Occlusal Traumatism: The Histologic Evaluation of the Periodontal Disease Process as Influenced by Local Irritants in Rhesus Monkeys

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OCCLUSAL TRAUMATISM: THE HISTOLOGIC
EVALUATION OF THE PERIODONTAL DISEASE
PROCESS AS INFLUENCED BY LOCAL IRRITANTS
IN Rhesus Monkeys

by

Michael D. Comar

A Thesis Submitted to the Faculty of the Graduate
School of Loyola University in Partial
Fulfillment of the Requirements
for the Degree of Master
of Science
May, 1968
DEDICATION OF THIS THESIS
TO MY PARENTS
DR. AND MRS. M. F. COMAR
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Last, but by no means least, many thanks and the greatest appreciation to my wife, Sandy, for her sacrifices during my two years of graduate study and the typing of this thesis.
CURRICULUM VITAE

Michael Dean Comar, eldest of three boys, was born September 4, 1938 in Three Rivers, Michigan.

Educational training began September, 1943 at Ruth Hoppin grade school in Three Rivers, Michigan, and in June, 1956 he graduated from Three Rivers High School. He then enrolled at Hillsdale College where in 1960 he completed the pre-dental curriculum and obtained the Bachelor of Science Degree. During this time he competed in varsity football and golf, and pledged the Delta Tau Delta social fraternity.

In September, 1960 he began the first of six years of professional study. In 1964 he completed the requirements for the Doctor of Dental Surgery degree at the University of Michigan's School of Dentistry where he was affiliated with the Delta Sigma Delta professional fraternity. He was awarded the Michigan State Dental License by being in the top third of his clinical class and excused from taking the state examination.

Upon completion of dental school he entered the United States Air Force Medical Corps and was awarded the commission of captain and served with the 2795th U.S.A.F. Hospital in Warner Robins, Georgia.

In August, 1966 he entered the Loyola University School of Dentistry to begin a two year program for a Master of Science
degree in the Department of Oral Biology and the certificate of specialization in periodontics.

In July of 1966 he successfully completed the requirements for state licensing in the State of Florida and, in November of 1967, in the State of Illinois.

In November, 1967 he became a member of the American Academy of Periodontics and in the spring semester of 1968 was a graduate instructor in oral pathology.
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CHAPTER I

INTRODUCTION

It is generally believed that a combination of local irritation and superimposed occlusal trauma will produce a more exaggerated tissue change than either the irritation or trauma alone. This has never been satisfactorily shown at the morphologic level and is of great practical significance to dentistry.

The unanswered question still remains in regard to the additional exaggerated effect if inflammation were present during these tissue changes. It has long been an unknown quantity as to the possibility of increased osteoclastic activity on the pressure surface, and if so, in what degree and direction. Also what effect inflammation and trauma have on the tension surface as far as the osteoblastic or osteoclastic activity is concerned.

The study also gives an additional insight into the possibility of altered pathways of inflammation and the role played by the transeptal and crestal fibers of the periodontal ligament and their importance in the crestal resorption phenomena in response to inflammation while a tooth is subjected to a superimposed occlusal trauma.
Of equal importance and often not observed or considered in trauma studies is the effect of these forces on the opposing dentition. This investigation shows that as great or greater an effect can be demonstrated.

Clinical, radiographic, and microscopic methods were employed to evaluate the findings of this study.
CHAPTER II

REVIEW OF LITERATURE

INTRODUCTION

The problem of relating occlusion to the various forms of periodontal disease has been a major point of concern in the area of periodontics since the turn of the 20th century. It was Karolyi\textsuperscript{30} who, in 1901, suggested that excessive occlusal forces lead to "pyorrhea." Since that time there has been voluminous amounts of literature presenting conflicting and sometimes confusing evidence as to the exact role of occlusion in the etiology and management of periodontal disease.

The experimental works have included: 1) experimental studies in animals, 2) studies in humans and 3) \textit{clinical impressions and opinions}.

The initial problem in the 1920's and 1930's was whether or not traumatic occlusion (occlusal forces that produce pathologic changes in the supporting structures) would produce periodontal pockets. Various research data in the past twenty years has fairly well eliminated this possibility.

In the last few years new developments have arisen regarding the possibility of altered pathways of inflammation in relation to occlusal trauma producing angular patterns (vertical) of osseous resorption. This is thought to be caused by a "spilling" of the inflammation response into the periodontal ligament space.
CLINICAL AND MICROSCOPIC STUDIES

Since Karolyi's original suggestion that excessive occlusal forces lead to "pyorrhea" numerous studies have come forth. The early works were carried out by Mosher, 1922; McCall, 1922; Stillman, 1923; Doxtater, 1923; Grove, 1924; Lerner, 1925; Hatfield, 1925; Scherer, 1925; Orban, 1928; Withycombe, 1931; Kronfeld, 1931; Stones, 1938; and others.

In 1925 Hatfield quoted a statement made by Cuvier in the 1800's that, "a proper harmony is needed between organs that act upon one another, not a perfect harmony...." This he used to illustrate that not all dentitions need equilibration or, as he referred to it at that time, grading.

Stillman (1925) believed that occlusal trauma was the original factor leading to periodontal destruction, bacterial invasion, and inflammation resulting in periodontal disease. His treatment was removal of traumatic factors.

Scherer (1925) said traumatic occlusion is generally recognized as the greatest primary factor in the etiology of pyorrhea (periodontoclasia) resulting in hyperemia of the marginal gingiva. He further states, "the presence of hypertrophy of both gingiva and underlying process around the margins of crowns has trauma as its probable causative factor."
Orban and Weinman (1933) studied material from human jaws obtained at autopsy and serially sectioned them. Their observations were: 1) in almost every case where missing teeth were not replaced there were signs of trauma, 2) areas of heavy pressure on the periodontal membrane produced necrosis and resorption of surrounding bone, 3) in most cases trauma did not seem to effect the cementum, 4) hemorrhage and thrombosis of blood vessels were noted, and 5) the most important finding was that there was no connection with inflammatory gingival disturbances or pocket formation.

Box (1935) cemented a crown on the lower incisor of a sheep and showed that excessive occlusal force can lead to calculus formation and the formation of a periodontal pocket.

Stone (1938) reported that excessive occlusal forces produced histologic signs of definite periodontal disease.

In contradiction to these early works a number of experimental studies have shown that excessive occlusal forces do not lead to the formation of pockets, calculus, or gingival disease.

Macapanpan (1952) using a rubber band that served as a separator and foreign body produced a gingivitis. In the presence of tooth movement exceeding its physiologic limits a damage to the periodontal ligament and an alteration of the inflammatory pathways were produced on the side of tension only.
Posselt$^{53}$ (1957) studied the clinical mobility and radiographic changes (maxilla only) associated with trauma. The first bicuspid and the second bicuspid were the areas most commonly involved. He found that mobility was seldom accompanied by radiographic signs and that not all radiographic signs were accompanied by mobility. He also noted a thickening of lamina dura and a widening of the periodontal ligament space simultaneously with mobility seventy-five percent of the time.

Stahl$^{58}$ (1957) studied occlusal stresses in relation to a protein diet. In protein deprived rats with occlusal stress the histologic changes were: 1) disorganization and degeneration of the periodontal ligament fibers 2) thrombosis of the vessels 3) a lack of repair of the periodontium. Occlusal stress with normal diet showed less severe degenerative changes and evidence of repair.

The interproximal alveolar crest height and the position of the epithelial attachment remained unaltered in all groups studied. Studies such as those of Weinmann$^{69}$ (1941), Orban$^{48}$ (1951), Glickman and Weiss$^{12}$ (1955, Bhaskar and Orban$^{2}$ (1955), and Wentz, Jarabak, and Orban$^{71}$ (1958) are just a few of many reportable studies. Consistant findings when occlusal forces act in one direction were as follows: 1) The free and attached gingiva remained unchanged. 2) The structures apical to the alveolar crest showed areas of tension and pressure. 3) The pressure areas are characterized by necrosis of the periodontal
fibers and thrombosis of the blood vessels. These changes are followed by marked resorption of bone, cementum, and some dentin, due to heavy osteoclastic activity. Once the pressure is relieved a reparative process begins and this consists of new ossous tissue and the formation of collagen fibers. 4) On the tension surface there is a stretching (elongation) of the collagen fibers and thrombosis of blood vessels. This area shows heavy osteoblastic activity as bone is rapidly being deposited on the alveolar bone proper. 5) With the occlusal force in one direction the tooth will "move" out of trauma as a new socket is being formed. 6) The epithelial attachment remains unaltered because the collagen fibers immediately apical to the lowest point of the epithelium are not affected by occlusal trauma. 7) The teeth are mobile in the early stages, but later become firm again. 8) There is no gingivitis, periodontitis, or pocket formation produced.

Wentz, Jarabak and Orban71 (1958) produced a buccal-lingual movement by actually jiggling premolars in monkeys. This established a situation different from previous trauma studies since the involved teeth could not move out of the traumatic state. This experiment showed a periodontal space widening to more than three times its original width. The traumatic changes as seen in earlier experiments completely disappeared in three months. There was no evidence of pocket formation, gingivitis, or calculus deposits, and the periodontal ligament appeared normal
except for the increased width. Clinically the teeth became loose within a week and remained so until the end of the experiment.

Glickman\textsuperscript{13} (1962) placed high crowns on six Rhesus monkeys and ran the experiment for 132 days. His results were destruction of transeptal fibers allowing the inflammation to pass into the ligament space on the pressure side \textit{only}. The most involved period appeared at ten days.

Glickman\textsuperscript{14} (1963) showed similar results to his 1962 report regarding altered pathways of inflammation on autopsy material.

Glickman\textsuperscript{14} (1963) stated that, "trauma combines with inflammation to cause destruction of the supporting tissues, thus acting as a co-destructive factor." He went on then to say, "microscopic changes produced by excessive occlusal forces differ from inflammation induced by local irritants from food debris, food impactions, or calculus."

Waerhaug\textsuperscript{67} (1966) and Lefkowitz\textsuperscript{35} (1945) showed that teeth in occlusal trauma by high crowns can be intruded to such an extent that a normal occlusal plane is established. Here again no periodontal pockets were introduced.

Glickman, Smulow, Vogel, and Passamonti\textsuperscript{18} (1966) studied the effects of occlusal forces upon the healing of mucogingival surgery. Surgical procedures were split flap, full mucoperiosteal flaps and gingivectomy. The occlusal effects showed "only"
on the periodontal ligament and alveolar bone. There was no effect on the gingival tissues. The ligament showed widening, increased density, and increased vascularity.

The osseous surface showed remodeled form by a new bone and osteoid in previously resorpted areas. There was an alteration of the labial plate of bone (gingival 1/3 was thinner and apical 1/3 thicker) but this is not believed due to the trauma because it was also seen in the hypofunction control animals.

Hiniker and Ramfjord26 (1966) studied the effect of anterior displacement of the mandible on the teeth and the temporomandibular joint of animals. They concluded that occlusal trauma produces significant adaptive changes in the periodontium but does not alter the temporomandibular joint.

Glickman19 (1967) publications on occlusal trauma are related to the possibility of altered pathways of inflammation in the presence of occlusal trauma. His findings and conclusions are as follows: 1) Periodontal disease introduces inflammation and traumatic changes which in addition to causing bone destruction often alters the angulation of the interdental septum. 2) Angular osseous defects on radiographs are strongly suggestive of occlusal trauma. 3) Traumatic occlusion is reversible, however, in the presence of inflammation it can produce angular and crater-like defects. 4) Furcations are the most susceptible area to occlusal trauma. 5) Occlusion does not effect the inflammation as long as it remains confined to
the gingival tissues. 6) In periodontal disease occlusion governs the condition of the supporting periodontal tissues around the inflammation. In summary he states, "Traumatic occlusion is an integral part of periodontitis rather than an unrelated disease entirely."

Stahl\textsuperscript{57} (1968) studied four jaws, from white males, surgically removed for treatment of oral cancer. All teeth showed clinical evidence of occlusal trauma. However, the inflammatory response progressed into the crestal septum and did not go into the periodontal ligament.

The literature today is constantly filled with various studies relating to occlusion utilizing such methods as radiographic studies, jaw tracings, histopathology studies, postural effects on teeth closure, faceting, centric relation and its relationship to centric occlusion, malocclusion, electronic implants and electromyographic findings.
CHAPTER III
MATERIAL AND METHODS

This investigation was carried out utilizing four adult Rhesus Macacus monkeys about four years of age with permanent dentitions. Monkeys were used due to their close species resemblance to humans and tendency for calculus accumulation when held in captivity.

High crowns without a mesial or distal contact were constructed on premolars to prevent contact between the maxillary and mandibular dentition and to produce a jiggling effect. This resulted in a prematurity closely simulating those seen during occlusal contact and lateral excursions in the human dentition thusly subjecting the supporting structures to an unphysiologic state of occlusal trauma.

The co-destructive factors created to produce this reaction were cast gold crowns with gross marginal overhangs completely around the teeth, and open contacts both mesial and distal of one to two millimeters to encourage food impaction.

The occlusal surface inclines of the cast gold crowns were prepared so that when in occlusal contact with the maxillary dentition the mandibular crowned premolars were forced mesially. The inclined plane also prevented any articular contact between the remaining maxillary and mandibular teeth. (Plate I Figure 2 and 3)
The mandibular right second premolars were used on two monkeys and the mandibular left second premolars on the other two. The reason for using opposite sides was due to previous damage to the mandibular right second premolars.

Teeth were prepared to receive a full cast gold crown. The copper band impression technique was utilized and a stone die constructed. The crown was then fitted, polished, and cemented the following week.

Various procedures such as crown preparation and casting cementation were done under a general anesthetic utilizing two to four cubic centimeters of sodium nembutal (25mg/cc) solution given interperitoneally.

Radiographs were taken to show osseous and periodontal space changes at 1, 2, 6, 7, 11, 12, 13 weeks.

Along with the radiographic study, clinical evaluations were made at weekly intervals as to:

1. Color of the gingival tissues
2. Depth of periodontal pocket (epithelial migration apically from C.E. junction).
3. Tooth mobility (rated from 1 to 4)
   A. Apically
   B. Bucco-lingually
   C. Mesio-distally
   D. Depression of tooth
   E. Any tooth migration that occurred

Kodachrome color clinical photographs were taken of the experimental areas at various times to record the clinical
change. Film with an ASA-25 at three inches, F-22 and 1/30 seconds were used.

Radiographs were also taken to visualize the structural changes that could be observed. The machine was set at KV 65 for 1/3 seconds using radiatized film.

Post entry sacrifice times of the experimental animals were:

Five days (Plate III Figure 6 A to Plate IV Figure 6 D)

Two weeks (Plate V Figure 7 A to Plate VII Figure 7 D)

Three weeks (Plate VIII Figure 8 A to Plate X Figure 8 E)

Fourteen weeks (Plate XIII Figure 10 A to Plate XV Figure 10 E)

The monkeys were sacrificed using fatal doses of nembutal injected into the heart. At this time the head was removed and the maxillae and mandible were immediately bisected, washed in clear water, immersed and allowed to remain in 10% formalin solution for a two week period.

Histologic specimens were obtained by the following method:

Fixation - 10% neutral formalin solution

Decalcification - large quantities of a 5% aqueous nitric acid solution

Observed - every two days until completely decalcified. Specimen washed in running water for twenty-four hours, then neutralized with 10% formalin solution to which an excess of calcium or
magnesium carbonate has been added, again washed in water for twenty-four to forty-eight hours.

Dehydration - 75% alcohol (twenty-four hours)
95% " " " "
100% " " " "
Ether "(50/50)" " "

Embedding - Celloidin
Thin - one week
Medium - " "
Thick - " "

The sections were prepared and stained with hematoxylin and eosin. Photomicrographs were then taken of the histologic preparations at various magnifications.
CHAPTER IV
FINDINGS

A. Five Days with High Crown

1. Macroscopic Findings

After five days the monkeys gingival tissues were redder than normal indicating an inflammatory response due to the crown margin overhang which has produced a home for bacteria and can act as an irritant. Careful probing with a scaled Fox probe showed no change in the pocket depth from either the epithelial attachment migrating apically or a pseudopocket formation due to gingival tissue hyperplasia.

The mobility of the crowned mandibular second premolar had increased. Using the method of from one to four, as presented in Orban's Periodontics, a reading of plus-two was obtained in a buccal to lingual direction. Clinically the crowned tooth already appeared slightly depressed into its alveolus.

2. Microscopic Findings

a. Mesial Root

The epithelium remains intact with its attachment on the cemental surface and its rete pegs have elongated. The vessels are compressed to a greater degree on the mesial surface than the distal, and the principal fibers of the periodontal ligament are compressed with some fibers taking a more parallel
orientation. Resorption occurs only in the crestal area. The marrow spaces of the mesial crest shows a moderate number of inflammatory cells with new osteoid being laid down. (Plate III Figure 6 A)

On the distal surface the vessels have dilated, some being filled with red blood cells and a clear eosinophilic edematous material. Here again the principal fibers have obtained a more parallel arrangement, and near the furcation a decrease of fibroblasts are seen. New osteoid is being laid down and the osteoblasts have lined up along the marrow spaces. The marrow spaces also contain a heavy round cell inflammatory response.

b. Apical Area

The apical area of the mesial root shows an increased widening of the periodontal ligament space which is now filled with dense fibrous tissue, fibroblasts, and a heavy vascular concentration replacing lost osseous tissue. Also present are numerous small islets of from six to nine cells which resemble epithelial rests.

c. Interradicular Area

The result of the osteoclastic activity is seen with resorption of the crestal bone and cementum. These tissue losses have been replaced by fibrous connective tissue and dilated blood vessels. The periodontal ligament fibers that remain show a complete disorientation and some areas have a
complete loss of nuclei and appear hyalinized. The osseous
crest appears in contact with the cementum due to a loss of
interradicular soft tissues. (Plate IV Figure 6 D)

d. Distal Root

The distal surface shows a widening of the
periodontal ligament space mainly in the apical and coronal
areas. The cementum is intact ending at the epithelial
attachment. The round cell inflammatory response is generally
walled off by the presence of the still intact transeptal
fibers which confines most of the inflammation to the sub-
epithelial area above. Some inflammation is seen adjacent to
the vascular canals between the transeptals and into the
periodontal ligament for a short distance before entering the
osseous structure. The presence of well organized horizontal
fibers is missing and a large number of transeptal fibers
have undergone a loss of fibroblasts.

Throughout the periodontal ligament are dilated
vessels filled with edematous fluid and erythrocytes. Epithelial
rests are present. Osteoblasts are organizing and lining up
along the alveolar bone proper, however, osteoclastic activity
is occurring at the alveolar crest. The general response is
greater here (tension side) than on the mesial surface of the
mesial root. (Plate III Figure 6 B)

The mesial surface shows dilated blood vessels
and epithelial rests in the periodontal ligament. Some areas
of fiber disorientation are seen. There is almost complete degeneration of the pulpal tissue, particularly in the pulp chamber proper.

B. Two Weeks with High Crown

1. Macroscopic Findings

After two weeks the gingival tissues have undergone a fibroblastic proliferation along with a change in color from the normal pinkish to a deep red and the formation of a periodontal pocket depth of one millimeter on the lingual and buccal, and two millimeters on the mesial and distal. The epithelium has not migrated apically rather the fibroblastic proliferation has produced a pseudopocket due to the irritating factors introduced. Mobility recorded as a plus-three in a buccal-lingual direction, and a plus-one in the mesial-distal direction. The tooth is also depressed approximately one to two millimeters into the alveolus. (Plate I Figure 3)

2. Microscopic Findings

a. Mesial Root

The mesial surface shows a generalized increase in the periodontal ligament space, especially in the creseal and apical areas, with hemorrhaging into the principal fibers due to vascular damage near the alveolar bone proper. The interdental papillae shows extensive enlargement due to a fibroblastic and endothelial proliferation in the gingival
fibers. The transeptal fibers appear disorganized with some hyalinized areas, hemorrhage into the fibers, and heavy fibroblastic proliferation. However, the moderate degree of round cell inflammation is maintained subepithelial and above the fibers. The inflammation has not passed into the periodontal ligament proper. The osseous response is of osteoclastic activity, mainly in the crestal area, with osteoid being laid down by osteoblasts within the narrow spaces and on the periodontal ligament surface near the apex. The crestal marrow spaces have lost their adipose tissue and loose connective tissue, being replaced with fibrous tissue and numerous small blood vessels. Surface resorption, in place of undermining resorption, is probably due to the extremely rapid and intermittent traumatic nature of the assuiting forces.

The distal surface shows the periodontal ligament fibers more parallel to the root surface in orientation with large dilated vessels. Near the furcation the ligament fibers tend to lose their nuclei and other characteristic features taking the form of ligament necrosis. Here again the marrow spaces are being replaced with fibrous tissue.

b. Interradicular Area

This area shows only a small islet of osseous tissue remaining as most of the furcal osseous tissue has undergone osteoclastic resorption and is replaced with endothelial and fibroblastic proliferation in the form of many
small dilated vessels, collagen fibers, and many fibroblasts. The residual piece of osseous tissue is atypical with empty lacunae. The distal furcation area has the tooth in near contact with the interradicular osseous tissue that is left, and the periodontal ligament of this area has undergone the process of hyalinization appearing without nuclei and as a homogenous pink staining mass. The surface of the osseous furcal tissue remaining shows both osteoclastic and osteoblastic activity while within the marrow spaces osteoid is being deposited. (Plate VII Figure 7 D)

c. Distal Root

The mesial surface shows the periodontal ligament compressed to the point of non-existence. The apical area shows an enlargement of the periodontal ligament. Osteoclastic activity is seen at the distal root apex along with cemental resorption. Hemorrhaging is seen as erythrocytes have escaped into the ligament tissue.

The distal surface shows a generalized enlargement of the periodontal ligament especially in the crestal area due to osteoclastic resorption. (Plate V Figure 7 A) Osteoclasts are seen on the alveolar bone proper surface with osteoblasts and osteoid lining the marrow spaces. The marrow spaces have undergone an increase in fibrous tissue replacing the loose connective tissue. The ligament vessels are dilated and some show tears in the vessel lining allowing the release
of erythrocytes into the ligament. Heavy fibroblastic proliferation is seen within the ligament, and near the crest the ligament fibers have lost their nuclei and started to undergo hyalinization. Also in the crestal area are small islets of osseous tissue that have gotten separated from the alveolar bone. Near the cementum are seen numerous epithelial rests.

The transeptal fibers, as on the mesial root, have taken a more oblique pattern due to the tooth being depressed in the alveolus with some of the fibers appearing to undergo hyalinization. However, the fibers are still generally intact and keeping the subepithelial round cell inflammation from reaching the periodontal ligament proper. (Plate V Figure 7) The pulpal tissue is unaltered wherein the five day specimen shows extensive degeneration.

C. Three Weeks with High Crown

1. Macroscopic Findings

At three weeks the gingival color is a deep red as an inflammatory response to the open contacts and crown overhangs. The pseudopocket formation due to fibroblastic proliferation in the gingival tissues has created a two millimeter pocket buccal and lingual while maintaining the two millimeters on the mesial and distal surfaces. The tooth depression appears slightly increased, but not a great amount over the two week specimen. (Plate VIII Figure 8) Tooth mobility
now measures a plus-two in all directions. The monkey has been able in just three weeks to almost re-establish an occlusal plane after having a high crown placed opening the bite two millimeters. This was accomplished by depressing both the mandibular and maxillary teeth at the expense of root, cementum, and alveolus.

2. Microscopic Findings
   a. Mesial Root

   The mesial surface shows a very thin remaining epithelium that has proliferated under the crown overhang left to act as an irritant. The transeptal fibers have lost some of their original orientation and new fibroblastic activity is occurring with collagen fibers being formed. These appear to be undergoing some degree of reorganization. The transeptals still appear to keep the subepithelial inflammation coronal. These fibers no longer enjoy their normal horizontal position but a more oblique pattern due to tooth depression. The fibers being on the pressure surface show more disorientation than those on the tension side of the distal root. (Plate IX Figure 8C)

   The crestal area shows osteoclasts producing crestal resorption with other areas of such activity along the ligament proper. However, the general pattern on the ligament surface is one of osteoblastic activity with new osteoid formed. Along with the increased fibroblast and endothelial activity the periodontal ligament fibers have obtained a more parallel
positioning. The cementoblasts have aligned and laid down a thin layer of cementoid.

The apical and of the mesial root is in close proximation with the alveolus. Here the fibers are disorganized showing an extreme cellular and vascular response. The microvessels are dilated and some have been injured releasing hemorrhage into the ligament of the apex. Osteoclasts are actively removing osseous tissue on the periodontal ligament surface. In the near marrow spaces osteoblastic activity is active with osteoblasts lined up and laying down osteoid. Some ligament fibers have undergone necrosis and hyalinization.

The distal surface shows where dentin and cemental resorption along with compression of the periodontal ligament fibers has occurred. Hemorrhage within the fibers of the ligament and new osteoid and cementoid are seen on the periodontal ligament surfaces.

b. Interradicular Area

The interradicular findings show the tooth depressed to the point it has compressed the ligament causing it to degenerate and now the cementum is nearly in contact with the osseous tissue of the furcation. Areas of dentin, cementum, and interradicular osseous resorption are seen on the ligament surface. (Plate X Figure 8 D) The resorbed tissues are replaced by a fibrous-vascular connective tissue. Hemorrhage and necrotic appearing islets of bone are present in the area.
Osteoblastic activity and new osteoid are seen within the fibrous marrow spaces. The pulpal tissues appear unaffected by the occlusal trauma.

c. Distal Root

The mesial surface exhibits excessive osteoclastic activity and numerous large multinuclear osteoclasts, whose osteoclastic activity has caused the periodontal ligament space to become enlarged. Osteoblastic activity is seen laying down osteoid in the interradicular marrow spaces. The marrow spaces have lost their normal loose connective tissue which is replaced by a fibrous tissue in areas near the furcation. This type surface resorption and marrow space response is again probably due to the excessive trauma.

The apical and distal surfaces show enlargement of the periodontal ligament proper. (Plate VIII Figure 8 and Plate IX Figure 8 B) The periodontal ligament's principal fibers are elongated due to the developed tension. Here the picture is truly one of proliferation and repair. The periodontal ligament is undergoing extensive fibroblastic proliferation, while the ligament surface of the alveolus is rapidly depositing new osteoid with many osteoblasts lined up along the alveolar bone proper forming new bone. (Plate IX Figure 8 B) This is also seen on the alveolar crest which is undergoing a remodeling. The transeptal fibers are undergoing fibroblastic proliferation and new collagen fibers are being laid down.
Here again both the transverse (horizontal) fibers of the periodontal ligament and the transeptal fibers are lined up more obliquely than normal due to the tooth's being depressed. (Plate VIII Figure 8 A and Plate IX Figure 8 C) The subepithelial round cell infiltration, consisting of plasma cells and lymphocytes, is still walled off from the ligament proper. (Plate VIII Figure 8 A) Here as on the mesial root the epithelial covering is just two to five layers thick and has proliferated under the overhanging crown margin. Pulpal tissue does not appear to have undergone degeneration due to the trauma.

D. Effects on Opposing Dentition - Three Weeks

1. Macroscopic

Although no attempt was made to accurately measure the depression in the maxillary arch, it appears clinically that the maxillary second premolar was depressed one to two millimeters into its alveolus at this time interval. (Plate XI Figure 9) Also the mandibular second premolar was depressed.

2. Microscopic

a. Mesial Surface (one rooted tooth)

The mesial surface of the second premolar shows a narrowing of the periodontal ligament space except at the alveolar crest. (Plate XI Figure 9A and Plate XII Figure 9 C) The epithelium remains intact with a moderate inflammatory
response between the epithelium and the transeptal fibers. The transeptals have maintained intactness with the cementum and now have taken a more oblique direction due to the premolar being depressed. Below the transeptal fibers a heavy fibroblastic and endothelial proliferation is seen. New collagen is being laid down with an increase in dilated blood vessels. The crestal osseous tissue on the periodontal ligament surface and the alveolar crest itself shows many large multi-nucleated osteoclasts resorbing the surface of the alveolar bone proper. (Plate XI Figure 9 A) Deep to the osteoclastic activity osteoblasts have oriented themselves and are depositing new osteoid along the surface of the marrow spaces. The principal fibers of the periodontal ligament appear compressed with some areas of nuclei loss. The general picture of the fibers is one of more parallel orientation to the root surface.

b. Apical Area

This area has an increase in the ligament space with numerous small dilated vessels and a lack of apical fiber orientation. The osseous picture is one of resorption and repair occurring together. (Plate XI Figure 9 B) The osteoclastic resorption is on the ligament surface of the alveolar bone proper, while within the marrow spaces osteoblasts have aligned themselves to the surface of the bony structures and are depositing new areas of osteoid. Many large, light staining nuclei of young fibroblasts are seen laying down young
reticular fibers.

c. Distal Surface

The response here is basically the same with a widening of the ligament space in the alveolar crest and apical area. (Plate XI Figure 9) The area of the periodontal ligament proper shows areas where cementum and dentin have undergone resorption and now some repair is occurring. (Plate XII Figure 9 D) The ligament fibers are elongated in some areas and unorganized and disoriented in others. The osseous tissue adjacent to the ligament proper shows heavy osteoclastic activity, as is also seen in the crestal areas except for a few areas of osteoblastic activity. The marrow spaces, adjacent again, shows new osteoid formation but to a greater degree. The principal fibers at the crest appear disorganized, however, they still maintain the inflammation coronally.

The general pattern of activity at three weeks is one of heavy osteoclastic surface resorption with corresponding osteoblastic activity within the adjacent marrow spaces. The reason for the heavy surface resorption is probably due to the extreme trauma applied so rapidly and in such a short time. The pulpal tissues appear to have not been effected by the excessive trauma.

E. Fourteen Weeks with High Crown

1. Macroscopic Findings
Fourteen weeks showed the gingiva had obtained a more pink color losing the red color seen at two and three weeks even though the cast crown was still in place. The gingival tissues have proliferated under the crown margins, (Plate XIII Figure 10 A and Plate XIV Figure 10 B) but the pocket depth is no longer present. The tooth mobility is now a plus-three in all directions. There does not appear to be any further depression of the mandibular tooth than with the three week animal. (Plate II Figure 4) It is believed any further depression probably took place in the maxillary dentition. This is shown nicely in the ninety-eight day microscopic jaw section of the opposing second premolar well depressed into its alveolus. (Plate XVI Figure 11)

2. Microscopic Findings

a. Mesial Root

The mesial surface shows the gingival interdental papillae proliferated to a greater extent than previous time intervals under the overhanging crown margin. (Plate XIII Figure 10 A) The papillae still maintains a thin epithelial covering which extends into the sulcus and appears attached to the dentinal tissues until it terminates with acellular cementum. The epithelium has undergone elongation of the rete pegs and proliferates so that various epithelial islets appear. The subepithelial connective tissue has taken on an increased vascularization with a heavy round cell infiltration.
(Plate XIII Figure 10 A) At the tip of the alveolar crest toward the adjacent first premolar some multinucleated osteoclasts are seen and active resorption of the crest is occurring. The crest area near the cementum and the periodontal ligament proper both show osteoblasts lined up parallel to the tooth depositing new osteoid and osseous tissue. The dominant picture of this area is one of repair.

The horizontal fibers of the periodontal ligament proper are undergoing rearrangement due to some loss of fiber orientation. (Plate XIV Figure 10 C) The vessels appear to have dilated more than previous time intervals and are filled with red blood cells and edema. Cementoblasts are lined up and laying down new cellular cementum in previous areas of cemental resorption.

The apical area shows an increase in width of the periodontal ligament space as does the alveolar crest area. Fibroblastic and endothelial proliferation of connective tissue has replaced the resorbed cemental and osseous tissue which is now undergoing repair.

b. Interradicular Area

Here is seen a gross destruction of the interradicular crestal osseous tissue which is replaced by small dilated blood vessels and a dense fibrous connective tissue showing a heavy fibroblastic proliferation. (Plate XV Figure 10D) There are numerous areas of cemental and dentinal resorption
in which the lost tissue is now being replaced by cellular cementum. (Plate XV Figure 10 E) This, along with the osteoblastic activity of the marrow spaces and surface of the periodontal ligament, is reestablishing a normal periodontal ligament space dimension as it was before the traumatic assault.

c. Distal Root

The mesial surface shows repair of cemental osseous resorption and a normal orientation of the periodontal ligament fibers. (Plate XV Figure 10E) The apical area shows a widening of the periodontal ligament space by osseous resorption of which some osteoclastic activity and the presence of large multinuclear osteoclasts exist removing islets of osseous tissue in the area. The cementoblasts are laying down cellular cementum in the apical area. The apical fibers have a disoriented appearance and there is some fibroblastic proliferation producing an increase in fibroblasts and collagen fibers.

The distal surface shows a widening ligament space which is returning to a normal width with osteoblasts depositing new osteoid. The alveolar crest area shows a widening of the ligament space with the transverse fibers of the ligament proper and transeptal fibers taking a more oblique direction due to depression of the premolar. (Plate XIV Figure 10 B) The crest is now undergoing osteoblastic repair. The
transeptals even though they have a more oblique pattern and show areas of hyalinization remain intact and keep the heavy inflammatory infiltration of round cells coronal and subepithelial except where some of the transeptals are torn from the cementum and here the inflammation has passed down to the intact fibers. (Plate XIV Figure 10 B) The interdental papillae has undergone hyperplasia in response to the crown margin irritation. The epithelial is much thicker than the mesial roots and there is some replacement of the subepithelial connective tissue with fibroblastic and endothelial proliferation of young collagen fibers and small dilated vessels although the inflammatory response is still extensive. (Plate XIV Figure 10 B) The general picture seen with the fourteen week monkey, except for a few exceptional areas, is of repair. Lost tissues are replaced by cementoblasts, osteoblasts, fibroblasts, and endothelial proliferation. Since the majority of response seen during this period demonstrates repair, it strongly suggests the second premolar with the high crown has reached a point where the traumatic forces are no longer being applied. This is accomplished by depression of both the involved maxillary and mandibular teeth.

F. Effects on the Opposing Dentition at Fourteen Weeks

1. Macroscopic

The maxillary second premolar appears clinically to be depressed about one or two millimeters. The exact measurements
are not available because the exaggerated effect on the opposing dentition was not expected and, therefore, no pre-operative markings were made to measure from. (Plate II Figure 4)

2. Microscopic
   a. Mesial Root

   The mesial surface shows the epithelium intact with a heavy subepithelial inflammatory infiltration which remains above the transeptal fibers. The transeptals have taken a more oblique direction due to depression of the premolar but they have remained intact to the cemental root surface. (Plate XVI Figure 11 and Plate XVII Figure 11 B) The crestal area shows the horizontal fibers of the ligament proper also in a more oblique direction with an increased vascular response. The vessels are dilated and filled with an edematous material and erythrocytes. The periodontal ligament space is widened in the crestal area due to osteoclastic activity but now new osteoid is being deposited. Near the apex excessive cemental and dentinal resorption has occurred due to the gross traumatic assault, which appears to have driven the mesial root through part of the distal root of the adjacent premolar. (Plate XVII Figure 11 A) This has isolated a piece of the alveolar bones between the roots which is now surrounded by osteoblasts and new osteoid. (Plate XVII Figure 11 B) The first premolar also has undergone resorption of cementum at its apex and is still active with numerous large multinucleated osteoclasts on the surface.
The apical area of the second premolar and the resorbed areas are undergoing repair with cementoblasts laying down new cementoid and osteoblasts laying down osteoid on the apical bone surface. The tissue of the ligament proper is extremely vascular with loss of the fiber arrangement and numerous dilated blood vessels filled with an edematous material.

The distal surface shows a loss of the periodontal ligament fiber orientation, osteoblastic activity of the alveolus, and many dilated vessels which are filled with erythrocytes and edema. The apical area has a thick area of cellular cementum which may or may not be associated with the traumatic forces.

b. Interradicular Area

There is a notching of the tooth surface due to cemental and dentinal resorption. The osseous tissue of the furcation has been resorpted so that only a fine point of tissue is left while other small islets of osseous tissue are seen adjacent to the ligament. (Plate XVI Figure 11) Vessel congestion and dilation has occurred and the crest and various islets of bone are now undergoing repair with osteoblasts lined up on their surface and laying down new osteoid.

c. Distal Root

The mesial surface shows enlargement of the periodontal ligament space near the apical area. The ligament vessels seem to be grossly dilated and the fibers have lost
their normal orientation. The cementum in the apical area is thick as similarly seen on the mesial root. The resorbed cementum and osseous tissue is undergoing repair by replacement with like-tissues. The apical end of the distal root shows the area of greatest ligament widening due to osseous resorption. This is being corrected by fibroblastic proliferation and osteoblasts laying down new osteoid on the apical area of the alveolar bone proper.

The distal surface shows a widening in the apical area of the ligament space with fiber disorientation and numerous dilated vessels and osteoid laid down on the periodontal ligament surface of the alveolus. (Plate XVI Figure 11) The transeptal fibers, even though these are more oblique, remain fairly well intact keeping the inflammatory response above the fiber arrangements, however, inflammation is seen passing through the transeptal adjacent to the blood vessels. Osteoblasts are laying down osteoid on the crestal surface and there are small islets of osseous tissue in the area. Cementoblasts are depositing new cementum along the resorbed root surfaces and the epithelial attachment remains intact on cementum. The epithelium is of more normal thickness with a heavy round cell infiltration between the epithelium and above the transeptal fibers. The general histological picture of the opposing dentition at fourteen weeks is basically one of repair. This suggests the teeth are no longer in a traumatic state as the
same soft and hard tissue responses are seen with the mandibular second premolar with the high crown at this period. It should be noted here that on the distal surface of the first premolar inflammation is seen passing into the periodontal ligament proper after passing through the transeptals, not due to fiber disorientation or destruction, but adjacent to the blood vessels in the loose connective tissue. This is seen in a non-traumatized tooth.

The maxillary second premolar's mesial root, though not into the antrum, has been depressed to such a degree that there is very little osseous tissue left protecting the antrum. Opposite the mesial root tip in the antrum is a large darkly staining mass resembling muscle which may or may not be a response to the trauma. (Plate XVI Figure 11 and Plate XVII Figure 11 A)

The elastic activity in the maxillary dentition seems to be more prevalent at this time period than in the mandibular arch. This suggests the possibility that any tooth depression after three weeks was probably in the maxillary arch.
CHAPTER V
DISCUSSION

Although many aspects of this investigation developed interesting findings, the most significant must be considered the response of the transeptal fibers. These fibers showed themselves to be possibly the most stable part of the periodontium in response to occlusal forces of a traumatic nature.

In all of the specimens involving the crowned teeth and their antagonists the subepithelial inflammation was maintained between the stratified squamous epithelium and the transeptal fibers (Plate VIII Figure 8 A) except where the inflammatory cells were adjacent to the blood vessels in the intracrestal osseous marrow spaces. (Plate III Figure 6 A, 6 B and Plate IV Figure 6 C) In general the transeptal fibers were intact except in the five day monkey where some disorientation was seen on the tension side. This is similar to the work of Macapanpan, except no alteration of the inflammation into the periodontal ligament space was observed due to fiber arrangement or reorientation.

The transeptal fibers during both the destructive and reparative periods appear to be the greatest protective mechanism the crestal osseous tissue has. The impression is
given from the histologic sections that without these fibers and their ability to withstand excessive forces a greater amount of osseous destruction could have occurred. Thus, it can be suggested that not only may these fibers have some supportive function and, as some suggest, may be responsible for the crestal osseous contour, but also that protection of the remaining periodontium may be their main function.

The fibers apparent durability was also shown by Ramfjord and Kohler's study on human teeth. They concluded that "the most stable periodontal structure with regard to functional changes in occlusal stress appear to be the Sharpey's fibers entering the cementum and the periodontal fibers coronal to the margin of the alveolar crest."

An investigation by Garguilo and Orban of the dentogingival junction in humans showed it to be a dynamic area with alterations throughout life except for the dentogingival transeptal fibers which maintained approximately a one millimeter width, and did not appear subject to change under either physiologic or stress circumstances. This has further been reaffirmed in this investigation where the integrity of the dentogingival junction has been maintained almost entirely.

This investigation in general supported Weinman's classical work regarding the distribution of the inflammatory cells along the blood vessels passing in the loose connective tissue. This study differs only in regard to the direction
the inflammatory cells are taking. Weinman suggests that inflammation travels from the marrow spaces to the periodontal ligaments, whereas, this study suggests that the majority of the inflammatory cells are produced locally from young peri-vascular cells and transverse from the gingiva to the marrow spaces or periodontal ligament in the loose connective tissue surrounding the blood vessels, or as Toto\textsuperscript{65} suggests, that the inflammatory cells are produced locally in the gingiva or ligament due to a foreign stimulus. A recent study by Akiyoshi\textsuperscript{1} also supports the concept of inflammation in relation to the blood vessels.

The second aspect of importance relating to the area of periodontics and occlusion was the histopathologic results obtained regarding the possibility of altered pathways of inflammation. Intraosseous pockets (angular resorption) and altered pathways, as reported by Glickman\textsuperscript{13, 14, 15, 17}, were not reproduced in this study. Inflammation was seen in the five day monkey entering the periodontal ligament in the crestal but limited only in the loose connective tissue surrounding the intra-gingival and crestal vessels. (Plate III Figure 6 A and Figure 6 B) No inflammation was seen leaving the blood vessels and "spilling" into the periodontal ligament space as previously reported. This finding was also supported by Stahl's report of four human jaws surgically removed because of cancer. The teeth were clinically diagnosed as being in a traumatic state, but when studied histologically inflammation in the
periodontal spaces was either very limited or non-existent, even though the crestal periodontal space was altered due to pressure.

The question then arises -- could the inflammation actually have been following the blood vessels into the periodontal ligament or produced locally in other studies reported instead of being due only to fiber disorientation? Also can the inflammation in the ligament space reported in this study be considered as altered pathways due to occlusal trauma that might produce vertical osseous resorption and intraosseous pocketing? In this study it probably should not be considered an altered pathway for three reasons: 1) Because it didn't produce an intraosseous pocket in the four specimens utilized. 2) One may expect some inflammation in the periodontal ligament space because of the blood vessels anastomosing from the periodontal ligament and gingival tissues allowing a loose connective tissue pathway. 3) Inflammatory cells were seen in the periodontal ligament closely following the blood vessels in a first premolar, not in occlusal trauma. (Plate IV Figure 6 C)

Glickman\textsuperscript{14} pointed out that necrosis of the fibers prevents pathway alterations by producing a physical blockage. In this investigation areas of fiber hyalinization were produced, but these were in isolated locations and not generally believed to have created a physical blockage problem.
Another revealing finding was the traumatic assault of the high crown on the supporting tissues in the opposing dental arch. (Plate XVI Figure 11 and Plate XVII Figure 11 A) The literature review uncovered little on the histologic effects of a high crown on the opposing dentitions, however, the effect appears to have been as great if not greater. The opposing dentition demonstrated a moderate degree of inflammation, probably due to the monkeys inability to establish oral hygiene. Here, even with the gross histological changes seen due to the traumatic insult and inflammation present, no altered pathways were produced. It would seem that with or without a high crown this also would be an ideal place to see angular resorption if such were to occur.

At the fourteen week interval the maxillary and mandibular teeth were depressed so that a functional occlusal plane was re-established allowing the traumatized tissues to undergo repair. (Plate II Figure 5) Depression of teeth in a traumatic state was also reported by Waerhaug\textsuperscript{67} and Lefkowitz\textsuperscript{35}.

The furcation areas showed gross destruction histologically with little or no inflammation present, (Plate IV Figure 6 D) strongly suggesting occlusal trauma as its etiology. Glickman's studies support this finding both histologically and radiographically.

The histologic changes seen regarding the epithelium, periodontal ligament, pressure, and tension surface were the
same as previously written in numerous articles on occlusal trauma. Changes seen along the cementum (cementoclastic) were common especially in the opposing arch. (Plate IV Figure 6D and Plate X Figure 8D) This doesn't agree with Orban and Weinman\textsuperscript{47}, who believed that trauma generally does not effect the cementum. The difference was probably due to the severity of the occlusal and directional stresses applied.

The crestal osseous involvement was different since almost all the areas of active osteoclastic resorption were on the alveolar bone proper surface. Undermining resorption as normally seen in a periodontitis was minimal. While the surface was undergoing resorption, heavy osteoblastic activity was depositing new osteoid along the osseous surfaces within the marrow spaces. This change in the resorptive pattern was most likely due to the excessive amount of occlusal stress.

The most destructive period appeared to be from the fifth to the fourteenth day. This is similar to previous animal investigations reported, and to Glickman's\textsuperscript{13} monkey study where the altered pathways appeared at ten days due to transeptal and periodontal ligament fiber disorientation.

It is observed that inflammation tends to follow the blood vessels regardless of whether or not trauma is present and has little, if any, effect on changing the "normal" path of inflammation.

This study shows that occlusal trauma can be a very damaging
process with or without inflammation and must be recognized clinically and radiographically so that proper treatment can be carried out. This does not mean prophylactic occlusal adjustment, but rather the removal of the pathology by establishing a functional occlusion when necessary. It must also be remembered that the occlusal forces in the investigation were constructed to be excessively traumatic, more so than would be found in the human dentition.

We now know from this and other investigations that the presence of inflammation and occlusal trauma does not appear to alter the integrity of the dentogingival complex. The destruction and detachment of the transeptal fiber and apical migration of the epithelium did not occur. To destroy the dentogingival complex it appears that local irritants such as bacterial toxins or calculus, and/or, systemic factors are needed -- not occlusal trauma.

Although research material is limited at this time regarding altered pathways, the possibility must be considered, proven, disproven, or explained such as this investigation has attempted to do. It is the hope that the results of this study will aid in a better understanding of occlusal forces plus the corresponding active inflammatory disease process.
CHAPTER VI
SUMMARY

The progress of inflammation and the type and degree of destruction of the periodontium was studied in four Rhesus monkeys utilizing a high cast gold crown on the mandibular right second premolar. A mesial force incline was established on the crown and a built-in means of producing local irritation was created using open mesial and distal contacts (one to two millimeters) and marginal overhangs.

Sacrifice times of five days, fourteen days, twenty-one days, and ninety-eight days were used for histological evaluation. The monkeys were also evaluated by clinical and radiographic means.

The investigation produced the following findings:

1) Clinically and histologically a depression of the maxillary and mandibular teeth was observed until a functional occlusal plane was re-established.

2) Clinical probing and histologic evaluation showed no apical migration of the epithelial attachment.

3) With excessive occlusal forces the transeptal fibers may take a more oblique position due to tooth depression, but in general remain intact and act as a wall protecting the osseous tissue from the subepithelial inflammation present due
to lack of the monkey's cleansing ability and built-in local irritants.

4) Inflammation in both traumatic and nontraumatic areas appeared in the loose connective tissue around the blood vessels, and in some cases entered the periodontal ligament for a short distance.

5) Neither the maxillary nor mandibular periodontiums showed a "spilling" of inflammatory cells into the periodontal ligament space due to fiber disorientation as has been reported in previous studies.

6) Gross destruction of the interradicular areas occurred due to occlusal trauma.

7) Occlusal trauma had as great an effect on the periodontium in the maxillary arch as in the mandibular arch where the high crowns were placed.

8) The five to fourteen day period is that of the greatest destruction whereas the ninety-eight day monkey showed predominantly repair of the periodontium. This strongly suggests that the involved teeth had reached a nontraumatic point.
CHAPTER VII
CONCLUSIONS

After careful evaluation of the clinical, radiographic, and histologic evidence the following may be concluded:

1) The inflammatory cells for the most part extend from the gingival tissues into the marrow spaces in the loose connective tissue around the blood vessels regardless of whether the teeth are in a traumatic or non-traumatic state.

2) Occlusal trauma does not produce periodontal osseous or soft tissue pockets.

3) Occlusal trauma does not appear to alter the pathway of inflammation into the periodontal ligament space due to fiber re-orientation.

4) The inflammation "may appear" to be altered when it follows a blood vessel that enters the alveolar crest from the periodontal ligament side rather than the very crestal tip or is produced locally in the ligament.

5) Excessive occlusal forces in the presence of inflammation can produce an active surface osseous resorption with osteoblastic activity in the marrow spaces instead of the undermining osseous resorption commonly found in periodontal disease.

6) The destructive response may be as great or greater to the teeth and their periodontiums opposite the high crowns.
7) Occlusal trauma alone can cause destruction of the interradicular crest area which on a radiograph may appear the same as furcation involvements due to inflammation.

8) In general the structures of the periodontium below the transeptal fibers are subject to possible destruction or damage by occlusal trauma. The transeptal fibers and above are influenced mainly by the inflammatory response and local irritants (such as calculus or bacteria toxins) not occlusal trauma.

9) A great deal more research is needed before it may be safely stated that traumatic occlusion plus inflammation can be cited as the etiology of intraosseous pockets.
CHAPTER VIII

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CHAPTER IX
APPENDIX

1. Clinical Photographs
2. Photomicrographs
3. Radiographs
PLATE I

Figure 1
Clinical photograph of preoperative site.

Figure 2
Clinical photograph of preparation and crown cementation at site of second premolar. Note overcontour and steep occlusal incline.

Figure 3
Clinical photograph at seventeen day interval. Note failure to contact non-involved posterior teeth.
PLATE II

Figure 4
Clinical photograph at ninety-one days. Note the gingival hyperplasia around the crown and depression of the upper and lower premolars. Functional occlusal plane is almost completely re-established.

Figure 5
Clinical photograph at one-hundred and eighty-nine days. Functional plane of occlusion is re-established.
PLATE III
Photomicrograph of five day specimen. (X100)

Figure 6 A
Mesial surface of the second premolar (Pressure side). Osteoblasts and new osteoid on the marrow space side and inflammation adjacent to the blood vessels in the alveolar crest area.

Figure 6 B
Inflammation adjacent to blood vessels in the periodontal ligament space and into the marrow on the distal side of the second premolar (tension side).
PLATE IV

Figure 6 C
Distal surface of the mandibular first premolar with inflammation in the ligament space (non-traumatized tooth). Note the transeptal fibers remain intact.

Figure 6 D
Interradicular area with osteoclasts and active osseous resorption. Loss of osseous tissue has been replaced by fibroblastic proliferation.
PLATE V

Figure 7
Survey photomicrograph of fourteen day specimen.
(X40)

Figure 7 A
Distal surface of the distal root showing crestal resorption and hyalinizing of ligament fibers.
Note loss of fibroblast nuclei. (X160)
Figure 7.

Figure 7 A.
PLATE VI

Figure 7 B
Distal root with vessel dilation and cell loss (lyalinizing) in the ligament space. Note increase in ligament space. (X100)

Figure 7 C
Mesial surface of mesial root with hemorrhage and periodontal ligament fiber disorganization. (X400)
Figure 7 D

Interradicular area and distal root. Note widened periodontal ligament apically and loss of osseous furcation tissue replaced by fibroblastic and endothelial proliferation. (X100)
PLATE VIII

Figure 8
Survey photomicrograph at twenty-one days. (X25)

Figure 8 A
Dentogingival junction of distal root. Note intact transeptal fibers maintaining inflammation coronally. (X160)
PLATE IX

Figure 8 B
Distal root demonstrates intact transeptal fibers.
(X160)

Figure 8 C
Mesial root of second premolar. Note minimal amount of epithelium and small pseudopapillae, which has formed under the crown's overhanging margin.
(X160)
PLATE X

Figure 8 D
Notice loss of cementum, dentin, and osseous tissue which are replaced by endothelial and fibroblastic proliferation. Tooth is closely approximated to the interradicular osseous tissue. (X100)

Figure 8 E
Apical area of distal root of second premolar. Osteoblastic and osteoclastic activity is present. (X400)
PLATE XI

Figure 9
Survey photomicrograph showing the effects of the opposing dentition at twenty-one days. (X25)

Figure 9 A
Mesial of maxillary second premolar. Transeptals are intact and crest is undergoing elastic activity. (X400)

Figure 9 B
Apical area showing resorption on the periodontal ligament side with osteoblastic activity and new osteoid within the adjacent marrow spaces. (X400)
PLATE XII

Figure 9 C
Crestal resorption with intact epithelial attachment and transeptal fibers. Moderate subepithelial inflammation is seen. (X40)

Figure 9 D
Periodontal ligament of distal root of second premolar. Note fiber disorganization, osteoclastic activity, cemental and dentinal resorption with a lack of inflammation. (X400)
PLATE XIII

Figure 10
Survey photomicrograph showing widening of periodontal ligament space. (X25)

Figure 10 A
Mesial of second premolar with pseudopapillae which has formed under the crown overhang. Epithelial proliferation of rete pegs and a moderate degree of inflammation. (X40)
Figure 10

Figure 10 A
PLATE XIV

Figure 10 B
Distal of second premolar with a widening of the ligament space and degeneration of the periodontal ligament. (X40)

Figure 10 C
Mesial of second premolar with new osteoid formation in the marrow spaces and fibroblastic proliferation. Note intact transeptals and absence of inflammation in the ligament space. (X100)
Figure 10 B

Figure 10 C
PLATE XV

Figure 10 D
New cementum and fibrous tissue have replaced lost tissues. (X40)

Figure 10 E
Mesial surface of distal root. Note new cementum. (X160)
Figure 11
Survey photomicrograph demonstrating the degree of tooth depression and encroachment of the maxillary antrum. These effects were at ninety-eight days.

(X25)
PLATE XVII

Figure 11 A
Resorptive effects of the high crown on the opposing dentition. Note resorption of cementum, dentin, and osseous tissue. (X400)

Figure 11 B
Transeptals on mesial of second premolar are oblique but remain intact. (X40)
Figure 11 A

Figure 11 B
PLATE XVIII

Figure 12
Preoperative radiograph of the opposing dentition. Note intact lamina dura and uniform ligament space.

Figure 13
Radiographic effects seen at seventy-seven days. Widening of the periodontal ligament space and crestal loss of lamina dura.
Figure 14
Preoperative radiograph of mandibular right side.

Figure 15
Cementation of high cast crown demonstrating open mesial and distal contacts and overhanging margins.
Figure 16
At the seven day interval a widening of the ligament space mesial and distal.

Figure 17
Widenig ligament space and loss of crestal density at fourteen days.
PLATE XXI

Figure 18
Twenty-eight days shows little radiographic change.

Figure 19
Radiographic changes at one-hundred and sixty-eight days shows a gross destruction of osseous tissue and widened periodontal ligament space.
PLATE XXII

Figure 20
Radiographic evidence of osseous loss at one-hundred and eighty-nine days. Note closure of previously open mesial and distal contacts.
has been incorporated, and that
is not given final approval.

Figure 20

The thesis is therefore accepted in partial fulfillment of the requirements for the Degree of Master of Science.

(Signature of Adviser)

(Date)
APPROVAL SHEET

The thesis submitted by Dr. Michael D. Comar has been read and approved by three members of the faculty of the Graduate School.

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

The thesis is therefore accepted in partial fulfillment of the requirements for the Degree of Master of Science.

5-21-48
(Date)

(Signature of Adviser)