Influence of Orthodontic Extrusion on Pulpal Vitality of Traumatized Maxillary Incisors

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Abstract

Introduction: The aim of this retrospective study was to examine the effect of orthodontic extrusion on the pulp vitality of maxillary incisors with a history of trauma. Methods: Pulpal condition was examined clinically (rating of crown color and sensitivity testing with a cryogenic spray) and radiologically (periapical and panoramic radiographs) after orthodontic extrusion of previously traumatized (Orthodontics/Trauma group, n = 77) and nontraumatized teeth (Orthodontics group, n = 400) and after previous dental trauma without subsequent orthodontic treatment (Trauma group, n = 193). Dental traumatia were divided into hard tissue injuries (fracture of enamel and enamel chipping, fracture of enamel-dentin without pulpal involvement, fracture of enamel-dentin with pulpal involvement, root fracture, crown-root fracture) and periodontal injuries (concussion, subluxation, intrusion, extrusion, lateral luxation, and avulsion). Results: Teeth in the Orthodontics/Trauma group showed a significantly higher frequency of pulp necrosis than teeth in the Orthodontics group (P < .001) or teeth in the Trauma group (P < .009). In addition, teeth in the Orthodontics/Trauma group with periodontal injuries showed a significantly higher rate of pulp necrosis than teeth in the Orthodontics group (P < .001) or the corresponding teeth in the Trauma group (P = .004). No significant differences were observed between teeth in the Orthodontics/Trauma group with previous hard tissue injuries and teeth in the Orthodontics group or the corresponding teeth in the Trauma group. In addition, no statistically significant differences were determined between central and lateral incisors. Conclusions: The results indicated that maxillary incisors with a history of severe periodontal injury have a higher susceptibility to pulp necrosis during orthodontic extrusion than nontraumatized teeth. (J Endod 2010;36:203–207)

Key Words
Dental trauma, orthodontic treatment, pulp necrosis

Previous investigations have found a high prevalence of traumatized permanent incisors among candidate teeth undergoing orthodontic therapy (1–3). Therefore, orthodontic movement of previously traumatized teeth represents a common problem in routine orthodontic treatment. Several studies have examined the incidence of root resorption after orthodontic treatment of traumatized teeth (4–10). However, only a few attempts have been made to date to analyze the influence of orthodontic treatment on the pulpal vitality of traumatized teeth (11–15). One previous study found that orthodontic intrusion of teeth, and especially of lateral incisors with severe periodontal tissue injuries, increases the risk of pulp necrosis compared with intrusion of nontraumatized teeth (11). However, to our knowledge, no previous investigation has examined the influence of orthodontic extrusion on the pulpal vitality of traumatized teeth.

Therefore, the aim of the present study was to examine the influence of orthodontic extrusion on the pulpal vitality of previously traumatized permanent maxillary incisors. Additional objectives were to assess the influence of different parameters such as the type of dental trauma, the type of incisor, extrusion period, and duration of orthodontic treatment on pulp vitality.

Materials and Methods

In this retrospective study, orthodontically treated patients with traumatized maxillary incisors (Orthodontics/Trauma group) were compared with orthodontically treated patients with nontraumatized maxillary incisors (Orthodontics group) and with patients with previous dental trauma to the maxillary incisors and no subsequent orthodontic treatment (Trauma group).

The orthodontically treated investigation groups (Orthodontics/Trauma and Orthodontics groups) were selected from the files of all patients who had completed the retention period after active orthodontic treatment with fixed appliances between 1994 and 2008 at 3 private orthodontic practices. All patients who had presented with an anterior open bite and had been treated orthodontically by using molar and incisor banding with a utility type of arch wire to extrude the maxillary incisors (16) were selected for further examination. According to the presence of previous dental trauma to the permanent maxillary incisors before onset of orthodontic treatment, these patients were divided into 2 groups, the Orthodontics/Trauma group and the Orthodontics group. The classification of Andreasen (17) was adapted to divide the dental traumatia into hard tissue injuries (fracture of enamel and enamel chipping, fracture of enamel-dentin without pulpal involvement, fracture of enamel-dentin with pulpal involvement, root fracture, crown-root fracture) and periodontal tissue injuries (concussion, subluxation, intrusion, extrusion, lateral luxation, and avulsion).

The presence of previous dental trauma was determined retrospectively from the dental records made at baseline. These included a standardized questionnaire (2) and...
the findings of the pretreatment clinical and radiologic examination before the initiation of treatment. During this clinical examination, the permanent incisors of all patients had been examined for evidence of traumatic injury, and a sensitivity test with a cryogenic spray (Provo-test; Hoechst, Frankfurt, Germany) had been performed in all traumatized teeth before the onset of orthodontic treatment. In addition, standardized pretreatment periapical radiographs had been obtained for all traumatized teeth, and pretreatment panoramic radiographs were available for all nontraumatized teeth.

**Orthodontics Group with Previous Trauma**

(Orthodontics/Trauma Group)

Additional criteria for subject selection in the Orthodontics/Trauma group were (1) complete dental records including the initial classification of dental traumatata, (2) positive sensitivity testing of the traumatized teeth before the onset of orthodontic treatment, indicating pulpal vitality, (3) presence of pretreatment and postretention periapical radiographs, and (4) results of the postretention sensitivity testing.

A total of 66 patients (45 males and 21 females) with 77 traumatized permanent maxillary incisors (50 central and 27 lateral incisors) were found to meet the inclusion criteria. Previous hard tissue injuries (fracture of enamel or fracture of enamel-dentin) were found in 32 teeth, and previous periodontal tissue injuries (subluxation, extrusive, lateral or intrusive luxation) were observed in the remaining 45 teeth (Table 1). The mean age of the patients at the time of trauma was 10.0 years (range, 7.3–16.7 years). The mean age at the end of orthodontic treatment was 15.5 years (range, 13.5–18.5 years).

**Orthodontics Group without Previous Trauma**

(Orthodontics Group)

The Orthodontics group comprised 100 randomly selected patients (64 females and 36 males) with 400 permanent maxillary incisors and no clinical or radiologic signs of pathosis or trauma and no history of dental trauma before the onset of orthodontic treatment. The mean age of the patients at the end of orthodontic treatment was 15.9 years (range, 13.5–19.0 years).

**Orthodontic Treatment**

In the Orthodontics/Trauma group, orthodontic treatment had been initiated at the earliest 3 months after hard tissue injuries or subluxation and 12 months after severe periodontal tissue injuries (lateral, extrusive, or intrusive luxation) (11). All patients were treated with a preadjusted appliance with 0.018-inch slot brackets. Orthodontic extrusion of the maxillary incisors was performed by means of a 0.016 × 0.016 inch Elgiloy (Rocky Mountain Orthodontics, Denver, CO) blue extrusion arch from the maxillary tube of the maxillary right first molar to the maxillary left first molar. First molars were usually consolidated with the premolars and canines with passive segmented arches. Force levels were adjusted to approximately 20 gm per tooth and usually checked at each visit. Calibration was performed in the mouth with a light force gauge (Haag-Streit, Bern, Switzerland). After correction of the vertical malocclusion, treatment was continued with continuous arch wire mechanics. The extrusion period averaged 4.8 months (range, 3.2–6.5 months) in the Orthodontics/Trauma group and 5.2 months (range, 3.4–7.0 months) in the Orthodontics group. The total treatment time with fixed appliances averaged 23.8 months (range, 11.7–31.2 months) in the Orthodontics/Trauma group and 24.0 months (range, 12.2–31.7 months) in the Orthodontics group. After termination of active orthodontic treatment, retention was performed with removable appliances. The mean retention period was 2.1 years in the Orthodontics/Trauma group (range, 1.0–3.6 years) and 2.3 years in the Orthodontics group (range, 1.3–2.8 years).

**Trauma Group**

The Trauma group included 173 patients (112 males and 61 females) with 193 traumatized teeth. Sixty-eight teeth had sustained hard tissue injuries (fracture of enamel or fracture of enamel-dentin), and previous periodontal tissue injuries (subluxation, extrusive, lateral, or intrusive luxation) were found in the remaining 125 teeth. All traumatized teeth in these patients showed a positive reaction to sensitivity testing after dental trauma. Only patients with a minimum follow-up period of 3 years after dental trauma were included in this study group. The mean age of the patients at the time of trauma was 9.3 years (range, 6.6–16.4 years). The mean age at re-examination was 14.7 years (range, 12.5–27.3 years).

**Evaluation of Pulpal Vitality**

In the Orthodontics/Trauma and Orthodontics groups, the final pulpal vitality was evaluated at the end of the retention period. In the Trauma group, the final pulpal vitality was evaluated during re-examination after an average follow-up period of 5.5 years (range, 3.1–9.5 years) after the dental trauma.

In the Orthodontics/Trauma and Trauma groups, the final evaluation of pulpal vitality included rating of crown color (normal or grayish), sensitivity testing with a cryogenic spray, and periapical radiographs. In the Orthodontics group, the final examination of pulpal vitality was performed by rating crown color and panoramic radiographs. However, additional sensitivity testing and periapical radiographs were performed in both orthodontically treated groups during orthodontic treatment if pulp necrosis (ie, gray color changes in the crown, pain, or swelling) was suspected.

The following clinical and radiologic criteria were used to define pulp necrosis: loss of pulpal sensitivity, gray color changes in the crown, and periapical radiolucency. Loss of pulpal sensitivity and at least one other clinical or radiologic sign were considered necessary before the diagnosis was made (11).

To estimate the reliability of radiographic assessment of periapical radiolucencies, 100 randomly selected periapical radiographs were re-evaluated within a 4-week interval by 2 examiners. Interexaminer agreement was calculated by using kappa statistics, which showed very good interexaminer agreement was found (κ = 0.90).

**Statistical Analysis**

The χ² test was used to detect significant intergroup differences and to determine significant relationships between pulp necrosis and type of incisors (central or lateral), extrusion period (≤4.8 months or >4.8 months), and duration of orthodontic treatment (≤23.8 months or >23.8 months). The χ² test was also carried out to determine significant intergroup differences between the various types of

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**TABLE 1. Distribution of Traumatized Teeth in the Orthodontics/Trauma and Trauma Groups According to Type of Trauma**

<table>
<thead>
<tr>
<th>Type of trauma</th>
<th>Orthodontics/Trauma group</th>
<th>Trauma group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enamel fracture</td>
<td>14</td>
<td>36</td>
</tr>
<tr>
<td>Enamel-dentin fracture</td>
<td>18</td>
<td>32</td>
</tr>
<tr>
<td>Subluxation</td>
<td>14</td>
<td>31</td>
</tr>
<tr>
<td>Lateral luxation</td>
<td>9</td>
<td>33</td>
</tr>
<tr>
<td>Extrusive luxation</td>
<td>10</td>
<td>30</td>
</tr>
<tr>
<td>Intrusive luxation</td>
<td>12</td>
<td>31</td>
</tr>
<tr>
<td>Total</td>
<td>77</td>
<td>193</td>
</tr>
</tbody>
</table>

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(continued...
Results

Pulp necrosis was detected in 9.1% of the teeth in the Orthodontics/Trauma group (n = 7), in 0.5% of the teeth in the Orthodontics group (n = 2), and in 1.6% of the teeth in the Trauma group (n = 3). No patient with more than 1 tooth with pulp necrosis was included in any of the investigated groups. Teeth in the Orthodontics/Trauma group showed a significantly higher frequency of pulp necrosis than teeth in the Orthodontics group (P < .001) or teeth in the Trauma group (P < .009). No significant differences were determined between the Trauma and the Orthodontics groups.

With respect to the correlation between pulp necrosis and type of incisor, no statistically significant differences were determined in the Orthodontics/Trauma group between central and lateral incisors. Pulp necrosis was observed in 8.0% (n = 4) of the traumatized central incisors and in 11.1% (n = 3) of the lateral incisors.

In the Orthodontics/Trauma group, 5 cases of pulp necrosis were diagnosed during orthodontic extrusion (71.4%), and the remaining 2 cases (28.6%) were diagnosed during later orthodontic treatment stages. No significant correlation was found between pulp necrosis and extrusion period (≤4.8 months or >4.8 months) or duration of orthodontic treatment (≤23.8 months or >23.8 months). In the Orthodontics group, both patients with pulp necrosis were diagnosed during active orthodontic treatment after extrusion.

In the Orthodontics/Trauma group, pulp necrosis was determined in 22.2% (n = 2) of the teeth after lateral luxation, in 20.0% (n = 2) of the teeth after extrusion, and in 25.0% (n = 3) of the teeth after intrusion injury. No cases of pulp necrosis were observed in teeth with fracture of enamel, fracture of enamel-dentin, or subluxation injury. In the Trauma group, the 3 cases of pulp necrosis occurred after previous subluxation and lateral and intrusive luxation injuries.

Teeth in the Orthodontics/Trauma group with periodontal tissue injuries showed a significantly higher rate of pulp necrosis than teeth in the Orthodontics group (P < .001). No significant differences were observed between teeth in the Orthodontics/Trauma group with previous hard tissue injuries and teeth in the Orthodontics group. In addition, significant differences were determined between teeth with periodontal tissue injuries in the Orthodontics/Trauma group and the corresponding teeth in the Trauma group (P = .004). No significant differences were determined between the Orthodontics/Trauma and the Trauma groups with respect to hard tissue injuries (Fig. 1).

Discussion

The results of the present investigation indicated that orthodontic extrusion of teeth with severe periodontal tissue injuries increases the risk of pulp necrosis as compared with extrusion of nontraumatized teeth.

Numerous investigations have shown that orthodontic tooth movement can affect the blood supply to the dental pulp (18–22). Of the possible force factors that can be applied to teeth during orthodontic treatment, intrusion is thought to have the greatest impact on the apical region (23–25). Several studies observed an obvious reduction of pulpal blood flow and marked histologic changes in the pulp as a result of intrusive forces (26–30). However, similar findings were reported after orthodontic extrusion of human premolars, including circulatory disturbances in the pulp with congested blood vessels, vacuolization, and edema of the pulp tissues (31). Nevertheless, the pulpal circulatory system of nontraumatized teeth seems to be capable of compensating for the reduction of pulpal blood flow, so that in these teeth, orthodontic extrusion does not usually result in pulp necrosis. In contrast, the findings of the present investigation suggest that previously traumatized teeth with severe periodontal tissue injuries are more susceptible to pulp necrosis during orthodontic extrusion. Although all traumatized teeth in the Orthodontics/Trauma group showed pulpal vitality before the onset of orthodontic treatment, it might be concluded that the capacity of the blood vessels supplying the pulp was insufficient for maintaining an adequate pulpal blood flow during orthodontic extrusion. A possible explanation could be that severe periodontal tissue injuries might cause permanent damage to and reduction of apical vessels, which might render these teeth more prone to pulp necrosis during orthodontic extrusion. Therefore, the combination of impaired pulpal blood flow during orthodontic extrusion with reduced capacity of the apical vessels might explain the significantly increased rate of pulp necrosis in teeth with previous periodontal tissue injuries.

No significant correlation was found between pulp necrosis and extrusion period or duration of orthodontic treatment. However, more than 70% of the cases with pulp necrosis in the Orthodontics/Trauma group occurred during the initial extrusion period. This emphasizes the detrimental effect of orthodontic extrusion on the pulpal blood flow of traumatized teeth.

Two teeth in the Orthodontics group showed signs of pulp necrosis during orthodontic therapy. Previous studies have reported pulp necrosis of nontraumatized teeth during orthodontic treatment (32–34). Possible explanations include anatomic variations of the apical foramen or the supplying vessels (30). In the present study, patients with previous dental trauma were identified by a standardized questionnaire and pretreatment clinical and radiologic examinations. However, it cannot be completely ruled out that both teeth had sustained unnoticed trauma and therefore developed pulp necrosis during orthodontic tooth movement.

This was a retrospective study with inherent limitations. Because sensitivity testing and periapical radiographs are usually not performed during regular orthodontic treatment, diagnosis of pulp necrosis in the Orthodontics group was based on clinical inspection and examination of panoramic radiographs after the retention period. However, all patients in this group were followed up for at least 1 year after active
orthodontic treatment. Therefore, it might be assumed that all cases of pulp necrosis were detected during this period.

The most commonly used tests for registering pulpal sensitivity in general practice are electrical and thermal tests such as the cold test with dichlor-difluormethane used in the present investigation. In contrast to the results of electric pulp testing, thermal pulp testing results are not reproducible in terms of graded intensity (35). In the present investigation, only a cold test was available for the examination of pulpal sensitivity. However, previous studies have reported a similar sensitivity and specificity of cold and electric tests to register pulpal vitality (35, 36), so that the use of a cold test most probably had no negative effect on the results obtained.

As a rule, the diagnosis of pulp necrosis is based primarily on 2 or more of the following signs: negative sensitivity testing, crown discoloration, and periapical radiolucency (37). However, loss of pulpal sensitivity or crown discoloration does not necessarily indicate pulp necrosis. A previous study observed higher sensitivity thresholds in teeth undergoing orthodontic treatment (38), and a lowered or absent response to sensitivity tests has been reported in teeth with progressive pulp obliteration after dental trauma (39). In addition, a pink crown discoloration indicates intrapulpal bleeding and is usually seen within 2–3 days after acute trauma, but it might disappear 2–3 weeks later, so that the crown regains its natural color (40). A yellow discoloration of the crown is seen in cases with progressive pulp obliteration and represents the reaction of a vital pulp to severe trauma (39). Finally, although a persistent gray discoloration usually indicates pulp necrosis and probably bacterial contamination of the pulp (37, 40), only temporary gray discoloration after dental trauma, followed by normalization of the crown color, has been reported in a previous investigation (41). Consequently, loss of pulpal sensitivity alone or coronal discoloration alone is not enough to justify a diagnosis of pulp necrosis. The development of periapical radiolucency has so far been considered the only sure sign of pulp necrosis (37). However, more recent studies have questioned the validity of the presently accepted diagnostic criteria, because in cases with transient apical breakdown, even the concomitant presence of all 3 classic signs of pulp necrosis could still be followed by pulp repair (37, 41, 42). In the present investigation, all 9 orthodontically treated subjects with pulp necrosis showed additional pain or swelling, so that the presence of a transient apical breakdown was considered to be very unlikely in these cases. Previous studies have shown that orthodontic movement of endodontically treated teeth has only a minor effect on the healing process of periapical lesions (43) and that root-filled teeth can normally be moved without extensive root resorption (44). Therefore, all 9 patients were immediately referred for endodontic treatment, and orthodontic movement of the affected teeth was resumed soon after termination of endodontic therapy and was completed successfully in all cases.

In contrast, no pain or swelling was observed in the teeth with pulp necrosis in the Trauma group, so that a conservative approach with further observation and without immediate endodontic intervention was considered to be appropriate (37). However, because of signs of acute inflammation, endodontic treatment was carried out in all 5 cases within the first 9 months after the diagnosis.

In conclusion, maxillary incisors with a history of trauma and severe periodontal tissue injuries have a higher susceptibility to pulp necrosis during orthodontic extrusion than do nontraumatized teeth. Most cases of pulp necrosis occurred during the initial extrusion period. Therefore, orthodontic extrusion of maxillary incisors with a history of trauma should be performed with lower extrusion forces than those used in the present investigation, and pulpal vitality should be screened regularly until the end of the retention period.

References