Effects of Intrusive Forces Upon the Microvasculature of the Dental Pulp

MICHAEL J. GUEVARA, D.D.S.
SAMUEL G. MCCLUGAGE, JR., PH.D.

Studies by several investigators suggest that intrusive orthodontic forces can adversely affect the pulpal microcirculation. In this regard, Stuteville,[1] Oppenheim,[2] and Marshall[3] believed that intrusive and other types of orthodontic forces can impede pulpal circulation resulting in damage to the pulp. Oppenheim[2] recommended the use of light forces with frequent rest periods to decrease the iatrogenic effects on the pulp. Schwartz[4] concurred with this concept, having stated that orthodontic forces should not exceed the capillary pressure of 20mm/Hg since strangulation of the vessels could ensue with subsequent necrosis. Butcher and Taylor[5,6] determined that excessive intrusive and extrusive forces caused necrosis of the pulp with no regeneration of the odontoblastic layer. They stated that teeth with open apices were more susceptible to trauma from intrusive forces. However, Stenvik and Mjor[7] felt that the effects on the pulp were dependent on the stage of root development and that teeth with open apices had a better prognosis. Oppenheim,[2] Strang and Thompson,[8] Stenvik and Mjor,[7] and Stuteville[1] found similar pulpal changes due to orthodontic forces. They described the following histologic observations in the pulp: hyperemia, diapedesis and margination of white blood cells, stasis, vacuole formation in the odontoblastic layer, cyst formation and hemorrhage. Graber[9,10] thought that hyperemia could occur even with light intrusive forces and that excessive forces could cause irreversible damage to the pulp. In this regard, Spector[11] referred to two cases in which devitalization of teeth resulted from orthodontic therapy. Butcher and Taylor[5,6] reported that vacuole formation, dilation of vessels, thrombosis, etc. could occur as a result of intrusive forces; however, they questioned their results because the controls also exhibited vacuole formation and congestion of blood vessels. Pohto and Scheinin,[12,13] Scheinin[14] and Kozam[15] questioned the validity of using vacuole formation and dilation of vessels as an index of trauma stating that fixation techniques could produce such artifacts.

From these previous studies it is apparent that many investigators share the opinion that intrusive orthodontic forces could iatrogenically affect the pulpal circulation. But, due to the questionable histologic methods used in some of these studies and because of the lack of any *in vivo* studies, convincing data demonstrating the actual changes upon the microvasculature of the pulp after application of orthodontic forces are lacking. Therefore, in an attempt to further elucidate this subject, an *in vivo* technique has been developed which permits sequential microscopic observations of the pulpal microvascular system after application of intrusive orthodontic forces.

MATERIALS AND METHODS

Twenty-one female Sprague-Dawley rats (250-300 gms) were used for the study. There were sixteen experimental and five control animals. The

From the Departments of Orthodontics and Anatomy, School of Dentistry, Louisiana State University, New Orleans, Louisiana.

Fig. 1. Rat incisor overlying quartz rod (R) with rubber dam and matrix band in place around incisor and first and second molars.

rats were randomly assigned to either of the following two groups: Group I served as the control group which underwent the same surgical procedure and two-hour microscopic observation period as Group II, but no intrusive orthodontic force was applied to the lower incisors. Group II had an intrusive orthodontic force of 80 gms applied to their lower incisors for a two-hour period. The surgical technique used to observe the pulp was modified after Pohto and Scheinin,[12,13] Kozam,[15] Taylor[16] and McClugage et al.[17] A triangular flap of skin was removed in an area bounded by the lateral aspects of the two clavicles, the corners of the mouth, and the lower lip at the junction between the alveolar mucosa and the attached gingiva. The subcutaneous tissues were reflected to expose the strap muscles of the neck. A midline incision was made in the sternohyoid muscle to permit cannulation of the underlying trachea (Fig. 1).

To prepare the tooth for removal of hard structure, all muscle attachments and alveolar mucosa were separated from the labial surfaces of the mandible to the level of the left third molar. The anterior belly of the digastric muscle was cut at its insertion, and the mandible was separated by cutting the ligament of the mandibular symphysis. A tofflemire matrix band was placed over the first and second molars to facilitate manipulation of the incisor.

Once all the gingival tissue was reflected to the level of the first molar, the medial half of the mandible was positioned under a binocular dissecting microscope. At this time a wing of a siamese bracket was directly bonded to the labial surface of the incisor, just coronal to the alveolar process (Fig. 2). Tooth and bone reduction of the incisor was accomplished with a high speed air-driven handpiece using a No. 57 carbide bur. To reduce any heat generated by the procedure the tooth was bathed with physiologic Ringer's solution kept at room temperature. Both the medial and the lateral surfaces of the tooth were reduced in an area immediately incisal to the crest of the alveolar bone until the pulpal vessels could be seen through a thin layer of dentin. At this point the animal was transferred to an optical bench where further reduction of the dentin was conducted under higher magnification using a stereobinocular microscope. To transilluminate the preparation the tip of the incisor was positioned over the end of a fused quartz rod which acted as a conduit to pipe light from a distant light source to the undersurface of the preparation (Figs. 1, 2). A scalpel blade was used to further reduce the dentin to a thickness of approximately 25 microns. At this thickness the underlying pulpal microvasculature could be observed microscopically.

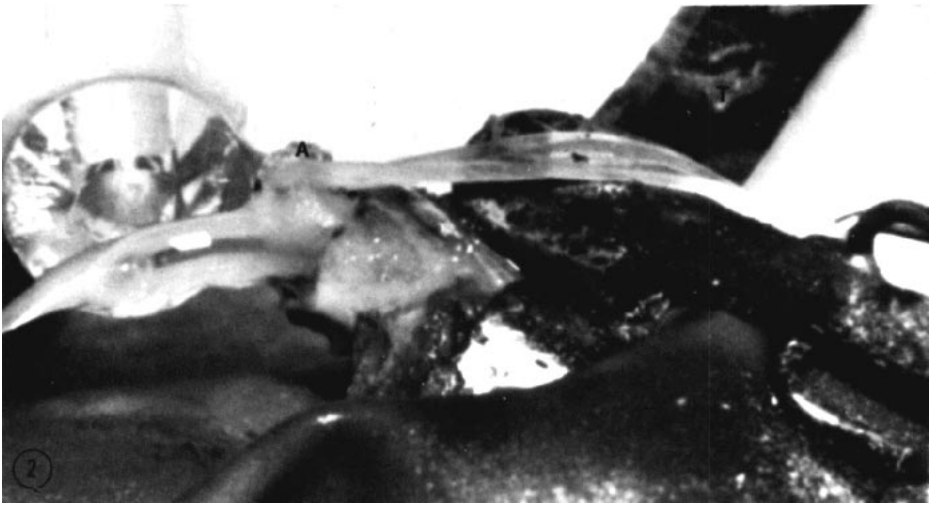

Fig. 2 Rat incisor after application of intrusive force. Note direct bonded attachment (A) and tofflemire matrix band holder (T).

During the final reduction the preparation was bathed with physiologic Ringer's solution warmed to the body temperature of the animal.

Observations were made by direct microscopy at magnifications of ×25 to ×160 using a Leitz stereobinocular microscope with appropriate oculars and objectives or alternatively the optical images were projected into a Bolex 16mm motion picture camera and cinemicrophotographed on Kodak Ektachrome film.

Application of Orthodontic Force

Before application of a force the internal diameters and flow rates of the blood vessels in the microscopic field were determined. Measurements of the internal diameters of the microvasculature were made by means of an eyepiece micrometer installed in one of the oculars of the microscope. The linear velocity of blood flowing through these vessels was recorded on a scale of 0 to 4, 0 representing complete stasis within a vessel and 4 representing very rapid flow permitting no definition of individual blood cells. Since observations were made before and after application of a force, each animal served as its own control; however, as mentioned earlier, some animals (Group 1) were observed for a two-hour period without any application of an orthodontic force. After measurements of the vessels in the microscopic field were recorded, a calibrated 80 gram intrusive force was attached from the tofflemire matrix band holder to the direct bonded attachment (Fig. 2). A Haldex gram gauge that was calibrated with gram weights was used to measure the elastic force. The force vector was parallel to the inferior surface of the incisor. The microvascular system of the dental pulp was then observed for two hours recording any changes versus time in the following parameters: 1) linear velocity of blood flow; 2) internal diameters of the blood vessels; 3) behavior of the white blood cells, i.e., increased margination, increased stickiness of white cells to the endothelial lining; and 4) any evidence of localized hemorrhage or thrombosis.

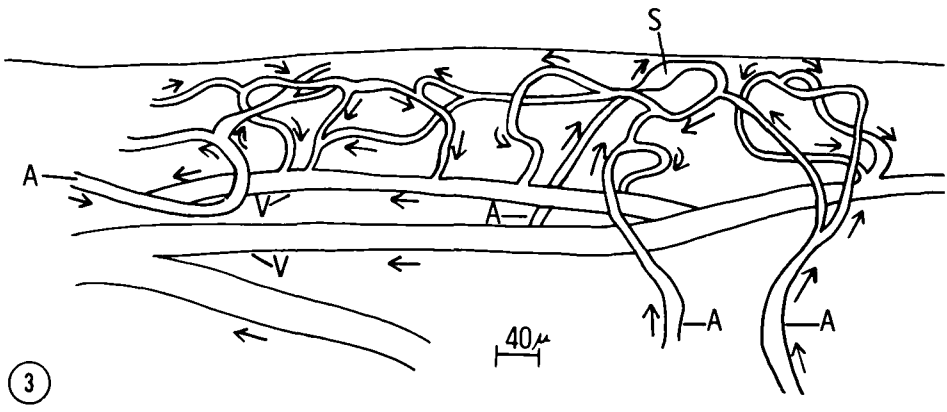

Fig. 3 A line drawing (drawn to scale) of a subodontoblastic plexus of capillaries on labial surface of the incisor representing a typical microscopic field from which in vivo observations were conducted. Note arterioles (A), venules (V), and a dilated "sinusoidal-like" capillary (S). Arrows indicate direction of blood flow. Size marker is 40 microns.

Results

In vivo microscopy of the pulp in control animals or before application of a force in experimental animals revealed four to five small veins (80 to 180 microns in internal diameter) and one to two arterioles (30 to 40 microns in internal diameter) coursing parallel to the dentinal surfaces. The linear velocity of blood flowing through these arterioles was scored at a flow rate of 4, while that of the small veins was somewhat slower. Occasional arteriovenous anastamoses were observed interconnecting the small veins and larger arterioles of the pulp. Smaller arterioles (8 to 15 microns) which branched obliquely from the centrally located arterioles coursed to the subodontoblastic plexus of capillaries (Fig. 3). These smaller arterioles had the same linear velocities of blood flow as the parent vessels from which they branched.

After application of the 80 gram intrusive force to the incisor, no changes were observed in the internal diameter of the microvasculature or in the linear velocity flowing through it in eight of the experimental animals. In four of the experimental animals the linear velocity of the blood flow of the larger vessels was reduced to a flow rate of 2 or less. Four other experimental animals exhibited complete stasis (0 flow rate) during application of the force. The circulatory arrest occurred about five minutes after application of the force. In those animals that had decreased flow rates or even complete stasis after a force was applied, complete recovery of flow occurred within five minutes after removal of the force.

Discussion

The results of this study suggest that the normal integrity of the pulpal microvascular system of rats may be vulnerable to various orthodontic forces during tooth movement. The variable reactions of the pulpal microvascular system exhibited by the experimental animals may be due to several reasons. Some of the variability could be due simply to anatomical variation of the pulp with some animals having more accessory circulation from the periodontal ligament and alveolus. It may also be possible

that in those animals where complete statis occurred, the periodontal support was not as strong as others, or that the distance from the apex of the lower incisor to the alveolus was smaller than in those animals which were unaffected. Furthermore, a variation in the anatomy of the lower incisor could have been a determinant for the differences observed. Rat lower incisors are curved, and the differences in the amount of curvature of the teeth could have altered the vector of the elastic force applied. Finally, small variations in the placement of the direct bonded attachment and matrix band holder could have also altered the vector of the force, even though steps were taken to avoid this possibility.

Spector[11] reported on a few cases where teeth were devitalized during orthodontic therapy; however, this small number of cases may be due simply to a lack of a comprehensive clinical analysis. There also have been several reports citing obliteration of pulp chambers after orthodontic therapy.[18] This could possibly be due to a compromised pulpal circulation during therapy leading to localized necrosis with subsequent mineralization of the pulp. Seltzer and Bender[19] felt that orthodontic forces may induce more rapid aging processes within the pulp due to circulatory interferences thereby reducing the ability of the pulps to withstand future insults. They also reported that orthodontically treated teeth show histologic findings similar to periodontally involved teeth.[19]

Based upon reports by other investigators and the results discussed in the present study, it is evident that the microvascular system of dental pulp may be compromised during the orthodontic movement of teeth. Most studies conducted by other investigators have been histologic studies; however, the *in vivo* study reported here supports some of their findings.

Summary

This *in vivo* study has demonstrated in rats that the nutritional blood flow of the pulp may be compromised after application of an intrusive orthodontic force. The results of this study and the findings of other investigators suggest that an in depth study analyzing the relationship between pulpal viability and orthodontic tooth movement may be indicated.

Department of Orthodontics
L.S.U. School of Dentistry
New Orleans, LA 70119

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