A Histologic Study of Pulpal Reaction to Orthodontic Tooth Movement in Dogs

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INTRODUCTION

Much has been written about the effects of orthodontic forces on the tissues surrounding the tooth. But very little is known about the effects of orthodontic forces on the pulp. Early researchers, such as Orban,[1] briefly stated that orthodontic forces had no effect on the pulp. Oppenheim[2] repudiated Orban's statement and described the phenomenon of diapedsis that occurs in the pulpal tissue after orthodontic tooth movement. More recently Seltzer and Bender[3] discussed the disturbances in the circulation of the pulp that resulted from the application of orthodontic forces. They believed there is a great similarity between the pulp of an orthodontically moved tooth and that of a periodontically involved tooth. They demonstrated radiographically that the orthodontically moved tooth seems to age much faster than the average tooth. There is an atrophy of the pulp eventually leading to an early obliteration of the pulp canal by reparative dentin.

This study examines the histologic changes occurring in the pulpal tissue of orthodontically moved teeth. The need for more comprehensive studies concerning the effects of orthodontic forces on the pulp is evident by a review of the literature.

MATERIALS AND METHODS

Six dogs were used in this study. These "conditioned" dogs (dewormed and inoculated against rabies and distemper) were in good health at least 15-20 days before being delivered to the animal farm. They were housed in regulation size dog cages and fed a diet of Purina Dog Chow (containing 27% protein) once each morning. Their ages were from about one to five years and their weights from thirty-five to fifty pounds.

Tooth movement was performed on the mandibular incisors only. A torquing force was applied to the mandibular incisors of three dogs and a bodily movement force to certain mandibular incisors of the remaining three. The mandibular canine teeth on all six dogs were used as anchorage teeth. A multiband technique was used. The bands were fitted indirectly on a Velmix model of the teeth involved. Preformed bands were adapted on the incisors with welded .018 inch slot edgewise brackets. Because of their small size, the mandibular central incisors were not banded. The canines were fitted with full gold cast crowns with an .018 inch rectangular tube soldered in place, level with the brackets.

The total procedure involved anesthetizing each dog three times over a period of three months.

In Step 1 each dog was anesthetized to obtain an alginate impression of the mandibular anterior teeth from canine to canine. Two models were poured in Velmix from each impression. One model was sent to a commercial dental laboratory for the fabrication of the cuspid cast crowns and the other model used for the fitting of preformed bands.

Step 2 was undertaken approximately

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two weeks later. Dog A had bands with welded .018 inch edgewise brackets cemented on the mandibular left second and third incisors and mandibular right second incisor. The crowns were cemented on the mandibular cuspid teeth.

The mandibular right third incisor was extracted. An .016 x .022 inch archwire was contoured to fit passively into the brackets and tubes, then tied in place with .010 ligature wire. A closed off-the-arch-coil was connected from the mandibular right cuspid to the mandibular right second incisor, after which a force of three ounces was applied to the incisor.

Dog B had bands on all the mandibular incisors except the central incisors, and crowns on the mandibular cuspids. An .018 x .022 rectangular wire was fitted with an estimated 20° to 30° of lingual root torque applied to the incisor teeth.

Dog C had bands on the mandibular left and right second incisors, after which the mandibular left and right third incisors were extracted. The mandibular cuspid crowns were cemented in place, then an .016 x .022 rectangular wire was fashioned passively to the arch. Two off-the-arch closed coils, exerting a pressure of three ounces each, were tied from the mandibular left and right cuspids to the mandibular left and right second incisors, respectively.

Dog D had bands on the mandibular left and right second and third incisors, and crowns cemented on the cuspids. An .018 x .022 archwire was adapted to the arch with an estimated 20° to 30° of lingual root torque applied to the incisor teeth.

Dog E had a band on the mandibular right second incisor and crowns on the cuspids. The mandibular right third incisor was extracted, and an .016 x .022 inch rectangular wire was placed passively into the bracket and tubes. An off-the-arch closed coil was attached from the mandibular right cuspid to the mandibular right second incisor exerting a force of three ounces.

Dog F had bands on the mandibular left and right second and third incisors, as well as cuspid crowns. An .018 x .022 inch rectangular wire was used to exert an estimated 20° to 30° of lingual root torque applied to the incisor teeth.

In all six dogs the appliances were activated exactly twenty-one days, after which time the dogs were again anesthetized and killed by an intracardiac injection of 10-15 cc of Nembutal. By sectioning alveolar bone the desired teeth were gently elevated out of the bony sockets and small longitudinal cuts made both mesially and distally on the root surface into dentin. These cuts were made while the teeth were submerged in a large basin of cold water. The teeth were then immersed in the fixative, a combination of formaldehyde and potassium bichromate and never used for more than a twenty-four hour period.

In every dog the mandibular incisor teeth affected by the forces were preserved. The central incisors which were not banded and not affected by the appliances were also preserved. A maximum time limit of forty-five minutes was allowed from the time of death of the animal to the time the specimen was placed in the fixative in order to avoid undesirable autolysis of the pulpal tissue.

The specimens were decalcified, sectioned, and stained with hematoxylin and eosin.

Findings

The control teeth for all dogs showed no abnormal histologic findings in their pulpal tissue. The odontoblastic layer was characteristically columnar shaped in the coronal portion, becoming cuboidal in the middle third, and somewhat squamous at the apex. The cellular elements were much more numer-

ous than the collagenous fibers. These cells, which are mostly fibroblasts, were more concentrated in the coronal portion of the pulp. The fibers, on the other hand, were more concentrated in the apical portion of the pulp (Fig. 1).

The pulp was highly vascular with red blood cells contained within most of the vessels. Scattered within all sections of the pulp of the control teeth were some extravasated red blood cells (in the central portion of the pulp). Red blood cells were not evident within the odontoblastic layer.

There were no calcific deposits, i.e., denticles, anywhere within the pulp. The lack of empty vacuoles or spaces eliminated the possibility of reticular atrophy. No brown pigment was evident in the subodontoblastic layers, nor was there an eosinophilic zone in the dentin. The zone of Weil, located in the coronal pulp of human teeth medial to the odontoblastic layer, was not apparent in any of the dog teeth.

All the teeth that had orthodontic forces applied to them had certain common characteristics within their pulps. There was a noticeable increase in the amount of collagenous fibers throughout the pulp, while the cellular concentration decreased. There was a decrease of fibroblasts, but an increase of round inflammatory cells, especially in the coronal portion of the pulp. The blood supply also seemed altered. The blood vessels were fewer in number and more constricted than in the control teeth. The extravasated red blood cells were still apparent.

Once again there was a lack of brown pigment in the subodontoblastic layer and no sign of an eosinophilic zone within the dentin. No reticular atrophy was indicated. The dentinal tubules did not contain any erythrocytes or nucleus-like bodies. The zone of Weil was not seen in any of the sections.

The odontoblasts maintained their characteristic morphology. In different portions of the pulp, however, the odontoblastic layer was disrupted. This cellular disruption varied consistently with the type of force applied to the teeth.

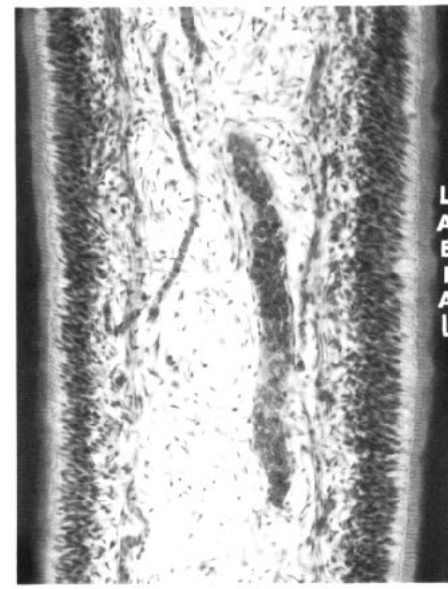

Fig. 1 Control specimen of pulp indicating normally aligned odontoblasts with normal cellular components.

Lingual root torque was applied to the teeth of three dogs. The pulps of these teeth showed a disruption or disorientation of the odontoblasts in the middle third of the root (Figs. 2, 3). The remaining three teeth demonstrated a broad area of disruption of the odontoblastic layer. This cellular disruption occurred on the labial side for almost the entire root portion of the pulp of dog A (Fig. 4). However, the same cellular disruption occurred on the lingual side of the dental pulps of dog C and dog E.

Discussion

The dogs were killed before removing any teeth. The teeth were carefully dissected out of the sockets to avoid any pulpal distortions. The blood supply to the pulp could easily be disrupted by

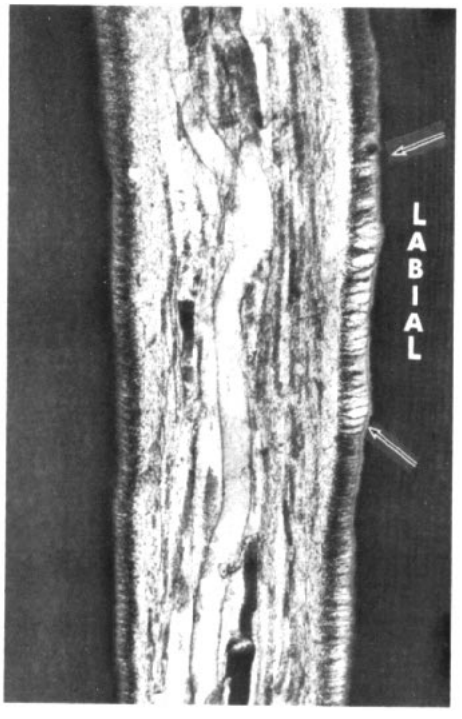

Fig. 2 Torqued tooth specimen of pulp; arrows indicate limits of vacuolization of the odontoblastic cells.

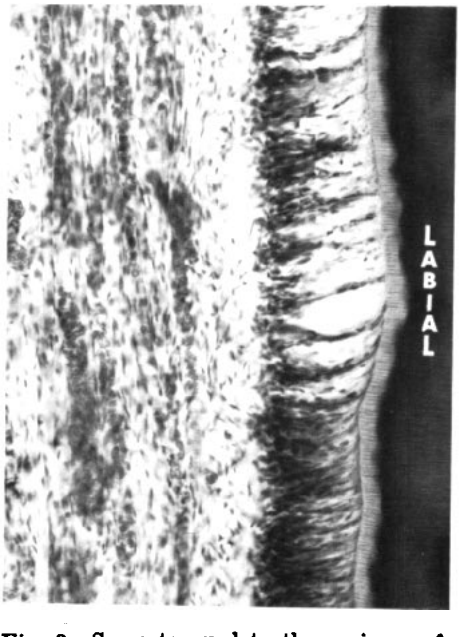

Fig. 3 Same torqued tooth specimen of pulp (Fig. 2) showing vacuolization of odontoblasts.

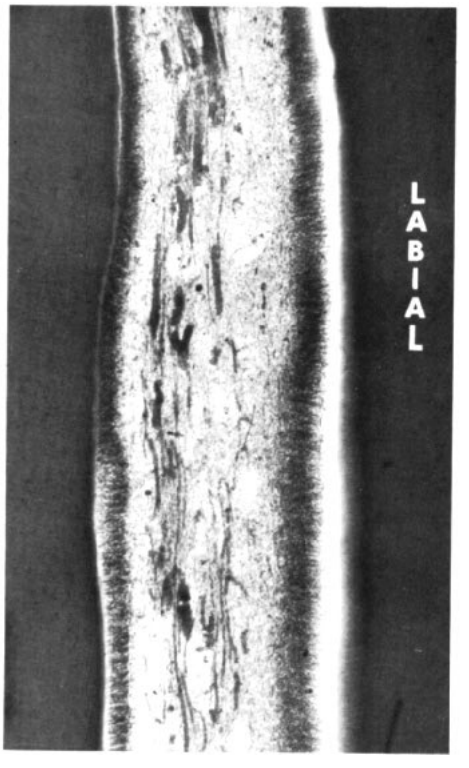

Fig. 4. Bodily moved tooth specimen of pulp; note the extent of the disorientation of the odontoblasts.

the extraction of the tooth. Forty-five minutes was the maximum time allowed to place the specimens in the fixative after the dogs were killed. The time period was limited to insure against any degenerative changes that might occur in the pulp. The time was based on the work done by Gatewood and Sorenson.[4] Empty vacuoles within the central portion of the pulp are a common degenerative change occurring in inadequately fixed pulps. These vacuoles represent reticular atrophy.

With few exceptions, the sections of the pulp obtained were very good. There was some tearing of the pulp which inadvertently occurred when the microtome passed from the harder decalcified tooth substance into the pulpal tissue. Very few pulp sections were en-

tirely mutilated. Some sections had portions which were torn but were still readable.

The periodontal membrane was not preserved because the tooth had to be penetrated in certain areas so that the fixative could effectively reach the pulp. Therefore, proof of tooth movements could not be obtained from the membrane. Since the activation period was only three weeks, radiographs could not be used either to determine any significant tooth movement.

All the teeth were sectioned in a mesiodistal direction. Therefore, the examination of the pulpal response was limited to labial and lingual views.

All of the dogs except one had good representative sections of pulpal tissue. Dog C was apparently much older than the other five dogs. The dentin appeared extremely scalloped. More collagenous fibers were present compared with the control teeth of the other dogs. The identification of collagenous fibers especially with hematoxylin and eosin stain was in question. However, the fibers observed were arranged in orderly bundles coursing throughout the pulp. The cellular elements were fewer in number. Because of the secondary dentin deposited with age, the pulp chamber and canal seemed quite narrow. Seltzer and Bender's[3] description of the premature aging of the dental pulp due to orthodontic tooth movement was confirmed in the present study. An increase of collagenous fibers occurred, with a corresponding decrease of the cellular elements in all the experimental sections of pulpal tissue.

In Sicher's edition of Orban's *Oral Histology and Embryology*,[5] the zone of Weil, a cell-free zone just medial to the odontoblastic layer, is well defined in the coronal portion of human dental pulps. There was no evidence collected from the dog's teeth that a zone of Weil existed in the coronal pulps of either the control or experimental teeth.

There were some positive correlations between Langelands[6] description of deviations from the normal histology of the pulp and the histologic findings within the experimental pulps of the six dogs. Evidence of inflammatory cells, which Langeland described, could be seen in the central portion of the pulp as well as adjacent to the odontoblasts. There was no obvious brown pigment indicating a deterioration of erythrocytes. Cavity formation containing some inflammatory cells or debris was not apparent. Langeland suggested that the initial pulpal reaction to injury would be a migration of the odontoblastic nuclei into the dentinal tubules. However, these nuclei would not be observed after three weeks post injury. The tubules in the experimental pulps did not show any nuclei. There was a possibility of some debris within the tubules of certain experimental sections, but this debris could not be positively identified because of the thickness of the sections.

The most obvious pulpal deviation observed within the experimental pulps was the disruption of the odontoblastic layer. The location and extent of the odontoblastic layer disruption varied depending on the type of force applied to the teeth. In the pulps of the torqued teeth, the disruption of the odontoblasts occurred in the middle third of the root. This disruption was consistently within the fulcral areas of the torqued teeth. The location of the odontoblastic disruption in the pulps of bodily moved teeth appeared to be either on the pressure or tension sides of the teeth. The span of this cellular disruption appears to correspond in length to the cellular activity that occurs in the alveolar bone adjacent to a bodily moved tooth. Since the pulps were sectioned in a labiolingual direction it is impossible to determine if this disruption of the odonto-

blasts occurred on the pressure or tension side.

Summary

This study was undertaken to observe the changes that occur in the dental pulp as a result of applying orthodontic forces to teeth. Six dogs were selected as the experimental animals; their mandibular incisors were used as controls, moved bodily, or torqued.

Besides noticing the apparent aging of the pulp, the most significant histologic finding was the disruption of the odontoblastic layer. The location and extent of the disruption varied with the type of orthodontic forces applied to the teeth. Within the pulpal tissue of the bodily moved teeth, the disruption of the odontoblastic layer extended the length of the root. But in the pulpal tissue of the torqued teeth the disruption was clearly limited to the middle third of the root.

The consistency of these histologic findings in all six dogs suggests that perhaps the internal structure of the tooth is not as impervious to the forces applied to it as previously thought. These forces seem to affect the internal histomorphology of the tooth. Future studies will determine if the disruption that occurs within the tooth due to these orthodontic forces may be reversible or temporary in nature. A different experimental animal, such as the Macaque rhesus monkey, may be helpful, since the dentition more closely resembles that of man.

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