasts were evident in the resorptive lacunae.

This study's intent was not to reduce the various stages of healing to a sequential outline but rather a morphologic description of what happened to a limited number of specimens during different time intervals after the C.S.F.

SUMMARY AND CONCLUSION

The purpose of this study was to gain information about the healing processes after circumferential supracrestal fibrotomy through the use of histological evaluation. Fifteen patients previously selected for orthodontic treatment were used in the study. The circumferential supracrestal fibrotomy procedure was performed on the maxillary first premolars, and the teeth with adjoining tissue were then extracted at various healing intervals from one day through 121. Tissues were then prepared for standard histologic evaluation.

The following conclusions may be drawn:

1. The basic healing response concurred with the broad scheme previously outlined for gingival healing and appeared to follow a characteristic pattern over time.
2. A new cemento-enamel junction attachment was restored after circumferential supracrestal fibrotomy.
3. Continuity of the periodontal membrane and supracrestal connective tissue was reestablished and fiber orientation was recognized by 28 days postsurgery.
4. Cementum resorption was a variable finding.

Long term (10+ years) analysis of the C.S.F. periodontium by conventional periodontal means would be of benefit for the evaluation of

clinical acceptability of the procedure. Study into the effect the fibrotomy may have in different regions of the dental arches would also be worthwhile.

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TABLE I

Slide #	Healing Time (Days)	Patient
4AZ & 4AW	Control	James Betts
8BW & 8BZ	Control	Lisa Hansen
EX & EZ	1	Sandra Lowe
P - X	1	Lynn Talent
R - X	1	Cassandra Brown
W - X	1	Donna Leslie
N - X	4	Lynn Talent
S - Z	4	Jan Leake
Q - X	4	Cassandra Brown
U - X	10	Jeanette Meling
Z - X	10	Scott Lee
DAX - DAZ	14	Terri LeGove
16AZ - 16AW	14	Elizabeth Hateli
O2AZ - O2AW	21	Roy Neal
BAX - BAZ	21	Tammy Jo May
18B2 - 18BX	28	Elizabeth Hateli
CAX - CAZ	28	Terri LeGove
MX - MZ	56	Marcia Ferguson
1BX & 1BZ	90	Amy Dinkler
LBX & LBZ	90	Tammy Taylor
2AZ & 2AW	120	James Betts
6AX & 6AZ	120	Lisa Hansen

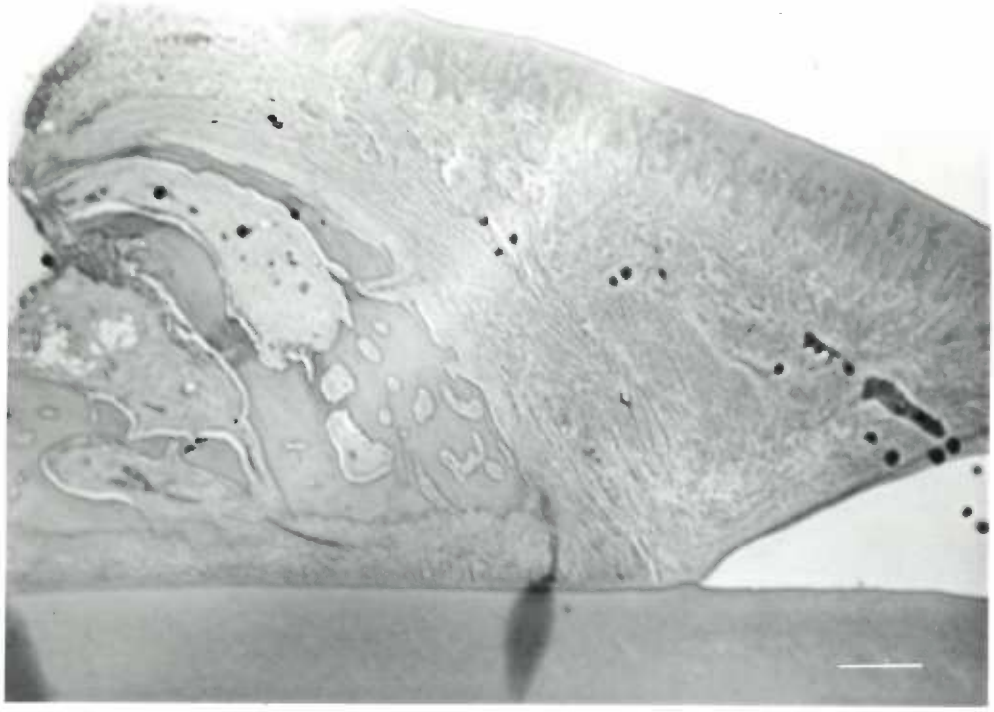

Fig. 1: Frontal view of control section. H & E scale equals 500 micron.

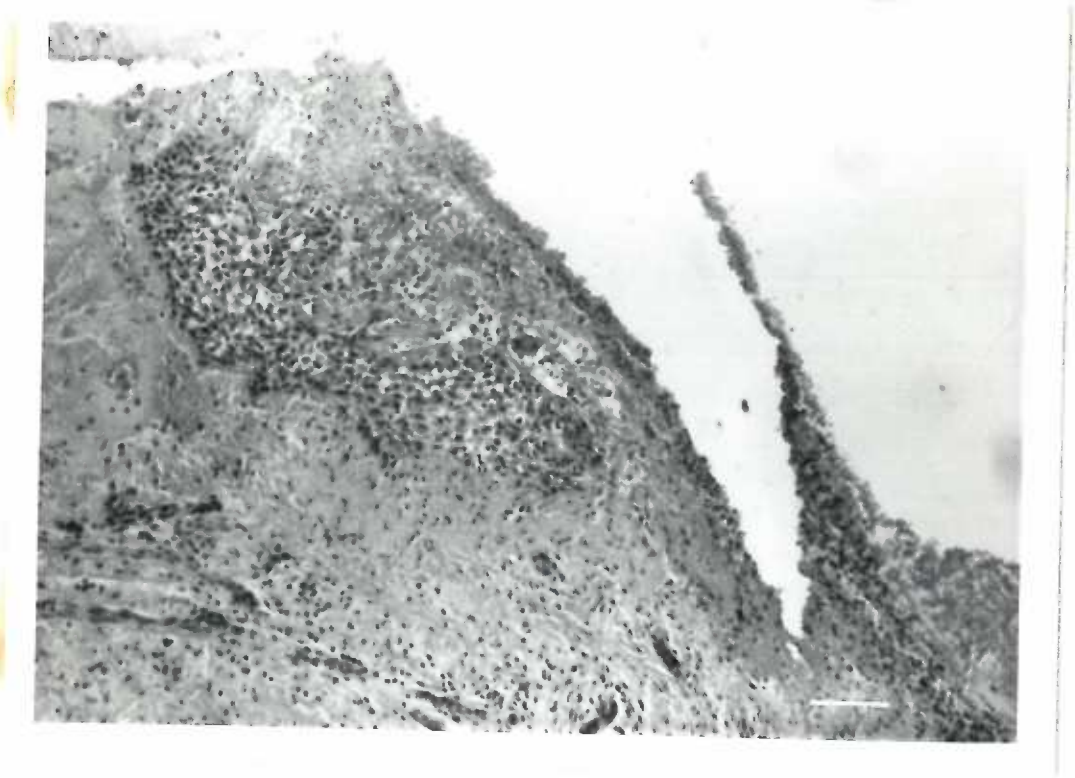

Fig. 2: Day 1. Frontal view showing circumscribed area of acute inflammatory cells. H & E scale equals 100 micron.

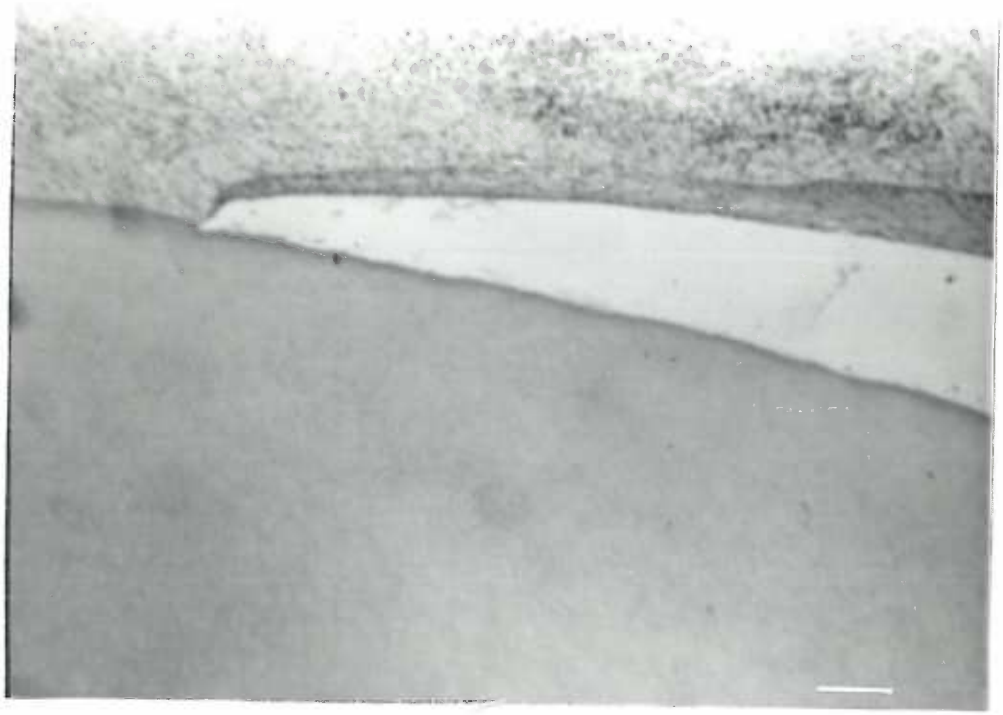

Fig. 3: Day 10. Frontal view showing newly formed dentogingival attachment. H & E scale equals 100 micron.

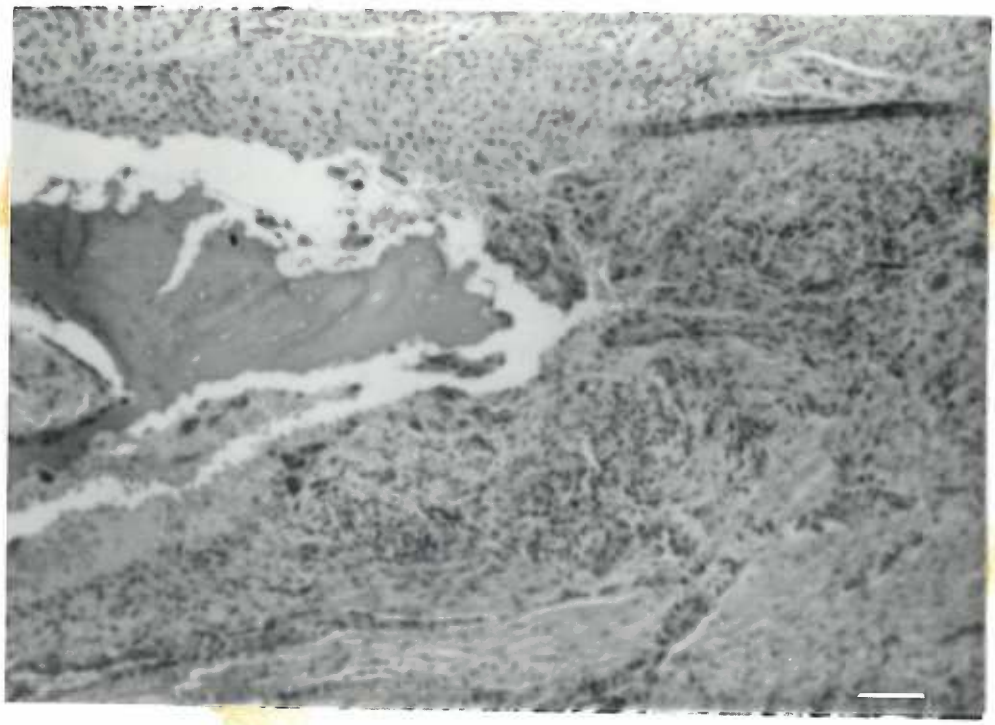

Fig. 4: Day 14. Frontal view showing alveolar bone with resorption lacunae on the PDL side. H & E scale equals 100 micron.

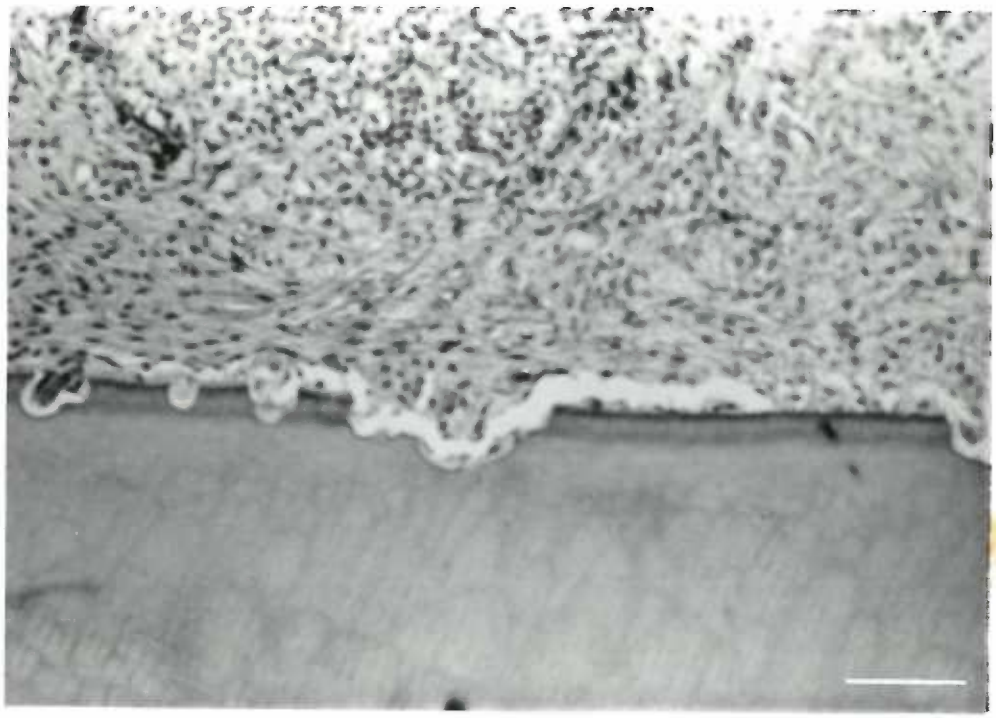

Fig. 5: Day 21. Frontal view showing area of cemental resorption. H & E scale equals 100 micron.

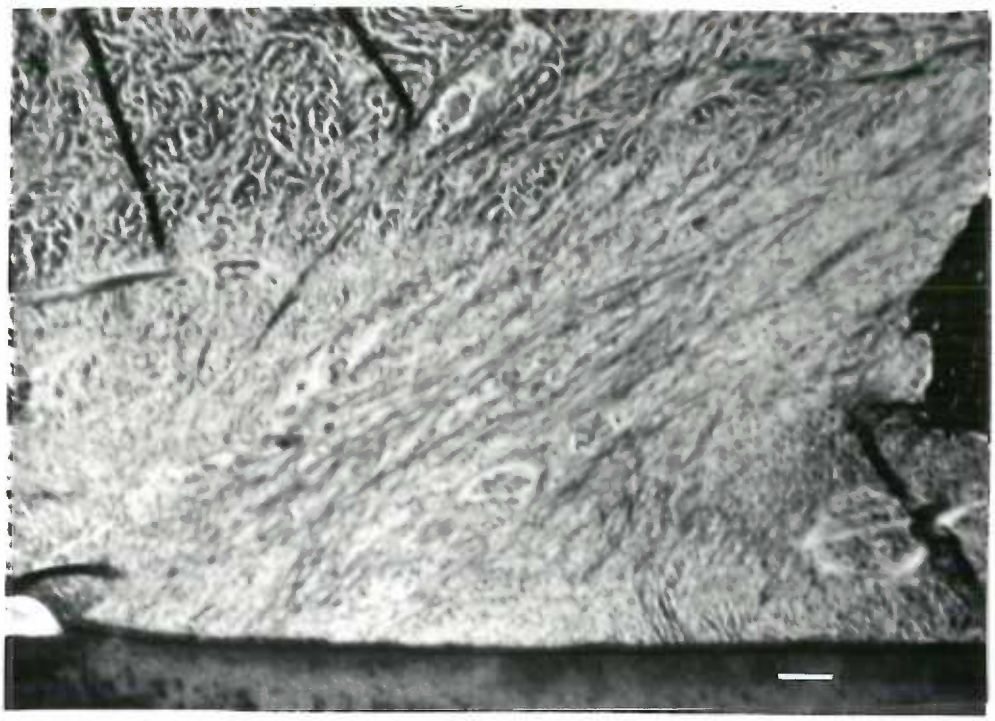

Fig. 6: Day 28. Frontal view showing fibers oriented similar to control. Mellory's stain scale equals 100 micron.