Traumatically Intruded Teeth

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Various combinations of orthodontic force, luxation, and observation are applied to 12 teeth traumatically intruded in 3 large dogs. All showed root resorption to some degree. Early orthodontic repositioning, with luxation if immobile, produced the best recovery.

KEY WORDS: • ANKYLOSIS • INTRUSION • LUXATION • ROOT RESORPTION • TRAUMA •

A traumatically intruded tooth is one that is forcefully and abruptly displaced from its position into the surrounding alveolar bone (Fig. 1). The resultant condition is also called intrusive luxation, and predominantly involves the maxillary anterior teeth (ANDREASEN 1970). Although intrusion of permanent teeth is infrequent (SNAWDER 1976), the sequelae compromise the longevity of the tooth and often include pulp necrosis, pulp obliteration, root resorption, ankylosis, and loss of marginal bone.

Different methods have been proposed for managing this type of dental injury. BRUSZT (1958) suggests that the best treatment is to do nothing, based on his observation of spontaneous re-eruption of intruded teeth. SKIELLER (1960), on the other hand, recommends immediate repositioning in most cases. Because abrupt repositioning of an intruded tooth could increase the likelihood of root resorption, ANDREASEN (1976) suggests immediate orthodontic traction to reposition the injured tooth. TAINTOR ET AL. (1977) recommend first leaving the tooth alone to allow it to re-erupt spontaneously, and in those instances where it does not, apply an extrusive orthodontic force.

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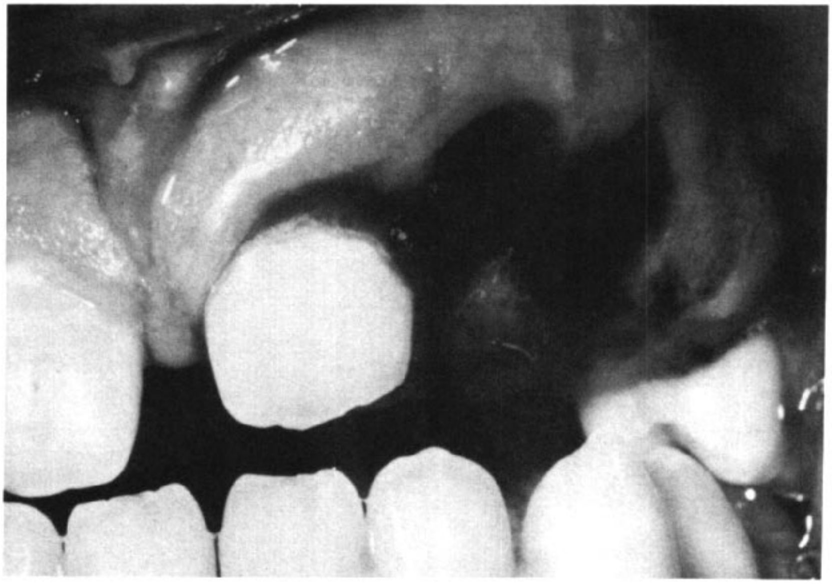


Fig. 1 Intrusive luxation of the left maxillary permanent central incisor (slight) and lateral incisor and cuspid (severe) in a 14yr-old boy.

Turley et al. (1984), investigating these latter two methods of managing traumatically intruded teeth, report that teeth intrusively luxated did not re-erupt on their own in cases of severe intrusion. Furthermore, the application of orthodontic forces often failed to extrude the intruded teeth and sometimes actually caused an undesirable intrusive movement of the anchorage teeth.

Histologic examination showed severely injured teeth to be ankylosed, suggesting that luxation may facilitate orthodontic repositioning in cases where mobility cannot be demostrated.

Certain factors, such as the amount of intrusion and the mobility of a tooth must be considered in the management of intrusive luxation, and the present study was designed to answer some of the questions about how these factors may affect the prognosis.

— Materials and Methods —

Three young adult collie type dogs were used for this project. Prior to each procedure, the dog was sedated with 2.0 cc of intramuscular acepromazine, 2.0cc demerol, and 1.0cc atropine. An intravenous injection of nembutal (30 mg/kg) was used as the anesthetic.

Alginate impressions were taken for study casts, and the dentition photographed. Amalgam markers were placed in the coronal portions of the canine and first and second premolars in each quadrant, and tantalum markers placed in the buccal cortical bone near the teeth to serve as radiographic reference points to assess tooth movement during the study.

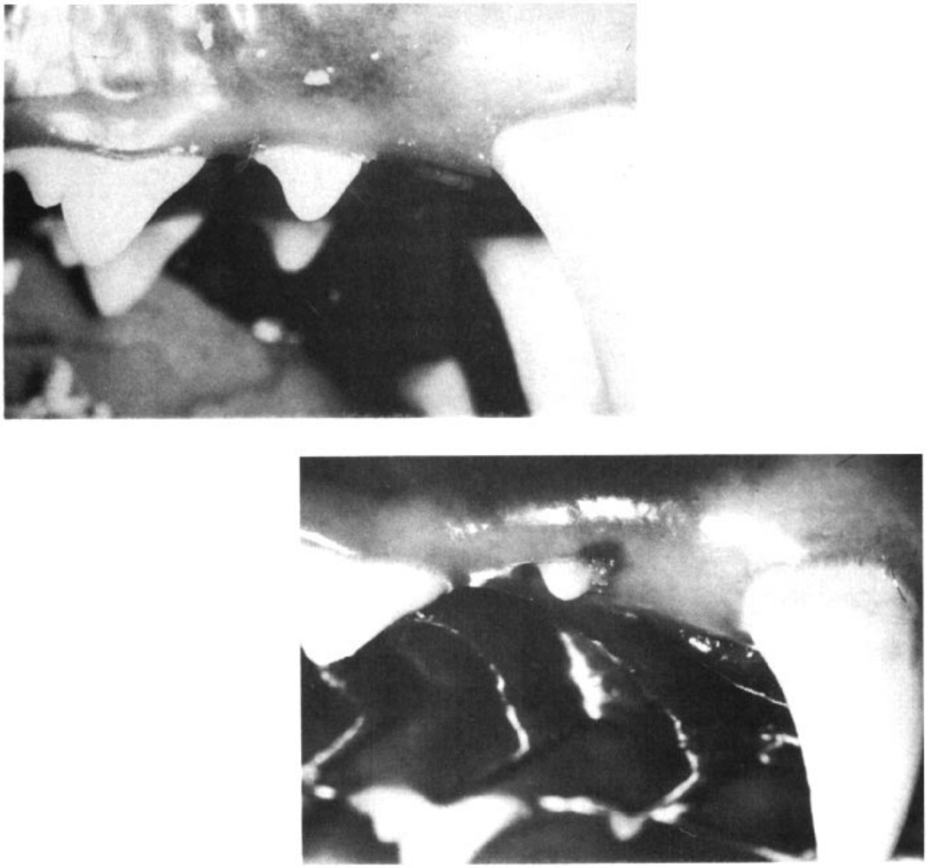


Fig. 2
top **Canine maxillary left first premolar (center) prior to intrusive luxation.**
bottom **Same tooth after traumatic intrusion.**

A positioning jig was fabricated from acrylic to facilitate consistent orientation for serial periapical x-rays using the bisected angle technique.

The four first premolars of each dog were selected for the experimental traumatic intrusion (Fig. 2). Initial periapical radiographs were taken to confirm complete root development, and to provide a baseline for later evaluations.

Prior to the intruding blow, cast gold crowns were placed over each tooth to prevent crown fracture. A C-clamp was then placed across each arch to support the cortical plates and avoid avulsion of the teeth as they were intruded. Finally, a holding instrument was placed over the crown, and a "gravity hammer" used to apply the intrusive blow along the long axis of the tooth.

One maxillary and one mandibular tooth was selected randomly for orthodontic traction, and the contralateral tooth left untreated. Orthodontic bands

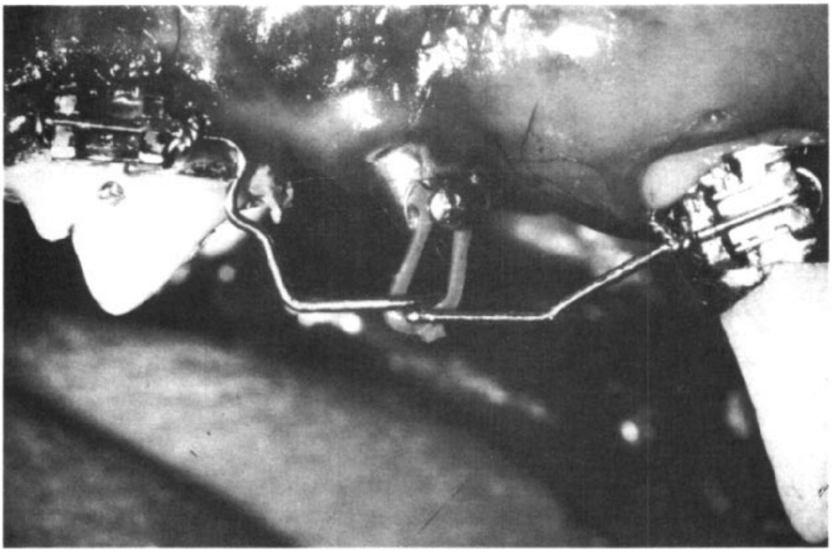


Fig. 3 Orthodontic appliance activated

were cemented to the canines and second premolars on either side of the experimental teeth. An undercut was prepared with a #34 inverted cone bur in each premolar selected for orthodontic traction, and a stainless-steel button attached with composite.

Extrusive orthodontic forces of 300gm were initated 2 to 14 days after the injury (Fig 3). Intruded teeth not demonstrating mobility were luxated to +2 mobility (mobility scale 0-3) with a small straight elevator before applying the orthodontic force.

Periodic evaluation of the experimental teeth included periapical radiographs, measuring and adjusting the orthodontic force, and assessment of tooth mobility. Radiographic and clinical measurements of tooth movement were also recorded.

Final radiographs and photographs were taken 12 to 13 weeks after the traumatic intrusion. The animals were then killed and the heads perfused with 10% formalin. Tissue blocks were taken to include the canine and first and second premolars. Histologic observations, using Mallory and hematoxylin and eosin stains under light microscopy, included the assessment of root resorption, ankylosis, pulpal status, and periapical pathology.

— Results —

Traumatic intrusion injuries and sequelae are summarized in Tables 1 and 2. Follow-up in two animals was without significant complications. In animal 3, one tooth previously selected for untreated follow-up was avulsed by the intrusive impact.

In spite of efforts to apply uniform intrusive blows, the amount of traumatic intrusion varied. The most intrusion was found in animal 2, ranging from 0.5mm to 5.0mm. It was less severe in animal 1

Table 1

Clinical observations and radiographic measurements

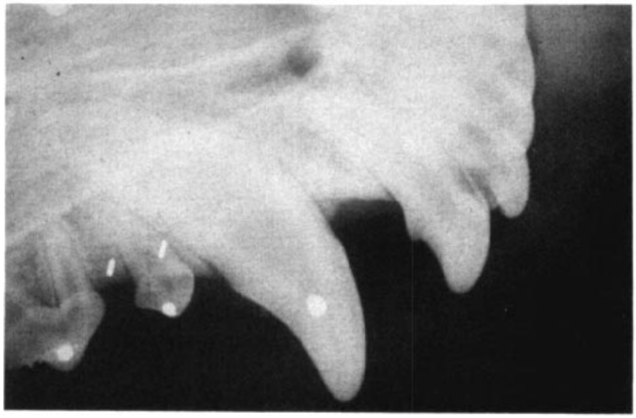
Treatment	Clinical Mobility	Traumatic Intrusion	Orthodontic Extrusion	Luxation Applied
		Dog 1		
Orthodontic	0	2.0	0	NO
	+2	1.0	1.5	NO
	+2	1.0	1.0	NO
Control	0	.5	0	NO
		Dog 2		
Orthodontic	0	5.0	4.0	YES
	+1	.5	.5	YES
	0	1.5	0	NO
	+1	.5	0	NO
		Dog 3		
Orthodontic	+2	.5	.5	NO
	0	.5	2.0	YES
	+1	1.0	0	NO

Table 2

Radiographic and Histologic Observations

Treatment	Ankylosis	Root Resorption	Pulpal Pathology
	Dog 1		
Orthodontic	+	+	V
	−	+	N
	−	+	V
Control	−	−	N
	Dog 2		
Orthodontic	+	+	N
	+	+	C
	+	+	V
	+	+	C
	Dog 3		
Orthodontic	−	+	?
	−	+	N
	−	+	N

C = Calcified pulp
N = Necrotic pulp
V = Vital pulp
? = Undetermined



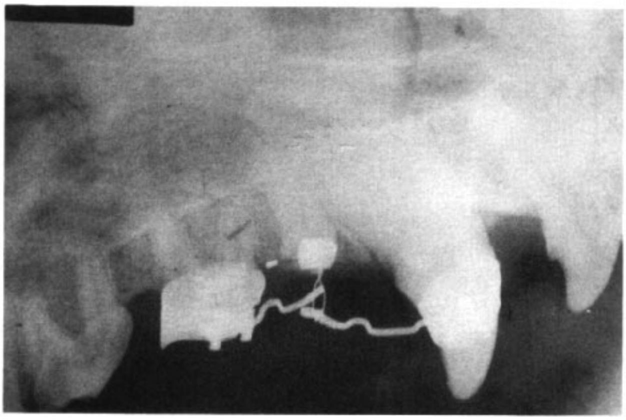


Fig. 4 **Animal 2.**
top **First premolar prior to traumatic intrusion.**
bottom **First premolar root following traumatic intrusion of 5.0mm. Orthodontic appliance is already in place. Note the maxillary sinus.**

(0.5mm to 2.0mm), and in animal 3 the intrusion was only 0.5mm to 1.0mm (Figs. 4 and 5).

Detailed observations are discussed below.

Animal 1

Clinical and radiographic evidence suggest that ankylosis might have occurred with one orthodontic and one untreated tooth that were immobile immediately following the intrusive luxation. Clinical evaluation four weeks after the injury demonstrated a lack of mobility, and radiographic examination did not reveal a discernible continuous periodontal ligament space around either of these teeth.

Subsequent histologic examination, however, revealed ankylosis associated with the orthodontic tooth but not the untreated tooth.

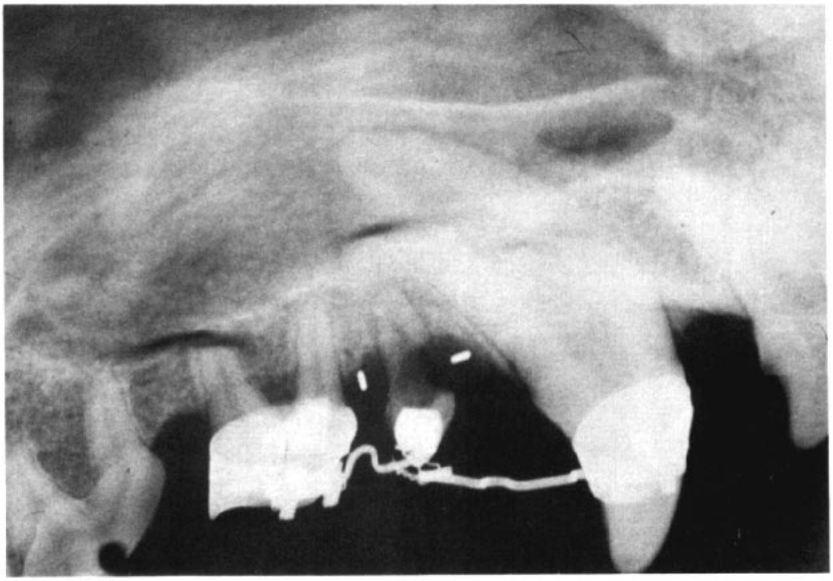


Fig. 5 Same tooth shown in Fig. 4, after 12 weeks of extrusive orthodontic force. Movement of 4.0mm has been attained. Note resorption of tooth and bone, and migration of implants.

The other orthodontic tooth in animal 1 was rendered mobile (+2) by the intrusive injury. Clinical and radiographic evidence suggest that these teeth did not ankylose. Mobility was present four weeks after the intrusive injury, and radiographic examination indicated a continuous periodontal ligament space.

Measurements made from superimposed radiographs reveal no therapeutic extrusion of the orthodontically managed tooth that was rendered immobile by the intrusive blow. This tooth was not luxated after the injury to produce clinical mobility. The untreated tooth also showed no eruption.

However, radiographic evidence of extrusion was apparent in both of the teeth that demonstrated mobility immediately after the traumatic intrusion, one with orthodontic intervention and one without.

Root resorption was apparent radiographically on only one tooth. This tooth was intruded 1.0mm, clinically mobile (+2) after the blow, and extruded 1.5mm orthodontically (0.5mm beyond its original position).

Histologic examination revealed a more detailed picture of the pulpal and periodontal status of each tooth. The teeth subjected to orthodontic extrusive force showed root resorption with and without accompanying ankylosis (Figs. 6 and 7).

Inflammatory resorption occurred randomly on the root surfaces of all three teeth. Replacement resorption was observed in two teeth, with multinucleated cells creating resorption channels along the root surface and within the

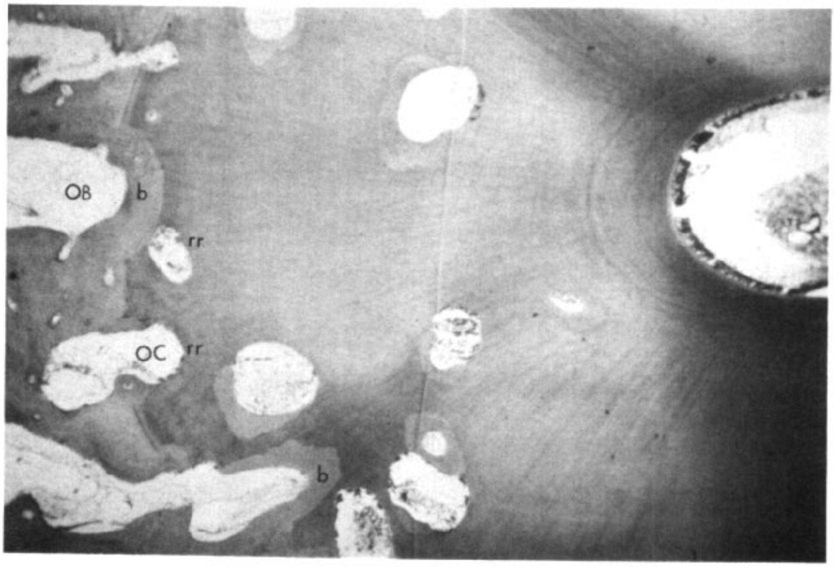


Fig. 6 **Horizontal section from experimental tooth in animal 1. Surface of the tooth shows odontoclasts resorbing dentin (rr) and calcified matrix fused to dentin. As odontoclasts (OC) resorb dentin, osteoblasts (OB) appear to be depositing bone to fill in the resorbed area. Extensions of alveolar bone (b) often filled these voids.**

dentin (Fig. 7). A calcified matrix, deposited by bone-forming cells, filled the resorbed root surface areas.

The untreated tooth which was initially mobile did not develop radiographically visible root resorption or ankylosis, but subsequent histologic examination revealed that all of the teeth, treated and untreated, had experienced some replacement resorption.

Pulpal examination revealed necrosis in one orthodontic and one untreated tooth. Necrotic pulps were devoid of matrix-forming cells and red blood cells. Two orthodontic teeth displayed vital pulps with matrix-forming cells lining the pulpal wall, and with blood vessels, red blood cells, and fibroblasts scattered throughout the pulpal tissue.

Animal 2

Clinical and radiographic examination indicated ankylosis only in the tooth which was intruded the most (5.0 mm). This tooth lacked initial mobility, and radiographic examination failed to demonstrate a definite, continuous periodontal ligament space. Orthodontic forces were ineffective until the tooth was luxated. This tooth showed radiographic evidence of replacement resorption (Fig. 5).

The other orthodontic tooth was only slightly intruded (0.5 mm), and mobile (+1) following the injury. Luxation was not performed at the initiation of orthodontic traction; however, luxation was applied later as the tooth became immobile and failed to move orthodontically.

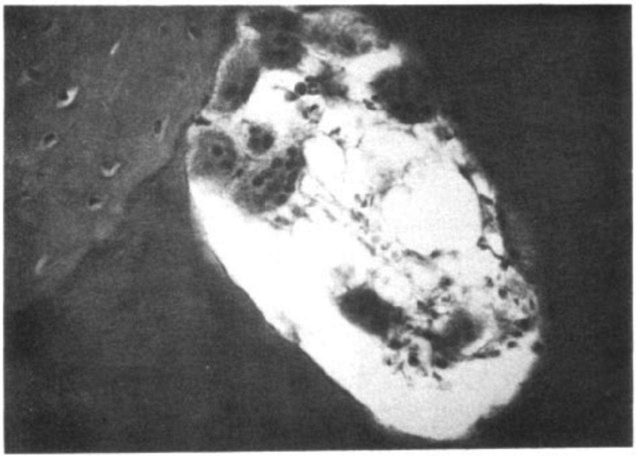


Fig. 7 Resorption channels deep into the dentin contain multinucleated cells.

Hematoxylin and eosin stain

Orthodontic extrusion occurred in both treated teeth, as determined by measurements made from superimposed radiographs. The untreated teeth showed no eruption.

A definitive picture of the pulpal and periodontal status was also provided by the histologic examination. Replacement resorption was evident in both orthodontic and untreated teeth. Dentinal resorption channels and generalized replacement resorption occurred similar to that in animal 1.

Pulpal examination revealed vital pulps in both orthodontic and untreated teeth. The pulp of one untreated tooth showed vital components, but partial pulpal obliteration was evident, with calcified tissue lining the canal. One orthodontic tooth showed signs of pulpal necrosis in the coronal portion, and a deficiency of matrix forming cells and blood cells.

Animal 3

In contrast to animals 1 and 2, ankylosis was not identified from clinical or radiographic evidence in any of the teeth in animal 3. However, radiographic examination did indicate that all the teeth, orthodontic and untreated, had undergone root resorption. The root resorption was inflammatory in nature, similar to that seen in animals 1 and 2. Replacement resorption was not evident in animal 3.

Measurements from superimposed radiographs reveal that orthodontic extrusion occurred in both of the treated teeth. As in animals 1 and 2, the teeth with no orthodontic force showed no eruption. One orthodontic tooth was intruded only slightly (0.5 mm), mobile (+2), and orthodontic forces were applied without further luxation. The other orthodontic tooth was also intruded 0.5mm, but immobile; orthodontic extrusion was successful only after luxation, as observed in animals 1 and 2.

Animal 3 was also similar to animals 1 and 2 in pulpal status. Both orthodontic and untreated teeth displayed necrosis as described earlier. No pulpal calcification was identified in any of these teeth, although the pulpal status of one orthodontic tooth could not be determined.

— Discussion —

Results of this study suggest that the effectiveness of orthodontic extrusion for repositioning traumatically intruded teeth appears to be dependent on the mobility of the tooth immediately following the injury. Traumatically intruded teeth which were clinically immobile did not respond to orthodontic traction. These were usually the severely intruded teeth, which were probably so tightly wedged into the surrounding alveolar bone that orthodontic forces could not overcome the mechanical resistance.

Such tight approximation of bone to tooth structure undoubtedly encourages the development of ankylosis. It is quite possible that the ankylosis was already under way well before the initiation of orthodontic traction.

In contrast to immobile teeth, those that were clinically mobile did respond to orthodontic extrusive forces. These teeth were presumably rendered mobile by the intrusive blow, although prior mobility was not evaluated.

Biederman (1962) states that the luxation technique for freeing an ankylosed tooth is almost identical with the first movements of tooth extraction, grasping it firmly with forceps and rocking it buccolingually, or applying mesiodistal luxation by means of an elevator. The objective is to break the bony bridge of ankylosis without injury to the nutrient vessels at the apex.

One important addition to Biederman's technique used in this study is the immediate application of orthodontic force. He recommends observing the tooth for eruption after luxation, and if no change is apparent after six months, to repeat the luxation. It is our experience that a luxated tooth will ankylose again if orthodontic forces are not applied and, in may cases, it may even ankylose with orthodontic forces.

Biederman suggested extracting the tooth if the second luxation is unsuccessful. We have seen ankylosed teeth extrude with orthodontic traction, even after a second luxation.

Numerous cases of spontaneous re-eruption following traumatic injuries have been reported (Ellis 1948, Andreasen, 1970), although this seems to be most common with deciduous teeth. Most reported cases of permanent tooth re-eruption have been those with incomplete root formation (Bruszt 1958, Andreasen 1970, 1976). Traumatically intruded permanent teeth with closed apices do not re-erupt as often.

Skieller (1960) suggests that teeth should be immediately repositioned following an injury. Andreasen (1970) observes that this appears to increase complications, stating that orthodontic extrusion may reduce the incidence of root resorption and ankylosis.

We concur that orthodontic traction should be used to reposition traumatically intruded permanent teeth. However, ankylosis and replacement resorption are still possible sequelae.

In this study, the orthodontic extrusive forces were initiated from 2 days to 2 weeks after the injury, so the time of onset of any ankylosis is unknown. Andreasen (1970) reports the initiation of ankylosis 2 weeks following avulsion and reimplantation of a permanent tooth.

An important factor in the onset of ankylosis and root resorption may be the stage of root development. Skieller (1960) found that teeth with complete root formation undergo resorption more frequently than those with incomplete root formation. Investigation of these factors may give clues to both root resorption and ankylosis.

Pulpal responses in this study were similar to those previously reported; both vital and necrotic pulps were observed. Some of the pulps that remained vital after traumatic intrusion developed pulpal calcification. These pulps, however, were undergoing degenerative changes that may have eventually led to necrosis. There were no differences in pulpal responses between untreated and orthodontic teeth.

— Conclusions —

1. Ankylosis, replacement resorption, pulpal necrosis and pulpal calcification are common sequelae of traumatic intrusion.
2. With immobile traumatically intruded teeth, orthodontic extrusion can be facilitated by luxation.
3. Even with luxation, ankylosis and replacement resorption are still possible sequelae.
4. With less severe injuries, orthodontic extrusion can facilitate repositioning of a displaced tooth without further luxation.

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