Dynamics of orthodontic root resorption and repair in human premolars: a light microscopy study

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SUMMARY The purpose of the study was to investigate the relationship between root resorption and repair in human premolars that had been orthodontically intruded. The objective was to examine these processes related to time and root development. Seventy-six premolars were divided into subgroups: 33 teeth were intruded and then extracted (G1); 25 teeth were intruded and then left in situ for varying periods before extraction (G2); 18 teeth served as the controls (G3). All teeth were examined by light microscopy. Using non-parametric statistical analysis, differences between the groups were examined with the Pearson chi-square test.

Teeth in G1 and G2 had significantly more resorptive lesions, 55 and 64 per cent, respectively, than the controls of 11 per cent. Resorption was observed over the whole root surface and increased with time. The occurrence increased to 100 per cent in both experimental groups after 36 days of intrusion. The appearance of lesions in relation to root development showed no differences between G1 and G2. In the apical part of the root, total resorption of the dentine was sometimes observed, but no resorptions extended into the predentine. Resorptive lesions undergoing repair were seen in both groups, with significantly more repair in G2 (58 per cent) than in G1 (32 per cent). Active resorption and repair were sometimes seen at the same resorption site. Deposition of cellular and acellular cementum was found to the same extent over the whole root when repair took place. With time, resorption appeared over the whole root surface. In some teeth, resorptive activity continued up to 10 days after removal of forces but on the other hand, repair of the resorbed area sometimes started during active movement. The individual variation in repair was much wider compared with resorption. The predentine layer in the apical area appeared not to be affected by the resorptive process.

Introduction

Root resorption is perhaps the most frequent adverse effect during orthodontic treatment. The type and magnitude vary substantially from mild apical root blunting in a large number of cases, to lateral root resorption, and infrequently, to extensive loss of the root (Breznika and Wasserstein, 2002; Abass and Hartsfield, 2007; Jönnson et al., 2007). Because of the potential for significant clinical and legal implications, assessment of the biological and mechanical basis of root resorption has been the subject of extensive research and discussion for almost a century. A comprehensive understanding of this problem has remained elusive because of the difficulty in comparing the results and conclusions of various studies that have utilized different experimental designs, patient populations, treatment mechanics, and analytical approaches. Additionally, the variability in radiographic techniques and material, which have been used, contributes to the discrepant findings. As a result of insufficient knowledge of the basic biological mechanisms of human root development and of the different appraisal of phylogenesis and odontogenesis, the findings in species other than humans, for example rodents, should be interpreted with caution (Reitan and Kvam, 1971; Bosshardt and Schroeder, 1996).

A positive association between active treatment duration and root resorption was demonstrated over 30 years ago (Mjör and Stenvik, 1969; Reitan, 1974). Since then, electron microscopy methods have become popular for demonstrating the third dimension of resorptive lesions (Kvam, 1972). Several studies have shown a spectrum of factors that may predispose patients to root resorption. A hereditary component is indicated by findings showing a significantly higher co-occurrence of root resorption among siblings than non-siblings (Harris et al., 1997). In the context of genetic predisposition, recent findings also support the notion of an association between interleukin-1β and tumour necrosis factor-α polymorphism and root resorption (Al-Quawasmi et al., 2003; Hartsfield et al., 2004).

The repair process of these resorptive lesions plays an important role in maintaining the functional integrity of a tooth that has been moved. Subsequent to odontoclast activity, an initial uncemented thin cementoid matrix on resting collagenous structures is deposited by repair cells, namely fibroblast-like and cementoblastic cells (Bosshardt and Schroeder, 1994; Brudvik and Rygh, 1995a,b). Probably, the activity of cementoblasts is influenced by growth factors such as epidermal growth factor, transforming growth factor-β, insulin-like growth factors or others (Sismanidou et al., 1996; Saygin et al., 2000).
To date, several histological light microscopic studies have been published on orthodontically induced root resorption in human teeth (Mjör and Stenvik, 1969; Reitan, 1974, Langford and Sims, 1982; Kurol et al., 1996; Lundgren et al., 1996; Owman-Moll et al., 1995a,b, 1996a,b; Kurol and Owman-Moll, 1998; Owman-Moll and Kurol, 1998, 2000). So far, only two have focussed on root resorption in intruded teeth (Mjör and Stenvik, 1969; Reitan, 1974). Therefore, the aim of this study was to investigate the occurrence and distribution of external root resorption caused by intrusion in relation to time and to the stage of root development, with emphasis on the dynamic relationship between the resorptive and repair processes.

**Materials and methods**

The material had been collected for previous investigations (Mjör and Stenvik, 1969; Stenvik, 1969, 1971; Stenvik and Mjör, 1970a,b). It consisted of 76 clinically intact premolars from 14 female and 16 male patients, aged between 10 and 13 years. All the teeth had been scheduled for extraction in connection with orthodontic treatment. None of the authors had any part in the diagnosis, treatment planning, or subsequent treatment of the malocclusions.

The appliance, which was used to intrude the teeth, has been described earlier (Mjör and Stenvik, 1970b). The intrusive force was measured at the start and end of active movement. The premolars were divided into three groups: group 1 (G1) comprised 33 teeth, which were intruded with forces of 35–250 g for 4–35 days and extracted immediately after the force had been removed. Group 2 (G2) consisted of 25 teeth, which were intruded with forces of 50–250 g for 5–28 days, left in situ for 4–104 days after the force had been removed and then extracted. Eighteen teeth were extracted without any exposure to orthodontic forces and served as the control material (G3). In 50 per cent, the teeth in the experimental and control groups were from different patients, 9 out of 18 teeth.

All teeth underwent routine histological procedures. Immediately after extraction, they were placed in 10 per cent neutral buffered formalin and fixed for 48 hours. They were then decalcified in 5.2 per cent nitric acid and embedded in paraffin. Serial sections, 5 μm thick, through the pulp in the axio-buccolingual direction were prepared and those with the longest corono-apical and widest buccolingual dimension were stained with haematoxylin and eosin. Eleven slides with six consecutive sections on each were prepared. Every second slide, in total five per tooth, was selected for examination. All together, 600 μm of the mesio-distal width of the root, 300 μm on each of the buccal and lingual sides, were examined by one author (BUW) by light microscopy (Labophot-2, Nikon, Tokyo, Japan). The amount and distribution of the resorptive lesions and repair, the resorption of the predentine layer in the apical area, and the occurrence of odontoclasts in the lesions were recorded in relation to the period of active intrusion and the subsequent rest period, to the stage of root development, and in which part of the root it took place. If small resorptive lesions were connected with a lesion in the neighbouring sections, they were counted as only one lesion. Resorptions were also recorded when the resorptive area was partly or totally repaired and a demarcation line in the dentine was seen.

The root development was categorized after Öhman (1965) into:

1. Partially developed root;
2. Fully developed root with open apex;
3. Fully developed root with closed apex.

Whether the repair tissue was of a cellular or acellular nature was recorded and the extent of repair in the resorptive lesions was categorized after Owman-Moll and Kurol (1998) into:

1. No repair;
2. Partial repair: parts of the surface of the resorptive lesion are covered with reparative cementum;
3. Functional repair: the total surface of the resorptive lesion is covered with reparative cementum without re-establishment of the original root contour;
4. Anatomical repair: the total surface of the resorptive lesion is covered with reparative cementum and the original root contour has been re-established.

**Statistical analysis**

Using non-parametric statistical analysis, differences between the groups were examined with the Pearson chi-square test. The significance level was $P \leq 0.05$. Ten randomly selected teeth were re-examined within a 2 months interval in order to test intra-examiner reliability. There was no significant difference compared with the first evaluation.

**Results**

Significantly, more resorptive lesions (Figure 1) were seen in both experimental groups than in the controls (Table 1). The occurrence of root resorption was independent of the stage of root development (Table 2). Odontoclasts were seen in most of the lesions (Figure 2) in both experimental groups (78% and 63%, respectively, in G1 and G2), but in none of the controls. The number of teeth with root resorption increased with time to 100 per cent after 36 days (Table 3), and the lesions were almost equally distributed over the root surface in both experimental groups. The predentine layer in the apical area was not affected by resorption, even when all the mineralized dentine was resorbed (Figure 3).

Repair took place and progressed with time during active intrusion. After 36 days, 33 per cent of the observed lesions
Significantly, more lesions were undergoing repair in G2 than in G1 ($P = 0.011$), revealing an increase in healing activity after the intrusive force had been removed (Table 4, Figure 4). After 70 days, 90 per cent of the lesions showed healing processes in G2. During this subsequent rest period, repair activity became more extensive, but anatomical repair was rarely seen (Table 5). In both experimental groups, the distribution of acellular and cellular reparative cementum were covered to some extent by reparative cementum in G1.
was similar and independent of the location of the lesion on the root.

**Discussion**

This histologic light microscopy investigation on intruded human premolars was performed in order to study the dynamics of orthodontically induced root resorption and, in particular, the reparative process in relation to periods of active tooth movement and rest. Having initially been collected for investigations concerning pulpal reactions on orthodontic movements (Stenvik, 1969, 1971; Stenvik and Mjör, 1970a, b), this material also provided information concerning the biological processes taking place on the surface of the root. The orthodontic appliance, which was used for intrusion, was somewhat different compared with other studies. For example, no bite block for excluding occlusal forces was utilized (Lundgren et al., 1996). However, intrusive tooth movements do not interfere with occlusal forces. In contrast to Reitan (1974), the interproximal contact areas between the test tooth and the neighbouring teeth were left without any manipulation to reduce friction. The test premolars were intruded with sectional springs on the buccal side, which also led to some labial tipping (Stenvik and Mjör, 1970b). The fact that no resorptions were detected on the cervical lingual third of the roots is in line with this assumption.

For clinical purposes, Reitan (1974) and Chan and Darendeliler (2005) advocated the use of light orthodontic forces during treatment in order to reduce the risk of root resorption. In the current investigation, force magnitude was measured twice, at the start and end of the active period of intrusion, but was of less importance for the objectives. Owman-Moll et al. (1996a, b) reported limitations with this approach, but essentially they found no correlation between force magnitude and the amount of root resorption. The present study supports the observation that force magnitude seems not to be important for the occurrence of resorption. Disparate findings between different studies on the effect of force magnitude may be due to individual variations in humans (Reitan, 1974; Harry and Sims, 1982; Owman-Moll et al., 1995a, b; Faltin et al., 1998; Acar et al., 1999; Harris et al., 2006).

The width of the histologically examined area comprised 600 μm of the lingual and labial surfaces of the root. In comparison with three-dimensional microscopy studies (Weiland, 2003; Chan and Darendeliler, 2004), the observed field may underestimate the actual amount of root resorption. However, this was not the main interest of the present research. The rationale for limiting the area of investigation was to study the relationship between the resorption and repair process in connection with active and rest periods and not to measure the extent and volume of resorptive lesions.

In contrast to radiographic investigations on intruded human incisors (Linge and Linge, 1983; Mavragani et al., 2002), no association was found between the stage of root

### Table 4

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>n under repair</th>
<th>P-value</th>
<th></th>
</tr>
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<tbody>
<tr>
<td>G1</td>
<td>44</td>
<td>14 32%</td>
<td>—</td>
<td>*</td>
</tr>
<tr>
<td>G2</td>
<td>48</td>
<td>28 58%</td>
<td>*</td>
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*P < 0.05.

![Figure 4](image)  
Resorptive lesion under partial repair with cellular cementum on the cervical buccal root surface of a lower first premolar (>10 original magnification, detail enlargement ×20 original magnification, average force 150 g, active intrusion 14 days, subsequent rest period 43 days). Black scale bar = 400 μm, blue scale bar = 200 μm.

### Table 5

<table>
<thead>
<tr>
<th>Period of active intrusion (days)</th>
<th>n</th>
<th>Categories of repair in G1 (see text)</th>
</tr>
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<tr>
<td></td>
<td>n</td>
<td>None</td>
</tr>
<tr>
<td>0–12</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>13–24</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td>25–36</td>
<td>27</td>
<td>16</td>
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<table>
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<tr>
<th>Subsequent rest period (days)</th>
<th>Categories of repair in G2</th>
</tr>
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<tbody>
<tr>
<td>0–35</td>
<td>36 19 9 3 5</td>
</tr>
<tr>
<td>36–70</td>
<td>10 1 6 1 2</td>
</tr>
<tr>
<td>71–105</td>
<td>2 0 1 0 1</td>
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n, number of resorptive lesions under repair.
development and the amount of root resorption. However, it cannot be excluded that incisors and premolars respond differently to orthodontic forces.

The increase of resorptive lesions in relation to time is comparable with findings when teeth are moved buccally (Kurol et al., 1996). The resorptive activity continued after force removal and, similar to the findings of Owman-Moll and Kurol (1998), odontoclasts were still present in resorptive lesions. This might be due to a remodelling process or to inherent individual disposition for resorption. When severe resorption is detected radiographically during orthodontic treatment, the period of rest should probably be quite extensive. The results of this study are in support of this concept as they show that the longer the rest period, the greater the repair. Resorptions in the apical area did not extend into the predentine. The morphology of the predentine formed during intrusion was affected in some teeth. Because of the uncalcified organic fibrillar content, predentine apparently is not targeted by odontoclasts (Reitan, 1974; Stenvik and Mjör, 1970b). These findings could explain root formation despite apical resorption, on the assumption that Hertwig’s epithelial root sheath remains intact and odontoblasts survive. This is in agreement with Reitan’s (1974) observation that apical root resorption does not prevent further development of roots in which there is a fairly wide predentine layer.

A main objective of this study was to investigate the repair process. Interestingly, the repair of some lesions started during active treatment, indicating on the one hand that the dynamics of the cellular environment and the potential for repair in humans may vary, and on the other that the process probably starts earlier in humans than in other species. A recent study (Jäger et al., 2008) showed that in rodents the repair processes did not start until the orthodontic force was released. In addition, it is noteworthy that repair by cellular cementum occurred as frequently in the cervical and apical parts of the root (Figure 4). After 5 weeks without force application, almost half of the lesions were undergoing repair and 90 per cent after 10 weeks. Other investigations on human teeth found similar rates (Langford and Sims, 1982; Owman-Moll and Kurol, 1998). This suggests that when resorption is recorded radiographically, cessation of active treatment or rest periods should be recommended as they initially lead to repair of the lesions. However, due to the limited material investigated, this interpretation has to be examined further.

Deposition of either acellular or cellular cementum was independent of the location of a lesion and of the category of repair. These findings are contrary to the results of some studies, which reported deposition mainly of cellular and acellular cementum only in the initial phase of repair (Langford and Sims, 1982; Bosshardt, 1994; Owman-Moll and Kurol, 1998). In future investigations on orthodontic root resorption, closer examination of the process of repair may contribute to the understanding of why resorption is progressive in some individuals but not in others.

Conclusion

With time, resorption appeared over the whole surface of intruded roots. In some teeth, resorptive activity continued after removal of the force. The predentine layer in the apical area appeared not to be affected by the resorptive process. Repair of the resorbed area might start during active movement, but there is a wide individual variation. During repair, both cellular and acellular cementum were distributed to the same extent in the lesions, independent of their location.

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