1980

Alterations in the Dimensions of the Dentogingival Junction in Marmosets as a Result of Occlusal Trauma

Lloyd V. Tilt
Loyola University Chicago

ALTERATIONS IN THE DIMENSIONS OF THE DENTOGINGIVAL JUNCTION IN MARMOSETS AS A RESULT OF OCCLUSAL TRAUMA

by

Lloyd V. Tilt, D.D.S.

A Thesis Submitted to the Faculty of the Graduate School of Loyola University of Chicago in Partial Fulfillment of the Requirements for the Degree of

Master of Science

April

1980

ACKNOWLEDGEMENTS

I wish to express appreciation to Dr. Anthony W. Gargiulo for his direction and friendship throughout the course of this endeavor.

Thanks also to Dr. Daniel A. Grant who instigated this research, provided immeasurable insight into the nature of its results, and is largely responsible for its completion.

And particularly to my wife am I most grateful. Without her encouragement and support many things would never have been begun, much less completed.

Lloyd V. Tilt

VITA

The author, Lloyd V. Tilt, D.D.S., is the son of Lloyd V. and Signe P. Tilt. He was born October 6, 1948, in Salt Lake City, Utah.

His elementary and secondary education was obtained in the public schools of Salt Lake City, Utah, where he graduated from South High School in 1966.

He attended the University of Utah in Salt Lake City from September, 1966 through June, 1970, graduating with a Bachelor of Science degree with a major in biology.

In September, 1970, he entered Loyola University School of Dentistry in Maywood, Illinois. He was graduated from that institution in June, 1974, receiving a Doctor of Dental Surgery degree.

From June, 1974 to August, 1975, he engaged in private, general dental practice in Green River, Wyoming.

In September, 1975 he was granted a two year residency position in periodontics at Loyola University School of Dentistry, Maywood, Illinois. In June, 1977 he was awarded a certificate of dental specialty in periodontics.

Frome June, 1977 to the present, he has engaged in the practice of periodontics in Ogden, Utah.

TABLE OF CONTENTS

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Clinical and histologic effects of traumatic occlusion upon the periodontium, and their resultant sequelae have long been a subject of study as well as controversy in the field of periodontics. Since Stillman coined the phrase "traumatic occlusion" in 1919, research has been carried out in an attempt to relate the role of trauma from occlusion to alterations in gingival contour, various inflammatory conditions, periodontal pocket formation and varying types of alveolar bone loss.

The preponderance of evidence to date indicates that periodontal changes due to inflammatory disease and periodontal changes due to occlusal trauma are distinct, discernible entities. It has been hypothesized that in combination, inflammation and traumatic occlusion may potentiate the pathologic effects of crestal bone loss and pocket formation.

Alterations in the disease-free structure and in the relationships of epithelial and connective tissue elements at the dentogingival junction due to occlusal trauma have been reported. The purpose of this research is to describe histologic responses to occlusal trauma in an animal model, Calitthrix jacchus (marmoset). This animal is found to have periodontal inflammation in the both the wild state and in captivity. The introduction of occlusal trauma

will permit the observation of any responses in the epithelial, fibrous and calcified compartments of the periodontium. More specifically, it is our intent to focus on alterations around the dentogingival junction with regard to the dimension of these structures as responses to trauma from occlusion superimposed upon inflammation are noted.

CHAPTER II

REVIEW OF THE LITERATURE

A. Traumatic occlusion in relationship to inflammation:

The effects of traumatic occlusion upon inflammation, and the interaction of trauma and inflammation have been reported in the literature, with varying interpretations.

Orban and Weinmann[3], in 1933, after studying human autopsy material by step-serial section reported damage to the periodontium including periodontal ligament necrosis, hemorrhage, blood vessel thrombosis and bone resorption at the histologic level. These responses agreed with earlier findings by Gottlieb and Orban[23] using dogs as the experimental model. Neither study, however, could observe interaction between the damage produced by trauma from occlusion and the overlying inflammatory condition of the gingiva.

Orban[6], in 1939, again reported on traumatic tissue damage and gingival inflammation. This study, performed in dogs, reported damage similar to that in humans, and showed that when the occlusal trauma was relieved, the damage was physiologically repaired and the periodontium returned to normal. There was an apparent reversal of the damage which had been produced by traumatic occlusion. He was unable to show any direct relationship between traumatic occlusion and gingival inflammation, concluding that they were two separate

pathologic entities frequently found in the same teeth and individuals.

This concept of separatism between occlusion and periodontal disease had previously been endorsed by Gottlieb and his followers[49]. In later studies, Orban[11] was able to distinguish two types of periodontal reaction to occlusal stress. In teeth with normal supporting structures, transitory changes occured in the tissues apical to the alveolar crest, with no apparent gingivitis or periodontitis present. However, in teeth with an already pathologically weakened periodontium, even normal occlusal forces were found to aggravate the pathology. This was substantiated in studies by Glickman[41] and Goldman[34].

Basic to the study of the combined effects of inflammation and occlusal trauma is a knowledge of the vascularity of the area. Many opinions have been expressed regarding the pathway of inflammation as it proceeds from the gingiva into the underlying tissues in periodontal disease. Early investigators reported the spread of inflammation via the lymphatic channels of the gingival connective tissue, directly into the periodontal ligament.

Reports published later by Box[2,4] purported that inflammation spreads directly into the alveolar bone, with the periodontal membrane rarely becoming involved.

Weinmann[24], in 1941, reported on the progress of gingival inflammation through the periodontium. In a study on human jaws, his findings showed inflammation to follow the course of the blood vessels into the marrow spaces of the alveolar bone. The periodontal ligament

was usually found to be free of inflammation. The periodontal ligament was at times secondarily involved by inflammation which followed the blood vessels out of the marrow spaces and into the periodontal ligament. But this occurence was independent of the severity of the gingival inflammation. The bulk of inflammatory cells and subsequent inflammatory processes was shown to be in the neighborhood of the blood vascular supply. Weinmann's concept of vascular supply was a construct based upon his and other workers' observations.

The first experimental evidence which related occlusal trauma and inflammation as cooperative factors in periodontal destruction came in 1962 when Glickman and Smulow[31] reported microscopic observations regarding the relationship of occlusal forces and the spread of gingival inflammation into the supporting periodontal tissues in human jaws. The discussion proposed a "backward propulsion" of inflammatory cells along vascular channels into endosseus marrow spaces and then into the periodontal ligament. They concluded that excessive occlusal pressures altered the transeptal fibers and the deep periodontal fibers with a resultant extension of inflammation into the periodontal ligament space, angular resorption of bone, and extension of the periodontal pocket between the tooth and bone. This mechanism, called the "altered pathway" required interaction of gingival inflammation and excessive occlusal trauma, and produced distinct patterns of bone loss.

Glickman[13,14] followed this study with papers in 1963 and 1965 which were constructs that attempted to provide an understanding

of the divergent opinions regarding the significance of trauma from occlusion by clarifying the roles of inflammation and occlusal trauma in chronic destructive periodontal disease. He presented a concept of "co-destruction" by inflammation and trauma from occlusion as a realistic explanation of the tissue destruction observed. It was alleged that trauma from occlusion does not cause inflammation or periodontal pockets in periodontitis, but it combines with the inflammation to cause the destruction of the supporting tissues, which is a most significant measure of periodontal disease.

In 1965, Glickman and others[41] published a further presentation of the "co-destructive factors" concept. Damage from occlusal trauma alone; widened periodontal ligament spaces, thickened lamina dura, etc. is reversible, repairable or can be adapted to. Inflammation, as long as it is confined to the gingiva, is not affected by occlusal forces. However, occlusal overload, in an already destructive periodontal situation, changes the alignment of the periodontal ligament fibers, thereby altering the pathway of inflammation into the periodontal ligament rather than into bone. This produces vertical, angular bone loss and infrabony defects which by themselves are not reversible. This later study was based upon experimental data previously obtained.

Comar, Kollar and Gargiulo[15] reported further on the question of whether a combination of local irritations and superimposed occlusal trauma will produce a more exaggerated tissue change than either factor alone. Using Rheses monkeys as models,

they showed that the interdental fibers may take a more oblique direction due to tooth depression, but in general remain intact, acting as a protective barrier against irritants to the osseus tissues. The structures of the periodontium below the interdental fibers were shown to be subject to damage from traumatic forces, while the interdental fibers themselves and the structures above them were mainly influenced by local and/or systemic factors. Because the interdental fibers remained intact, there was also no apical migration of the epithelial attachment, therefore no pocket formation. Vertical and angular patterns of bone loss were not produced.

A study reported in 1972 by Polson and others[17] tested the hypothesis that a traumatic injury subjacent to a marginal periodontitis alters the progression of the periodontal lesion, resulting in angular bony defects. The experiment, performed in monkeys, found no significant differences in the severity or patterns of bone loss either with or without the presence of the traumatic influence. Their conclusion was that when the inflammation was resolved, the changes were reversible.

Continuing the theme of his previous study, Polson[21] was later able to mechanically produce a severe lesion in the coronal periodontal ligament immediately subjacent to a chronic marginal periodontitis, thus fulfilling the essential requirements for the co-destructive factor effect. He was able to show the possibility of altered pathways of inflammatory progression, but was unable to demonstrate their sequelae: angular bone loss, infrabony pockets.

A study on the effect of mechanical injury to the periodontal ligament on gingival fluid flow rate, a marker of inflammation, was presented in 1971 by Bowles and Muhleman[16]. Their study showed the desmosomal trauma evidence clinically by increased mobility does not enhance the gingival fluid flow rate, indicating that continuous abnormal forces applied to the teeth, as well as resultant periodontal ligament injuries, are without effect on the degree of marginal inflammation. It was therefore concluded, that occlusal trauma neither produces marginal gingivitis nor increases its severity.

B. Effects of traumatic occlusion upon the structures of the periodontium:

A study on tissue changes in experimental traumatic occlusion conducted by Gottlieb and Orban[33] in 1931 reported a great deal of damage which they attributed to occlusal trauma. Periodontal ligament compression, some areas of necrosis of the periodontal ligament, and blood vessel thrombosis were the most prominent.

Orban and Weinmann[3], in 1933, using human autopsy material displayed virtually the same damage as Gottlieb and Orban earlier reported: periodontal ligament crushing, necrosis, hemorrhage, and blood vessel thrombosis.

In 1939, Orban[6] reported the effects of traumatic occlusion. Using dogs, he noted compression of the periodontal membrane on the pressure side and widening of the membrane on the tension side. If compression was complete, hyalinization and necrosis of the periodontal membrane occured, and areas of bone resorption began. He noted that

when pressure was relieved, the tissues soon recovered and returned to normal, all pathology being repaired.

Macapanpan and Weinmann[18] analyzed the spread of gingival inflammation in instances of coexisting trauma to the periodontal ligament and reported the tissue pathology produced. They indicated that trauma causes damage to the periodontal ligament on both the pressure and tension sides, and that damage to the periodontal ligament on the tension may diminish the resistance of the normal periodontal ligament to direct infiltration from a gingival inflammation, leading to periodontitis. Their results supported the view that traumatism of the periodontal ligament will not, by itself, cause periodontitis.

In 1955, Bhaskar and Orban[8] studied experimental trauma using Rheses monkeys with high fillings. Their reported showed that the most damage occured within three weeks of initiation of trauma, and repair coexisted with pathology, gradually becoming dominant. They also were able to observe adaptation to the trauma, the teeth moving as a result a lateral bone resorption widening the periodontal ligament space, until the trauma was eliminated or physiologic compensation occured. Findings indicated that occlusal trauma does not produce gingivitis, periodontitis, pockets, clefts, festoons, recession, pulp stones, erosion or caries. Changes seen in occlusal trauma were found to be specific, in their most extreme consisting of: necrosis of periodontal tissue, resorption of bone, root resorption, and bone apposition on tension areas. All of these changes which were attributed solely to traumatic occlusion, were reversible.

Wentz, Jarabak and Orban[11], trying to create an experimental situation which better simulated the human condition, created jiggling forces on teeth. The combined effects of pressure and tension alternately applied were recorded as thrombosis of blood vessels, necrosis of the periodontal ligament, resorption of bone and cementum. The resorption of bone and removal of necrotic connective tissue led to an increased width of the periodontal ligament until it was more than three times as wide as a non-traumatized tooth. Finally, the damaging effect of the jiggling force was nullified by the extreme width of the periodontal ligament space and no further crushing necrosis occured. Once the pathologic cycle was completed, the periodontal ligament was repaired, bone tissue was replaced, and cementum repaired, but a wider periodontal ligament space was maintained. As a consequence of the wider periodontal ligament, there was increased mobility of the tooth. The mobility, however, was not a sign of an inflammatory periodontal disease state, it was a functional readjustment of normal tissues to continued occlusal stress. These findings were substantiated by Yuodelis[12] and Breitner[48] in subsequent studies.

Glickman and others[32] in a study of the reactions of splinted and non-splinted teeth to occlusal trauma, demonstrated the capacity of the periodontium for compensation and adaptation to extreme load. Non-splinted teeth were depressed into their sockets, causing great damage to the periodontal ligament in pressure areas. Supporting earlier studies, periodontal ligament necrosis and undermining alveolar bone resorption was demonstrated.

Bifurcation and trifurcation areas were shown to be affected earlier and to a greater extent than other areas along the root surface. The long term subjects reestablished complete vertical closure by intrusion of the teeth, and subsequent readjustment of the alveolar housing and periodontal ligament. Although tooth depression occured, no changes were observed in the location and nature of the epithelial attachment apparatus, or the transeptal and gingival fiber attachment location, and no pockets could be observed. Once equilibrium of occlusal force had been reestablished by periodontal adaptation, pathology was quickly repaired, supporting the concept that damage produced by trauma from occlusion is reversible.

In a similar study reported in 1964, Safavi and coworkers[22] reported on periodontal traumatism produced by sustained increase in vertical dimension. Using monkeys, splints and crowns were placed high in occlusion to produce trauma. Necrosis of the periodontal ligament and alveolar bone was consistently demonstrated at axial, apical and interradicular areas. Some tipping lesions were produced on lower molars, creating infrabony osseus defects by intrusion. Teeth were quickly intruded, with approximately fifty per cent closure of the increase in vertical dimension within fifty-five days. There was no detectable influence upon gingival inflammation or upon transfer of exudates from the gingival inflammation through the attachment apparatus or into the periodontal ligament. Even with extreme intrusion, gingival fibers remained intact, epithelial downgrowth did not occur, and no periodontal

pockets were created.

In a study specifically aimed at bone alterations produced by occlusal trauma, Itoiz and others[25] showed increased bone resorption and decreased bone formation between within one and two weeks after initiating trauma, which continued for the duration of the experiment. Immediately after initiating the trauma there was a lag phase in which there was no observable resorptive response. It was postulated that the damage was still occuring, it just took time for the damage to be apparent histologically. Once again, it was observed that gingival fiber elements remained intact, there was no alteration in the level of the epithelial attachment, and no periodontal pockets were produced.

Kemper, Johnston and Huysen[26] in 1968 studied periodontal tissue changes in response to high artificial crowns. They demonstrated, as had previous studies, that bone resorption occured on the pressure side, bone apposition occured on the tension side. Bone resorption was direct on the periosteal and periodontal ligament surfaces, while the periodontal ligament was still vital, and undermining resorption occured when the periodontal ligament was necrosed. All lesions attributed to traumatic occlusion were repaired and healed without inflammation or scarring.

C. Traumatic effects upon periodontal pocket formation:

The concept of periodontal pocket production by trauma from occlusion has long been discussed and widely reported. Basically, three possibilities have been proposed: that periodontal pockets are not produced by traumatic occlusion, that periodontal pockets

may be produced by traumatic occlusion, and that periodontal pockets probably are not produced by traumatic occlusion alone, but may be produced by traumatic occlusion and inflammation in combination with each other.

A large number of researchers, differing in background and over a long period of time, using a variety of experimental models have supported the concept that periodontal pockets are not produced by traumatic occlusion. From Orban[3] in 1933 through Polson[17] in 1972, encompassing investigators such as Macapanpan and Weinmann[18], Bhaskar[8], Safavi[22], Wentz, Jarabak and Orban[11], and Comar[15] reports indicate that though the tooth may be intruded, periodontal ligament necrosis and alveolar bone resorption may take place, that interdental fibers, and gingival fibers remain intact. Although they may assume a more oblique direction due to depression of the tooth in the socket, their insertions remain constant and the fibers provide a protective barrier that prevents inflammation from reaching osseus structures below. And once the trauma has ceased, or the periodontium has accomodated to the excessive forces, the trauma-induced pathology is repaired and the supporting structures of the periodontium reestablish themselves. No epithelial downgrowth along the root surface occurs and no periodontal pockets result. A functional adaptation occurs in the periodontium.

A second group of researchers have reported that periodontal pockets can be produced by traumatic occlusion[2,4]. Waerhaug[9] provided the most notable experimental evidence that periodontal

pockets may be produced through trauma in a study reported in 1955. Using high crowns on dogs, Waerhaug studied the effects of long-standing, repeated occlusal overload. His results showed that under extremely unfavorable conditions, a deepening of the clinical periodontal pocket below the cementoenamel junction can be produced by occlusal stress. A combination of horizontal and vertical overload may cause an intrusion of the tooth to such an extent that it touches the alveolar crest in the deepest point of the epithelial attachment. In this manner, the periodontal ligament may be destroyed all the way to the most apical attachment of epithelium. Epithelial downgrowth along the root surface occurs after the periodontal ligament fibers have been destroyed and before new periodontal ligament attachment can occur. He opined that this epithelial downgrowth, once present, is irreversible. More coronal detachment of the extended epithelial attachment thus created a periodontal pocket. However, in a later experiment[47] trying to produce pockets in monkeys via the same mechanism, Waerhaug was unsuccessful in reproducing his previous results. Although some epithelial downgrowth did occur, it was similar in dimension to the control specimens and traumatic damage was found to be totally repaired when trauma was relieved.

The third group of investigators[13, 14, 32, 41, 43, 45] in the trauma/periodontal pocket controversy agrees with the first group in that periodontal pockets are not produced through traumatic occlusion alone. They believe that trauma from occlusion in combination with an already present gingival inflammation of

"sufficient" severity may produce periodontal pockets. Through a single series of experiments and many subsequent publications, Glickman and coworkers proposed the concept of traumatic occlusion and inflammation acting together as "co-factors of destruction". They state that as long as the inflammation is confined to the gingiva, it is not affected by occlusal forces. However, once a simple marginal periodontitis has become established, occlusal force which is excessive serves to potentiated the pathology that is produced. Excessive occlusal pressures alter the transeptal fiber and the deep periodontal fiber arrangement and direction, with a resultant extension of inflammation into the periodontal ligament space and production of angular patterns of bone resorption and extension of the periodontal pocket between the tooth and bone. They state that when infrabony pockets and osseus defects are present, the possibility that they have been produced by the combined effect of trauma from occlusion and inflammation must be considered. This does not mean that the combination of inflammation and trauma from occlusion always results in infrabony pockets and angular bone destruction. Infrabony pockets and osseus defects by themselves are not lesions of trauma from occlusion, but they occur on teeth with evidence of trauma in the supporting periodontal tissues. Inflammation must be present to produce the periodontal pockets. The presence of angular osseus defects on the radiograph is no pathognomonic of trauma from occlusion, but it is highly suggestive. No other local factors have been so consistently identified with angular defects.

The findings of Glickman and coworkers were largely uncorroborated for many years. More recently, however, Polson[21] in 1974 was able to produce mechanically a severe lesion in the coronal periodontal ligament immediately adjacent to a chronic marginal periodontitis, fulfilling the essential requirements for the channelization of inflammation into the periodontal ligament space, the co-destructive factor effect. He was unable to show angular patterns of bone resorption associated with this, but did in part substantiate Glickman's hypothesis. However, Polson found that once the inflammation was resolved, pathology was repaired.

Lindhe and others[19] studied the effects trauma from occlusion and permanent tooth hypermobility would have on the rate of periodontal bone destruction in dogs. Their histologic sections showed that the degree of apical proliferation of the pocket epithelium was more pronounced in the test subjects in traumatic occlusion than in the control subjects not in trauma. They also reported increased bone destruction in sections where inflammation and trauma coexisted.

CHAPTER III

MATERIALS AND METHODS

Ten healthy, young adult marmoset monkeys (Calitthrix jacchus) were selected as experimental models. Under sodium barbital anesthesia, shallow grooves were cut into the occlusal surface for retention and quick-cure acrylic was flowed onto the occlusal surface of the mandibular right first molar to an approximate depth of one millimeter above the occlusal plane. The left side was left untouched and served as control model for the experiment. The animals were then replaced in cages with food (Purina monkey chow) and water ad libitum. The marmosets were then sacrificed according to the following schedule: one animal was sacrificed at one day following placement of the acrylic overlay, two animals at three days, two animals at five days, two animals at ten days, two animals at seventeen days, one animal at thirty days.

Immediately after sacrifice, the specimens were fixed in a solution of alcohol, formalin and acetic acid, decalcified in nitric acid-formalin and prepared for celloidin enbedding in the routine manner. Mesio-distal sections were then cut at fifteen micron thickness. Sections were stained using hemotoxylin and eosin, PAS-hemotoxylin, Mallory's connective tissue stain, and

Pearson's silver nitrate gel impregnation.

Sections were then studied under light microscopy. All measurements were taken at a magnification of 100X using an eyepiece grid calibrated in microns. Measurements were taken on both mesial and distal surfaces recording magnitude of the epithelial attachment, connective tissue attachment, and the location of the epithelial attachment with relation to the cementoenamel junction. These measurements were made on the maxillary right first molar which was the experimental tooth in traumatic occlusion, and on the maxillary left first folar which served as the control.

In addition to dimensional measurements, histological assessment was made for the sulcular epithelium, epithelial attachment, connective tissue attachment of the corium, periodontal ligament and alveolar bone. The condition of the tissue, alterations and pathology, if any, was noted.

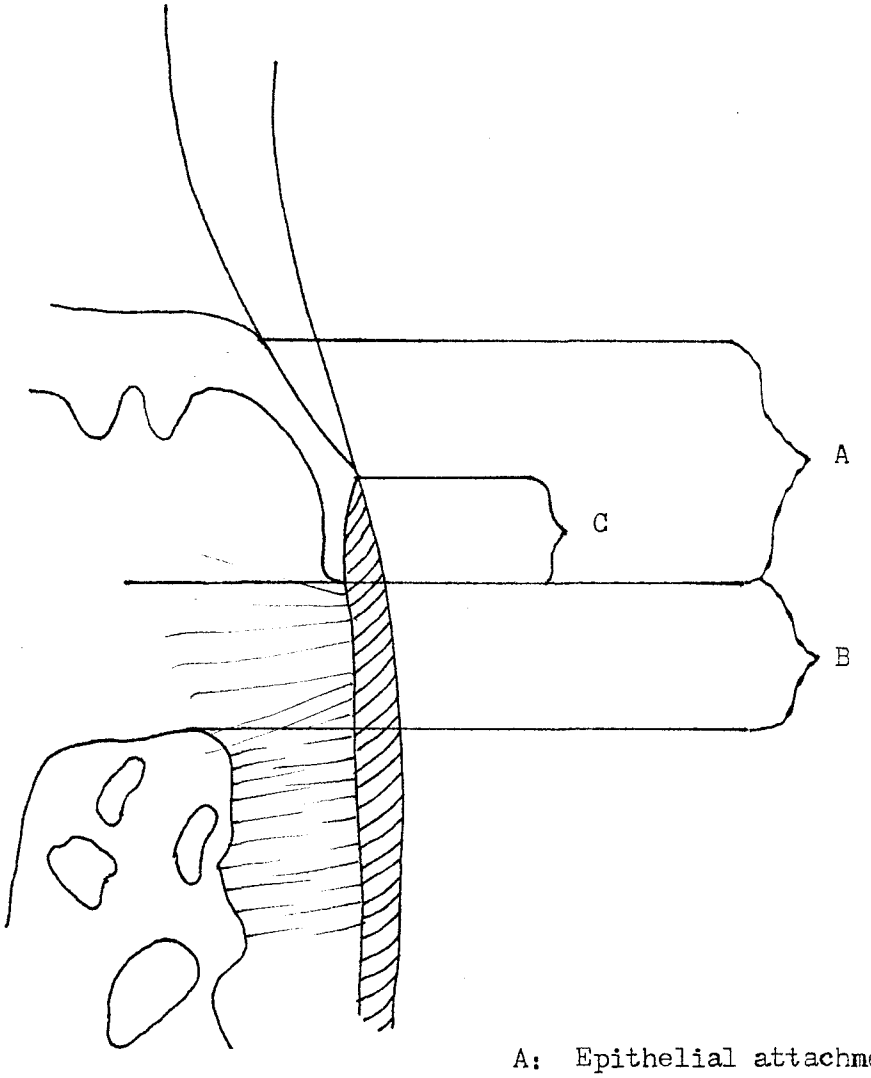
From these measurements and histologic evaluations, cellular and dimensional changes were recorded for the experimental period from one to thirty days.

Figure I depicts the levels at which histologic measurements were taken using the calibrated eyepiece grid. In order to maintain consistency and uniformity in measurement procedures, all measurements were made in a plane parallel to the long axis of the root.

Figure I

Zones for Dimensional Study of Structures

of the Dentogingival Junction



A: Epithelial attachment

B: Connective tissue attachment

C: Cementoenamel junction to epithelial attachment

CHAPTER IV

OBSERVATIONS

Histologic observations and measurements of the epithelial, fibrous, and osseus compartments are reported according to their chronologic sequence: control, one day, three day, five day, ten day, seventeen day and thirty day specimens.

A. Control specimens: In all cases, the left side, or contralateral side was preserved for control purposes. Although the controls were measured and observed in the same time references as the experimental, the findings for all controls were very similar and will be reported as one group.

1. Epithelial compartment: Control specimens displayed a histologically demonstrable gingivitis. The epithelial surface layer was intact, there was no ulceration of the sulcular epithelium, but the surface was covered with plaque and exudate. The epithelial attachment was fairly constant in size between samples and was attached at the level of or just coronal to the cementoenamel junction. The epithelial attachment averaged four cell layers in length and two to three cell layers in depth.

2. Fibrous compartment: There was a mild subepithelial inflammatory process in the immediate lamina propria. Mononuclear

inflammatory cells were scattered throughout the lamina propria and polymorphonuclear leukocytes could be seen in the lamina propria as well as making their way through the junctional and sulcular epithelium. The connective tissue elements were well organized. Gingival fibers were well defined, horizontal in direction and the entire connective tissue zone above the alveolar crest exhibited densely collagenous fiber elements. Characteristically, fiber groups of the periodontal ligament were dense, appeared highly cellular, compact and well-ordered. Periodontal ligament fiber insertions on both the cemental and osseus surfaces were easily discernible. The periodontal ligament was constant in width on all observable surfaces: mesial, distal and apical. The PDL space had a moderate degree of vascularity with vessels originating from the alveolar wall coursing throughout the ligament. The periodontal ligament component from the alveolar crest to the root tip apically was free of any inflammatory elements.

3. Osseus compartment: The osseus structures showed normal topography on both the alveolar crest and periodontal ligament surfaces. Distal and disto-crestal surfaces had a sequence of orderly layered resting lines describing some physiologic mesial migration of the tooth, with bony apposition along the distal surface. Large marrow spaces were, the area being highly vascular. The osseus compartment was free of any inflammatory involvement. The osseus structures showed no evidence of resorption.

4. Histologic measurement of periodontal for control specimens:

a. Mean length of epithelial attachment: 112.000 microns.

b. Mean length of connective tissue attachment: 150.666 microns.

c. Mean distance epithelial attachment to cementoenamel junction: 84.500 microns.

B. One day specimens: Figures 2, 3.

1. Epithelial compartment: The sulcular surface appearance was similar to that of the control specimens. The epithelial surface was intact, but heavily layered with plaque and exudate. Epithelial ridges within the gingival papilla were elongated, projecting deeper into the lamina propria than control specimens exhibited. In one specimen an extensive chain of enlarged epithelial cell rests was seen extending half-way down the distal root surface, through both the connective tissue and the periodontal ligament. Epithelial attachment dimensions were the same as for the control specimens. Epithelial attachment level was either at or just slightly coronal to the cementoenamel junction.

2. Fibrous compartment: Subepithelial inflammation in the lamina propria was evident, with a mild infiltration of round cells. Mononuclear cells and other inflammatory cells were present in the same proportion as control specimens. The connective tissue was densely collagenous. The gingival and dental-alveolar fiber groups were well developed, intact, and maintained their horizontal orientation. The fibers of the periodontal ligament appeared well organized, with normal orientation. Vascularity was not remarkable.

Periodontal ligament width remained fairly constant from mesial to distal, however some sections appeared narrower on the mesial than on the distal. This may have been the early expression of pressure compression on the mesial surface.

3. Osseus compartment: No alteration from the control specimens was noted. The bone was mature, well organized, showing previous resting lines along the distal surface. The marrow spaces were large and normal in appearance. Inflammatory elements or alterations were not present.

4. Histologic measurement of periodontal structures for one day specimens:

a. Mean length of epithelial attachment: 72.070 microns.

b. Mean length of connective tissue attachment: 216.983 microns.

c. Mean distance from epithelial attachment to cementoenamel junction: 20.517 microns.

C. Three day specimens: Figures 4, 5.

1. Epithelial compartment: The epithelial surface appeared intact, though layered with plaque and exudate. The epithelial attachment had notably altered. The epithelial attachment had increased in length along both mesial and distal surfaces, now averaging approximately six to eight cell layers in length. The epithelial attachment also increased in depth, now averaging four to six cell layers deep laterally. Some sections showing epithelial proliferation also showed the long epithelial attach-

ment with finger-like extensions, projecting laterally and apically into the adjacent connective tissue. Many sections showed the apical extent of the epithelial attachment to be nearly at the same level as the alveolar crest, describing epithelial proliferation along the root surface. The epithelial attachment level was now located at the cementoenamel junction or apical to it. Slightly over one-half of the sections studied demonstrated epithelial attachment apical to the cementoenamel junction.

Another observation of great interest concerned the zone of connective tissue immdiately apical to the epithelial attachment. The normally dense, collagenous nature of the connective tissue had been altered just below the proliferating epithelial attachment. There now appeared a clear zone, a zone of hyalinization of connective tissue extending for approximately fifty to one hundred microns apical to the epithelial attachment. This zone contained no cellular elements, no discernible fibrous elements, and consisted of a clear-staining area like that of the hyalinization in the periodontal ligament from traumatic injury reported in other studies.

2. Fibrous compartment: Inflammation of the lamina propria was of the same dimension as earlier specimens, with virtually the same histologic appearance and cellular infiltrate.

The dentogingival and dentoalveolar fiber groups appeared intact. Their orientation was now oblique rather than horizontal, indicating depression of the tooth.

The periodontal ligament space appeared to be of the same

approximate width mesially and distally, but apically had narrowed by about one-half, giving the appearance of increased cell density in the apical areas. Though mesial and distal periodontal ligament dimensions were similar, the mesial side appeared to be under compression, while the distal side showed fiber elongation and signs of tension. Periodontal ligament fiber directions were only slightly vertically oblique, their inserations into bone and cementum remained intact and vascularity was not altered. No inflammatory elements were present in the three day specimens.

3. Osseus compartment: The mesial surface of the alveolar bone, and the mesio-crestal surface displayed some osteoclastic activity. Direct surface resorption could be seen in resorption bays forming along the mesial surface. One subject contained a well developed lacunae with osteoclasts at the mesio-crestal corner. The mesial and mesio-apical surfaces were under obvious pressure. The tension side, the distal, displayed no alterations in bony contour or in osteoblastic or osteoclastic activity. No inflammation or inflammatory elements were observable.

The nature of the cementum along the distal tooth surface had also changed in appearance, becoming thinner near the cemento-enamel junction, with incremental lamellae becoming more visible along the middle and apical areas of the distal root surface.

4. Histologic measurement of periodontal structures for three day specimens:

a. Mean length of epithelial attachment: 107.014 microns.

b. Mean length of connective tissue attachment: 131.806 microns.

c. Mean distance from epithelial attachment to cementoenamel junction: 58.819 microns.

D. Five day specimens: Figures 6, 7, 8, 9, 10.

Five day specimens showed signs of acute occlusal trauma, with the tooth greatly intruded, the apical and furcal root structure forced against the alveolar bone.

1. Epithelial compartment: The surface of the epithelial layer appeared intact with surface debris and exudate as reported in previous specimens.

The epithelial attachment in the five day specimens showed the most dramatic alteration yet seen. The epithelial attachment, along the distal surface had proliferated three-quarters of the way apically along the root length, with finger-like extensions projecting laterally into the widened periodontal ligament space. In some sections, the epithelium in the periodontal ligament space appeared discontinuous with the epithelial attachment coronally. However, inspection of serial sections displayed continuity with the distal epithelial attachment. This mass of epithelial cells along the distal root surface was at some points twenty to thirty cell layers in depth.

The connective tissue immediately surrounding the enlarged epithelial attachment presented a cell-free zone adjacent the epithelial cells and an area of apparent hyalinization of connective tissue extending apically from the epithelial attach-

ment along the root surface.

The epithelial cell mass was intact and remained attached from its normal coronal level completely down the distal root surface to the apical end of it proliferation. No coronal detachment of the epithelium was observed. No periodontal pocket was observed.

Inflammation did not accompany the apical proliferation of epithelium and remained localized to the subepithelial lamina propria.

2. Fibrous compartment: Slight inflammation in the subepithelial connective tissue and exudation continued as in previous specimens.

The periodontal ligament was crushed and destroyed in the furcation areas and the apical areas. The ligament space was greatly narrowed mesially and widened by approximately two times distally. Along the mesial and apical surfaces were many areas of blood vessel thrombosis, periodontal ligament necrosis and cell-free areas of hyalinized connective tissue. The occlusal force was clearly in a mesio-apical direction. Compression was evident along the mesial and apical surfaces, tension along the distal surfaces. The distal portion of the periodontal ligament remained intact, the ligament fibers being stretched and depressed apically, but undergoing no observable histologic change. These histologic changes occured in the absence of any inflammatory change.

3. Osseus compartment: Osteoclastic activity continued

at an increased rate along the entire mesial and apical surfaces. Numerous resorption bays with osteoclasts were observed. The alveolar crest remained intact, with no resorption or apposition occuring. The distal surface of the alveolar housing showed no evidence of either osteoblastic or osteoclastic activity. Fatty marrow appeared to be replacing hematologic marrow in the most coronal marrow spaces. Inflammatory elements were not present.

4. Histologic measurement of periodontal structures for five day specimens:

a. Mean length of epithelial attachment: 111.250 microns.

b. Mean length of connective tissue attachment: 53.125 microns.

c. Mean distance from epithelial attachment to cementoenamel junction: 55.125 microns.

E. Ten day specimens: Figures 11, 12, 13.

Signs of acute trauma from occlusion persisted, with root structure pressed against the alveolar bone apically.

1. Epithelial compartment: The picture in the surface epithelium remained virtually unchanged from that of the control.

The epithelial attachment had shortened considerably from the five day specimens but was still longer and deeper than the control and one day specimens. The level of the epithelial attachment was at or below the cementoenamel junction. The length of the attachment had shortened, but the clear zone of connective tissue observed earlier below or apical to the epithelial

attachment was still present. The cell-free zone of apparent hyalinization of connective tissue was of approximately the same dimension as in earlier specimens.

The attachment of epithelium to root surface was intact along the entire length of the proliferative epithelium. There was no coronal detachment of epithelium. There was no periodontal pocket produced.

2. Fibrous compartment: There was a mild inflammation in the subepithelial lamina propria with infiltration of inflammatory elements, round cells, and some exudate present on the surface.

The dentogingival and dentoalveolar fiber groups were observed to be intact, though still oriented in an apically oblique direction.

The fibers of the periodontal ligament were also in an apically oblique direction, as well as under tension on the distal surface. The mesial and apical surfaces contained zones of periodontal ligament necrosis, areas of hyalinization, and a fiber direction nearly parallel to the root surface.

3. Osseus compartment: The alveolar bone along the mesial and apical surfaces demonstrated marked osteoclastic activity. In most areas, the resorption was direction, along the periodontal ligament surface. However, in some areas adjacent periodontal ligament necrosis, undermining resorption was occuring within the marrow spaces. The alveolar crests were intact, and neither they nor the distal alveolar surface showed signs of

osteoblastic or osteoclastic activity.

4. Histologic measurement of periodontal structures for ten day specimens:

a. Mean length of epithelial attachment: 65.000 microns.

b. Mean length of connective tissue attachment: 89.936 microns.

c. Mean distance from epithelial attachment to cementoenamel junction: 27.281 microns.

F. Seventeen day specimens: Figures 14, 15.

1. Epithelial compartment: The epithelial surface was intact, covered with plaque and some exudate from the underlying inflammatory process.

The epithelial attachment was longer than the control group and had proliferated down along the cementum in over one-half of the sections. The thickness in cell layers had diminished from previous days down to approximately three to four cell layers in depth. Just apical to the epithelial attachment there persisted a small zone of apparent hyalinization, a clear-staining, cell-free, collagen-free zone.

2. Fibrous compartment: The lamina propria was mildly invaded with mononuclear cells. The inflammation was confined to the area immediately adjacent to the epithelial layer.

The dentogingival fiber bundles on the distal side of the tooth had maintained their horizontal relationship to the alveolar crest. The mesial dentogingival fibers were found to be still at

an apically oblique relationship to the alveolar crest, expressing a mesio-apically applied force and subsequent tooth depression.

The periodontal ligament fiber groups retained a fairly normal appearance and arrangement along the distal surface. The mesial, however, was greatly disrupted. Zones of cell-free, clear hyalinization were present where the mesial root surface had contacted or nearly contacted the alveolar bone. Areas of ligament repair were present with the reticular fibers aligned parallel to the root surface.

3. Osseus compartment: The mesial, or pressure surface was still undergoing moderately active osseus resorption. The resorption was predominantly direct, along the periodontal ligament surface of the alveolar housing. Areas were noted, however, associated with periodontal ligament necrosis, where undermining or endosseus resorption was taking place.

The distal surface of the alveolar housing was undergoing considerable osteogenic activity. Resting lines were visible along the entire distal surface, with very active new bone formation present, particularly in the coronal one-half. The entire distal bone surface was lined with osteoblasts. The root surface was lined with cementoblasts, with active cementum deposition taking place.

4. Histologic measurement of periodontal structures for seventeen day specimens:

a. Mean length of epithelial attachment: 29.405 microns.

b. Mean length of connective tissue attachment: 27.024 microns.

c. Mean distance from epithelial attachment to cementoenamel junction: 59.881 microns.

G. Thirty day specimens: Figure 16.

1. Epithelial compartment: The surface layer of the epithelium appeared intact, though layered with plaque and some spots of exudate.

The epithelial attachment appeared to be longer than the control group. In some sections the epithelial attachment was quite long, while in other sequential sections it was smaller in length and depth than at the previous sequential intervals. The epithelial attachment was on the cemental surface in slightly less than one-half of the sections.

2. Fibrous compartment: The subepithelial inflammation occupied approximately the same area as that of the control group and was equivalent in severity. There was a mild infiltrate of round inflammatory cells, primarily polymorphonuclear leukocytes and mononuclear cells.

The appearance of the fibrous elements of the dentogingival junction was virtually the same as at seventeen days. The distal surface including the dentogingival, dentoalveolar, and periodontal ligament fiber groups had normal fiber density and arrangement. The mesial surface was still greatly narrowed, with an oblique direction. There were zones of hyalinization and periodontal ligament repair, with the newly forming fibers assuming a

relationship more parallel to the root surface, rather than perpendicular.

3. Osseus compartment: The osseus elements also appear to be closely similar to the appearance at seventeen days. The distal surface is undergoing active osseus apposition with resting lines and files of osteoblasts aligned along the distal bony surface and cementoblasts displayed along the distal root surface. The mesial surface still displays active osseus resorption, with underming resorption occuring in several areas associated with periodontal ligament necrosis.

4. Histologic measurement of periodontal structures for thirty day specimens:

a. Mean length of epithelial attachment: 77.237 microns.

b. Mean length of connective tissue attachment: 81.316 microns.

c. Mean distance from epithelial attachment to cementoenamel junction: 31.053 microns.

CHAPTER V

DISCUSSION

The epithelial compartment from zero to the thirty day interval consistently showed the greatest alteration of any area.

Specimens at all intervals including control had histologically demonstrable gingival inflammation. The inflammatory state remained at a constant level throughout the experimental period and was localized in the subepithelial lamina propria. The epithelial surface above remained intact, though covered with plaque and inflammatory exudate.

The gingival papilla remained constant in appearance, displaying moderately enlarged epithelial ridges, presumably in response to the mild inflammation below.

It was at the level of the epithelial attachment that the most notable changes took place. At the zero and one day intervals, the epithelial attachment was normal in appearance, four cell layers in length and located at or coronal to the cementoenamel junction. By three days, changes had taken place. The epithelial attachment had doubled in length and width, displaying lateral projections into the connective tissue, and the attachment level had progressed apically to the cementoenamel junction or slightly apical to it. In the five day specimens, epithelium had progressed

two-thirds of the way apically along the root surface on the distal and had grown in depth to approximately thirty cell layers. The large epithelial mass also sent numerous projections laterally into the connective tissue. Surrounding the epithelial mass along the root surface there was a cell-free, clear-staining zone, similar in appearance to an area of hyalinized connective tissue as may appear in the periodontal ligament as a result of injury. In subsequent intervals up to thirty days while there were some sections showing deep epithelial proliferation parallel to bone, the mean dimensions of this epithelial attachment mass gradually decreased back to a relatively normal level. The mean location of the epithelial attachment returned to the cementoenamel junction or just slightly apical along the cemental surface.

The mechanisms of this epithelial proliferation deserve some discussion. Connective tissue and fibrous insertions just below the level of the epithelial attachment have been considered a barrier to apical epithelial proliferation. In specimens beginning at three days and continuing for the length of the experiment, a clear zone just apical to the epithelial attachment was present. This zone was free of cells, having a clear-staining, homogenous appearance. That fibrous and connective tissue, from traumatic injury, is hyalinized and then repaired has been shown in numerous studies[3, 6, 8, 15, 33]. There may be a relationship between this hitherto undescribed hyalinization located just apical to the junctional epithelium and the apical proliferation of junctional epithelium noted in subsequent chronological sections. If this

protective zone of fibrous and connective tissue beneath the epithelial attachment were removed, and repair delayed due to continuing traumatic insult, the barrier to apical epithelial proliferation would have effectively been eliminated.

The cellular origin for epithelial proliferation has two distinct possibilities: the epithelial attachment itself, and epithelial cell rests along the root surface.

It is most probable that the origin of cells for proliferation was the epithelial attachment itself. The close time intervals for sectioning provided a continuous view of the hyperplasia of the attachment area. From day three at the beginning of noticeable proliferation, through day thirty, the epithelial attachment is always observed to have complete continuity. In one section at day five, the epithelial cell mass along the distal surface appears discontinuous with the epithelial attachment more coronally. This can be explained in terms of sectioning. This section is off to the side of the bulk of the proliferating epithelial mass, probably taken through one or more of the large projections which must be presumed to extend not only laterally into the periodontal ligament space, but buccally and lingually around the root surface as well. The cells of the epithelial attachment itself are probably the primary source for proliferative capability.

An alternative source and possibly working in conjunction with the attachment cells is the cells of the epithelial rests located along the root surface. In specimens at the one, three,

seventeen and thirty day intervals, extensive epithelial cell rests, sometimes appearing in chains, were noted. That these cells are stimulated by trauma and other disturbances is well known[8, 15], and the possibility of these cell rests serving as proliferative foci, enhancing epithelial progression along the root surface cannot be discounted[35]. Though possibly not a primary source for progression of the epithelial attachment along the root surface, their anatomical position and proliferative capabilities lend them as a mechanism of enhancement of epithelial progression.

Despite the extreme dimension of epithelial attachment proliferation along the root surface, there was histologically no detachment of the epithelium coronally, and thus no periodontal pocket formation. Results as far as tooth depression, connective tissue fiber damage, and periodontal ligament damage are the same as were produced by Waerhaug[9] in 1955. In his study, Waerhaug also had epithelial proliferation along the surface of the root. He, however, stated that there was coronal detachment and periodontal pocket formation. We were unable to reproduce those findings within the time sequence of our study. There is one tantalizing difference: extensive epithelial proliferation along cementum and parallel to bone occured. This may be a prelude to infrabony pocket formation. However, there is no evidence that long epithelial attachments will always detach coronally. Indeed, the converse may be true. It may be that the junctional epithelium is actually displaced coronally during healing. This possibility

is supported by data showing the return in mean junctional epithelium length in the thirty day specimen to a length close to that in the control specimens. Results of this study substantiate what many previous researchers[3, 8, 11, 15, 17, 18, 22] have stated; that periodontal pockets are not produced by traumatic occlusion.

Histologic evaluation of alterations in the other compartments, fibrous and osseus, provides virtually the same results as have been found in the past[3, 6, 8, 15, 33]. In the periodontal ligament, mesio-apical compression and crushing were observed as the tooth was intruded against the alveolar bone. By five days, necrosis and thrombosis in the periodontal ligament was occuring in the furca and apical areas. By ten days there were large areas of hyalinization within the periodontal ligament, breakdown persisting through thirty days. Areas of tissue breakdown and areas of repair were seen to coincide in many sections. Repairing periodontal ligament fibers were more parallel to the root surface in their orientation than mature, healthy fibers.

Osteoclastic activity began at three days on the pressure surface, peaking at the ten day interval and involving the entire mesial surface and the apical surface. Resorption was predominantly direct, along the periodontal ligament surface, but in areas where the periodontal ligament was necrosed, undermining resorption was observed. Cemental resorption in areas of trauma was not remarkable.

At seventeen days, osteoblastic activity was noted along the distal surface which continued through the thirty day interval. In the later specimens, osteoclastic activity along the mesial or

pressure surface and osteoblastic activity along the distal or tension surface coincided.

The sequence and chronology of these events in the fibrous and osseus compartments compares very favorably with studies done by Wilderman[49, 50] and Staffileno[51] on the repair of osseus and fibrous connective tissues.

In spite of the severity of the damage produced in the epithelial, fibrous and osseus compartments, and the chronic gingival inflammation which was present throughout the experiment, there was no alteration in the degree of inflammation, the location of inflammation, or production of inflammation. The mild inflammation which was present remained localized to the lamina propria of the gingival papilla area. All histologic damage produced in the periodontium took place in the absence of any inflammatory influence, nor did the damage have any effect upon the inflammatory process. At no time were there any inflammatory elements present in the epithelial attachment, gingival fiber groups, periodontal ligament, or osseus structures. It must, therefore, be said that traumatic occlusion in this case did not alter the pathway, severity or location of inflammation. This is in contradiction to some reports[13, 14, 31], but is in accord with the majority[3, 6, 8, 11, 15, 16, 25, 26]. This would substantiate the view that occlusal trauma by and of itself does not cause gingivitis or periodontitis. And in this instance, despite drastic apical migration of the epithelial attachment, periodontal pocket formation did not occur and progression of

periodontal disease was not enhanced.

The significant event observed throughout the experiment was the remarkable apical proliferation, through cellular growth, of the epithelial attachment. The histochemical nature of the process that cleared the way for epithelial cell progression is, however, also of great interest. The clear-staining, cell-free zone of apparent hyalinization appearing apical to the epithelial attachment from the third day on throughout the experiment needs to be explored. Two possibilities are proposed: that there was an epithelial cell secretion of some order creating cellular dissolution to clear the way; that tissue lysozymes and enzymatic factors released by cell death along the mesial surface destroyed remaining cellular elements in the area to remove cellular barriers to epithelial migration. In any case, the nature of this apparently hyalinized area bears further analysis.

The disappearance of the new, elongated epithelial attachment should also be further explored. While it is possible that what was observed was just a singular event, it is equally possible that this apical proliferation of the epithelial attachment is a frequent event under situations of great occlusal trauma. There may be a physiologic repair mechanism involving epithelial retraction or dissolution once excessive occlusal forces are relieved, allowing the attachment apparatus to return to a so-called normal level without prolonged pathologic effects. This is another area which certainly requires further study.

The applicability of the animal model used in this

experiment must also be considered along with the results. Marmosets (Calitthrix jacchus) have been used experimentally in the past, with marked correlations to the human condition. The development of the dentition and periodontium, presenting primary, succadaneous and permanent teeth, and similar periodontal elements to those of man have been noted[38]. Marmosets display a clinically and histologically demonstrable gingival inflammation virtually throughout life, in middle and older age becoming subject to alveolar bone loss, tooth mobility and tooth loss as in periodontitis[39]. These similarities, together with the histologic response to occlusal trauma so similar to those in previous studies already noted, makes the marmoset a valuable tool in the study of periodontal conditions.

CHAPTER VI

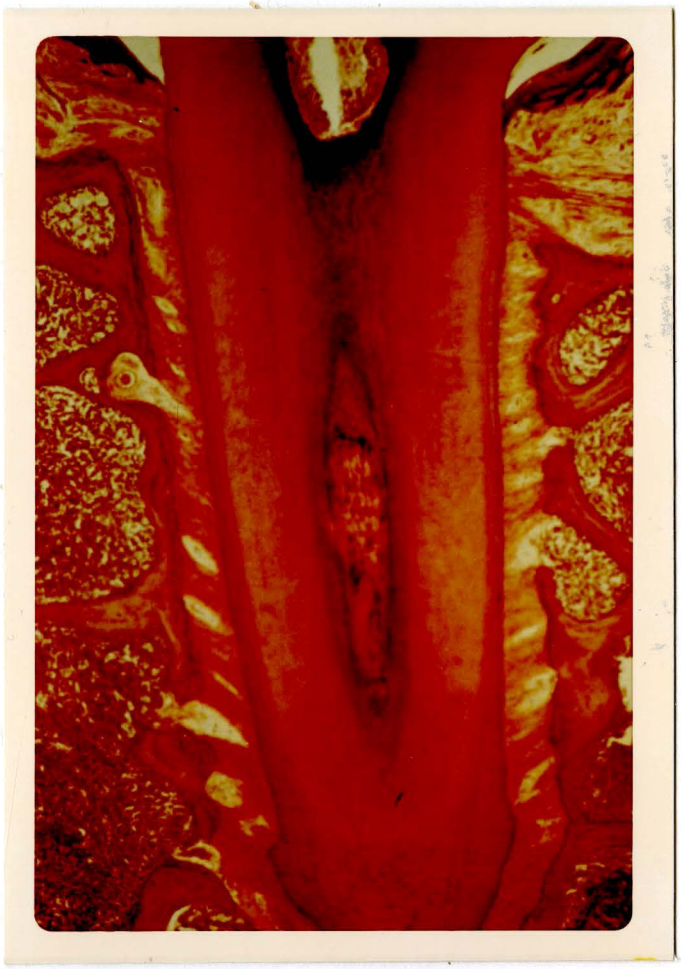
SUMMARY

Research was undertaken to report histologic responses to occlusal trauma in the periodontium of an animal model, marmoset (Calitthrix jacchus), which is naturally subject to periodontal inflammation and its sequelae. Responses were observed and noted in the epithelial, fibrous and osseus compartments of the periodontium. More specifically, experimental efforts concentrated on alterations around the dentogingival junction, dimensional changes in these structures in response to trauma from occlusion which was superimposed upon existing gingival inflammation.

Over a thirty day experimental period, dramatic changes were reported in the size and also the location of the epithelial attachment apparatus. The histologic nature of the changes was examined. The dimensions of the changes were recorded. The nature and significance of these changes was discussed.

Figure 2

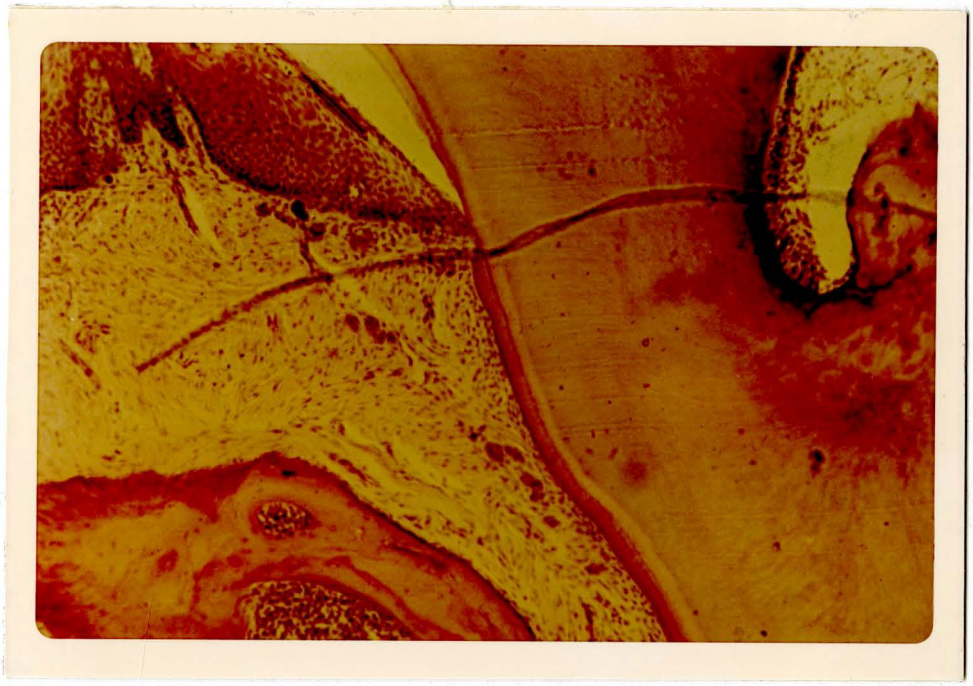
One day specimen



Sulcular epithelium intact. Epithelial attachment at or just coronal to cementoenamel junction. Constant periodontal ligament width. Mature, well organized osseus structures.

Figure 3

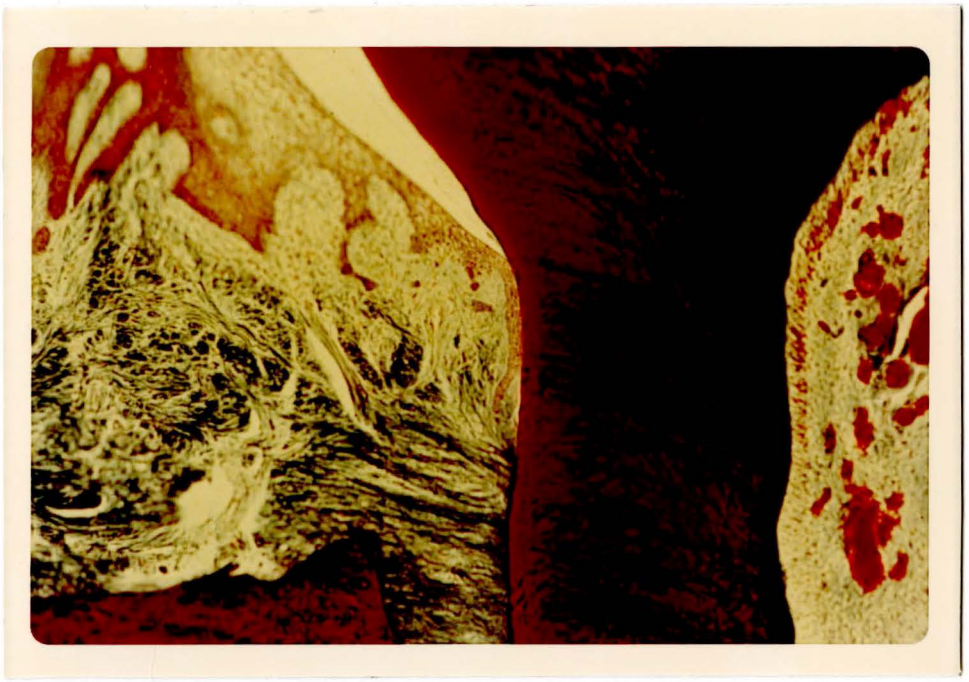
One day specimen



Epithelial surface intact. Note chain of enlarged cell rests extending through connective tissue and periodontal ligament along distal root surface.

Figure 4

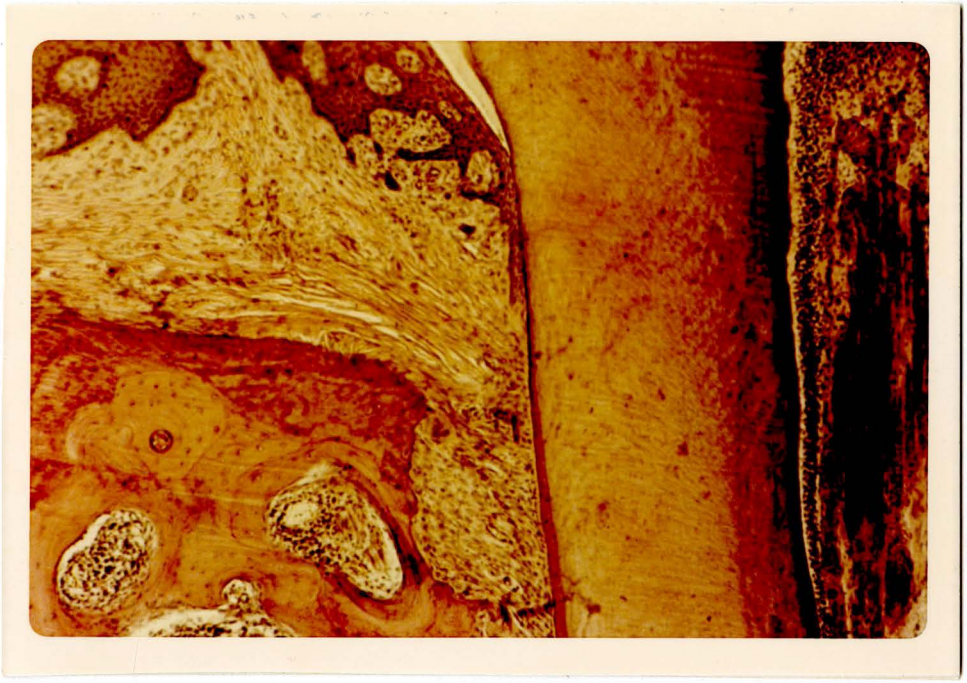
Three day specimen



Lengthening epithelial attachment with laterial finger-like projections extending into connective tissue.

Figure 5

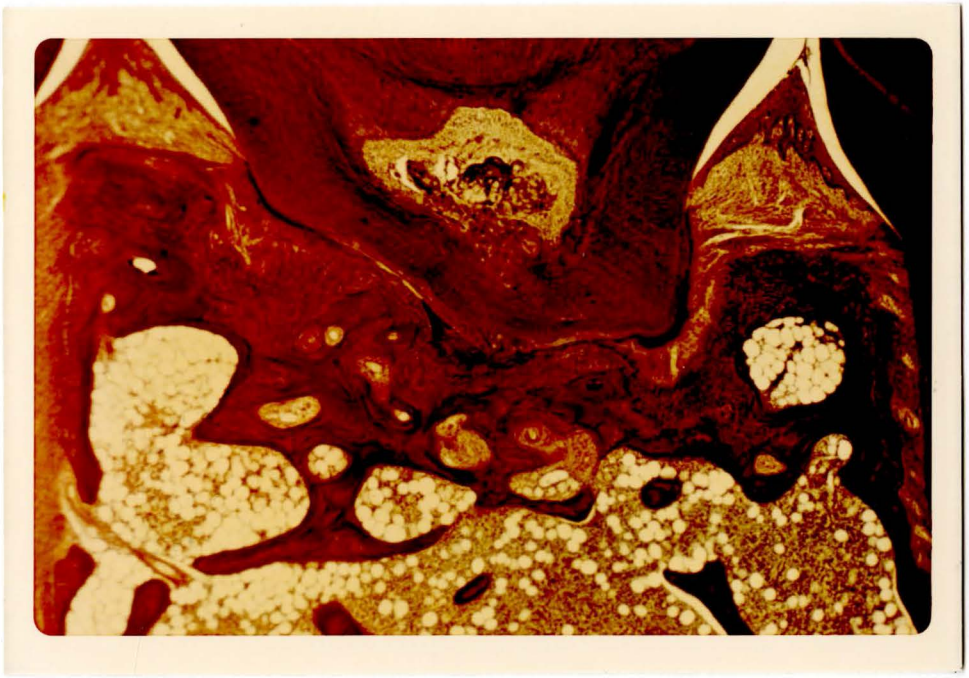
Three day specimen



Apical progression of the epithelial attachment nearly to the top of the alveolar crest. First appearance of cell-free clear zone of apparent hyalinization at apical border of epithelial attachment.

Figure 6

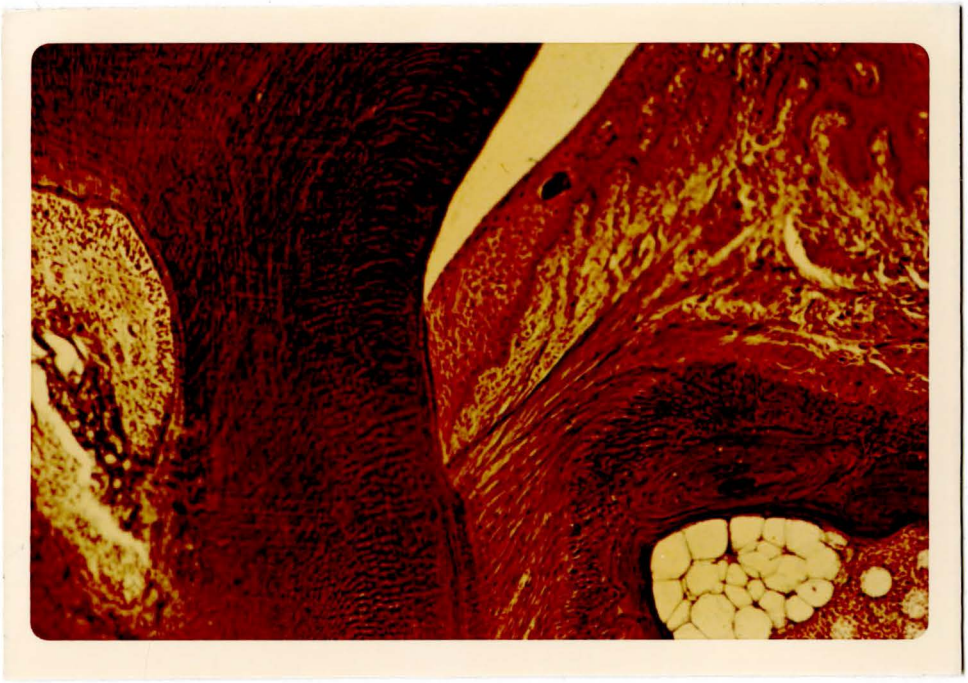
Five day specimen



Acute occlusal trauma. Furcation area of tooth in contact with alveolar bone.

Figure 7

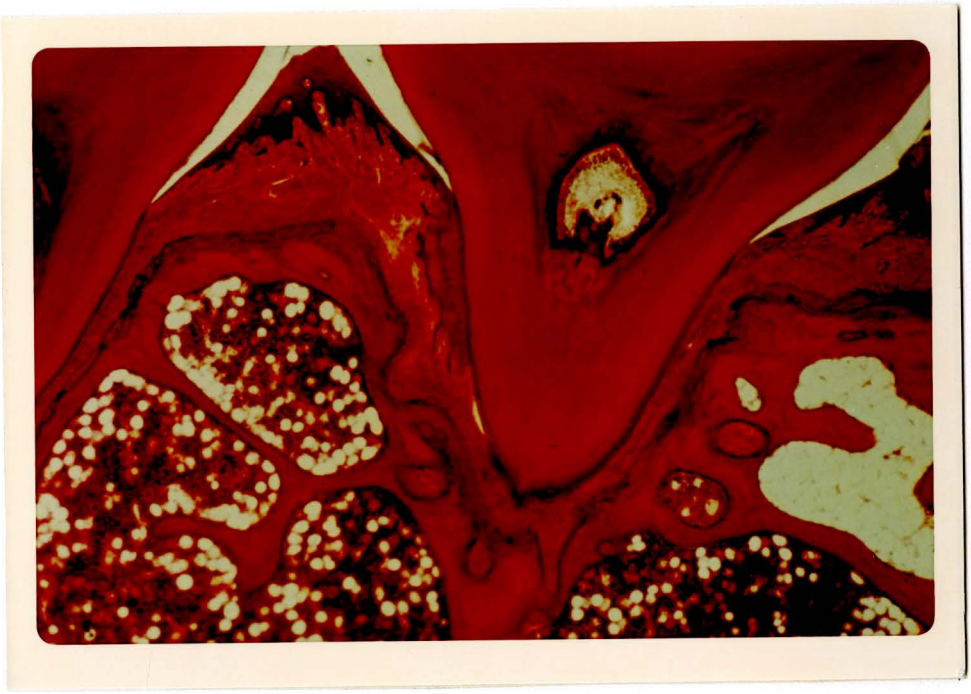
Five day specimen



Tremendous thickening and deepening of the epithelial attachment, progressing apically along cementum.

Figure 8

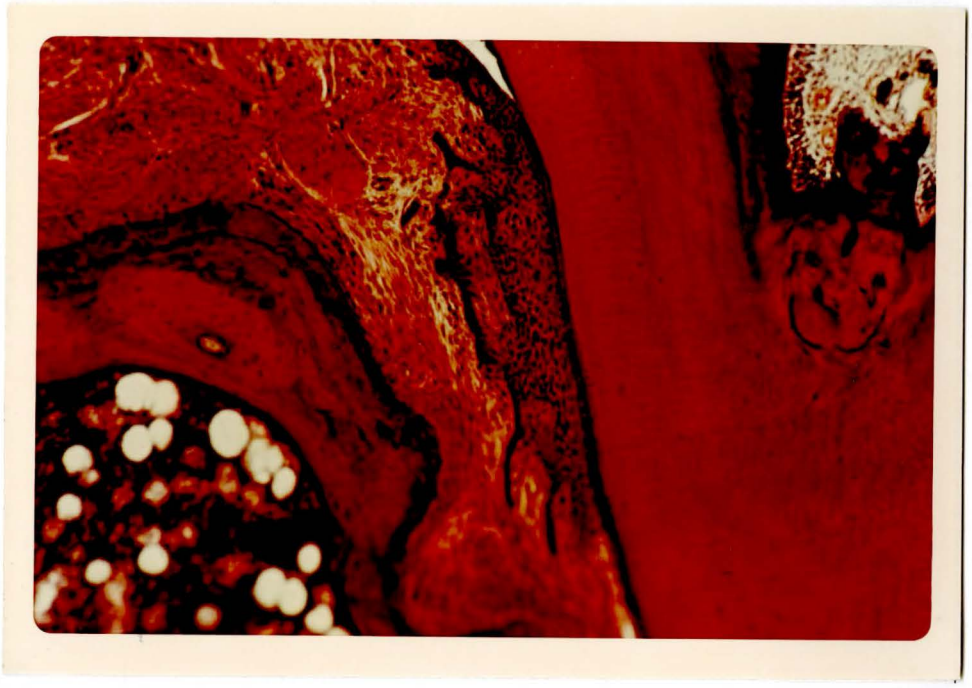
Five day specimen



Dramatic proliferation of epithelial attachment along distal surface, twenty to thirty cell layers thick sending projections laterally into periodontal ligament space.

Figure 9

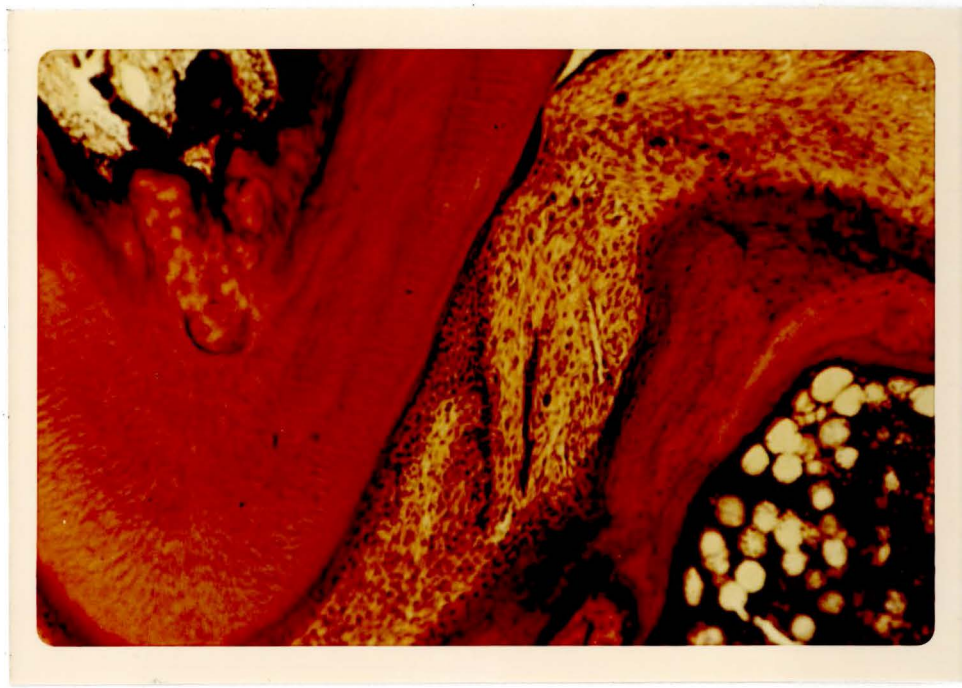
Five day specimen



Hyperplastic changes in the epithelial attachment progressing laterally and apically along the distal root surface.

Figure 10

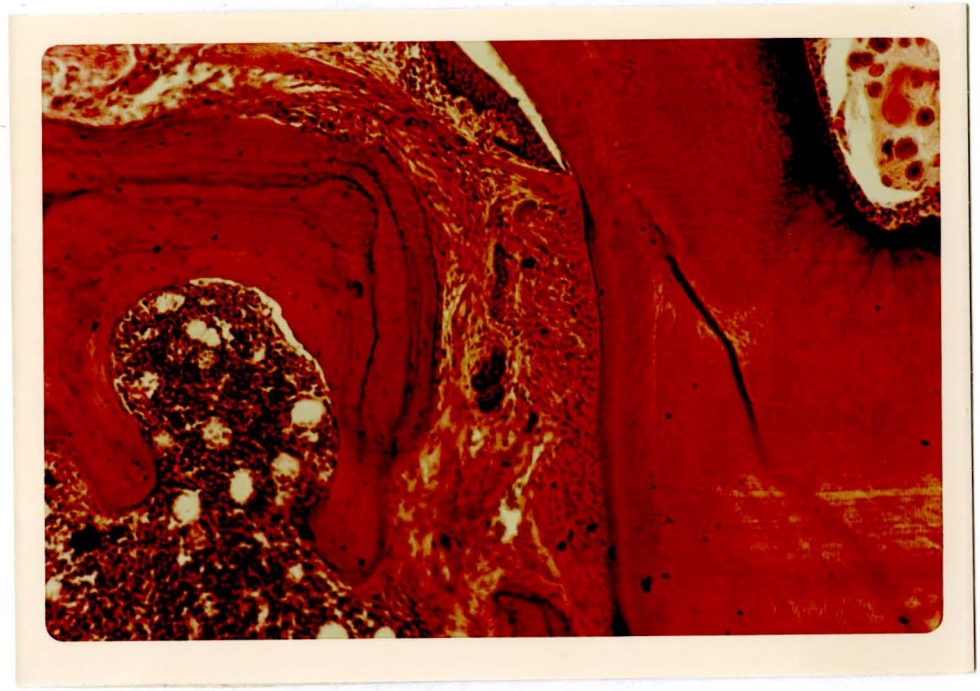
Five day specimen



This is the most apical extension of the epithelial attachment noted. The periodontal ligament space along the distal surface is approximately three times normal width. No detachment of epithelium from root surface is noted.

Figure 11

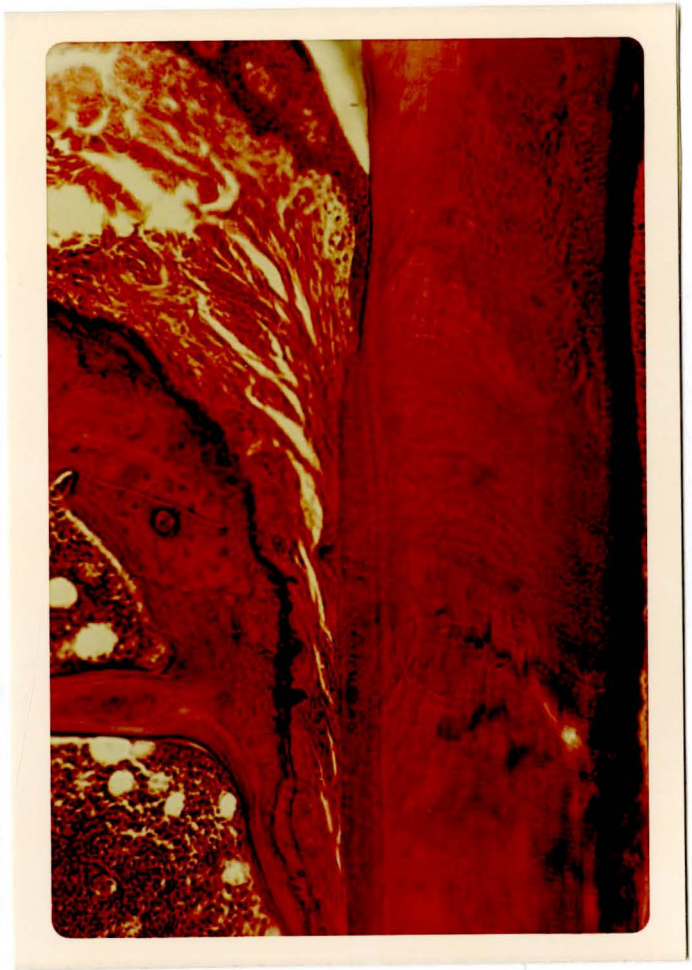
Ten day specimen



Elongated epithelial attachment persists, with numerous large epithelial cell rests. Clear zone of hyalinization still present apical to epithelial attachment. Connective tissue fiber bundles oriented in an apically oblique direction.

Figure 12

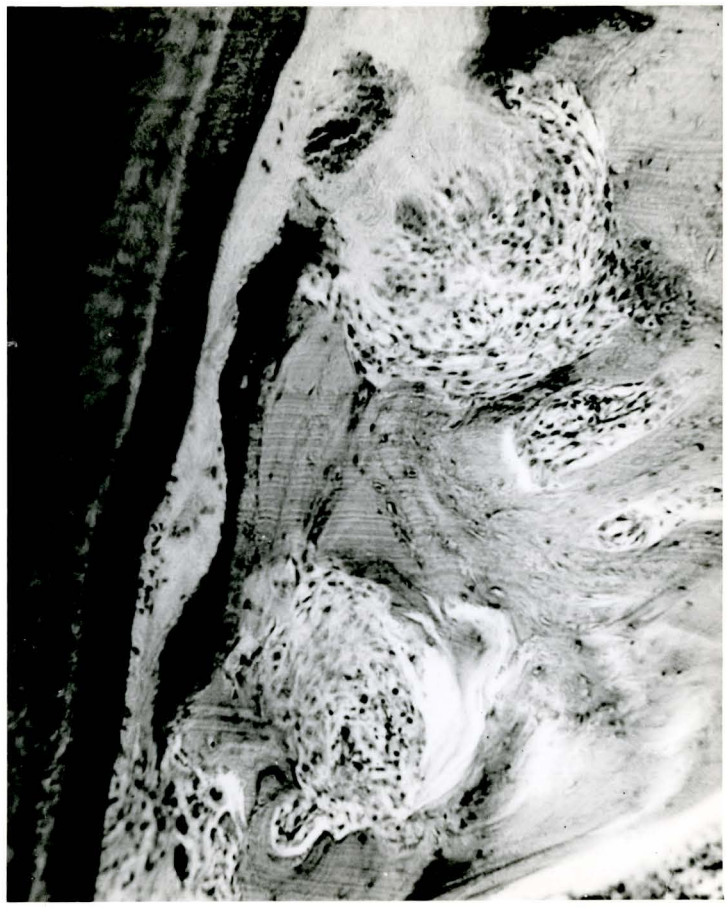
Ten day specimen



Epithelial attachment is smaller than five day specimen, but remains larger than control. Oblique direction of fiber bundles is apparent.

Figure 13

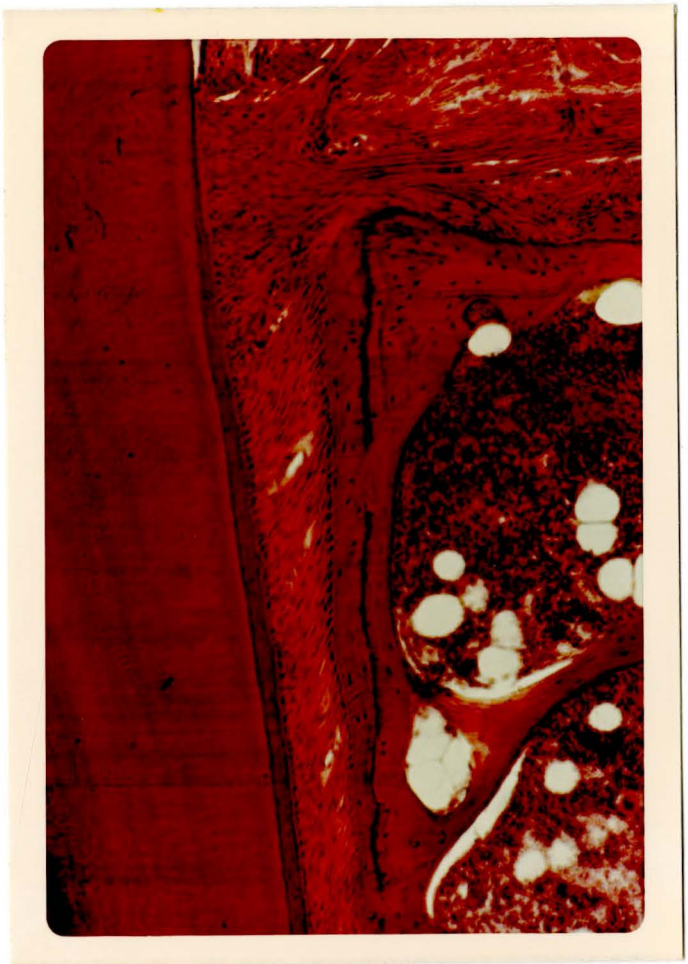
Ten day specimen



Osteoclastic activity is dramatically increased, with both direct and undermining resorption taking place.

Figure 14

Seventeen day specimen



Distal surface displays marked osteoblastic and cementoblastic activity. Files of osteoblasts line the osseus surface, with new osseus formation visible. Lighter staining cementoid indicates cementoblastic activity.

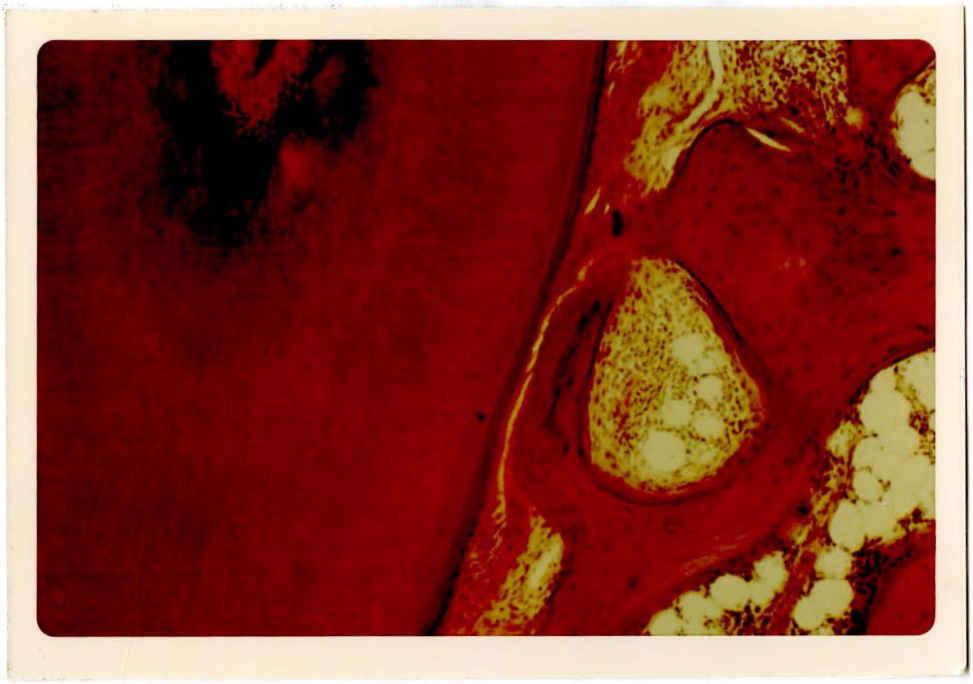
Figure 15

Seventeen day specimen

Osteoclastic activity continues along mesial, pressure side.

Figure 16

Thirty day specimen



Persistent zones of periodontal ligament necrosis and hyalinization coinciding with areas of repair along mesial root surface.

Mean Dimensions for Structures of the

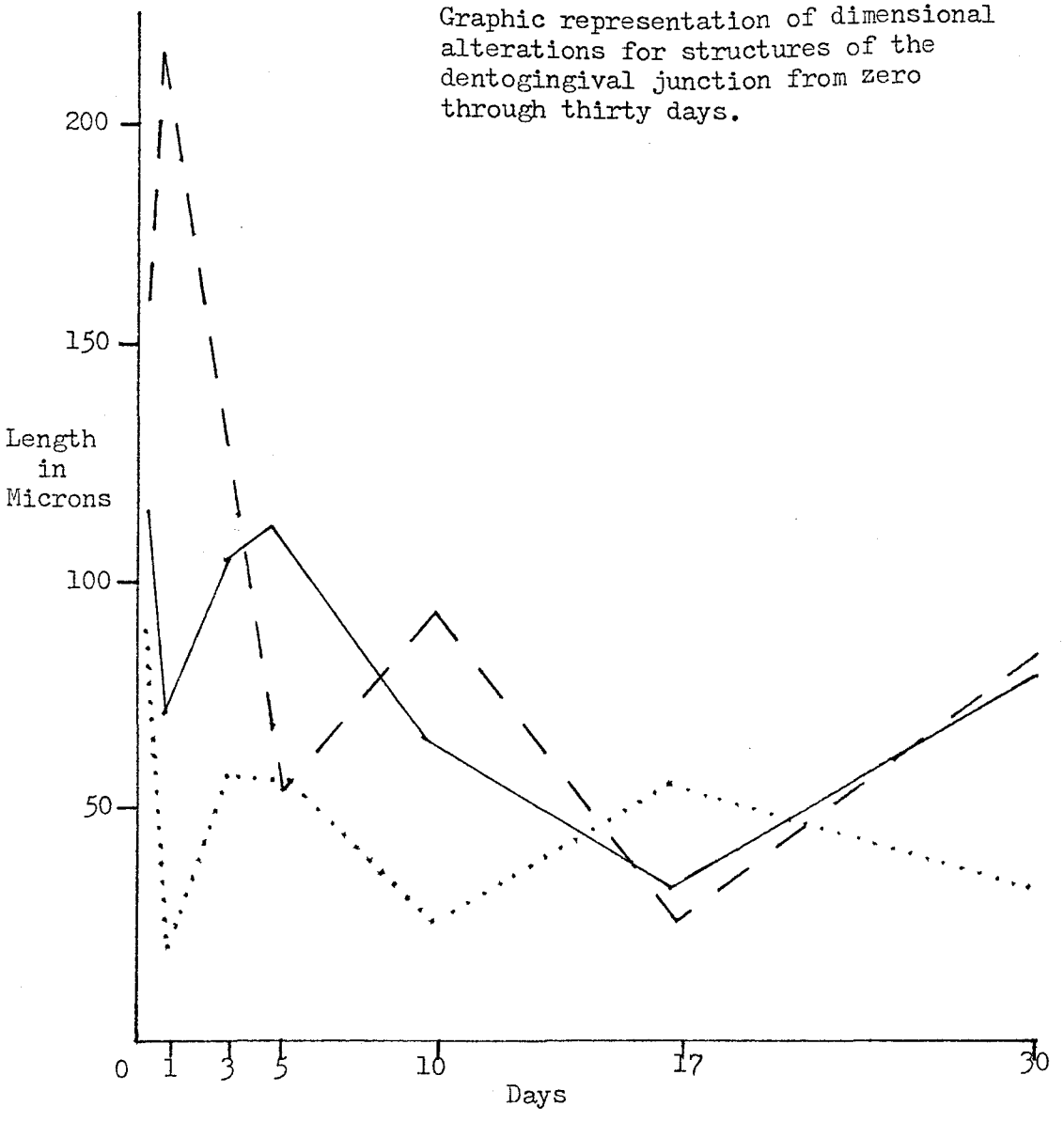
Dentogingival Junction - in microns

Structure	Control	1 One Day	3 Three Days	5 Five Days	10 Ten Days	17 Days	30 Days
Epithelial Attachment	115.333	72.070	107.014	111.250	65.000	29.405	77.237
Connective Tissue Attachment	160.500	216.983	131.806	53.125	89.936	27.024	81.316
Cementoenamel Junction to Epithelial Attachment	87.833	20.517	58.819	55.125	27.821	59.881	31.053

Figure 17

Figure 18

Graphic representation of dimensional alterations for structures of the dentogingival junction from zero through thirty days.



——————— Epithalial attachment

— — — Connective tissue attachment

. . . . . Cementoenamel junction to epithelial attachment

BIBLIOGRAPHY

1. Karolyi, M., Beobeachtungen Uber Pyorrhea Alveolaris, Uieteljschr. Zahnhielk, 17:279, 1901.

2. Box, H. K., Traumatic Occlusion and Traumatagenic Occlusion, Oral Health, 30:642, 1930.

3. Orban, B. and Weinmann, J. P., Signs of Traumatic Occlusion in the Average Human Jaw, Jnl. Dent. Res., 13:216, 1933.

4. Box, H. K., Experimental Traumatagenic Occlusion in Sheep, Oral Health, 25:9, 1935.

5. McCall, J. O., Traumatic Occlusion, Jnl. Amer. Dent. Assoc., 26:519, 1939.

6. Orban, B., Traumatic Occlusion and Gum Inflammation, Jnl. Perio., 0:39, 1939.

7. Gottlieb, B., Traumatic Occlusion and Rest Position of the Mandible, Jnl. Perio., 18:7-21, Jan., 1947.

8. Bhaskar, S. N. and Orban, B., Experimental Occlusal Trauma, Jnl. Perio., 26:270-284, Oct., 1955.

9. Waerhaug, J., Pathogenesis of Pocket Formation in Traumatic Occlusion, Jnl. Perio., 26:107-118, 1955.

10. Stahl, S. S., Miller, S. C. and Goldsmith, E. D., The Effects of Vertical Occlusal Trauma on the Periodontium of Protein Deprived Young Adult Rats, Jnl. Perio., 28:87-97, April, 1957.

11. Wentz, F. M., Jarabak, J. and Orban, B., Experimental Occlusal Trauma Initiating Cuspal Interferences, Jnl. Perio., 29:117-127, April, 1958.

12. Yuodelis, R. A. et al., The Prevalence and Possible Role of Nonworking Contacts in Periodontal Disease, Periodontics, 3:219-223, Sept.-Oct., 1965.

13. Glickman, I., Inflammation and Trauma from Occlusion: Co-destructive Factors in Chronic Periodontal Disease, Jnl. Perio., 34:5-10, 1963.

14. Glickman, I., Effect of Excessive Occlusal Forces Upon the Pathway of Gingival Inflammation in Humans, Jnl. Perio., 36:141-147, Mar.-April, 1965.

15. Comar, J. D., Kollar, J. and Gargiulo, A., Local Irritation and Occlusal Trauma as Co-factors in the Periodontal Disease Process, Jnl. Perio., 40:193, April, 1969.

16. Bowles, D. et al., Effects on Gingival Fluid of Periodontal Ligament Trauma, Helv. Odontol. Acta, 15:58, April, 1971.

17. Polson, A. M., Effect of Traumatic Injury on the Progression of Marginal Periodontitis, Jnl. Perio. Res. Suppl., 10:17, 1972.

18. Macapanpan, L. C., and Weinmann, J. P., The Influence of Injury to the Periodontal Membrane on the Spread of Gingival Inflammation, Jnl. Dent. Res., 33:263, April, 1954.

19. Lindhe, J. et al., Influence of Trauma from Occlusion on Progression of Experimental Periodontitis in the Beagle Dog, Jnl. Clin. Periodontology 1 (1):3-14, 1974.

20. Orban, B., Bhatia, H., Kollar, J., Wentz, F., The Epithelial Attachment (The Attached Epithelial Cuff), Jnl. Perio., 27:167, 1956.

21. Polson, A. M., Effect of Traumatic Injury on the Progression of Marginal Periodontitis, Jnl. Perio. Res. Suppl., 9:108-113, 1974.

22. Safavi, H., et al., Periodontal Traumatism Produced by Sustained Increase in Vertical Dimension: A Histopathologic Study, Jnl. Perio., 45:207, 1964.

23. Gottlieb, B., Traumatic Occlusion, Jnl. Amer. Dent. Assoc., 14:1276, 1927.

24. Weinmann, J. P., Progress of Gingival Inflammation into the Supporting Structures of the Teeth, Jnl. Perio., 12:71, 1941.

25. Itoiz, M. E., Carranza, F., and Cabrini, R., Histologic and Histometric Study of Experimental Occlusal Trauma in Rats, Jnl. Perio., 34:305, 1963.

26. Kemper, W., Johnston, J., and Van Huysan, G., Periodontal Tissue Changes in Response to High Artificial Crowns, Jnl. Pros. Dent., 20:160-164, 1968.

27. Orban, B., Oral Histology and Embryology, Sixth Ed., C. V. Mosby, 1966, Harry Sicher (ed.).

28. Orban, B., Histology and Physiology of the Gingiva, Jnl. Amer. Dent. Assoc., 44:624, 1952.

29. Orban B., Clinical and Histologic Characteristics of the Gingiva, Oral Surg., 1:827, 1948.

30. Goldman, H., The Topography and Role of the Gingival Fibers, Jnl. Dent. Res., 30:331, 1951.

31. Glickman, I., Smulow, J., Alterations in the Pathway of Gingival Inflammation into the Underlying Tissues Induced by Excessive Occlusal Forces, Jnl. Perio., 33:7, 1962.

32. Glickman, I., Stein, R., Smulow, J., The Effect of Increased Functional Forces Upon the Periodontium of Splinted and Non-splinted Teeth, Jnl. Perio., 32:290, 1961.

33. Gottlieb, B., Orban, B., Tissue Changes in Experimental Traumatic Occlusion with Special Reference to Age and Constitution, Jnl. Dent. Res., 11:505, 1931.

34. Goldman, H. M., Occlusal Trauma, Dent. Clin. No. Amer., March, 1966, p. 79-87.

35. Grant, D. A., Bernich, S., A Possible Continuity Between Epithelial Rests and Epithelial Attachment in Miniature Swine, Jnl. Perio., 40:87, 1969.

36. Goldman, H. M., Histologic Topographic Changes of Inflammatory Origin in the Gingival Fibers, Jnl. Perio., 23:2:104, 1952.

37. Aisenberg, M. S., Aisenberg, A. D., A New Concept of Pocket Formation, O. O. O., 1:1047, 1948.

38. Grant, D. A., Bernich, S., Levy, B. M., Dreizen, S., A Comparative Study of Periodontal Ligament Development in Teeth With and Without Predecessors in Marmosets, Jnl. Perio., 43:162, 1972.

39. Grant, D. A., Chase, J., Bernich, S., Biology of the Periodontium in Primates of the Galago Species, Jnl. Perio., 44:540, 1973.

40. Levy, B. M., Dreizen, S., Bernich, S., Grant, D. A., Comparative Study of the Periodontium of Aged Marmosets and Humans, I. A. D. R. abstract no. 604, Program and Abstracts, 1970.

41. Glickman, I., Clinical Significance of Trauma from Occlusion, Jnl. Amer. Dent. Assoc., 70:607, 1965.

42. Levy, B. M., and Bernich, S., Studies on the Biology of the Periodontium of Marmosets: II Development and Organization of the Periodontal Ligament of Deciduous Teeth in Marmosets, Jnl. Dent. Res., 47:27, 1968.

43. Glickman, I., Weiss, L., Role of Trauma from Occlusion in Initiation of Periodontal Pocket Formation in Experimental Animals, Jnl. Perio., 26:14, 1955.

44. Glickman, I., Smulow, J., The Combined Effects of Inflammation and Trauma from Occlusion in Periodontitis, Int. Dent. Jnl., 19:393, Sept., 1969.

45. Glickman, I., Smulow, J., Further Observations on the Effects of Trauma from Occlusion of Humans, Jnl. Perio., 38:280-293, 1967.

46. Zander, H., and Muhlemann, H., The Effect of Stresses on the Periodontal Structures, O. O. O., 9:380, April, 1956.

47. Waerhaug, J. and Hansen, E., Periodontal Changes Incident to Prolonged Occlusal Overload in Monkeys, Acta. Odont. Scand., 24:91.

48. Breitner, C., The Tooth-Supporting Apparatus Under Occlusal Changes, Jnl. Perio., 13:72-90, 1942.

49. Wilderman, M., Pennel, K., King, K., and Barron, J., Histogenesis of Repair Following Osseus Surgery, Jnl. Perio., 41:551, 1970.

50. Wilderman, M., Wentz, F., and Orban, B., Histogenesis of Repair after Mucogingival Surgery, Jnl. Perio., 31:283, 1960.

51. Staffileno, H., Levy, S., and Gargiulo, A., Histologic Study of Cellular Mobilization and Repair Following a Periosteal Retention Operation Via Split Thickness Mucogingival Flap Surgery, Jnl. Perio., 37:117, 1966.

APPROVAL SHEET

The thesis/dissertation submitted by Dr. Lloyd V. Tilt has been read and approved by the following Committee:

Doctor Anthony W. Gargiulo, Director
Clinical Professor and Chairman,
Periodontics, Loyola

Doctor Patrick D. Toto
Professor and Chairman
General and Oral Pathology, Loyola

Doctor Hal D. McReynolds
Associate Professor,
Histology, Loyola

The final copies have been examined by the director of the thesis/dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the thesis/dissertation is now given final approval by the Committee with reference to content and form.

The thesis/dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Master of Science in Oral Biology.

4-14-80
Date

Anthony W. Gargiulo DDS, M.S
Director's Signature