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Archives of Oral Biology 46 (2001) 891–900

Archives of Oral Biology

www.elsevier.com/locate/archoralbio

Histomorphometric study of the periodontal vasculature during and after experimental tipping of the rat incisor

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Accepted 9 April 2001

Abstract

The periodontal vasculature encircling the entire length of the rat lower incisor was studied at the time of tipping movement and 3 months later. In 12 rats (212 ± 4 g b.w.), loads (0.19 ± 0.016 N) were applied to the lower left incisor in a linguointrusive direction. After 2 weeks of loading, six experimental animals were killed with the loading springs in place. The springs were removed in the six remaining rats, which were killed 12 weeks later. Six additional rats with intact teeth served as control. All incisors were fixed, demineralized, embedded in glycol methacrylate and cross-sectioned perpendicular to the long axis of the tooth. The distance of each section (2 μm) from the apex was calculated. A computerized image-analysis program was used to measure the width and area of the labial and lingual periodontal ligament to establish whether the measured segments corresponded to the compressed or expanded zones. In each cross-section, the various types of blood vessels were counted and the cross-sectional area of all venous vessels was measured. The results showed that after 2 weeks of loading (1) the general trend of vascular changes was similar under pressure and tension; (2) the large-diameter vessels were unaffected by loading; (3) the mean number of terminal arterioles had decreased significantly, while the number of capillaries and postcapillary venules had increased significantly in the apical tooth part; (4) the venous blood vessel area had decreased significantly in the apical tooth part; (5) the intensity of the vascular reaction was dependent on the degree of tissue distortion; and (6) after 12 weeks' recovery the vascular changes were still present, demonstrating a rebound effect. The findings suggest that microvascular alterations following tooth loading are not directly related to the spatial effect of loading itself and are of a much longer duration than expected.

Keywords: Periodontal vasculature; Rat incisor; Tipping movement; Tissue recovery

1. Introduction

The manifold functions of the periodontal vasculature explain the extensive body of investigations of this system. Many such studies have been on rodent teeth, using the molar or the continuously erupting incisor. The detailed anatomy of periodontal vascularization has been obtained mainly by applying corrosion casts or by using various perfusion methods (Kindlova and Matena, 1959, 1962; Adams, 1962; Carranza et al., 1966; Matena, 1973). Magnification angiography has been used to reveal the architecture of the mandibular vessels in dogs (Jeffcoat et al., 1982). Carranza et al. (1966) found that, in general, the periodontal ligament of the rat, mouse, hamster, cat and dog molars, as well as the cementum-related part of the ligament in continuously erupting rodent incisors, have similar modes of

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[1] Based on research submitted in partial fulfilment of the requirements for the degree of Master of Science in Dental Sciences.

0003-9969/01/$ - see front matter
PII: S0003-9969(01)00058-9

vascular supply. Thus, in all these species, the blood supply derives from arterial branches feeding the bone marrow and periosteum. In addition to the main afferent arteries, the blood supply is carried by small arteries (originating in the alveolar bone), which enter the periodontal ligament via vascular canals and branch into terminal arteries to form ultimately a capillary network. The venous outflow is collected into a rich, occlusoapically directed network of collecting venules, which, in turn, drain into large efferent veins in the bone. The enamel-related periodontal ligament of the incisor has, in addition, a rich vascular plexus related to the enamel organ.

The types of blood vessels that comprise the periodontal microvascular bed in teeth of limited eruption have been described extensively (Sims, 1983; Weekes and Sims, 1986; Freezer and Sims, 1987; Lew, 1987; Wong and Sims, 1987; Tang and Sims, 1992; Kannari et al., 1993; Selliseth and Selvig, 1994; El-Agroudi et al., 1998) and less so in teeth of continuous eruption (Moxham et al., 1985; Blaushild et al., 1992; Kannari et al., 1993). In both molars and incisors, arterial vessels form a relatively small proportion of the total periodontal vascular volume; most of the vessels belong to the venous system and are classified as capillaries or postcapillary venules. In the incisor, the vascularity in the apical half is much richer than in molars; incisors have fewer postcapillary venules and instead, venous vessels of large caliber and capillary-like structure (called sinusoids) prevail near the apex. All vessels are characterized by an abundance of fenestrations (Moxham et al., 1985).

One of the important functions of the periodontal vascular complex is in tissue remodeling, as in orthodontic force application. The initial response of the mechanically compressed or expanded periodontal tissue is release of vasoactive neuropeptides by the stimulated sensory nerve endings (Vandevska-Radunovic et al., 1997). The blood vessels respond with increased permeability through the endothelial gaps (Iida et al., 1992; Tang et al., 1993; Chintakanon and Sims, 1994). Moxham et al. (1985) and Clark et al. (1991) report numerous fenestrae also in vessels of the rat incisor and molar; Clark maintains that these have also been noted in mice and man. Mechanical loading induces an increase in number and size of the fenestrae (Lew et al., 1989), enabling extravasation of leukocytes into the interstitial tissue (Rygh et al., 1986), which itself is the major source of a variety of cytokines that activate osteogenic cells, thus initiating bone remodeling and tooth movement (Davidovitch, 1991).

Two main experimental methods have been applied to test the response of the gingival, pulpal and periodontal blood flow to strain/stress changes in the periodontal tissue: perfusion with fluorescent microspheres (Kvinnsland et al., 1989; Vandevska-Radunovic et al., 1994) and laser-Doppler flowmetry (Yamaguchi et al., 1991; McDonald and Ford, 1994; Barwick and Ramsay, 1996; Beightler et al., 1998). The increase in blood flow following orthodontic force application was found to continue for a week, after which it subsided to control values (Vandevska-Radunovic et al., 1994).

Studies of the periodontal vasculature of teeth subjected to short-term loads have produced controversial results. Thus, Göz et al. (1992) found that tipping the teeth of dogs for <2 h did not affect the circulation. The intrusion of rat teeth for 30 min caused an increase in the volume of capillaries and postcapillary venules (Clark et al., 1991); the same effect was also observed in tooth extrusion lasting 30 min (Lew et al., 1989). On the other hand, in another experiment Lew (1989) noted that a 30-min extrusion produced degenerative changes in postcapillary venules, whereas Tang and Sims (1992) observed an increase in the number of capillaries, but a reduction in the amount of postcapillary venules.

Studies on the periodontal vasculature of teeth subjected to long-term loading (not less than 7 days) report an increase in vascular activity in the rat (Rygh et al., 1986), sprouting of capillaries and postcapillary venules in marmosets (Parlange and Sims 1993) and rats (Murrell et al., 1996), an increase in the density of blood vessels concomitant with increased density of the regulatory sensory nerves in the rat (Vandevska-Radunovic et al., 1997) and increased vascularity within the periodontal ligament in dogs and monkeys (Khouw and Goldhaber, 1970).

Many of the studies on the correlation between mechanical loading and the periodontal vasculature have been made on limited sections of the ligament only, representing either tissue compression or expansion, depending on the movement investigated. However, forces applied to the tooth crown generate interchanging areas of pressure/tension/quiescence along the bone-embedded root. The vascular supply in each area is influenced not only by local factors, but also by the changes in other areas. In view of this, we have now sought to depict a comprehensive, quantitative representation of the periodontal vasculature around and along the entire rat incisor that had undergone tipping movement for 2 weeks.

2. Materials and methods

2.1. Histological preparation

The study was carried out on 12 experimental and six control female rats of the Hebrew University Sabra strain (aged 13 weeks, mean weight 212 ± 4 g). In the experimental rats, a continuous mechanical linguointrusive load of 0.19 ± 0.016 N was applied to the lower left

incisor by means of a closed-coil spring stretched between the incisor and the lower left molar (Steigman et al., 1981). After 2 weeks of force application, six experimental animals were killed with the springs in place (spring group), while in the remaining six rats, the springs were removed to enable the periodontium to recover over a 12-week period (recovery group). Six animals matched by strain, sex, age and weight served as baseline control for all experimental rats. The controls were killed at the same time as the spring group.

Animals were killed by ether overdose, their left mandibles were dissected and fixed in Bouin–Holland solution, radiographs of the specimens were taken in the lateral plane and they were then demineralized in 0.27 M/l EDTA (pH 7.4) at room temperature.

The intrabony part of the incisor was divided into six equal segments (designated A–F; Fig. 1) by a method that eliminates the potential distortion caused by transverse sectioning of curved teeth (Steigman et al., 1983). In short, each radiographed incisor and its surrounding bone was traced on an acetate sheet and the centre of the arch formed by the labial border of the bone-embedded part was determined geometrically. From this centre, radii were drawn, dividing the tooth into short segments. The mandible was placed on the tracing and cut along the radii. This procedure ensures that all cross-sections lie perpendicular to the long axis of the tooth, obviating the distortion inherent in the curved rat incisor. The segments were embedded in glycol methacrylate (JB-4; Polyscience Inc., Warrington, PA) and serial transverse sections (2 μm) were cut perpendicularly to the long axis of the tooth. The distance of each cross-section from the posterior alveolar wall (referred to here as 'apex') was calculated. The most apical section was 2.8 mm from the apex and the most coronal 18.7 mm. Segment A, which contained the alveolar crest, was discarded because bone continuity is frequently disrupted there. The mean distance of the remaining segments from the apex is presented in Table 1.

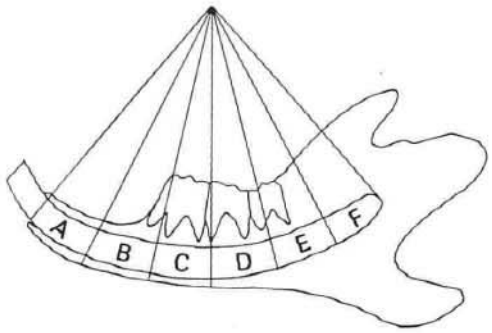

Fig. 1. Schematic diagram of a dissected rat mandible demonstrating the division of the incisor into six segments.

Table 1
Mean (±S.E.M.) distance (in mm) from the apex of the investigated segments in the control, spring and recovery groups

Segment	Group		
	Control	Spring	Recovery
F	3.28 ± 0.15	3.15 ± 0.05	3.26 ± 0.22
E	6.52 ± 0.23	5.45 ± 0.34	6.70 ± 0.33
D	9.77 ± 0.29	9.19 ± 0.42	10.38 ± 0.31
C	13.28 ± 0.33	13.26 ± 0.44	14.17 ± 0.61
B	16.67 ± 0.30	16.61 ± 0.29	18.02 ± 0.30

2.2. Computerized image analysis

One section from each segment was chosen for examination by computerized image analysis (CVE-2 system; Galay Production Ltd., Israel). The combined data from all sections in each of the control, spring and recovery groups (a total of 30 sections per group) provided a representative view of the periodontal vasculature along the entire tooth. The periodontal ligament of each section was divided by a line joining the mesial and lateral cementum–enamel junctions into cementum- and enamel-related parts (Fig. 2).

The blood vessels were classified histomorphologically (Lew, 1987; Simionescu and Simionescu, 1988) into small arterioles, terminal arterioles, capillaries, postcapillary venules, sinusoids and collecting venules

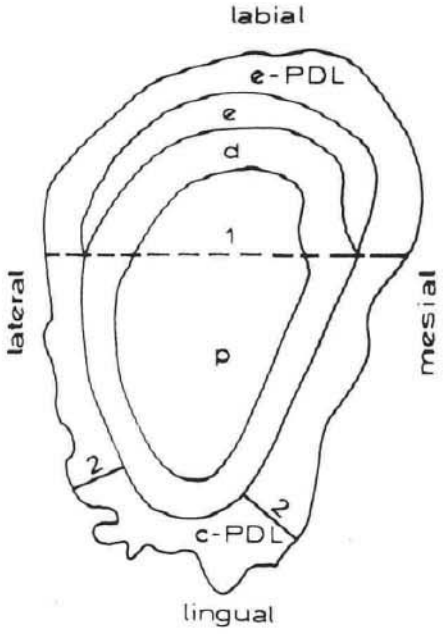

Fig. 2. Schematic presentation of the division (line 1) of the periodontal ligament (PDL) into enamel (e) and cemental (c) PDL. P, pulp; d, dentine; e, enamel space.

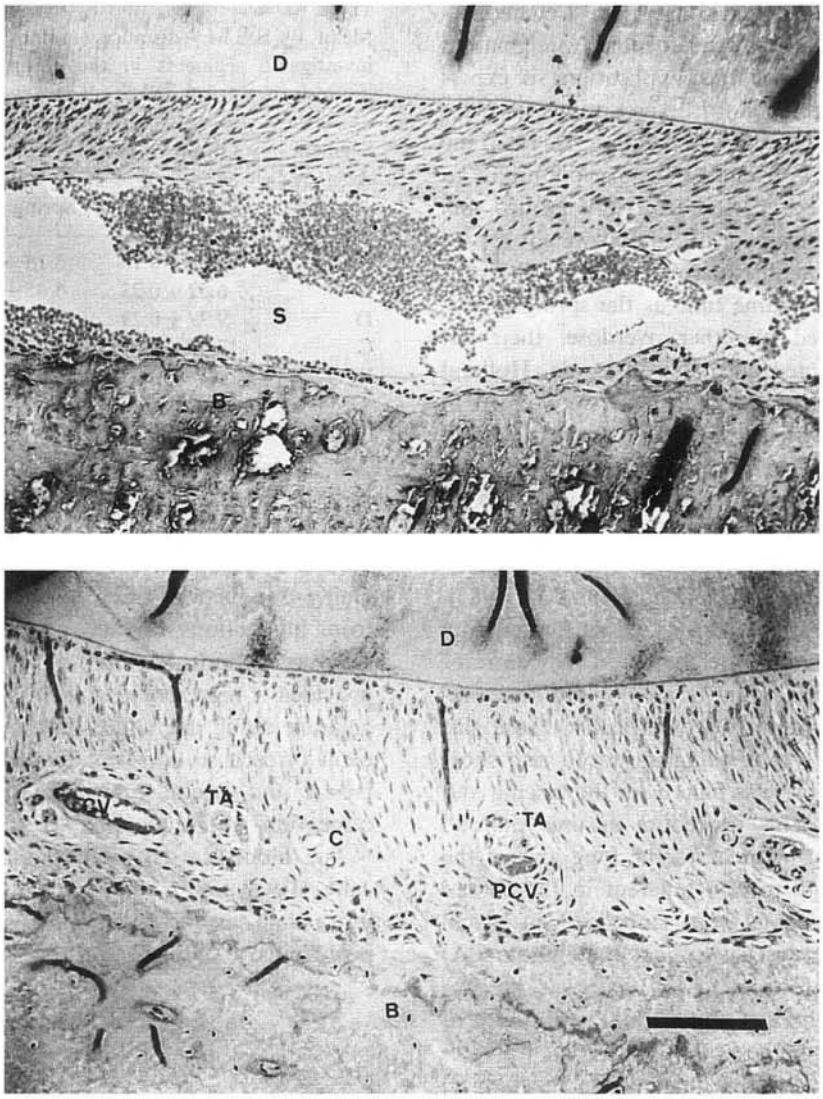

Fig. 3. The various types of blood vessels in the apical (top) and incisal (bottom) parts of the periodontal ligament of the rat incisor. B, bone; D, dentine; S, sinusoid; PCV, postcapillary venule; TA, terminal arteriole; C, capillary; CV collecting venule. Haematoxylin and eosin. Bar = 75 μm.

(Fig. 3). The classification is described in greater detail in our previous work on periodontal vascularization (Blaushild et al., 1992). The various types of blood vessels were examined according to this classification.

2.3. Measurements and calculations

The following variables were investigated: (1) the area of cementum-related (between bone and the cemental line) and enamel-related ligament (between bone and the outer perimeter of the ameloblastic layer); (2) the mean lingual and labial periodontal widths (calculated by dividing the cementum- and enamel-related areas by the length of the cemental and ameloblastic lines, respectively); (3) the percentage of contraction/expansion of the ligament in the spring group compared with the controls—the contracted areas were designated as 'pressure' sites and the expanded areas as 'tension' sites, even if the difference was not statistically significant; (4) in each cross-section all types of blood vessels (except sinusoids) were counted; and (5) the luminal areas of venous vessels only were measured, as routine histological procedures cause contraction of arteriolar vessels. To enable inter- and intragroup comparisons of the vascular area, it was presented as a percentage of the total periodontal area.

2.4. Data evaluation

The intra- and interobserver reproducibility of the measurements was assessed by: (a) double determinations of 20 different sections performed by the same investigator at an interval of 3 months; (b) double determinations of 18 different sections performed by two independent examiners. In both instances the error did not exceed 3%.

The vascular variables were grouped and analyzed according to their location in either a pressure or tension site. The mean ($\pm$S.E.M.) was determined for each site in every segment. Comparison was made between the compressed and expanded areas in each of the two experimental groups and between them and the baseline controls. The significance of differences between the various measurements and calculations was established using the Sheffé F-test (level of significance, 95%).

3. Results

3.1. Body weight

The animals withstood the experimental procedures well and remained healthy throughout the entire experiment. Weight loss (17 ± 4 g) occurred only during the first 2 days of spring application; thereafter the weight remained stable in the spring group and increased in the recovery group.

3.2. Morphometry of the periodontal ligament

The enamel-related periodontal ligament (Table 2; Fig. 4) presented a mirror image of the cementum-related ligament. Force application caused a significant ($P < 0.01$) decline in the mean area and width of segments D–F and enlargement in segment B. In the recovery group, all measurements expressed a rebound effect in that they rose above the control values.

The area and width of the cementum-related ligament (Table 2; Fig. 4) were not uniform along the tooth. In the controls, the smallest measurements were obtained in the apical and coronal segments. In the spring group, the mean area and width in segments D–F showed significant ($P < 0.01$) enlargement, whereas in segments C and B a tendency to reduction was observed. In the recovery group, the width and area of the cementum-related ligament in all segments (except B) were below those of the control group, owing to the rebound effect.

From these findings, the cementum-related ligament in the spring group was designated as a pressure area in segments B and C and as a tension area in segments D–F; these designations were interchanged for the enamel-related periodontium.

3.3. Area of venous blood vessels

In the control group, the venous blood vessels and especially the sinuses, occupied about half of the periodontal space near the tooth apex, their relative area dwindling in the coronal direction (Table 3). After spring activation, the proportional vascular area decreased significantly ($P < 0.01$) in the pressure site of segments D–F and in the tension site of segments E and F. The difference between pressure and tension was prominent in the coronal segments, particularly in segment B. While in the tensed ligament, the vascular area differed only slightly from that of the control, this area increased 4-fold in segment B of the compressed ligament. Similar trends were noted in the recovery group, albeit with less pronounced differences ($P < 0.05$) (Table 3).

3.4. Number of blood vessels

3.4.1. Small arterioles

Only six arterioles were observed in the entire enamel-related ligament in the control and recovery groups, the number rising to 13 in the spring group. In

Table 2
Mean ($\pm$S.E.M.) cross-sectional areas (in mm^2) of the enamel-related (e-PDL) and cementum-related (c-PDL) periodontal ligament

Segment	Control group	Spring group[a]	Recovery group[a]
e-PDL			
F	0.56 ± 0.03	0.48 ± 0.02 (−14)	0.66 ± 0.10 (+18)
E	0.57 ± 0.02	0.39 ± 0.03 (−32)	0.66 ± 0.05 (+16)
D	0.48 ± 0.02	0.42 ± 0.01 (−13)	0.52 ± 0.06 (+8)
C	0.41 ± 0.03	0.42 ± 0.04 (+2)	0.51 ± 0.09 (+24)
B	0.36 ± 0.03	0.45 ± 0.06 (+25)	0.48 ± 0.03 (+33)
c-PDL			
F	0.66 ± 0.02	0.84 ± 0.04 (+27)	0.61 ± 0.02 (−8)
E	0.90 ± 0.02	1.13 ± 0.07 (+26)	0.75 ± 0.08 (−17)
D	0.91 ± 0.04	1.14 ± 0.08 (+25)	0.86 ± 0.10 (−5)
C	0.76 ± 0.02	0.73 ± 0.05 (−4)	0.65 ± 0.05 (−14)
B	0.63 ± 0.03	0.59 ± 0.02 (−6)	0.67 ± 0.05 (+6)

[a] Numbers in parentheses denote percent change compared with control values.

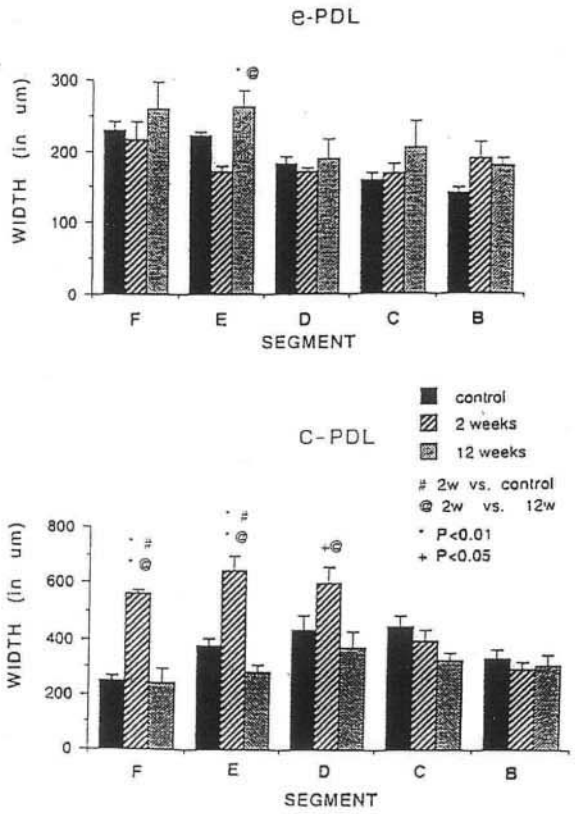

Fig. 4. Mean (±S.E.M.) width of the labial (e-PDL) and lingual (c-PDL) periodontal ligament.

the cementum-related ligament, the number of arterioles in the control group amounted to three per cross-section, diminishing to two per cross-section in the spring and recovery groups. Statistical evaluation was not feasible.

3.4.2. Terminal arterioles

The mean number of these arterioles in the control group was 13.6 ± 1.8 and 8.6 ± 1.7 per cross-section in the pressure and tension areas, respectively. In the spring group, the mean number of these vessels decreased to 3.4 ± 0.8 and 4.4 ± 1.6 in the pressure and tension sites, respectively. The decrease was significant in all segments ($P < 0.01$), except in segment B of the tension site. In the recovery group, the quantity of arterioles remained less than that of the control (9.2 ± 2.6 and 5.1 ± 1.4 for the pressure and tension sites, respectively; difference not significant).

3.4.3. Capillaries

The number of capillaries increased notably in both experimental groups (Table 4). In the control group, their mean number per cross-section increased gradually, starting at the apex. In the spring group, these values were elevated in the cementum-related ligament both at pressure and tension sites, the increase being significant in segments F, E and D ($P < 0.05$). The amount of capillaries in the periodontium of the recovery group did not differ statistically from the control.

3.4.4. Postcapillary venules

The distribution of these venules in both the control and spring groups was similar to that of the capillaries (Table 5). In the controls, the postcapillary venules were concentrated mainly in the coronal half of the tooth. In the spring group, their number increased significantly in segments F, E and D at the pressure site ($P < 0.01$) and in segment E at the tension site ($P < 0.05$). In the recovery group, this increase continued to

Table 3
Mean (±S.E.M.) area of venous blood vessels in the pressure and tension sites as a percentage of the total periodontal area

Location	Segment	Control group	Spring group[a]	Recovery group[a]
Pressure site				
e-PDL[b]	F	64.11 ± 1.18	39.10 ± 4.20 (−39)	54.80 ± 6.70 (−15)
	E	59.22 ± 2.74	40.00 ± 3.80 (−32)	36.40 ± 3.60 (−39)
	D	55.08 ± 4.44	27.00 ± 2.90 (−51)	38.30 ± 5.90 (−30)
c-PDL[c]	C	15.69 ± 3.87	16.73 ± 2.42 (+7)	19.80 ± 1.45 (+26)
	B	3.27 ± 1.42	12.70 ± 1.40 (+388)	9.10 ± 3.04 (+278)
Tension site				
c-PDL	F	49.89 ± 3.92	23.22 ± 8.59 (−53)	28.55 ± 6.81 (−43)
	E	47.37 ± 2.30	29.28 ± 4.43 (−38)	37.72 ± 3.13 (−20)
	D	42.57 ± 1.69	39.02 ± 4.75 (−8)	38.45 ± 2.21 (−10)
e-PDL	C	36.76 ± 1.37	32.90 ± 4.30 (−11)	45.60 ± 9.20 (+24)
	B	22.86 ± 3.43	21.90 ± 2.70 (−4)	30.30 ± 3.10 (+33)

[a] Numbers in parentheses denote percent change compared with control values.
[b] Enamel-related periodontal ligament.
[c] Cementum-related periodontal ligament.

Table 4
Mean (±S.E.M.) number of capillaries per section in the pressure and tension sites

Location	Segment	Control group	Spring group[a]	Recovery group[a]
Pressure site				
e-PDL[b]	F	3.02 ± 0.74	12.50 ± 3.50 (+391)	19.80 ± 6.29 (+619)
	E	9.83 ± 1.79	20.90 ± 4.15 (+213)	14.30 ± 3.51 (+145)
	D	11.00 ± 2.38	15.40 ± 2.61 (+140)	13.40 ± 2.54 (+122)
c-PDL[c]	C	15.67 ± 2.04	18.40 ± 5.75 (+117)	18.90 ± 2.99 (+120)
	B	25.00 ± 6.19	21.60 ± 7.40 (−14)	22.20 ± 6.19 (−11)
Tension site				
c-PDL	F	11.20 ± 2.31	22.50 ± 4.50 (+201)	19.40 ± 5.90 (+83)
	E	7.00 ± 2.13	15.40 ± 3.26 (+220)	16.00 ± 2.86 (+229)
	D	9.20 ± 1.60	14.90 ± 2.70 (+162)	9.20 ± 2.36 (0)
e-PDL	C	24.70 ± 3.67	22.20 ± 4.63 (−10)	8.50 ± 2.04 (−66)
	B	16.30 ± 3.07	14.20 ± 1.79 (−13)	14.20 ± 8.76 (−13)

[a] Numbers in parentheses denote percent change compared with control values.
[b] Enamel-related periodontal ligament.
[c] Cementum-related periodontal ligament.

be significant on the pressure sites in segments F ($P < 0.05$), E ($P < 0.01$) and D ($P < 0.01$).

3.4.5. Collecting venules

This type of blood vessel was rarely observed in the control group (i.e. 27 in cementum-related and none in the enamel-related ligament). In the spring group, 19 and three venules were present in the cementum- and enamel-related ligament, respectively and in the recovery group, these vessels amounted to 35 and three in the respective parts of the periodontium. Owing to the scarcity of these vessels, statistical analysis was not feasible.

4. Discussion

In this three-dimensional study, we used tipping of the rat incisor to examine the effect of orthodontic forces on the periodontal vascular system. The advantage of using this particular movement is that it engenders regions of pressure and tension along the entire tooth, bringing in their wake tissue distortion of varying intensity. The morphometry of the periodontal area and width disclosed lingual areas of pressure opposite labial areas of tension at the incisal tooth edge and vice versa in the apical part of the incisor. The fulcrum of the tipping movements, as determined by the least changes in tissue size, was between segments C and D (9–13 mm from the apex). The tissue distortion increased proportionally to its distance from the fulcrum. These findings are in agreement with our previous investigation of a similar sample (Brin et al., 1990).

The periodontal vascular system responded to the mechanical stimulation by a decrease in the venous area and an increase in the number of microvessels, the intensity of the reaction being dependent on the degree of tissue distortion. On the face of it, the findings for venous area and number of microvessels seem contradictory. However, Blaushild et al. (1992) report that, in the rat incisor, the larger part of the total venous area derives from blood sinusoids, whereas the contribution of the microvessels is negligible. It follows, therefore, that our results represent mainly the decrease in sinusoid cross-sectional area.

The decrease in the proportional vascular area in the apical tooth half in both pressure and tension zones is explained by the increased permeability of capillaries, postcapillary venules and chiefly, sinusoids. The significant increase in the number of fenestrations in capillaries and postcapillary venules (Moxham et al., 1985; Lew et al., 1989) and in the number of endothelial junctions (Tang et al., 1993) enhances extravasation of fluid into the interstitial tissue. This 'leakage' might have affected the total area of the apical vasculature in our material. In contrast, in the apical ligament of the rat molar, where the vascular volume is represented mainly by postcapillary venules, an increase in vascular volume of the compressed and tensed periodontium has been observed (Lew et al., 1989; Clark et al., 1991).

In the coronal part of the ligament, on the other hand, the response of the compressed and expanded zones to loading was found to differ: in the expanded sites the vascular area remained unaffected, whereas in the compressed sites it increased 4-fold. It is possible that the apically extravasated blood was squeezed, via tissue channels, to the farthest incisal segment (Tang and Sims, 1992), supporting the concept that a significant fluid exchange exists across the vessel walls in both directions. Furthermore, the enlargement of the vascu-

lar area in segment B may well be a reflection of increased blood flow in the gingival plexus (Beightler et al., 1998).

Although the number of small arterioles and collecting venules counted was insufficient for statistical analysis, mere observation gave the impression that their quantity remained unaffected by loading. This notion is in agreement with the findings of Ng et al. (1981), Lew (1989) and Sims (1999) in dog and rat molars. A possible explanation is that as the walls of these vessels contain one to two layers of smooth muscle, their relative thickness renders them less susceptible to external stimuli.

The significant decrease in the number of terminal arterioles in both the tension and pressure zones along the entire tooth length is in keeping with the findings of Clark et al. (1991). They reasoned that a slight dilation of the lumen of the terminal arterioles might promote excessive filling of the venous bed. It is possible that such dilation occurs and thus compensates for the decreased number of terminal arterioles. This issue demands further investigation.

The notable increase in the number of capillaries and postcapillary venules at the pressure and tension sites has been reported in nearly all investigations on the periodontal vasculature under loading. This phenomenon is attributed to the inflammatory processes occurring in the mechanically stimulated tissue (Rygh et al., 1986; Cooper and Sims, 1989; Vandevska-Radunovic et al., 1997). The angiogenesis begins with the sprouting of endothelial cells in arterioles, venules and capillaries, with the proliferating cells then migrating and forming new capillaries (Nakamura et al., 1986; Chang et al., 1997; Derringer and Linden, 1998). This course of events is characteristic of wound healing and of severe periodontal disease (Jeffcoat et al., 1982) and emphasizes the inflammatory background of tissue remodeling in orthodontic tooth movement.

Analysis of the circulation in the mechanically expanded and stressed periodontal loci clearly demonstrates the similarity of the vascular response to pressure and tension stimuli. This finding had already been observed (but not commented upon) by Khouw and Goldhaber (1970), Rygh et al. (1986) and Murrell et al. (1996) in monkeys, dogs and rats. The cause of such similarity is attributable to the fact that the response of the periodontal vasculature (in particular of capillaries and postcapillary venules) is not dictated by the spatial changes, but by the inflammatory processes in the periodontal ligament. As these are very much alike in both pressure and tension areas, it makes the vascular response nearly identical in these opposite sites. The only exception is found in those areas subjected to excessive stress with consequent necrosis (Lindskog and Lilja, 1984).

The reconstitution of the periodontal vasculature appeared to take place at a much slower rate than expected. Nakamura et al. (1986) found disarrangement and an increase in the number of capillaries 31 days after load cessation in rat molars, while Parlange and Sims (1993) reported on the same condition in marmoset incisors after 9 weeks of retention. Even after the prolonged recovery period of 12 weeks applied here, the morphometric data pertaining to the periodontal ligament had still not returned to normal. Such retarded return to normal has been observed also in periodontal lesions consequent to mechanical loading (Steigman et al., 1993).

It is of interest that at 3 months after loading, the vasculature of the animals was still in a disturbed state,

Table 5
Mean (±S.E.M.) number of postcapillary venules per section in the pressure and tension sites

Location	Segment	Control group	Spring group[a]	Recovery group[a]
Pressure site				
e-PDL[b]	F	7.20 ± 1.77	16.00 ± 2.00 (+222)	29.00 ± 5.84 (+403)
	E	14.80 ± 2.00	20.90 ± 3.75 (+141)	39.10 ± 8.79 (+264)
	D	28.80 ± 3.46	31.70 ± 5.44 (+10)	53.40 ± 5.59 (+85)
c-PDL[c]	C	18.70 ± 2.14	28.20 ± 2.15 (+51)	30.60 ± 4.97 (+64)
	B	26.80 ± 2.69	26.25 ± 9.42 (−2)	30.60 ± 3.89 (+14)
Tension site				
c-PDL	F	17.40 ± 5.33	19.00 ± 5.00 (+9)	31.20 ± 5.69 (+79)
	E	8.20 ± 2.15	23.00 ± 4.55 (+282)	15.10 ± 1.61 (+85)
	D	13.20 ± 1.14	20.40 ± 3.26 (+55)	24.30 ± 4.88 (+85)
e-PDL	C	30.80 ± 2.62	22.60 ± 4.18 (−27)	29.00 ± 8.33 (−6)
	B	32.50 ± 2.60	20.30 ± 3.00 (−38)	32.40 ± 5.74 (0)

[a] Numbers in parentheses denote percent change compared with control values.
[b] Enamel-related periodontal ligament.
[c] Cementum-related periodontal ligament.

notwithstanding the intense metabolism of the periodontal tissues in the rat incisor. The slower tissue turnover of the human periodontium means that, in all probability, reconstitution of its vascular system lasts considerably longer than in the rat. Our results therefore support the recommendations of Parlange and Sims (1993), calling for a long (in excess of 1 year) retention period after orthodontic treatments.

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