

Periodontal healing following orthodontic movement of rat molars with intact versus damaged periodontia towards a bony defect

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SUMMARY The aim of this research was to determine whether orthodontic tooth movement influences periodontal healing.

In 16 male Wistar rats, 12 week of age, a bony defect was created mesial to both maxillary first molars, not including the attachment apparatus (group 1), and in 15 animals, the defect included the periodontal ligament (group 2). In both groups, the right first molar was moved mesially (orthodontic side) for 2 weeks followed by a 1-week retention period; the contralateral molar was not moved (control side). Histomorphometric analysis was performed. The results within and between the different treatment groups and sides were statistically compared by *t*-test and analysis of variance with repeated measures on logarithmic transformation.

Junctional epithelium was significantly larger at the control than at the orthodontic side of both groups ($P = 0.024$), and significantly larger in group 2 than in group 1 ($P < 0.001$). A significantly ($P = 0.034$) larger pocket depth was found at the control side in group 1. Supracrestal connective tissue was larger at the control than at the orthodontic side for both groups and significantly larger in group 2 than in group 1 ($P = 0.004$). Root resorption was found infrequently only at the orthodontic side in both groups (five out of 31 cases).

The principal findings suggest favourable effects of orthodontic tooth movement on restraining epithelial apical down-growth and decreasing pocket depth. Orthodontic treatment could not completely avoid formation of a long epithelial attachment. Therefore, periodontal regenerative surgery might be indicated prior to orthodontic tooth movement. Orthodontic movement, shortly after periodontal surgery, had no detrimental effect on periodontal soft tissue healing or on diminished but non-inflamed periodontal tissues.

Introduction

Healing following successful treatment of chronic periodontal lesions includes maturation of the gingival connective tissue, formation of a long epithelial attachment along the exposed root surface, and minimal new connective tissue attachment with bone regeneration (Polson and Heijl, 1978; Froum *et al.*, 1982; Bowers *et al.*, 1989; Wikesjö *et al.*, 1992). The influence of orthodontic tooth movement as a possible enhancing factor for periodontal regeneration has not been thoroughly investigated and is subject to controversy (Brown, 1973; Polson and Reed, 1984; Polson *et al.*, 1984; Geraci *et al.*, 1990; Nevins and Wise, 1990; Salama and Salama, 1993; Wennström *et al.*, 1993; Nemcovsky *et al.*, 1996; Mantzikos and Shamus, 1999).

After orthodontic tooth movement, osteoblasts responsible for bone formation originate from the periodontal ligament (PDL) cell population (Roberts and Chase, 1981). In rats, orthodontic tooth movement enhances bone apposition 6.5-fold in surgical bony defects with no direct contact with the periodontal tissue (Vardimon *et al.*, 2001). However, when

this defect type was compared with a periodontal bony defect including PDL damage, greater healing was found in the latter (Nemcovsky *et al.*, 2004). Although the mesiodistal dimension of a bony defect may be reduced when a tooth is orthodontically moved into the infrabony defect, reports are inconsistent regarding the nature of the resultant periodontal attachment. There have been conflicting results after tooth movement into areas with diminished bone level, tooth sockets following extraction, and surgical bone defects (Polson and Heijl, 1978; Polson and Reed, 1984; Polson *et al.*, 1984; Geraci *et al.*, 1990; Nevins and Wise, 1990; Wennström *et al.*, 1993; Liou and Huang, 1998; Vardimon *et al.*, 2001). Enhanced periodontal and bone regeneration by orthodontic tooth movement towards a bony defect (Geraci *et al.*, 1990; Nevins and Wise, 1990; Liou and Huang, 1998) and intrusive movement (Melsen, 1986; Cardaropoli *et al.*, 2001) have been reported. However, others did not find this effect (Polson *et al.*, 1984; Wennström *et al.*, 1993).

Human studies have shown that teeth with reduced but healthy periodontium can be orthodontically moved with no

enhanced periodontal destruction, provided pre-orthodontic hygiene treatment is undertaken and forces are maintained within physiological limits (Eliasson *et al.*, 1982). Orthodontic tooth movement into edentulous areas with reduced bone height and into extraction sites can be performed while the height of the connective tissue attachment of the supporting apparatus is maintained (Reed *et al.*, 1985; Lindskog-Stokland *et al.*, 1993), however, with a reduced alveolar bone crest level. There was no detrimental effect on connective tissue attachment when teeth were moved towards intrabony defects and subgingival infection was eliminated before orthodontic movement. However, enhanced periodontal destruction and connective attachment loss were observed when teeth were moved into inflamed defects (Wennström *et al.*, 1993).

The aim of the present study was to evaluate the influence of orthodontic tooth movement on periodontal soft tissue healing adjacent to two types of surgical bony defects, i.e. with or without PDL/root surface damage.

Materials and methods

The Committee for Animal Care Unit of the Faculty of Medicine, Tel Aviv University approved the study. The study comprised of 31 adult male Wistar rats, 12-weeks-old, weighing 350–450 g. The animals were divided into two groups according to the type of surgical defect produced: group 1 ($n = 16$) with a bony defect adjacent but not directly

associated with the maxillary first molar mesial root and without damage to the PDL (Figure 1) and group 2 ($n = 15$) with a bony defect including the maxillary first molar mesial root with damage to the PDL and root surface (Figure 2). Before each procedure, the animals were weighed and anaesthetized with an intramuscular injection of ketamine chlorhydrate (Rhône Mérieux, Lyon, France, 90 mg/1 kg body weight) and xylazine 2 per cent (Vitamed, Bat-Yam, Israel, 10 mg/1 kg body weight). During all treatment procedures, the animals were placed in a custom-made head-restraining device to allow correct access to the maxillary first molar region.

The experiment protocol has been previously described (Vardimon *et al.*, 2001; Nemcovsky *et al.*, 2004). Briefly, the protocol was carried out in five phases over 4 weeks.

Phase 1

A bony defect was created using a water-refrigerated high-speed-mounted 1-mm diameter round diamond bur, with similar dimensions in both groups (2 mm mesiodistal and buccolingual width and 2 mm depth). In group 1, the bony defect was prepared 1.5 mm anterior to the first molar with the aid of a specially constructed stent without root damage (Figure 1A); in group 2, the mesial aspect of the first molar mesial root and the proximal bone were involved (Figure 2A). The exposed root surface was root planed. One operator (CEN) performed all surgical procedures.

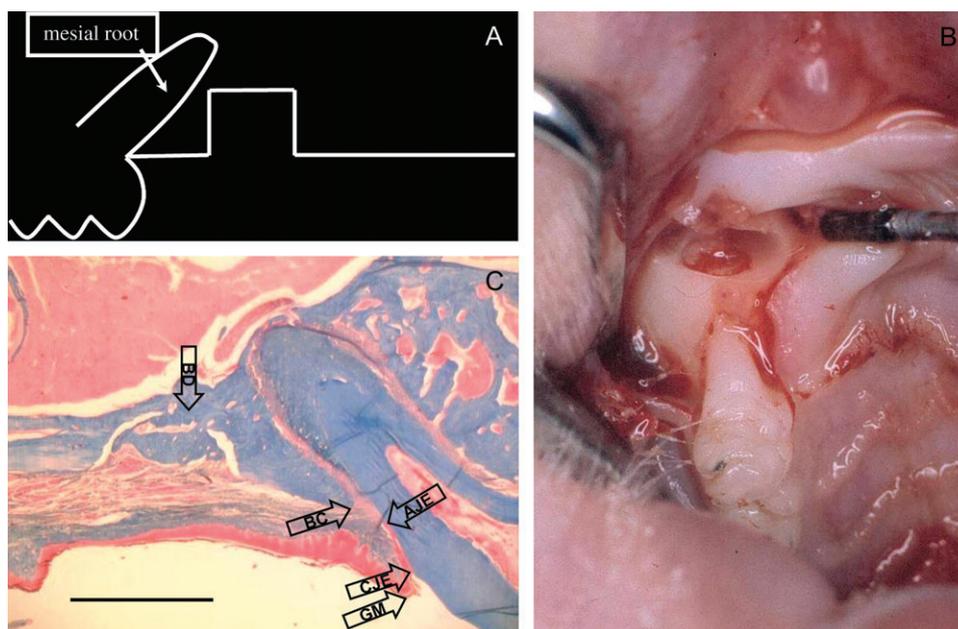


Figure 1 (A) Schematic drawing of the bone defect made in group 1, 2.0 mm anterior to both maxillary first molars with no involvement of the mesial root. (B) Clinical aspect of the prepared defect, note there is no contact between the defect and root surface. (C) Histological specimen from group 1 at the end of the study, from orthodontic side—bone defect (BD) does not include the root surface. Gingival margin (GM), coronal (CJE) and apical aspects (AJE) of the junctional epithelium, and bone crest (BC) are noted with arrows. Bar = 1 mm.

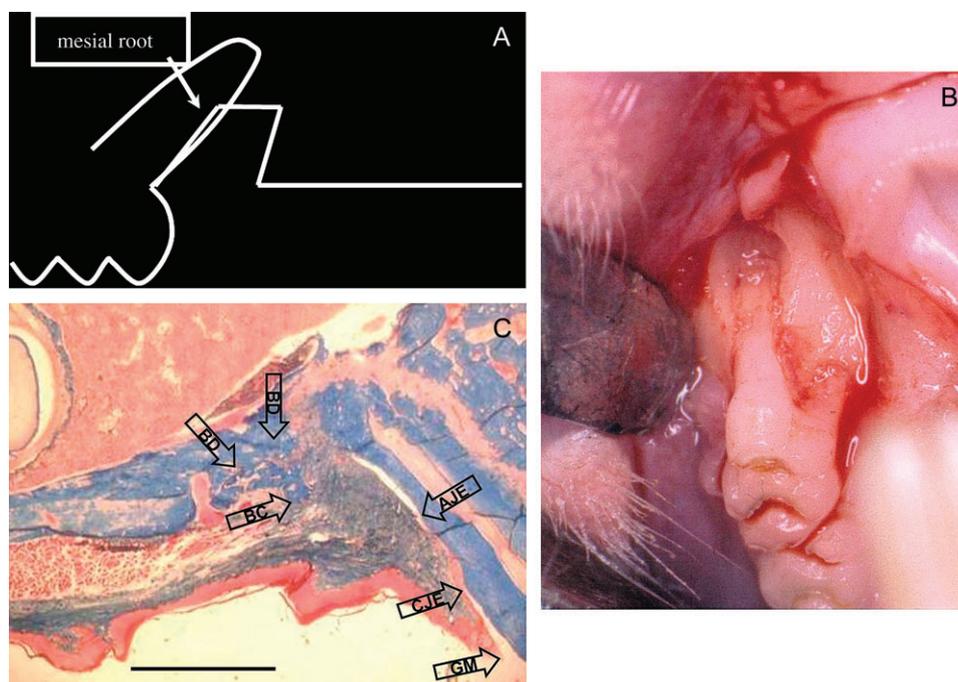


Figure 2 (A) Schematic drawing of the defect made in group 2 involving the bone and attachment apparatus of the mesial root of both maxillary first molars. This included the proximal bone and root planing of the exposed root surface. (B) Clinical aspect of the prepared defect, note that the defect involves the proximal root surface. (C) Histological specimen in group 2 at the end of the study, from the orthodontic side—bone defect (BD) includes root surface and periodontal attachment apparatus. Gingival margin (GM), coronal (CJE) and apical aspects (AJE) of the junctional epithelium lining part of the instrumented root surface, which had been prepared, and bone crest (BC) are noted with arrows. Bar = 1mm.

Phase 2

After a 1 week healing period, an orthodontic appliance was inserted and the maxillary right first molar was mesially moved for 1 week (orthodontic side); no orthodontic appliance was placed on the control side, thus, no orthodontic forces were applied to the left molar. For the orthodontic appliance, a mesiodistal hole was made along the two maxillary incisor crowns at the gingival margin. This hole moved from an initial gingival position to the incisal third of the crown by the end of the experiment due to the continuous eruption nature of the rat incisor. Most likely if pulp exposure has occurred, then it was sealed by a dentine bridge formation during the next 3 days (Inoue and Shimono, 1992). Throughout the study, the animals continued to eat and drink normally, as shown through weight gain, and no evident signs of suffering due to pulp damage were noticed. The appliance consisted of two elastomeric rings with no span (GAC International Inc., Central Islip, New York, USA) stretched between the first molar and incisors. A stainless steel ligature wire (0.09 inch) was threaded through one ring of the chain and wrapped tightly around the maxillary right first molar. A similar ligature wire was threaded tightly through the second elastic ring and the hole of the maxillary incisors (Figure 3). An initial force of 80–120 g was applied which diminished by 30 per cent after 30 minutes (Nattrass *et al.*, 1998).

Phase 3

The appliance was reactivated and mesial movement was continued in all animals independently of the amount of orthodontic tooth movement achieved (third week). As the first molar moved mesially, a space, approximately 0.7 mm after the first week, with a further 0.7 mm increase after reactivation, developed between the first and second molars. After 2 weeks of orthodontic tooth movement, tipping and displacement of 1.4 mm occurred. The space was calculated by measuring the distance between incisor tip and the mesial cusp of the first molar with help of a digital calliper (Mitutoyo Digimatic Electronic Caliper, Tokyo, Japan; 0.03 mm accuracy, 0.01 mm resolution) after each phase.

Phase 4

Retention phase (fourth week): active treatment was terminated and the elastomeric chain was replaced with a passive stainless steel ligature wire.

Phase 5

After 1 week of retention, the animals were anaesthetized with an intramuscular injection of ketamine chlorhydrate and xylazine and killed with CO₂, and the maxillary tissue blocks retrieved.

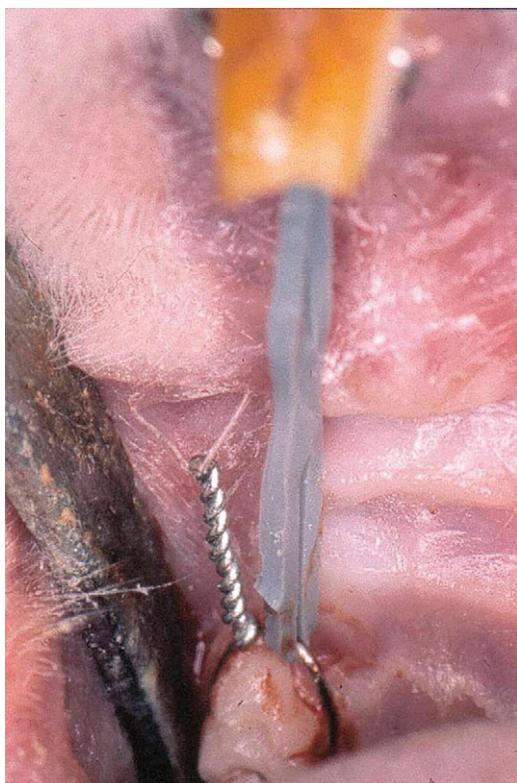


Figure 3 Orthodontic appliance, consisting of two elastomeric rings, with no span, stretched between the first molar and incisors. A stainless steel ligature wire was threaded through one ring of the chain and wrapped tightly around the maxillary right first molar.

Hemimaxillary blocks were fixed in 10 per cent neutral phosphate-buffered formalin and processed using routine histologic techniques for decalcified tissue. After embedding in paraffin, the blocks were serially cut parasagittally into 4–6 μm sections for light microscopy. The sections were stained with Mallory's trichrome. From each hemimaxilla, three slides from the central area of the defect, presenting the largest mesial root length and showing all measured variables, besides root resorption, were selected for computer-assisted histomorphometric analysis (Bioquant Nova Image Analysis System, R&M Biometrics, Nashville, Tennessee, USA). The mean from the three sections was calculated for all measurements. Since both groups differed in the nature of the surgical defect produced, a double-blind protocol was not used.

The measurements were made on the mesial aspect of the mesial root of the first molar, as follows (Figure 4):

- 1 Length of the junctional epithelium (JE), measured as the distance between the most coronal to the most apical aspects of the epithelium along the root.
- 2 Pocket depth, measured as the distance between the most coronal aspects of the JE on the root surface and gingival margin.

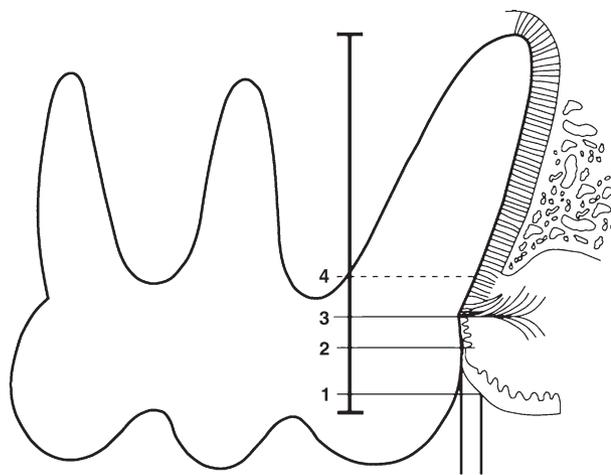


Figure 4 Schematic illustration of the measured variables: 1–2, pocket depth; 2–3, junctional epithelium; 3–4, supracrestal connective tissue.

- 3 Thickness of the supracrestal connective tissue, measured as the distance between the most apical aspects of the JE to the level of the alveolar bone crest.
- 4 Root length, measured as the distance between the cemento-enamel junction to the root apex of the mesial root.
- 5 Root resorption, presence or absence of root resorption areas along the mesial and distal sides of all roots.

Statistical analysis

Root resorption could not be statistically analysed due to the small number of animals that demonstrated this feature. For each variable, within groups, the difference, orthodontic minus control side (Δ), was calculated. Orthodontic and control sides within groups, and the Δ for each variable between groups, were compared by Student's *t*-test. The results were also analysed using analysis of variance (ANOVA) with repeated measures on logarithmic transformation to approach normal distribution, where the measured variables were the dependent variables and defect type (groups) and orthodontic/control (sides), the independent variables.

Results

The results are summarized in Table 1 and Figure 5.

Junctional epithelium

Within both sides, JE was larger at the control than at the orthodontic side of both groups. Δ JE (within groups) was similar for both groups. ANOVA showed a statistically significant effect of orthodontic tooth movement on JE ($P = 0.024$) with no interaction between orthodontic tooth movement and groups, i.e. orthodontic tooth movement had

Table 1 Measurements for all variables in both groups.

	Group 1		Group 2		<i>P</i>			
	Orthodontic (A)	Control (B)	Orthodontic (C)	Control (D)	A versus B	C versus D	A versus C	(A–B) versus (C–D)
Junctional epithelium	391 ± 109	451 ± 107	738 ± 191	812 ± 168	0.095	NS	0.001	NS
Pocket depth	210 ± 36	250 ± 83	225 ± 77	230 ± 44	0.034	NS	NS	NS
Supracrestal connective tissue	557 ± 262	577 ± 132	717 ± 207	844 ± 250	NS	0.077	0.004	NS
Root length	2428 ± 902	2373 ± 208	2415 ± 355	2493 ± 200	NS	NS	NS	NS
Root resorption	60037 ± 13765	0	26620 ± 19057	0				

Values in microns and root resorption in square microns; NS = not significant.

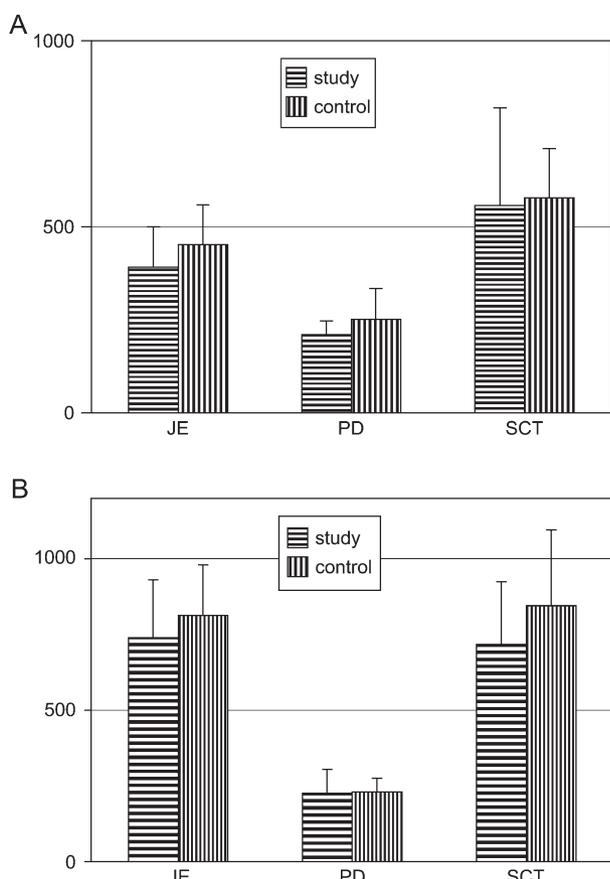


Figure 5 Graph comparing the three variables: junctional epithelium (JE), pocket depth (PD), and supracrestal connective tissue (SCT) between the study and control sides in (A) group 1 and (B) group 2 (values in microns).

a similar effect on JE in both groups. JE was statistically significantly larger in group 2 (where the defect included the periodontal attachment apparatus) than in group 1 for both study and control sides ($P < 0.001$).

Pocket depth

A statistically significantly ($P = 0.034$) larger pocket depth was found for the control side in group 1, but similar for

both sides in group 2. ANOVA showed no statistically significant effect of orthodontic tooth movement on pocket depth and no interaction between orthodontic tooth movement and groups. No statistical difference for Δ in pocket depth was found in either group.

Supracrestal connective tissue

Within both groups, supracrestal connective tissue was larger at the control than at the orthodontic side of both groups; however, it only approached statistical significance in group 2 ($P = 0.077$). ANOVA showed an effect of orthodontic tooth movement on supracrestal connective tissue that approached statistical significance ($P = 0.089$) with no interaction between orthodontic tooth movement and groups. A significant difference in supracrestal connective tissue between groups was found ($P = 0.004$); supracrestal connective tissue was larger in group 2 than in group 1.

Root length

The values for root length were similar between and within groups (Table 1). No statistical difference for Δ in root length was found between either group.

Root resorption

Root resorption was present in two of the 16 specimens in group 1 and in three of the 15 specimens in group 2. However, in these five animals, out of the 31 that comprised the study ($5/31 = 16$ per cent), root resorption was found only at the orthodontic side. Nevertheless, no statistical conclusion could be established due to the small number.

Discussion

The defect type in group 1 resembles a tooth adjacent to a bony defect, such as an old extraction site in which the adjacent tooth has an intact root surface with no association between the bony defect and the tooth. The defect type in

group 2 presented reduced bone level, initial periodontal attachment loss, and non-inflamed periodontal tissues. The validity of the measurements in this histomorphometric study is supported by the fact that root length was similar within and between groups.

The main finding of the present investigation was a significantly shorter JE at the orthodontic treated side in both groups. This response was similar, consistent in both groups, and statistically significant as analysed by ANOVA. Apparently, orthodontic tooth movement can enhance connective tissue healing and restrain the extent of the JE apical down-growth. However, in the present study, orthodontic tooth movement could not completely avoid formation of a long epithelial attachment along the planed root surface in group 2, since the JE at the orthodontic side was greater in group 2 than in group 1. This agrees with a previous study in monkeys (Polson *et al.*, 1984) in which reduction in width of an intrabony periodontal defect following orthodontic tooth movement was accompanied by a long JE. Nevertheless, the present findings could suggest that orthodontic tooth movement may enhance results following periodontal regenerative surgery (Nemcovsky *et al.*, 1996).

A positive statistically significant decrease in pocket depth was found at the orthodontic side of group 1. ANOVA showed no statistically significant difference in the effect of orthodontic tooth movement on pocket depth between groups, which means that this effect was actually similar in both groups. In previous research, a similar model was used to examine bone regeneration (Vardimon *et al.*, 2001; Nemcovsky *et al.*, 2004). Recently, a clinical study has shown that combined periodontic–orthodontic therapy results in significant probing depth reduction and radiographic bone fill (Corrente *et al.*, 2003); however, periodontal regeneration can only be assessed through histologic evaluation. The present study partially supports these clinical findings. Another recent investigation, with the same type of defects, demonstrated that following orthodontic tooth movement, bone healing of surgical defects directly associated with the PDL was greater than bony defects with no direct contact with the PDL (Nemcovsky *et al.*, 2004). This suggests that PDL originated cells may be involved in both the soft and hard tissue periodontal healing mechanism.

The dimensions of supracrestal connective tissue were smaller at the orthodontic side of both groups; however, this effect was greater in group 2. Supracrestal connective tissue reorganization can lead to a non-functional fibres orientated parallel to the root surface (Caton and Zander, 1976; Caton *et al.*, 1980). It is feasible that an extended retention period than that used in the present study could be required for supracrestal connective tissue reorganization.

The present findings agree with a previous report that orthodontic tooth movement performed shortly after surgical intervention has no negative effect on diminished

but non-inflamed periodontal tissues (Wennström *et al.*, 1993).

Root resorption appeared infrequently (16 per cent) at the orthodontic side but never at the control side. A higher incidence of root resorption has previously been reported (Polson *et al.*, 1984; Andreasen, 1985; Geraci *et al.*, 1990; Vardimon *et al.*, 1991; Karring *et al.*, 1997). This could be related to the fact that only severe root resorption was measured (cementum + dentine) and that only the mesial and not the distal root was assessed. In group 1, the root surface remained intact. However, in several specimens in group 2, minimal amounts of new cementum at the most apical extent of the planed root were observed. In the present report, this variable was not included.

Conclusions

The findings of the present study suggest a favourable orthodontic tooth movement effect on restraining epithelial apical down-growth and a decrease in pocket depth at the orthodontically treated side. Orthodontic tooth movement could not completely avoid formation of a long epithelial attachment along the planed root surface. Therefore, periodontal regenerative surgery prior to orthodontic tooth movement is indicated. Orthodontic tooth movement shortly after periodontal surgery produced no side-effects on periodontal soft tissue healing.

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