ORIGINAL ARTICLE

The effect of functional occlusal forces on orthodontic tooth movement and tissue recovery in rats

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The influence of physiologic occlusal forces on tissue response to orthodontic tooth movement and its subsequent recovery was investigated in rats. The mandibular incisors were subjected to tipping and intrusive loads for 2 weeks. In 27 animals, the teeth remained in occlusion, while the incisors were shortened out of occlusion in 29. In each group, some rats were killed at the end of the tooth movement process, and the rest were kept alive for a 13-week recovery period. The subgroups were matched with corresponding controls. Tooth intrusion and eruption were monitored throughout the experiment; then histomorphometric and histopathologic evaluations were performed on the incisors and their periodontal tissues. Tooth movement was barely affected by the functional forces. However, immediately upon 2-week load application, damage to the pulp was more extensive in the occluding teeth; this might be due to the combined intrusive vectors of functional and mechanical loads. The effect of the physiologic function was expressed primarily during the recovery period, in which the eruptive function and distorted periodontal ligament space returned to normal significantly faster in the occluding teeth. This was also true for the healing of dental and periodontal lesions. The incidence of tooth resorption was also reduced. Normal occlusal function is imperative for rapid reconstitution of the damage caused by orthodontic forces. (Am J Orthod Dentofacial Orthop 2002;121:620-8)

The intimate relationship between masticatory function and craniofacial morphology has been widely investigated in both animals and humans.[1-3] It has been found that function affects the morphology of the rat incisor socket and its location in the jaw borders.[4] Moreover, it is well known that functional stimulation is imperative to maintain the integrity of the periodontal tissues. In extreme cases of nonfunction or prolonged hypofunction, the tooth-supporting tissues undergo atrophic changes—the so-called disuse atrophy[5]—with the severity of the degeneration depending on the duration of the reduced occlusal function. The degree of atrophy can be ameliorated by nonmasticatory functional forces acting on the teeth.[6]

Experimental hypofunction is achieved by either shortening teeth out of occlusion or extracting their antagonists. In experiments in which hypofunction lasted between 2 and 90 days, the following quantitative and qualitative alterations in the tooth-supporting structures have been noted: increased bone deposition at the alveolar crest, concomitant with a decrease in the thickness of the transseptal fibers[7,8]; a decrease in number and diminished mineralization of Sharpey's fibers with an increase in the diameter of these fibers[6]; a rise in quantity and density of the periodontal fibroblasts accompanied by a reduction in the amount of ground substance in the extracellular matrix[9-11]; an increase in the number of microtubules in the fibroblasts[9] and alterations in the morphology and orientation of these cells in the continuously erupting incisor[12]; and an increase in the total volume of the periodontal ligament (PDL) of the hypofunctional rat incisor by 24% after 21 days and by 53% after 90 days of lowered occlusal function.[13] Some of these changes are conducive to a considerable decrease in the mechanical strength of the periodontium in the continuously erupting tooth[14] and in teeth of limited growth.[15]

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Submitted, July 2001; revised and accepted, November 2001.

0889-5406/2002/$35.00 + 0 **8/1/123342**

doi:10.1067/mod.2002.123342

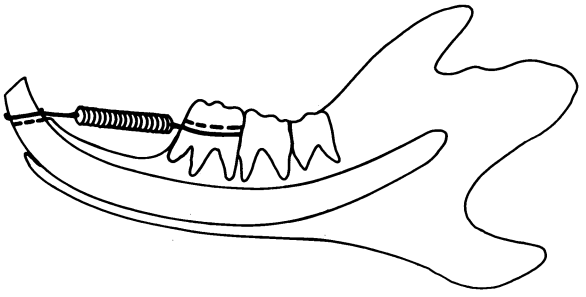

Fig 1. Schematic drawing of experimental appliance.

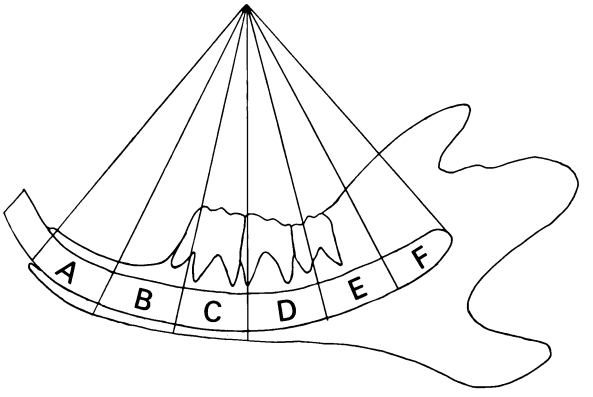

Fig 2. Schematic drawing of dissected rat mandible showing division of incisor into 6 equal segments.

It has also been reported that removing occlusal function affects healing of periodontal injuries; the healing process is modified and decelerated because of lessened bone and cementum formation and frequently occurring ankylosis.[16]

In orthodontic tooth movement, the tissue changes are dictated by the magnitude, the duration, and the direction of the mechanically applied forces. No information is available, however, about the influence of functional loads when combined with mechanical forces. Better insight into the latter issue would have practical implications because it would allow more scientifically based planning of mechanotherapy.

The purpose of this study was to investigate the influence of occlusal forces on orthodontic tooth movement and on tissue recovery after such movement.

MATERIAL AND METHODS

Sample handling

The study sample comprised 56 young adult rats of the Hebrew University Sabra strain (initial mean weight, 204 ± 4 g). The animals were divided into group A (27 rats) and group B (29 rats). In group A, the teeth were allowed to remain in occlusal contact, and, in group B, the lower left incisor was kept constantly in hypofunction by twice weekly shortening the occlusal edge. In each group, 10 rats served as controls. In the 36 experimental animals, a continuous linguo-intrusive load (mean load, 19 ± 1.6 g; range; 18.8-19.2 g) was applied to the lower left incisor (Fig 1) for 2 weeks. The method of force application by means of a closed-coil spring stretched between the left first molar and the incisor has been described previously in detail.[17]

At the end of the 2-week spring activation, 7 rats from group A and 10 from group B were killed (by an overdose of ether) with the springs in situ; at the same time, 5 control rats from each group were killed. In the remaining rats in both groups, the springs were removed, and the teeth were allowed to recover for 13 weeks. The division into the various subgroups is given in Table I. The amount of tooth eruption was measured in all incisors throughout the experimental period.[18] The measurements were made twice weekly at noon on the same days of the week, with the animals under ether anesthesia.

Table I. Distribution of sample

	Group A (n = 27) In occlusion		*Group B (n = 29) Hypofunction*	
Duration	*Experimental*	*Control*	*Experimental*	*Control*
2 weeks spring	7	5	10	5
13 weeks recovery	10	5	9	5

Histomorphometry

The mandibles were dissected, cleaned of soft tissue, separated into right and left halves, and fixed in Bouin's fluid The left hemi-mandibles were used for the histomorphometric evaluation by a method described in detail elsewhere.[19] Briefly, it entails taking a nondistorted lateral radiograph of each mandible and tracing the outlines of the incisor and mandibular bone on acetate paper. The center of the arc formed by the labial border of the bone-embedded part of the incisor is geometrically determined on the tracing. From this center, radii are drawn to the labial border of the incisor, dividing the tooth into 6 equal segments (Fig 2).

Two consecutive 100 μm-thick transverse ground sections were cut (Isomet low-speed saw, Buhler Ltd, Lake Bluff, Ill) from the incisal edge of each of the 6 segments, parallel to their bordering radii. The exact distance of each section from the posterior wall of the alveolar socket (hereafter called apex) was established.

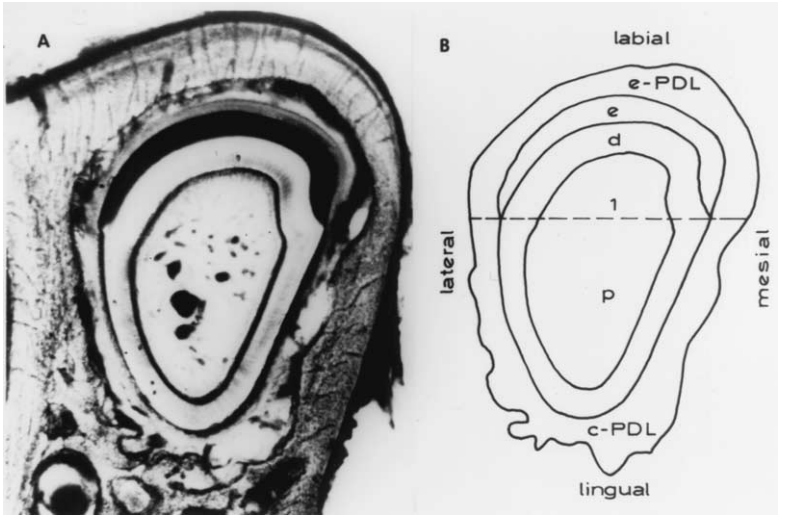

Fig 3. Cross section of rat incisor. **A**, Photograph; **B**, tracing. *P*, pulp; *d*, dentin; *e*, enamel; *e-PDL*, enamel-related periodontal ligament; *c-PDL*, cementum-related periodontal ligament; *1* line joining mesial and lateral cementoenamel junctions.

The sections were viewed and traced on a Reichart Visopan microscope (Vienna, Austria). The tracings were fed into a computer by means of an EDT-114 digitizer (Houston Instruments, Belgium). A specifically designed computer program was used to measure the following parameters (Fig 3): (1) the cross-sectional area of the socket, the enamel-related PDL (e-PDL), and the cementum-related PDL (c-PDL), marked by the line joining the lateral and mesial cementoenamel junctions; (2) the width of the e-PDL (along the median perpendicular to the cementoenamel junction plane); (3) the mean width of the mesial and lateral c-PDL (calculated by dividing the areas of the mesial and lateral sectors by their measured length[19]); and (4) the volumes of the e-PDL, the c-PDL, and the socket (calculated for the apical 3-18 mm of the bone-embedded tooth).

Histopathology

After removing the 2 transverse ground sections from each of the 6 tooth segments, the rest of the tooth parts were decalcified in 10% EDTA (pH 7.4) at room temperature, embedded in glycol methacrylate (JB-4, Polyscience Inc, Washington, Pa), and cut (Reichart-Jung Microtome, C. Reichart, AG, Vienna, Austria) serially and perpendicularly to the long axis of the tooth into 2-μm sections. The cross-sections were stained with hematoxylin-eosin, and the exact distance of each section from the apex was calculated. Randomly chosen representative sections (6 sections from each segment) were examined by light microscopy. The following parameters were recorded: (1) tooth: enamel and dentin resorption, enamel and dentin folds, and damage to the ameloblasts, odontoblasts, and pulp; and (2) PDL: necrotic lesions, cell death, edema, infiltration by inflammatory cells, and hemorrhages.

Data analysis

The mean daily eruption rate (± SE) was calculated for each of the 4 subgroups and expressed as a percentage of the corresponding control values. The Student *t* test was used to evaluate the intergroup differences (level of significance, $P < .05$). The measurements obtained for all ground sections were plotted on a 3-dimensional system of coordinates according to the distance of the section from the apex. Whenever the tissue parameter depended on the distance of the tissue from the apex, second-order polynomials were fitted to the plotted points, and the adequacy of the polynomial fit was checked with the chi-square test. Tissue volumes of the socket and the PDL were calculated as an integral of the area of the polynomials. For variables that remained constant at all tooth levels, mean values (± SE) were established, and their intergroup differences were calculated with the Student *t* test. The data obtained for the PDL width and area are presented as a percentage of the corresponding control values.

The frequency of dental and periodontal lesions was calculated in each subgroup as a percentage of teeth showing the same type of injury in that particular subgroup. The lesions were mapped according to their location on the long axis of the tooth.

Table II. Mean (± SE) weekly rate (in μm) of intrusion/eruption during spring application

	Group A In occlusion	*Group B Hypofunctional*
Week 1	−156 ± 44	−167 ± 56
Week 2	54 ± 58	72 ± 64
Total	−102 ± 51	−95 ± 60

RESULTS

The animals withstood the experimental procedures well, and their gingival tissues remained healthy throughout the experimental period. They showed a slight weight loss during the 2-week spring application, but this was offset by a mean weight gain of 59.0 ± 6.0 g during the recovery period.

The degree of active tooth intrusion during the first week of spring application and the cessation of eruption during the second week did not differ significantly between groups A and B (Table II). The fulcrum of rotation of the tipping movement (determined as the area with the least changes of the PDL) was located at a mean distance of 14 mm (range, 11-16 mm) from the apex in the functional teeth and at 11 mm (range, 9-13 mm) in the hypofunctional incisors. The difference between the locations of the 2 fulcrums was not significant.

At the end of the 3-month recovery period, the highest rate of eruption in the functional teeth was 85% and that in the hypofunctional teeth was not more than 58% of the values recorded for their respective controls (Fig 4). The absolute end values of tooth eruption differed significantly between groups A and B ($P < .05$).

Upon the 2-week spring activation, the distortion of the PDL in all parameters examined was similar in the functional and the hypofunctional teeth. The differences between the 2 groups for each parameter had no statistical significance (Fig 5). In both groups, the width and the area of the e-PDL were diminished, and, in the c-PDL, these parameters were enlarged; the differences between the loaded teeth and their respective controls were statistically significant (for *P* values, see Fig 5).

At the end of the 3-month recovery period, the PDL of the occluding teeth had returned to control values; ie, the c-PDL parameters were reduced, and those of the e-PDL were expanded; an exception was the labial width that had rebounded to exceed the control width (Fig 6). The PDL values of the nonoccluding teeth, on the other hand, failed to return to normal. The differences between experimental and control values were statistically significant (for *P* values, see Fig 6). The delay in recovery of the PDL in the hypofunctional teeth was notable also for the calculated volumes of the e-PDL and the c-PDL (Fig 7).

The frequency of the dental and periodontal lesions in groups A and B is shown in Table III. The addition of occlusal forces to the mechanical loading (group A) did not appreciably affect most of the dental and periodontal tissues. Exceptions were increased frequency of pulp damage (Fig 8, *1*), decreased frequency of disordered enamel formation/calcification (Fig 9, *1*), and decreased dentin resorption (Fig 10, *1*). In group A, the enamel lesions were dispersed along the entire tooth, and, in group B, they were concentrated in the apical 8 mm (Fig 9, *1*). Dentin resorption was evident mainly in the apical and coronal tooth parts in both groups (Fig 10, *1*), following the pattern of distribution of the necrotic areas (Fig 11, *1*).

During the recovery period, the occlusal forces exerted a beneficial effect on the healing of all dental lesions and decreased the incidence of necrotic lesions in the PDL (Table III). The damage to pulp and enamel in the teeth of group A was less than half of that observed in group B (Figs 8, *2* and 9, *2*); no new dentinal or enamel folds were found in the apical tooth part that was created after removing the springs. The occlusal forces also reduced the frequency of dentin resorption and necrotic lesions (Figs 10, *2*, and 11, *2*).

Lesions connected with the extremely sensitive enamel organ and lesions linked with inflammation (ie, infiltration by inflammatory cells, hemorrhages, edema, and cell death) were present in all teeth of groups A and B upon spring removal and at the end of the recovery period.

DISCUSSION

The results of this study indicated that functional occlusal forces play only a negligible role in orthodontic tooth movement. In this context, it is of interest that, in finite models of implants, the biting forces do not affect the mechanical stress distribution.[20]

In fact, the combination of mechanical and functional loads must be evaluated on the basis of the direction and magnitude of each force. The functional occlusal forces acting on the rat incisor have 2 vectors: biting loads in the intrusive direction and grinding loads in the anteroposterior direction. The biting loads have a magnitude between 200 and 2500 g (but mainly below 800 g) and are of relatively short duration (40-160 ms).[21] The grinding loads, defined by Robins[21] as "complex loading profiles," are of low magnitude (400 g), but of a relatively long duration (160-680 ms). With respect to the mechanical forces, as used in the present

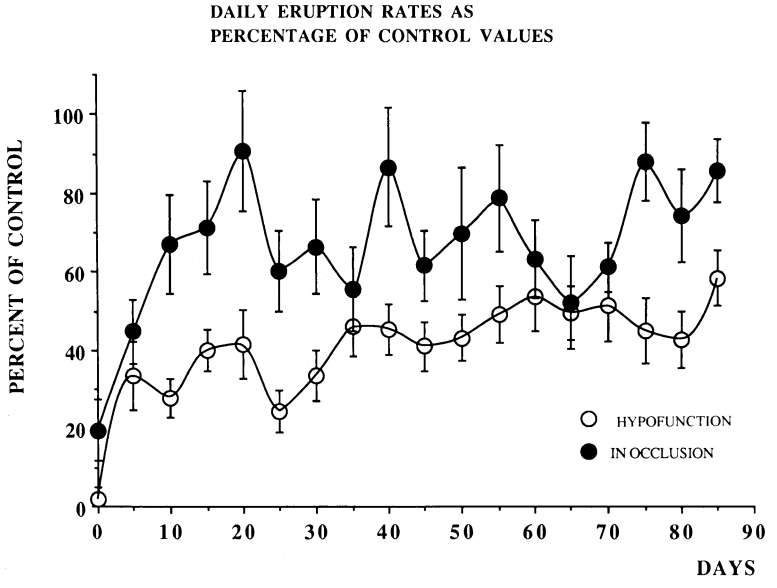

Fig 4. Mean (± SE) daily eruption rate during recovery period as percentage of eruption rate of corresponding controls. *Closed circles*, group A; *open circles*, group B.

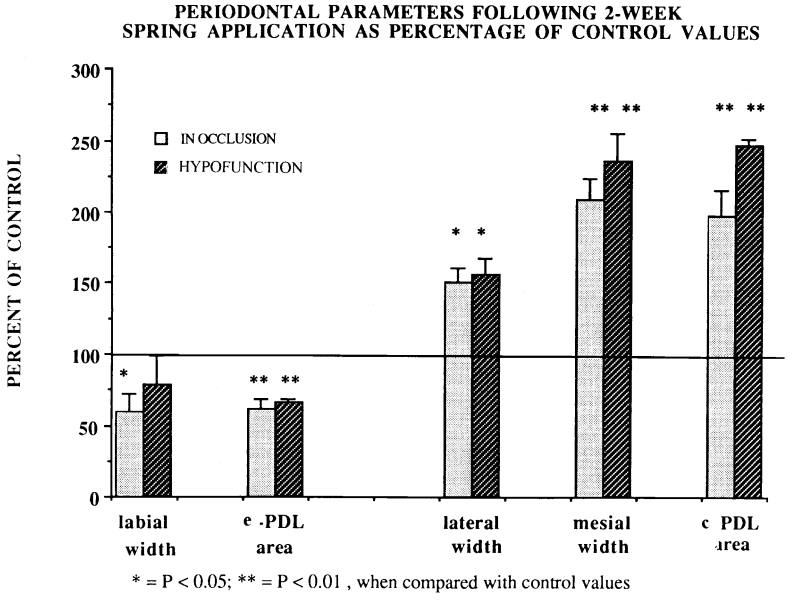

Fig 5. Periodontal parameters after 2-week spring application as percentage of control values (100%). *Lightly shaded columns*, group A; *heavily shaded columns*, group B (raw data available upon request).

study, these also consisted of 2 vectors:[17] in the intrusive direction and in the posterior direction. The latter vector caused the lingual tipping of the incisor, which in turn brought about the expansion and compression of the c-PDL and the e-PDL, respectively.

The results of this study showed that functional occlusal forces do not affect orthodontic tooth movement. An analysis of the effects of the 2 vectors of the functional forces may explain this lack of influence. The alternating posterior/anterior mandibular grinding movement generates forces that act on the tooth in opposite directions, thus canceling each other. The

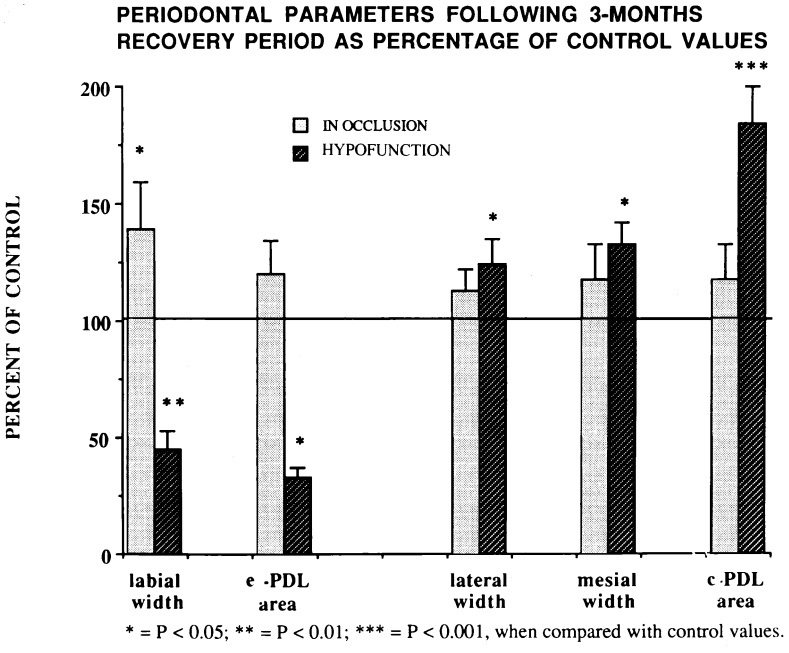

Fig 6. Periodontal parameters after 13-week recovery period as percentage of control values (100%). *Lightly shaded columns*, group A; *heavily shaded columns*, group B (raw data available upon request).

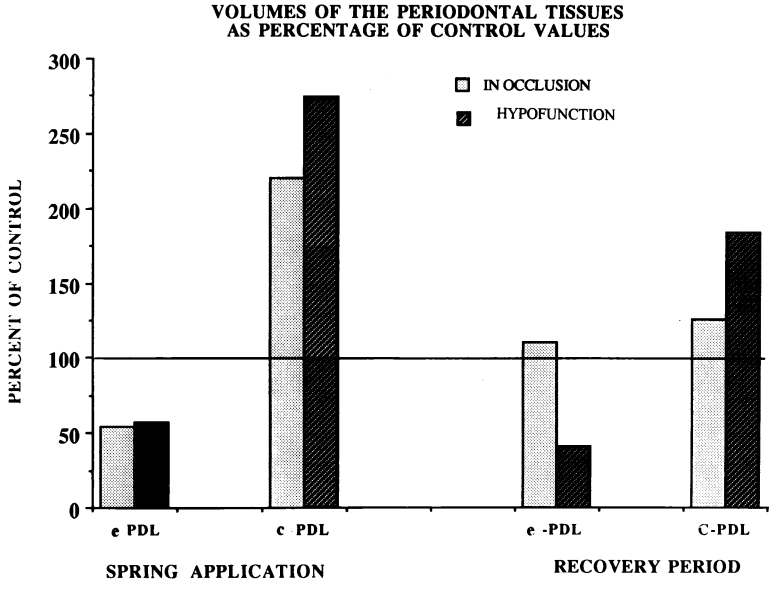

Fig 7. Volume of periodontal tissues in 4 subgroups as percentage of corresponding control values (100%). *Lightly shaded columns*, group A; *heavily shaded columns*, group B.

biting movement, which augments the mechanical vertical vector, does not affect tooth movement because its duration is extremely short. The detrimental influence of the functional forces was expressed mainly by the high frequency of pulpal damage. At the same time, the reduced incidence of enamel disorders and dentin resorption indicated a beneficial effect of function. It is suggested that the multidirectional functional load dis-

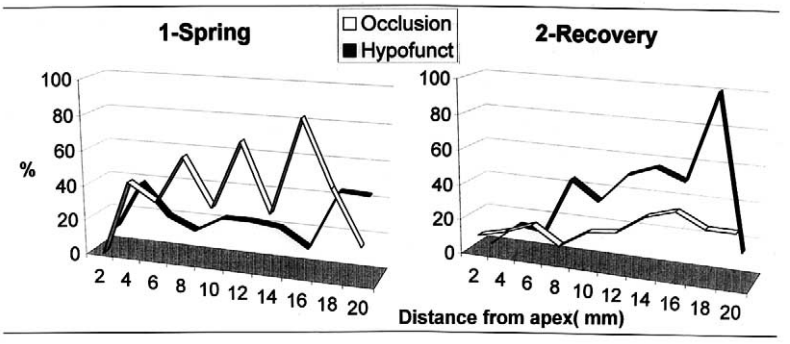

Fig 8. Frequency distribution of pulpal lesions after 2 weeks of spring application **(1)** and after 13 weeks of recovery **(2)** as it relates to their location along tooth axis.

Table III. Frequency of dental and periodontal lesions expressed as percentages of afflicted teeth

	Spring		*Recovery*	
Lesion	*Group A In occlusion*	*Group B Hypofunction*	*Group A In occlusion*	*Group B Hypofunction*
Tooth				
Pulp and odontoblast	100	80	30	78
Enamel folds	86	80	10	33
Dentinal folds	67	60	40	56
Damaged ameloblastic and papillary layers	100	90	90	100
Formation/calcification of enamel	43	90	40	67
Resorption of enamel	57	60	50	56
Resorption of dentin	43	70	60	78
Periodontal ligament				
Necrotic lesions	86	90	40	56
Inflammatory cells	100	100	100	100
Hemorrhages	100	100	100	100
Edema	100	100	100	100
Cell death	100	100	100	100

rupts the continuous mechanical force, rendering it intermittent and thus less damaging.[22,23]

Although the occlusal forces played only a relatively minor part in the orthodontic tooth movement, they were a major factor in the subsequent tissue recovery process. Except for the phenomena connected with periodontal inflammation, the various types of damage to the teeth and the PDL healed more rapidly and more extensively in the functional teeth compared with the nonfunctional incisors. Especially notable was the return to normal dimensions of the periodontal space. During spring activation, the size and the volume of the PDL are determined by the applied mechanical force (and are thus barely affected by the relatively weak functional loads); during the recovery period, the return of the periodontal dimensions to normal values is regulated by the rate and the direction of alveolar bone turnover. This remodeling (ie, adequate bone resorption in the formerly compressed areas and adequate bone apposition in the formerly expanded regions) was retarded in the hypofunctional teeth; the distortion of the PDL in these teeth remained comparable with that caused by spring activation. On the other hand, the PDL dimensions in the occluding teeth tended to return to normal, with the labial PDL rebounding to exceed normal values. The rebound phenomenon is defined as temporary excess above control level.[24] The underlying causes of this phenomenon are obscure, and one may speculate whether the excessive labial bone resorption is connected with the increased vascularization of the labial PDL in the rat incisor.

The morphological recovery of the PDL may be attributable not only to the beneficial effect of function, but also to the absence of the detrimental effect of

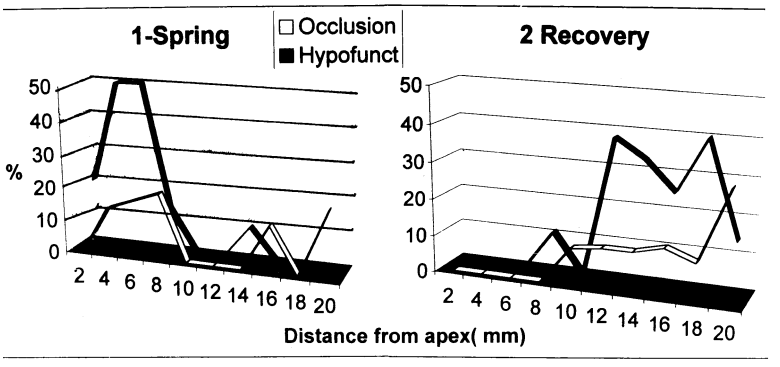

Fig 9. Frequency distribution of enamel lesions after 2 weeks of spring application **(1)** and after 13 weeks of recovery **(2)** as it relates to their location along tooth axis.

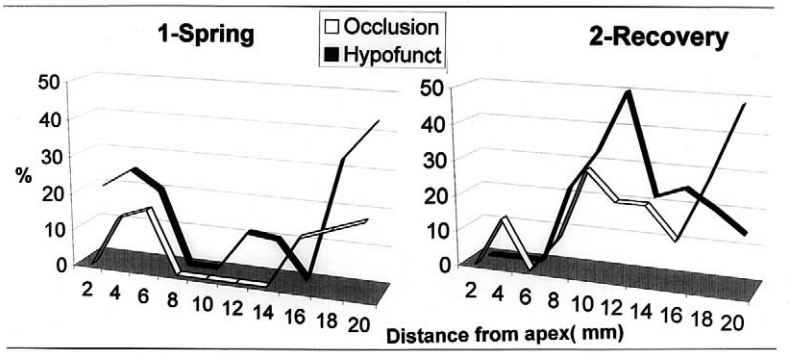

Fig 10. Frequency distribution of sites of dentin resorption after 2 weeks of spring application **(1)** and after 13 weeks of recovery **(2)** as it relates to their location along tooth axis.

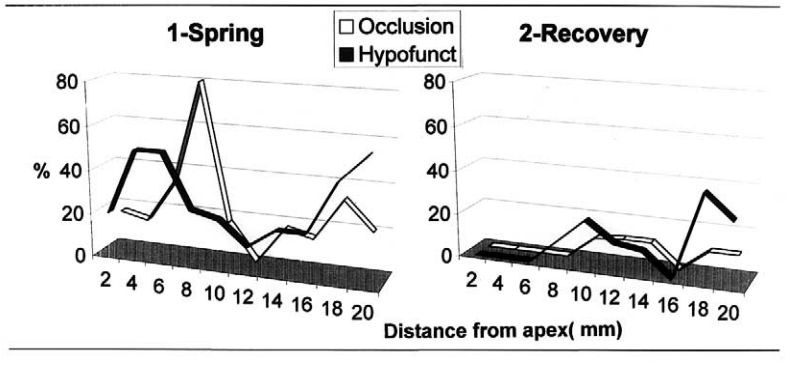

Fig 11. Frequency distribution of necrotic lesions after 2 weeks of spring application **(1)** and after 13 weeks of recovery **(2)** as it relates to their location along tooth axis.

hypofunction. In this respect, it is widely accepted that occlusal forces contribute to the maintenance of normal PDL space. In contradistinction, hypofunction causes many disorders, such as increased collagen synthesis and decreased collagen degradation,[8,11] thereby promoting widening of the PDL space; decreased bone mineralization[8]; increased bone formation at the alveolar crest[7]; and bone resorption.[5,25] Considering these

disorders, it becomes obvious why the PDL dimensions of the hypofunctional teeth lagged behind their occluding counterparts in returning to normal.

The detrimental effect of hypofunction was also linked to the delayed functional recovery of the eruptive potential of the nonoccluding incisors and to the retarded healing of the mechanically induced lesions. The deleterious effects of altered occlusal function include disorganization and disorientation of the collagenous fibers,[26] quantitative and qualitative changes of the fibroblasts,[9,11,12] changes in the extracellular matrix,[11] formation of necrotic lesions, and root resorption.[25] When the untoward consequences of hypofunction are superimposed on the mechanically induced tissue changes that are inherent in orthodontic treatment, tissue integrity is compromised, thereby impeding or preventing tissue recovery.

CONCLUSIONS

On the basis of the results from this study, the following conclusions can be drawn:

1. During active orthodontic treatment, neither presence nor absence of occlusal forces had a notable effect on the moving tooth. Nevertheless, in planning mechanotherapy, it is advisable to analyze the relationships between the vectors of the occlusal and the mechanical forces.
2. After tooth movement, ie, the retention period, the occlusal forces were of major importance in fostering and expediting periodontal recovery.
3. As far as the behavior of the human periodontium can be extrapolated from animal models, the retention (ie, recovery) period of teeth that are kept out of occlusion should be longer than the time allotted to teeth in functional occlusion.

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