

**A LONGITUDINAL OUTCOME  
STUDY OF LATERAL LUXATION INJURIES TO THE  
PERMANENT MAXILLARY INCISORS  
OF CHILDREN AND ADOLESCENTS**

**by**

**Mandana Nikoui**

**A thesis submitted in the conformity with the requirements  
for the degree of Master of Science Faculty of Dentistry  
Department of Paediatric Dentistry  
University of Toronto**

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**Abstract**

A longitudinal outcome study was undertaken to assess variables that influence tooth survival, pulpal and periodontal outcomes of laterally luxated permanent maxillary incisors of children and adolescents. A sample of 42 patients (26 male and 16 female) with 58 laterally luxated incisors was identified. The mean age at the time of injury was 11.3 years (range: 6.3-17.8 years). Pulpal necrosis (PN) and pulp canal obliteration (PCO) were common healing complications. Cox and logistic regression analysis demonstrated that root development and extent of lateral luxation were not significantly related to PN and PCO ( $p > 0.05$ ). This study represents the first to report incisor survival of children and adolescents following lateral luxations. In the entire sample ( $n = 58$ ) no incisors were observed to fail. Therefore, parents can be informed that the survival prospects for laterally luxated incisors in children and adolescents are excellent.

**Key words:** trauma, permanent incisor, lateral luxation, survival analysis, pulpal necrosis, pulp canal obliteration, prognosis

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## **Introduction**

A lateral luxation injury involves displacement of tooth root(s) through bone and affects both pulpal neurovasculature and periodontium (Andreasen and Andreasen, 1994). To date, there are no evidence based outcome studies that support current treatment protocols for the management of a lateral luxation injury. The amount of tissue damage can only be estimated since animal studies that might provide insights into this particular injury have not been undertaken. The few clinical studies that describe lateral luxations have all combined this injury with other types of luxation injuries. In addition, their samples include mixed populations of adults and children and lack standardized treatment protocols.

A longitudinal outcome study with a standardized treatment protocol would provide prognostic information for both clinicians and parents, serve as the foundation of an evidence-based clinical protocol and set a standard for future clinical studies.

## Literature Review

### Classifications and Definition

Tooth luxation describes a broad spectrum of displacements. Five different types of luxation injuries are recognized: concussion, subluxation, extrusion, lateral luxation and intrusion (Andreasen and Andreasen, 1994). Prior to 1981, there was inconsistency in both the description and classification of these injuries. This lack of consistency led to misdiagnosis and inclusion of intrusions and extrusions into lateral luxation samples (Andreasen and Andreasen, 1985). As a consequence, lateral luxations may have been over-reported in the early literature. However, due to increased usage of the classification developed by Andreasen and Andreasen (1981, 1994) and subsequently revised by the World Health Organization (WHO) codes for Dentistry and Stomatology (1993, 1995) (Appendix I), luxations may now be accurately described. Andreasen and Andreasen (1994) defined lateral luxation as the displacement of a tooth in a direction other than axially that occurs in conjunction with comminution or fracture of the alveolar socket. This review concentrates exclusively on *lateral luxation injuries* to permanent maxillary incisors.

### Epidemiology

The full spectrum of luxation injuries to maxillary permanent incisors of children are only exceeded in prevalence by crown fractures (Andreasen, 1970a; Andreasen and Ravn, 1972; Oikarinen and Kassila, 1987). Luxation injuries occur most commonly between the ages of 8 and 12 years (Andreasen and Ravn, 1972; Ravn, 1974; Robertson, 1997). Males have more frequent injuries to permanent maxillary incisors than females (Andreasen and Ravn, 1972;

Josefsson and Karlander, 1994; Luz and DiMaz, 1994). The majority of luxation injuries affect the maxillary permanent central incisors followed by the maxillary permanent lateral incisors (Andreasen, 1970b; Andreasen and Ravn, 1972; Ravn, 1974; Josefsson and Karlander, 1994).

There are no epidemiological studies that have focused on the causes of lateral luxation injuries. However, relative to all categories of luxation injuries, falls during play followed by contact team sports (i.e. ice-hockey), traffic accidents and acts of violence are the main cause of luxation injuries (Oikarinen and Kassila, 1987). Five studies described the proportion of lateral luxation injuries compared with all dental injuries (Table 1).

**Table 1.** Summary of studies of the proportion of lateral luxation injuries to the dentition in children and adults

<b>Authors</b>	<b>Year</b>	<b>Ages(yrs)</b>	<b>Sample Size</b>	<b>Proportion%</b>
Sane & Ylipaavalniemi	1988	6-50	220	7.9
Crona-Larsson & Noren	1989	6-19	196	3.0
Schatz & Joho	1994	1-16	228	7.9
Caliskan & Turkun	1995	6-35	470	3.8
Sae-Lim <i>et al.</i>	1995	1-18	766	4.1

### ***Summary***

Lateral luxation injuries occur most commonly in children and adolescents. The peak age of occurrence is between 8-12 years when root formation is incomplete and the alveolus is elastic. This injury occurs more frequently in males than in females and most commonly involves the maxillary permanent central incisors. Although few epidemiological studies have considered the lateral luxation injury as a separate entity, its proportion appears to range between 3 to 8 per cent of all dentoalveolar injuries.

## Diagnosis

Diagnosis of lateral luxation injuries is based upon both clinical and radiographic observations (Andreasen and Andreasen, 1985). Clinically, inspection and palpation are essential. The crown of the laterally luxated incisor is usually forced either lingually or labially. However due to the direction of the force, the crown tends most commonly to be displaced *lingually* and the root displaced labially. As a result, the apical portion of the root may fracture the labial alveolar socket wall (Andreasen and Andreasen, 1990). In its displaced position, the root is usually locked *in situ*. Percussion of the affected tooth may produce pain and will elicit a resonant sound. Luxated teeth rarely respond to neural conduction testing at the time of injury (Andreasen, 1986). If the crown is displaced *labially*, the associated labial root movement fractures the labial alveolar socket wall and tears the junctional epithelium of the lingual aspect of the incisor.

A radiographic examination is used to determine the apical location of the luxated tooth, the stage of root formation and the presence of fracture of the alveolar socket. Radiographs usually reveal an increase in the width of the periodontal ligament (PL) space (Andreasen and Andreasen, 1985; Newton, 1992). Andreasen and Andreasen (1985) noted that in addition to a periapical radiograph, an occlusal radiograph is required to disclose the displacement. However, they did not mention the use of a lateral radiograph that may provide additional information about the orientation and extent of this injury. Furthermore, it has been reported that the use of film holders provide accurate and reproducible radiographs of lateral luxation injuries (Andreasen and Andreasen, 1985). Lateral luxation is a difficult diagnosis to establish without

adequate radiographs and as a result, the luxation could be incorrectly classified as an intrusion or extrusion.

### **Pathogenesis**

A lateral luxation affects the blood supply to pulpal tissues and periodontium. Localized ischemia may be produced by rupture of the pulpal neurovascular supply and compression of the root surface PL cervically and laceration of the PL apically (Andreasen, 1985; Andreasen and Andreasen, 1990). Two laboratory studies attempted to assess morphologic and histologic changes in the pulp and PL following experimental luxation of immature rat teeth. (Miyashin *et al.*, 1990, 1991). Unfortunately, in both studies the investigators ignored human epidemiological findings and chose experimental subluxation of molars rather than lateral luxation of incisors. Consequently, there are no animal studies specific to lateral luxation injuries.

### **Summary**

In the early literature, lateral luxations were probably over-reported due to the inclusion of intrusions and extrusions. Both clinical and radiographic examinations are required for the diagnosis of lateral luxation injuries. Since no animal studies have been attempted that reproduce equivalent lateral luxation injuries, our understanding of the development of pathosis of pulpal tissues and periodontium in humans is limited.

### **Treatment**

There are no outcome-based protocols for the treatment of laterally luxated incisors. However, the guidelines of Andreasen and Andreasen (1994) are the basis of management of

luxation injuries for the majority of clinicians. These guidelines have not been tested in a longitudinal outcome study and furthermore, there is a lack of information on specific outcomes, economic costs, risks and benefits. Nevertheless, lateral luxation injuries continue to be managed by a combination of reduction, splinting, clinical and radiographic observation and, when required, endodontic treatment or extraction. This management strategy is based upon methods used for other luxation injuries and extrapolation from animal replantation studies.

### ***Reduction***

It is generally accepted that a laterally luxated tooth should be reduced to its original position as soon as practically possible (Andreasen, 1981a; Andreasen and Andreasen, 1994; Dumsha, 1995). If treatment is delayed, the laterally luxated incisor may be actively repositioned (i.e. orthodontically) or in minor injuries, become repositioned under the influence of soft tissue pressure (Mackie and Warren, 1988; Roberts and Longhurst, 1996; Curzon, 1999). The choice of method will be influenced by level of co-operation, extent of displacement, time since injury and each clinician's choice.

When the crown is driven lingually, the incisor may require forceful manual repositioning due to displacement of the root tip through the labial alveolar socket. Andreasen and Andreasen (1994) suggest that the root apex should be disengaged by digital pressure over the root tip in an incisal direction. Once the incisor is repositioned, it is further suggested that the fractured labial alveolar socket wall be repositioned to its normal state.

### ***Splinting***

Semi-rigid splints are commonly used to stabilize laterally luxated incisors (Oikarinen, 1987; 1990) and are recommended for a minimum of 3-4 weeks (Andreasen *et al.*, 1999). This splinting duration is suggested to allow bone repair to take place and was based on clinical and experimental findings derived from fractures of the shaft of long bones and jaw fractures (Andreasen, 1981a). Rishman and Laskin (1964) reported that the mobility of jaw fragments may affect the rate of bony callus formation and furthermore, when fragments are mobile, more time elapses before bony bridging is observed.

There are no animal studies to demonstrate the effects of splinting on PL repair or pulpal survival following lateral luxation injuries. Nonetheless, in a clinical study of 42 laterally luxated teeth, Oikarinen *et al.* (1987a) considered the duration of immobilization (mean: 52 days) with a rigid splint and a follow-up time that varied from 6 to 38 months (mean: 22 months). The authors reported that the duration of splinting significantly ( $p < 0.05$ ) increased the marginal alveolar bone loss in 31 per cent of laterally luxated teeth. The authors did not make a distinction between different types of splint and/or duration of splinting and used only a rigid splint (metal bar) for a mean duration of 52 days. Experimental and clinical studies of avulsed and autotransplanted teeth have shown, however, that rigid splinting may affect periodontal healing by increasing the risk of ankylosis (Kristerson and Andreasen, 1983).

### ***Antibiotics***

The value of systemic administration of antibiotics following lateral luxation injuries has not been tested in humans. Nevertheless, antibiotics are prescribed empirically in order to reduce

or control bacterial contamination following rupture of the junctional epithelium and to decrease the likelihood of inflammatory root resorption (IRR). Croll (1991) suggested the use of antibiotics following luxation injuries because the PL is inoculated with salivary microorganisms. Treatment recommendations do not define an antibiotic of choice, duration or dosage.

### ***Endodontic Treatment***

There are no specific guidelines for the timing of endodontic treatment of a laterally luxated tooth. Despite a range of opinions and lack of clinical trials, endodontic treatment is based on the diagnosis of pulpal necrosis (PN).

In teeth with immature apices where there may be increased potential for pulpal revascularization, Andreasen and Andreasen (1994) recommend delaying pulpectomy. It is presumed that an immature apical foramen favors the possibility of anastomoses between pulp vessels as well as the ingrowth of new vessels. However, studies on replanted and transplanted teeth with mature apices have shown that the vascular supply to the dental pulp may be compromised and as a result, ischemia and necrosis may develop (Öhman, 1965, Kristerson and Andresen, 1984). As a result, radiographic and clinical evidence of PN or associated periodontal pathosis (i.e. inflammatory root resorption) are indications for pulpectomy (Skiller, 1960; Andreasen, 1970a, Andreasen and Vestergaard Pedersen, 1985). In teeth with mature apices, the likelihood of revascularization is decreased and therefore it is recommended that root canal therapy be initiated as soon as PN is diagnosed (Andreasen *et al.*, 1999).

When inflammatory root resorption (IRR) is diagnosed, non-setting calcium hydroxide paste may be used as an interim medication following pulpectomy (Cvek *et al.*, 1974). The effectiveness and duration of treatment with calcium hydroxide is unclear and is extrapolated from recommendations for avulsion injuries. In replanted teeth, the duration of calcium hydroxide treatment recommended by the American Association of Endodontists (AAE) (1983, 1994) has been reduced from a period of 6 to 12 months to an interval of 7 to 14 days. As soon as signs of periodontal pathosis have ceased, root canal therapy followed by obturation with gutta percha and sealer should be completed (Andreasen and Andreasen, 1994).

### ***Summary***

Guidelines of Andreasen and Andreasen (1994) are widely used for management of lateral luxation injuries. Since human experimentation is not possible and animal studies have not been undertaken, current treatment recommendations for lateral luxation injuries are empirically-based and derived from management of other luxation injuries. The guidelines suggest immediate repositioning followed by splinting. As soon as clinical and radiographic diagnosis of PN or periodontal pathosis is made, pulpectomy is indicated. The usage of non-setting calcium hydroxide to control IRR is based on replantation studies (Andreasen, 1975a; Andreasen and Andreasen, 1994; AAE guidelines, 1994) and is followed by conventional root canal therapy.

### **Prognosis**

#### ***Healing***

The extent of damage to the pulp and periodontium following luxation injuries is unknown and current knowledge is derived from animal replantation studies (Nasjleti *et al.*,

1978; Andreasen, 1980; Skoglund and Tronstad, 1981). Injury to the pulp and periodontium is followed by wound healing. As a result, the outcome of healing is either complete restoration of tissue architecture, structure and function (*regeneration*) or restoration with scar tissue and distortion of the normal architecture (*repair*) (Ten Cate, 1994).

### ***Healing Complications-Pulp***

Pulp survival following luxation injuries remains unpredictable and current knowledge has been derived from studies of replantation and other luxation injuries (Lee, 1996; Humphrey, 1999). Following luxation injuries the neurovascular supply to the pulp may be compromised and lead to healing complications (Andreasen, 1981a).

Maintenance of pulp vitality is dependent upon the healing capacity of pulp tissues. Pulpal healing capacity is related to the *stage of root development* (Arwill *et al.*, 1967; Magnusson and Holm, 1969; Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985; Oikarinen *et al.*, 1987a). Andreasen *et al.* (1986) studied the relationship between diameter of the apical foramen and pulp survival. They postulated that an increase in apical diameter would result in a larger surface area and increase the likelihood of revascularization. Moreover, they suggested that teeth with a constricted apical foramen would have a limited opening for ingrowth of new vessels. However, findings in this study were based on radiographic techniques that may limit the value of the measurements made. An often-cited laboratory study reported that, in dogs, repair was observed through ingrowth of well-vascularized connective tissue in immediately replanted incisors and single rooted premolars with immature apices. (Skoglund and Tronstad, 1981)

Studies have reported that pulp survival is also related to *the extent of injury* (i.e. severity of trauma) (Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985). Following luxation injuries, the neurovascular supply may be severed and as a result, lead to pulpal ischemia (Skieller, 1960; Andreasen, 1970; Stalhane and Hedegard, 1975). Andreasen (1989) speculated that luxated teeth with minimal displacement may favor either end-to-end anastomosis of ruptured vessels and nerves in the apical region or gradual revascularization of the damaged pulp. In addition, experimental and autotransplantation studies in dogs (Skoglund and Tronstad, 1981) and humans (Öhman, 1965) reported pulpal repair following replantation of immature teeth. In this process, some authors stated that pulpal repair in the presence of bacterial invasion was unlikely (MacDonald *et al.*, 1957; Reeves and Stanley, 1966). Cvek *et al.* (1990) reported that pulpal revascularization represented a barrier against bacterial invasion due to increased immunological defense and/or presence of pulpal hydrostatic pressure. Furthermore, the Andreasen (1981b) study in monkeys showed that revascularization was inhibited when ischemic pulpal tissue was infected.

### ***Transient pulpal healing complications***

#### ***Transient Coronal Discolouration***

Transient coronal discoloration may be observed following lateral luxation injuries (Andreasen, 1989). This can range from pink to bluish or gray discolouration (Arwill *et al.*, 1967; Auslander, 1967). Stanley *et al.* (1978) postulated that rupture of the apical vessels due to trauma leads to ischemia with breakdown of capillaries and subsequent escape of erythrocytes into the dentinal tubules. This is more likely to occur in the case of moderate injury (i.e. lateral luxation) compared with a severe injury (i.e. extrusion or intrusion) where apical vessels are

instantly ruptured with no extravasation of blood into the pulpal tissue (Andreasen and Andreasen, 1994). Furthermore, Marin *et al.* (1997), in an *in vitro* experimental study that used single-rooted premolar teeth from orthodontic patients (10 to 18 years), reported that the cause of discolouration in non-infected traumatized teeth was related to the accumulation of haemoglobin in dentinal tubules.

In a clinical study, Andreasen (1986) observed a transient gray discolouration possibly related to pulp vascular damage and subsequent hemorrhage in the healing period following luxation injuries. She reported that in the absence of infection, this transient change could lead to pulpal healing and follow-up observation was recommended. However, Andreasen (1989) reported that in the presence of bacterial infection of the pulp, permanent coronal discolouration related to autolysis and extravasation of cellular breakdown products into dentinal tubules may be observed.

#### *Transient Apical Breakdown*

Transient apical breakdown (TAB) is defined by Andreasen (1986) as a periapical rarefaction that can be diagnosed radiographically within the first year following a luxation injury. Andreasen (1986) postulated that it may be related to the type of luxation injury and stage of root development. This author found that TAB develops in mature teeth with fully-formed roots and was more likely to occur following moderate luxation injuries such as lateral luxations compared with intrusions. Furthermore, in her sample of 27 maxillary permanent incisors, Andreasen (1986) found that TAB developed in 12 per cent of laterally luxated teeth compared with none in intrusions. Based on Andreasen's study (1986), the

radiographic manifestation of TAB in lateral luxation injuries is either an increase in PL space or an increase in the diameter of the apex of a mature tooth. Andreasen, (1989) reported that TAB might reflect a repair process in teeth with complete root formation that allows for ingrowth of new vessels into the constricted apical foramen.

According to Andreasen and Andreasen (1994), like transient coronal discoloration, TAB might reflect an intermediate phase of wound healing that returns to normal when repair is complete. Thus, they recommend delaying endodontic treatment for up to one year in the absence of symptoms.

### ***Permanent pulpal healing complications***

The sequence of events that lead to pulpal healing complications after lateral luxation injuries remains unclear (Andreasen and Andreasen, 1994). Only four clinical studies have described the outcome of lateral luxation injury (Table 2) and they represented only 3 separate samples (n = 170) of laterally luxated teeth. All four papers included lateral luxation injury with the other types of luxation injuries. Furthermore, all four case series had heterogeneous sample populations that combined a growing population and a stable adult population, and differed with respect to both design and statistical analysis. Nevertheless, these studies represent the only clinical data available.

**Table 2.** Clinical studies of lateral luxation injuries to the permanent dentition

<b>Authors</b>	<b>Number of Lateral luxations</b>	<b>Age Range</b>
Andreasen & Vestergaard Pedersen(1985)	122 teeth	Not reported
Andreasen <i>et al.</i> , (1987a)	122 teeth	Not reported
Oikarinen <i>et al.</i> , (1987)	42 teeth	6-64 years
Crona-Larsson <i>et al.</i> ,(1991)	6 teeth	6-19 years

The retrospective studies of Andreasen and Vestergaard Pedersen, (1985) and Andreasen *et al.* (1987a) used the same sample population of 122 laterally luxated teeth and neither provided an age range nor specified the teeth included in the sample. Furthermore, the authors questioned the reliability of their own data since information collected at the time of injury and follow-up was documented by unknown raters. Multivariate survival analyses were performed for all types of luxation injury as a group rather than for each individual type of injury. They reported that complete root formation had a significant effect on the development of PN ( $p < 0.05$ ) following lateral luxation injury. Furthermore, they stated that both stage of root development ( $p < 0.007$ ) and type of fixation ( $p < 0.05$ ) were significantly related to the development of PCO.

The sample of the Oikarinen *et al.* (1987a) study included mostly adults although the ages ranged from 6 to 64 years and did not give any information about the teeth included in the sample. They reported that among 42 laterally luxated teeth, 35 per cent developed external root resorption while 33 per cent developed PCO and 31 per cent developed marginal bone loss. The authors noted that their findings may have been affected by the heterogeneity of their sample including a mixed sample of children and adults. The Crona-Larsson *et al.* (1991) sample of 171 teeth included only 6 laterally luxated teeth and provided no additional information.

### *Pulpal Necrosis*

Pulpal necrosis (PN) follows interruption of the vascular supply and is the most frequent complication of all types of luxation injuries (Skieller, 1960; Stalhane and Hedegard, 1975; Andreasen and Vestergaard Pedersen, 1985). Diagnosis of PN should be based upon lack of neural conduction in response to thermal and electrical testing, gray discolouration and periapical radiolucency (Jacobsen, 1980). Andreasen (1988) reported that tenderness to percussion at the time of diagnosis could be related to an infected necrotic pulp. More than one clinical sign is required for definitive diagnosis of PN and radiographic appearance is considered the most important criterion (Magnusson and Holm, 1969; Jacobsen, 1980). Radiographic changes are not apparent during the first 3 weeks but can be observed within 3 months of luxation injuries (Jacobsen, 1981; Andreasen and Vestergaard Pedersen, 1985).

Studies have reported that PN is dependent upon the extent of injury and the healing capacity of the pulp that is in turn related to the stage of root development (Arwill *et al.*, 1967; Magnusson and Holm, 1969; Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985;

Oikarinen *et al.*, 1987). Severe luxation may cause more damage to the pulpal blood supply and PN may develop more often in teeth with constricted apical foramen. The clinical study by Andreasen and Vestergaard Pedersen (1985) is the only one to report the relationship between PN and root development in laterally luxated teeth. They reported that 77 per cent of teeth with complete root formation demonstrated PN compared with 8 per cent of teeth with immature roots. Furthermore, they reported that one year following lateral luxation injury, pulp survival ranged from 90 per cent in teeth with immature apices to 20 per cent in teeth with mature apices. They did not provide information on the source of these observations. They simply stated that this data was derived from clinical investigations.

In a multivariate analysis of luxation injuries ( $n = 637$  teeth), Andreasen and Vestergaard Pedersen, (1985) reported that among 122 laterally luxated teeth followed for 10 years, 58 per cent developed PN. Similarly, Oikarinen *et al.* (1987) reported that in a (mostly adult) sample of 42 laterally luxated teeth followed for 38 months, 50 per cent developed PN. However, in the Oikarinen study, diagnosis of PN was based solely on sensibility tests (thermal and electrical) and consequently, assessment of pulp status did not meet clinical standards that include radiographic examination.

### *Pulp Canal Obliteration*

Pulp canal obliteration (PCO) is a term commonly used to describe the radiographic observation of hard tissue formation within the root canal (Jacobsen and Kerekes, 1977). Pulp canal obliteration is characterized by a reduction in the size of the pulp chamber and narrowing of the root canal (Andreasen, 1970a). Some investigators have reported that obliteration of the

pulpal space begins coronally and progresses apically (Herbert, 1953; Azaz *et al.*, 1980).

Andreasen *et al.* (1987a) observed that PCO may appear between three months to one year after lateral luxation injuries but they provided no indication of how the diagnosis or analysis of time of onset was determined. Clinical studies have reported that constricted pulpal tissue is capable of neural conduction in response to thermal and electrical tests but the diagnostic value of these indirect tests of vascular integrity remain inconclusive (Andreasen, 1970a; Fulling and Andreasen, 1976a, 1976b; Jacobsen and Kerekes, 1977).

Some studies refer to this hard tissue formation as secondary dentin (Herbert, 1953; Fischer, 1974; Andreasen, 1989) while others refer to it as irregular secondary dentin, reaction dentin or tertiary dentin (Kuttler, 1959; Sayegh *et al.*, 1968; Bhaskar *et al.*, 1969). Avery *et al.* (1974) have proposed the term "response dentin" to describe a generalized dentin deposition rather than the terms reparative dentin or tertiary dentin that are used to describe localized dentin deposition in response to a noxious stimulus such as caries. Furthermore, hard tissue deposition within the pulp chamber and canals is a progressive physiological process that continues throughout life (Sayegh *et al.*, 1968, Philippas and Applebaum, 1967). This hard tissue deposition can, however, be accelerated in case of dental trauma (Andreasen, 1970a; Jacobsen and Kerekes, 1977), autotransplantation or orthodontic therapy (Andreasen *et al.*, 1987a). While the exact cause of PCO is still unclear, different hypotheses have been suggested. Experimental studies in rabbits have attempted to explain that accelerated hard tissue deposition may be caused by a sympathetic nervous system response to trauma that will directly affect the odontoblasts or indirectly produce blood vascular changes (Avery *et al.*, 1971; Biesterfeld *et al.*, 1979; Avery, 1981). These studies have reported that the diminished blood flow to the pulp

observed during sympathetic nerve stimulation could result in “pulpal respiratory depression” that in turn could stimulate PCO. In contrast to this *neural model* (sympathetic/parasympathetic) control of blood flow to odontoblasts, Tønder (1980) using a dog model, hypothesized that hard tissue formation may be controlled by blood-borne substances (*vascular model*) and thus, could be instituted if pulpal revascularization was achieved. In this regard, Andreasen *et al.* (1988) observed that during pulpal repair, pulp tissue was invaded by macrophages, new vessels and pulpal progenitor cells. However, the source of these repopulating progenitor cells was not known. Andreasen (1988) stated that there may be progenitor cells within the pulp that are capable of surviving periods of hypoxia following trauma until revascularization has occurred. Furthermore, the Skoglund *et al.* (1981) experimental study in dogs observed the presence of enzyme activity throughout pulpal repair in replanted and autotransplanted teeth. Extrapolating from these experimental models, the accelerated hard tissue formation observed after luxation injury may reflect either *neural* or *vascular* repair of the dental pulp (Andreasen, 1989).

Following luxation injuries, dentin formation varies histologically depending upon the origin and differentiation of cells involved in its production (Robertson, 1997). Fischer (1974) reported that histological studies of hard tissue that obliterated the pulp chamber following trauma demonstrated vital soft tissue inclusions. However, the author did not give any further detail about the nature of this tissue but stated that fibroblasts were the main pulpal cells responsible for hard tissue formation. Johnson and Bevelander (1955) reported, based on a histochemical study, that “pulpal calcifications” required a complex precursor that consisted of reticular tissue and a protein matrix that was also found in bone, dentin and cementum.

Andreasen and Andreasen, (1994) stated that although the source of repopulating progenitor cells is not known, osteo-dentin, bone or cementum-like tissue was formed in response to pulpal injury, yet the reasons for this variety of tissue responses is not known.

Sayegh *et al.* (1968) reported that hard tissue formation within the pulp chamber and root canals could be either pathological or physiological. Burstone (1953) reported in the case of physiological calcification, cells such as odontoblasts were associated with a protein matrix to form calcospherites that subsequently fused to produce a homogenous calcified structure. In the case of pathological calcification, the protein matrix was covered by necrotic cells of the dental pulp. Furthermore, Fish (1938) distinguished between “calcified repair tissue” and “calcified scar tissue” and reported that both consisted of calcified collagenous matrix. The former resembled bone or cementum while the latter was acellular or contained debris of disintegrated odontoblasts.

Andreasen *et al.* (1987a) reported that PN and PCO appeared to be complimentary with regard to the stage of root development. Furthermore, they stated that there was a greater frequency of PCO after lateral luxation of teeth with immature apices (70 per cent) compared with teeth with mature apices (11 per cent). Presumably, this is due to the requirement of vital pulp tissue for development of PCO.

In contrast to PN, where no treatment-related variables could be established, studies by Andreasen *et al.* (1987a) and Oikarinen *et al.* (1987) reported that PCO was related to the type of splint used. Andreasen *et al.* (1987a) reported that a rigid splint of orthodontic

bands/brackets bonded with cold cure acrylic significantly increased ( $p<0.05$ ) the frequency of PCO compared with a semi-rigid acid-etch/resin splint. They postulated that this might be due to the additional trauma caused by insertion of an orthodontic band on a traumatized tooth.

A late complication (1-16 per cent) of trauma is the development of PN secondary to PCO (Halcomb and Gregory, 1967; Andreasen *et al.*, 1987a; Robertson *et al.*, 1996). Different hypotheses have been postulated regarding the pathogenesis of PN following PCO. Pulpal necrosis might be the result of progressive hard tissue deposition that in turn reduced the vascular supply and produced pulpal ischemia (Andreasen, 1970a; Andreasen *et al.*, 1987a). Some authors have reported that PCO may be regarded as a potential site of bacterial invasion due to the presence of vital pulp tissue and in that way may lead to PN (Patterson and Mitchell, 1965; Fischer, 1974). In addition, it has been reported that the development of PN subsequent to PCO may increase with subsequent injuries, orthodontic or restorative procedures (Robertson *et al.*, 1996; Jacobsen and Kerekes, 1977). Earlier studies have recommended prophylactic endodontic treatment (Patterson and Mitchell, 1965, Fischer, 1974) while more recent studies do not support such measures due to the low frequency of this complication (Lundberg and Cvek, 1980; Andreasen *et al.*, 1987a, Curzon, 1999).

### ***Summary***

Following lateral luxation injuries, continued pulpal vitality may be illustrated by transient phases of discolouration and/or apical breakdown. Pulp tissue can become necrotic or become progressively calcified (PCO) but remain vital. Less frequently, teeth with PCO may

develop PN and require endodontic treatment. Pulp canal obliteration is more frequently observed in immature teeth while PN is more commonly observed in teeth with mature apices.

### ***Healing Complications-Periodontium***

#### ***Root Resorption***

The most common periodontal healing complication following a lateral luxation injury is external root resorption (external RR) (Andreasen and Vestergaard Pedersen, 1985). External root resorption is a pathological process that is associated with the damage to both the pulp and periodontium (Andreasen and Andreasen, 1994). Andreasen and Hjørtting-Hansen (1966) developed a radiographic classification for external root resorption according to their histological and radiographic appearance. Based on their avulsion study, the radiographic appearance of external RR has been classified as inflammatory root resorption (IRR) or replacement root resorption (RRR).

Andreasen and Hjørtting-Hansen (1966) reported that IRR is initiated by damage to the PL followed by rapid resorption of the root surface. As long as the cementum remains intact, bacteria and bacterial metabolites from a necrotic dental pulp remain largely confined to the root canal. However, if the cementum is resorbed, fractured or disrupted, the underlying dentin will be exposed to bacteria and toxic products from the infected pulp that pass towards the root surface. This will lead to inflammatory responses within the PL and cause IRR (Andreasen, 1981b). Andersson (1988) observed that IRR was significantly higher in young populations (range: 8-17 yrs.) compared with an older group (range: 17-39 yrs.). He explained that these findings might be related to the presence of wide dentinal tubuli and a higher

permeability of root dentin in immature teeth. Inflammatory root resorption can be treated by extirpation of the dental pulp and treatment with calcium hydroxide paste followed by conventional root canal therapy (Andreasen and Andreasen, 1994).

Dentoalveolar ankylosis is a term used to describe fusion of alveolar bone with a tooth root (Tronstad, 1988). Ankylosis can be observed histologically in animal models 2 weeks following replantation (Andreasen, 1980). Andreasen and Kristerson (1981) reported that ankylosis may be either transient or permanent depending upon the amount of injured cementum. Andreasen and Kristerson (1981) stated that transient areas of ankylosis could be related to minor damage to the root surface. In contrast, when the damaged area is large, the PL will not be able to proliferate over the entire root surfaces, permanent ankylosis may develop and the root will be progressively replaced by bone (i.e. RRR). Andersson (1988) reported that a necrotic PL may stimulate superficial resorption of the root surface and furthermore, cellular removal of the necrotic PL will lead to ankylosis between the cementum and bone. Replacement root resorption may ultimately lead to destruction of the root or fracture of the crown.

If ankylosis develops before growth of the alveolar process has been completed, progressive infra-occlusion of an affected incisor can occur (Andreasen and Hjørting-Hansen, 1966). In a study of replanted human teeth with necrotic PL, Andersson (1988) observed that the rate of RRR was higher in the group of 8-16 years group compared with an older age group (17-39 yrs.). Furthermore, the author reported that a replanted tooth with a necrotic PL would be resorbed within 3 to 7 years in the 8-16 years group whereas in older

patients they were more likely to remain functional for a longer period. This was postulated to be due to a higher bone turnover rate in children and adolescents compared with adults.

Recently, Andreasen *et al.* (1999) reported a relationship between the development of IRR and RRR and the stage of root formation following lateral luxation injuries. They used a sample of 122 laterally luxated teeth reported in earlier clinical studies (Andreasen and Vestergaard Pedersen, 1985; Andreasen *et al.*, 1987a) to show that the development of IRR was similar for incisors with immature or mature apices (3 per cent). However, the frequency of RRR in teeth with immature apices was nil compared to 1 per cent in teeth with mature apices. Development of either IRR or RRR is not dependent upon the stage of root formation.

Andreasen and Vestergaard Pedersen (1985) reported in their study of 122 laterally luxated teeth that external RR may be observed radiographically in 27 per cent of cases. This study did not provide diagnostic criteria for external RR, except for a descriptive table. The clinical study by Oikarinen *et al.* (1987) noted that among 42 laterally luxated teeth, 10 and 7 per cent were affected by IRR and RRR respectively. In their study, all luxated teeth were immobilized by a rigid splint for a mean period of 52 days. Several studies in monkeys have shown that long, rigid splinting of avulsed and auto-transplanted teeth increased the frequency of IRR and RRR (Nasjleti *et al.*, 1982; Kristerson and Andreasen, 1983).

### *Marginal Bone Loss*

According to Andreasen *et al.* (1999) loss of marginal bone is a transient periodontal healing complication observed following intrusion and lateral luxation where compression of PL

has occurred. Subsequently, damaged tissue (i.e. necrotic PL tissue remnants and bone) is removed by macrophage/osteoclast activity. This may lead to formation of granulation tissue at the site of compression and as a result, rarefaction of bone may be observed radiographically.

Oikarinen *et al.* (1987) reported that loss of marginal bone is more likely to develop with increased age of the patient ( $p < 0.05$ ). The authors also observed that the duration of splinting significantly affected marginal alveolar bone loss ( $p < 0.05$ ) (31 per cent of 42 laterally luxated teeth). They proposed that longer immobilization periods may increase bacterial deposition at the marginal gingiva. However, Oikarinen *et al.* (1987) used a rigid splint and did not consider the effect of various types of splint on marginal bone loss. Ngassapa *et al.* (1986) found that marginal bone loss is more likely to develop with a rigid splint compared with a non-rigid or semi-rigid splint. Andreasen and Vestergaard Pedersen (1985) stated that among 122 laterally luxated teeth, 5 per cent were affected by marginal bone loss. However, they did not describe the method of measurement, age group, type or duration of splint.

### ***Summary***

External RR and marginal bone loss are two complications of periodontal healing observed in populations of adults and children following lateral luxation injuries. External RR can be either IRR or RRR. Inflammatory root resorption is infrequently observed and is usually associated with PN. Replacement root resorption is unlikely to develop following lateral luxation. Marginal bone loss is a radiographic observation that may be affected by the age of patient and/or duration of splinting and occurs following lateral luxation in 5-30 per cent of cases.

**Statement of the problem**

There is a paucity of literature on healing outcomes for laterally luxated permanent maxillary incisors in children and adolescents. The four clinical studies that describe lateral luxations used combined populations of adults and children and lack standardized treatment protocols. Two of the clinical studies combined lateral luxations with the other types of luxation injuries thus confounding the results. Since the incidence of this injury peaks in children between 8-12 years and ranges into adolescence, it may also be complicated by ongoing growth of teeth and jaws (Andreasen and Ravn, 1972). As a result, ankylosis and infraocclusion are expected healing complications in this population of children and adolescents who have not achieved complete alveolar growth.

A longitudinal outcome study of lateral luxation injuries to permanent maxillary incisors in children and adolescents treated according to a standardized treatment protocol would provide quantitative prognostic information for clinicians and parents. Such a strategy would provide a reference point for future studies and potential protocol revisions for lateral luxation injuries of children and adolescent populations.

## **Hypotheses**

Stage of root development (mature vs immature) and extent ( $\leq 2$  mm vs  $>2$  mm) of lateral luxation injury are predictors of healing complications.

## **Objectives**

1. To examine the clinical outcomes of lateral luxation injuries to permanent maxillary incisors of children and adolescents initially treated at The Hospital for Sick Children between June 1988 and June 1998.
2. To determine which of the following independent variables influence the development of healing complications (PN, PCO, external RR {IRR and RRR}) following lateral luxation injuries in children and adolescents:
  - a. Extent of lateral luxation injury
  - b. Stage of root development
3. The frequencies of healing outcomes (PN, PCO, external RR{IRR and RRR}) for lateral luxation injuries to permanent maxillary incisors of children and adolescents are different from those reported in studies of mixed populations of children and adults.

## **Materials and Methods**

### **Database**

Since June 1988 information on all patients who presented to The Hospital for Sick Children, Toronto (HSC) with dentoalveolar injuries has been entered into a database. The establishment of the database coincided with the introduction of protocol-based clinical management and treatment of dental traumatic injuries at HSC. Input to the database is ongoing and it contains selected information about patients and their dental injuries that had been collected on standardized data collection forms (Appendix II).

### **Inclusion/Exclusion Criteria**

Patients were identified from the HSC trauma database and their eligibility for the study decided according to the following inclusion criteria: (1) no medical complications, (2) initial examination at HSC between June 1988 and June 1998, (3) lateral luxation injuries to maxillary permanent incisor(s) and (4) minimum of 6 months follow-up. Patients were not included if they had incomplete records, had sustained previous trauma to their permanent incisors, received emergency treatment elsewhere, or had incisors with associated root fractures or untreated caries.

### **Sample and Treatment Protocol**

After application of the inclusion/exclusion criteria, a total of 72 eligible patients were identified. Standardized protocol for the clinical management and treatment of traumatic injuries at the HSC dental clinic was first introduced in June 1988. The protocol for lateral luxation

injuries (Appendix III) was derived from clinical studies published in the literature and the treatment recommendations of Andreasen (1981).

### **Recall Protocol**

A research chart was prepared for all eligible patients. Each chart was a duplicate of the original HSC dental chart and included the original radiographs. Patients were contacted for clinical and radiographic follow-up according to a recall algorithm described by Barrett (1995), (Appendix IV). If the patient did not wish to come to HSC for a follow-up visit, a signed consent form (Appendix V) was obtained from the patient/parent for release of radiographs and clinical information from their private dentist.

Standard clinical and radiographic examinations were performed for patients who presented for follow-up. A single examiner (MN) recorded the clinical information on a standardized follow-up form (Appendix VI) and obtained the appropriate radiographs.

### **Clinical Examination**

#### ***Tooth mobility***

Mobility was recorded according to the scale described by Lindhe (1983). The assessment was performed by gently moving the incisor between the index finger on the lingual of the tooth and the mirror handle on the labial surface.

**Table 3.** Rating scale used to estimate tooth mobility (Lindhe, 1983)

Score	Estimated Movement
0	No abnormal looseness
1	Horizontal movement of the crown of not more than 1 mm
2	Horizontal movement of the crown of more than 1 mm
3	Horizontal and vertical movement of the crown of more than 1 mm

***Tooth colour***

Tooth crown colour was evaluated by comparison with the unaffected maxillary incisors. Categories were yellow, gray or normal.

***Percussion tone***

This test was performed by tapping on the incisal edge of the tooth with the end of mirror handle held parallel to the long axis of the incisor. The tone was classified and recorded as either normal or ankylosed. The latter elicited a characteristic resonant sound compared with a typical incisor. To establish a basis for comparison, unaffected maxillary incisors were also tested.

***Percussion sensitivity***

Pain was recorded as either present or absent compared with the unaffected maxillary incisors by tapping the incisal edge of the tooth with a mirror handle.

***Fistulae***

Fistulae were recorded as either present or absent.

### ***Pulp vitality tests***

Pain sensation was recorded as either present or absent compared with the unaffected maxillary incisors. Neural conduction was tested with both electrical (Siemens Sirotest® II) and cold (ice stick) stimuli.

### **Radiographic Examination**

Maxillary occlusal and periapical radiographs were obtained at the follow-up visit. All exposures were made at 65 kVp and 15 mA for 0.8 second for the periapical and 1 second for the occlusal exposure. This combination of radiographs was chosen based on the findings to Brynolf (1970 a b c) who demonstrated that from a combination of two radiographs, it was possible to diagnosis correctly in 90 per cent of normal and pathological cases. A large film (Kodak® Ultra speed, size 4) and the bisecting angle technique were used for occlusal radiographs. Periapical radiographs were taken using the paralleling technique with a Rinn XCP® kit and a small film (Kodak® Ultra speed, size 2). The orientation of the central beam for periapical radiographs depended on which teeth were injured. In the case of a central incisor, the central beam was directed between the central incisors. If a lateral incisor was injured, the beam was directed between the central and lateral incisor on the affected side.

### **Radiographic Assessment**

The stage of root development at the time of injury was assessed based on radiographs taken at the time of initial presentation. The scoring system of Moorrees *et al.* (1963) was employed to classify the stage of root development where stages 6,7 were classified as mature and stages =5 were classified as immature. The scoring system of Andersson *et al.* (1984) was

used to quantify external RR. Both of these indices have previously been described in detail by Barrett (1995) and Lee (1996).

Assessment of healing complications utilized radiographs taken at follow-up visits. Evidence of PN, PCO and external RR was recorded as either absent or present. If a tooth was quantified as having external RR, then it was classified as IRR or RRR.

Since radiographic assessment is subjective and variation may be expected between individuals, the reproducibility of their observations was assessed. By having two paediatric dentists assess the radiographs independently, it was possible to study the variation between them (Fleiss and Shrout, 1977). This is defined as inter-rater reliability. Raters were presented with radiographs in which the first of the series was a periapical or occlusal view taken at the time of injury. This was followed by a periapical and occlusal view taken at the latest follow-up visit. Raters were blinded to the identity of the patients.

The reproducibility of assessments by the same dentist (intra-rater reliability) was determined by having each of the two dentists evaluate randomly-selected radiographs one week later. If there was disagreement between raters, an expert consensus panel (two paediatric dentists) made the final decision. Data were transferred to a computer spreadsheet program for statistical analysis and Kappa scores were calculated.

## Statistical Analyses

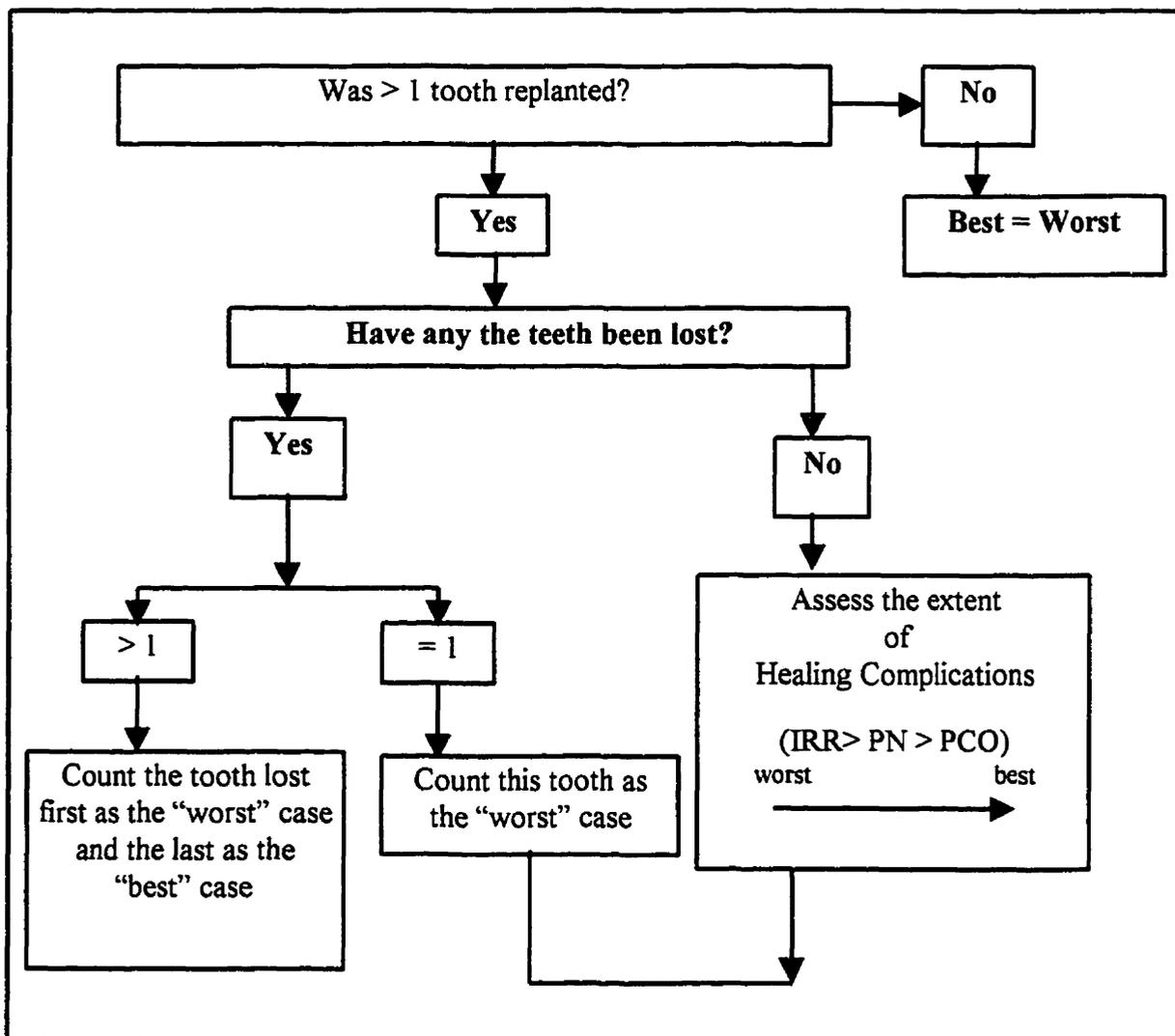
Kappa statistics were used for inter- and intra-rater measures to determine agreement beyond chance for radiographic assessments of root development and healing outcomes (PN, PCO, external RR{IRR and RRR}) (Fleiss, 1981; Hunt, 1986).

Proportional Hazards Regression (Cox, 1972) was used to determine if apical development and /or extent of lateral luxation were significantly related to development of PN. Logistic regression was used for PCO and external RR since the exact time of initiation of PCO or external RR could not be determined from radiographs taken at follow-up visits. Logistic regression was performed to evaluate if apical development and /or extent of lateral luxation were related to the development of PCO or external RR. The extent of lateral luxation was defined as mild if displacement was  $\leq 2$  mm or severe if  $>2$  mm.

In addition to analyses of individual teeth, the data were sorted into best and worse case scenarios because some patients had more than one laterally luxated incisor. For this purpose, an algorithm similar to that described by Barrett (1995) was used (Figure 1).

A critical value of 0.05 was used for all statistical tests. All statistical calculations were performed using the software package SAS® version 6.12 for the IBM-PC.

**Figure 1.** Algorithm used for the determination of best and worst case scenarios when a patient had more than one tooth replanted (modified from Barrett, 1995).



## Results

### Sample

A potential sample of 72 patients (27 females; 45 males) that represented 97 laterally luxated permanent incisors was identified according to inclusion and exclusion criteria. The mean patient age at the time of trauma was 11.3 years (range: 6.3 to 17.8 yrs.; S.D.=2.8). Follow-up information was available for 42 subjects (26 males; 16 females) that represented 58 laterally luxated maxillary permanent incisors (49 centrals; 9 laterals). The mean age for this group at the time of trauma was 11.4 years as well (range: 6.3 to 17.8 yrs.; S.D.=2.7). When necessary, the mean time until initiation of root canal treatment (RCT) was 193 days (range: 18-910 days; S.D.=225) and the mean follow-time was 4 years (range: 6 months –10 yrs.).

There were several reasons for the decrease in number of subjects available at follow-up. Some patients could not be found (i.e. moved from the area), did not consent to follow-up or refused the appointment.

There was no significant difference between the available and unavailable sample with regard to their age ( $p = 0.65$ ) or extent of lateral luxation ( $p = 1.0$ ). Among available subjects, 62 per cent had mild ( $\leq 2$  mm) and 38 per cent had severe ( $> 2$  mm) injuries. Among unavailable subjects, 84 per cent and 16 per cent had mild and severe injuries respectively.

Tables 4 and 5 present summaries of healing complications and their occurrence based on the stage of root formation at the time of trauma.

**Table 4.** Occurrence of healing complications at follow-up (n=58).

<b>Healing Complications</b>	<b>PN</b>	<b>PCO</b>	<b>IRR</b>
Absent	35	35	43
Present	23	23	15

**Table 5.** Occurrence of healing complications according to stage of root formation at follow-up (n=58).

<b>Healing Complications</b>	<b>PN</b>	<b>PCO</b>	<b>IRR</b>
Immature	3	6	1
Mature	20	17	14

### **Radiographic Assessment**

Kappa scores for inter-rater agreement for radiographic assessment are summarized in Table 6.

**Table 6.** Kappa scores for inter-rater reliability from radiographs collected at the time of injury and at follow-up.

<b>Variable at the time of injury</b>	<b>Kappa</b>	<b>p-value</b>
Root formation	0.8	0.001
<b>Variables at follow-up time</b>	<b>Kappa</b>	<b>p-value</b>
PN	0.9	0.001
PCO	0.9	0.001
External RR	0.3	0.1

The inter-rater Kappa scores for PN and PCO were in the range of perfect agreement ( $K = 0.81-1.0$ ) and substantial agreement ( $K = 0.61-0.80$ ) for root formation according to the classification defined by Landis and Koch (1977) (Appendix VII). Both values were significant ( $p < 0.05$ ). However, the Kappa score for external RR was only in the range of slight agreement ( $K = 0.0-0.20$ ) and the value was not significant ( $p > 0.05$ ).

The intra-rater Kappa scores for both raters showed perfect agreement for PN, PCO and stage of root formation and all values were significant. However, the Kappa scores for identification of external RR for both raters were in the range of slight agreement ( $K = 0.0-0.20$ ) and not significant ( $p > 0.05$ ). Therefore, use of external RR data was excluded from further analysis.

### **Proportional Hazards Regression**

Proportional Hazards Regression (Cox, 1972) was used to determine whether stage of root development or extent of injury were related to development of PN.

Both PN and PCO occurred equally in this study. Among 58 incisors, 23 laterally luxated incisors were affected by PN (39 per cent). Of these teeth, 3 had immature apices and 20 had mature apices. The distribution of incisors with PN for the best and worst case scenarios as well as the entire sample is presented in Table 7. The results of the Cox regression for the best and worst case scenarios are presented in Table 8 and 9, respectively.

**Table 7.** Distribution of incisors with PN and censored incisors for best and worst case scenarios and the entire sample.

<b>Scenario</b>	<b>Total teeth</b>	<b>PN</b>	<b>Censored</b>	<b>Per cent Censored</b>
<b>Entire</b>	58	23	35	68
<b>Best</b>	42	12	30	71
<b>Worst</b>	42	19	23	54

**Table 8.** Results of the Cox regression for the best case scenario for PN.

<b>Variable</b>	<b><i>p</i>-value</b>	<b>Relative Risk</b>
Extent of lateral luxation injury (>2 mm vs ≤ 2 mm)	0.08	2.9
Root development (mature vs immature)	0.5	1.5

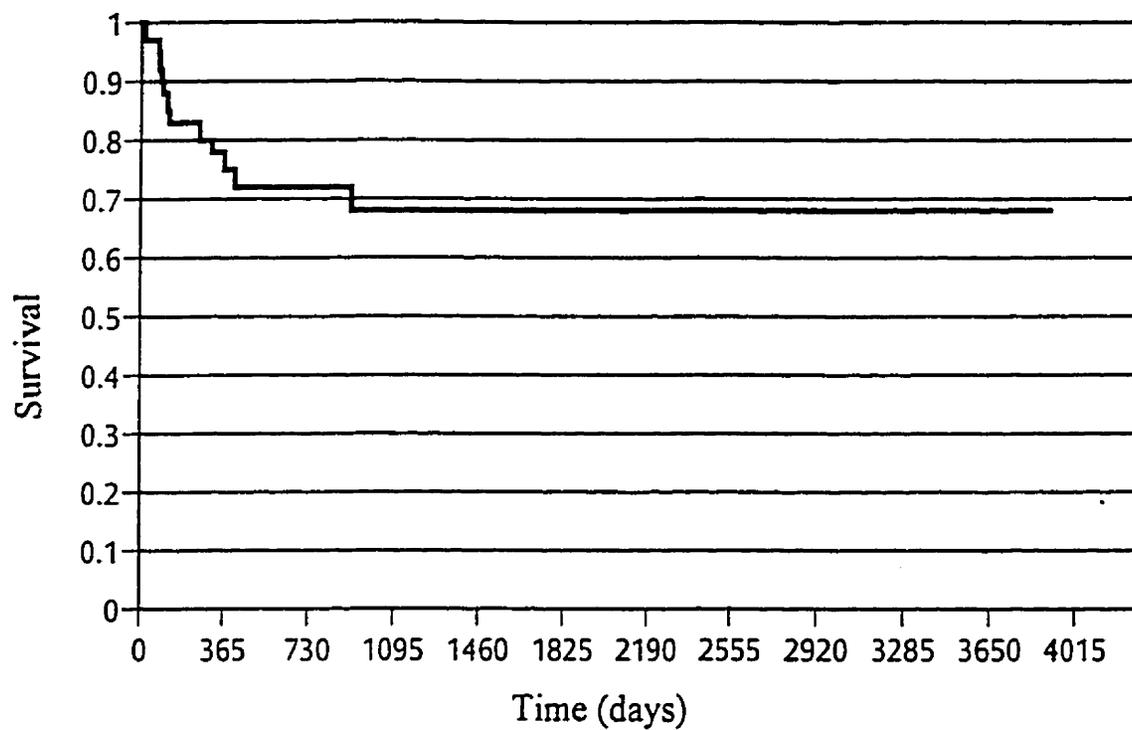
**Table 9.** Results of the Cox regression for the worst case scenario for PN.

<b>Variable</b>	<b><i>p</i>-value</b>	<b>Relative Risk</b>
Extent of lateral luxation injury (>2 mm vs ≤ 2 mm)	0.5	1.3
Root development (mature vs immature)	0.3	1.9

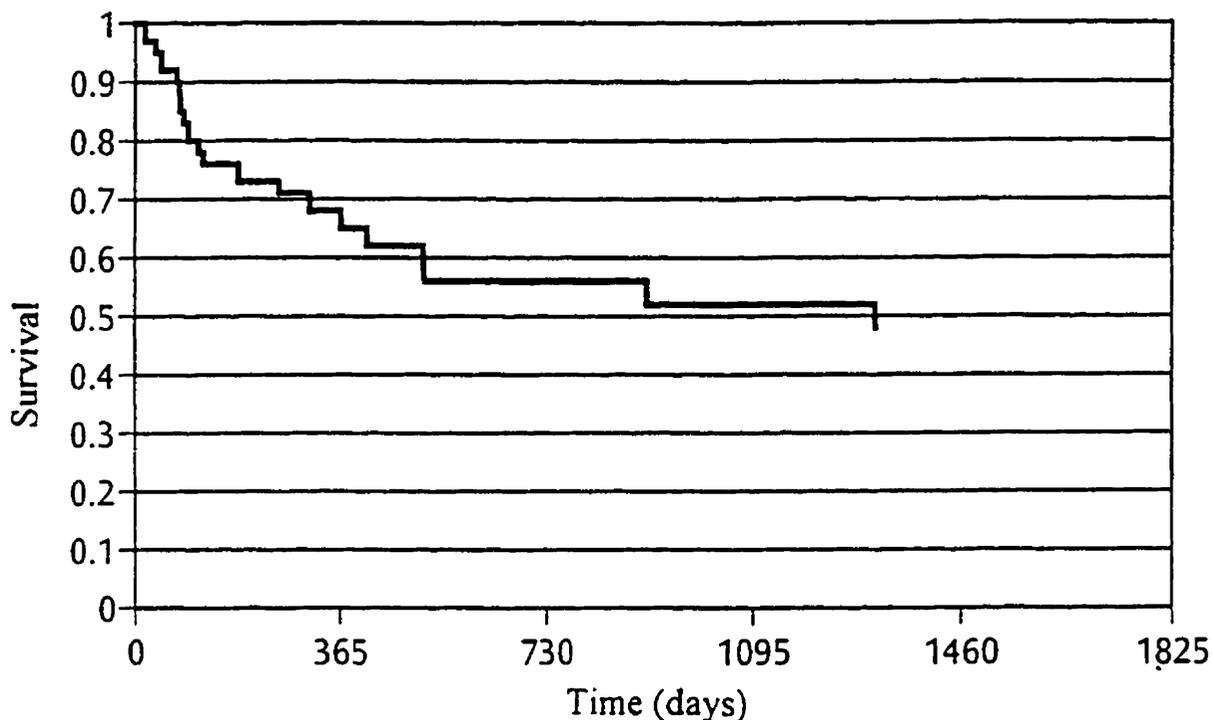
For both best and worst case scenarios, the stage of root development or extent of injury were not significantly related to the development of PN following lateral luxation injuries ( $p > 0.05$ ).

Kaplan-Meier survival curves for best and worst case scenarios are presented in Figures 2 and 3. After one year, in the best case, the probability of pulp survival is 72 per cent compare

Figure 2. Kaplan-Meier pulpal survival curve for best case scenario



**Figure 3.** Kaplan-Meier pulpal survival curve for worst case scenario



### Logistic Regression Analysis

Logistic regression analysis was performed for PCO. Logistic regression analysis was used to determine whether stage of root development or extent of injury were significantly related to the occurrence of PCO.

In this study, 39 per cent of laterally luxated incisors were affected by PCO (23 of 58 incisors). Among these incisors, 6 had immature roots and 17 had mature roots. The distribution of incisors with PCO for the best and worst case scenarios as well as the entire sample is presented in Table 10.

**Table 10.** Distribution of incisors with PCO and censored incisors for best and worst case scenarios and the entire sample.

<b>Scenario</b>	<b>Total teeth</b>	<b>PCO</b>	<b>Censored</b>	<b>Per cent Censored</b>
<b>Entire</b>	58	23	35	68
<b>Best</b>	42	18	24	57
<b>Worst</b>	42	15	27	64

The results of the Logistic regression for the best and worst case scenarios are presented in Table 11 and 12, respectively.

**Table 11.** Results of the Logistic Regression for the best case scenario for PCO.

<b>Variable</b>	<b><i>p</i>-value</b>	<b>Odds Ratio (95 per cent CI)</b>
Extent of lateral luxation injury (>2 mm vs ≤ 2 mm)	0.5	0.63 (0.10-3.84)
Root development (mature vs immature)	0.9	1.0 (0.28-4.37)

**Table 12.** Results of the Logistic Regression for the worst case scenario for PCO.

<b>Variable</b>	<b><i>p</i>-value</b>	<b>Odds Ratio (95 per cent CI)</b>
Extent of lateral luxation injury (>2 mm vs ≤ 2 mm)	0.86	0.89 (0.14-5.39)
Root development (mature vs immature)	0.81	1.2 (0.30-6.55)

As in the case of PN, the extent of lateral luxation and stage of root development were not significantly related to the development of PCO following lateral luxation injuries for both best and worst case scenarios ( $p > 0.05$ ).

## **Discussion**

This was the first longitudinal outcome study specifically focused on a sample of lateral luxation injuries to the permanent incisors of children and adolescents. The sample of 42 patients that represented 58 laterally luxated maxillary permanent incisors was unique because it consisted exclusively of a population of children and adolescents. Potential heterogeneity was further minimized by using a standardized treatment protocol. Demographic characteristics of the sample were consistent with the epidemiological literature (Andreasen, 1970a; Andreasen and Ravn, 1972). The mean age at the time of injury (11.4 yrs.) was in the peak range for dental injuries, more males were injured than females (1.6:1) and maxillary permanent central incisors (85 percent) were predominant.

Follow-up data was available for 58 per cent of the original sample of 72 patients. Despite the use of a regimented recall protocol (Appendix IV), 14 patients could not be traced, 9 moved out of the region and 7 refused the appointment because they were adolescents who chose not to comply. Patients available for follow-up were not significantly different from those unavailable with regard to age, gender, type of incisors or severity of injury ( $p > 0.05$ ). Thus, there was no sampling bias between the two groups.

### **Radiographic Assessment**

During radiographic assessment, variation may be expected between individual clinicians. Therefore, Kappa statistics for inter- and intra-rater reliability were used to determine the reproducibility of radiographic assessment by two raters (Fleiss and Shrout, 1977). In this

regard, the variation between the two raters is defined as inter-rater reliability whereas the reproducibility of assessments by the same rater is referred to intra-rater reliability. These estimates represent not only the reliability of a rater between successive evaluations but also determine the consistency of an individual rater. Whenever there was disagreement between the two raters, an expert consensus panel of two calibrated paediatric dentists made the final decision. Standards for acceptable reliability on dental measures have not been developed but ranges have been proposed for interpretation (Appendix VII).

Radiographic assessment was completed to evaluate the stage of root development and the presence or absence of healing complications (PN, PCO and external RR {IRR and RRR}). The Kappa scores demonstrated that the raters had a *perfect* degree of agreement for identification of stage of root development, PN and PCO (Table 7). However, the Kappa scores for external RR were not significant. Reasons for the variation in raters scores for external RR have been reported. (Andreasen *et al.*, 1987b; Andersson *et al.*, 1989). Root resorption cavitations may not be possible to detect on radiographs when they are small and/or located on the labial or lingual root surfaces. Additionally, Andersson *et al.* (1989) reported that in some radiographic views it is difficult to differentiate between alveolar bone and root surface radiolucency in the resorption areas and this might be due to resorption areas of the labial or lingual root surfaces superimposed over a radiolucent root canal. Moreover, Goldman *et al.* (1974) stated that there was great variability in the interpretation of radiographs among trained observers and this was even found when the same observer examined the radiograph on different occasions. Recently, Goldberg *et al.* (1998) simulated external RR on maxillary central and lateral incisors of human skulls. They reported that radiographs were not very accurate for

diagnosis of external RR and observed that the resorption areas were more difficult to diagnosis if they were located on the apical third compared with the middle and cervical third of the root surface. This may be due to the increased thickness of alveolar bone near the root apex. In the current study, we suggest that the low Kappa for identification of external RR was due to the low quantity or absence of root resorption. This is consistent with a single report in the literature (Andreasen *et al.*, 1999).

### **Survival analysis**

Survival analysis is widely applied to studies of life expectancy of patients with cancer (Peto *et al.*, 1977) and was more recently introduced into long-term dental trauma investigations (Andreasen and Vestergaard-Pedersen, 1985; Andreasen *et al.*, 1987a, Barrett and Kenny, 1997). This analysis permits staggered entry of patients into a trial over time up to a closing date and provide outcome (i.e. prognostic) information during a given observation period (Norman and Steiner, 1994). In previous clinical studies of lateral luxations, survival analysis was used to observe the relationship between the injury or treatment-related variables and the development of healing complications such as PN (Andreasen and Vestergaard Pedersen, 1985) or PCO (Andreasen *et al.*, 1987a). This has led to a better understanding of the variables that affect outcomes but has not provided information on prognosis.

### **Healing Complications**

Proportional Hazards Regression (Cox, 1972) was used to determine whether the stage of root development or extent of injury were related to development of PN. This analysis takes into consideration the *time* to a well-defined event; i.e. development of PN or root canal

therapy. Since some patients injured more than one incisor, the sample was divided into best and worst case scenarios to ensure that observations were independent (Barrett and Kenny, 1997). Alternatively, Andreasen *et al.* (1995) employed a random draw strategy to eliminate the problem of interdependence in a study of 400 avulsed permanent incisors.

The best and worst case strategy was used because it would be the most likely to result in a significant difference between groups if in fact, there was one. In this regard, the strategy took the form of a sensitivity analysis where the findings are influenced by our initial assumptions. The results showed that the best case did not differ significantly from the worst case scenario. In this sample, only 40 per cent of laterally luxated incisors developed PN and most PN occurred within the first year. This finding indicates that PN is at greater risk during the first year following this injury.

The occurrence of PN (40 per cent) was less than expected when compared with the four existing clinical studies of lateral luxation injuries (Table 2). This might be related to the possibility that a number of extrusive and intrusive injuries may have been falsely categorized as lateral luxations. This would have produced lateral luxation samples that included injuries associated with increased incidences of PN and PCO. Pure samples of intrusions (Humphrey, 1999) and extrusions (Lee, 1996) have shown PN to be the most common complication and in both cases greater than this study.

In the current study, the stage of root development was not significantly related to the development of PN following lateral luxation injuries ( $p > 0.05$ ) (Table 6). The results of the

current study differ from Andreassen and Vestergaard Pedersen (1985) who reported that mature apices were significantly related to the development of PN ( $p < 0.0002$ ) following lateral luxation injury. Based on the current study and the unresolved discrepancy in these results with the study of Andreassen and Vestergaard Pedersen (1985), the effects of root development remain unsettled.

No previous clinical or animal studies have used amount of coronal dislocation to quantify the severity of lateral luxation injuries. In this sample a lateral luxation was defined as mild if displacement was  $\leq 2$  mm or severe if  $>2$  mm. In this study, the extent of lateral luxation injury was not significantly related to the development of PN following lateral luxation ( $p > 0.05$ ).

Logistic regression analysis was used to determine if stage of root development or extent of injury were significantly related to development of PCO. Since a specific time to end-point is required for proportional hazards regression analysis, this model was inappropriate for PCO because the exact time the event occurred cannot be determined. The results of the current study showed that 40 per cent of laterally luxated incisors developed PCO. This finding was higher when compared with other studies (Andreassen *et al.*, 1987a; Oikarinen *et al.*, 1987). As was the case for PN, misdiagnosis of lateral luxations or inclusion with other types of luxation injuries would lead to a skewed sample distribution.

Andreassen *et al.* (1987a) stated that stage of root development was significantly related to the development of PCO. Again, this finding was based on a sample that combined lateral

luxations with other types of luxation injuries. In the current study, 73 per cent of incisors with mature apices experienced PCO in contrast to 11 per cent of laterally luxated teeth with mature apices reported by Andreasen *et al.* (1987a).

The extent of lateral luxation injury was not significantly related to the development of PCO following lateral luxation ( $p > 0.05$ ). Furthermore, none of the laterally luxated incisors became necrotic after evidence of PCO. In the literature, the development of PN secondary to PCO is a late complication that occurs in 1 to 16 per cent of affected teeth (Halcomb and Gregory, 1967; Andreasen, 1979; Robertson *et al.*, 1996; Andreasen *et al.*, 1987a). Investigators have discussed whether evidence of PCO is predictive of PN and if subsequent prophylactic endodontic treatment should be considered (Lundberg and Cvek, 1980; Robertson *et al.*, 1996). Recent studies do not support such measures due to the low frequency of this complication (Lundberg and Cvek, 1980; Andreasen *et al.*, 1987a, Curzon, 1999).

In this study, no cases of infraocclusion were observed in the entire sample. In contrast to extrusions where PL is stretched or torn, and intrusions where the PL is severely crushed, in lateral luxations, the PL is compressed on one side and torn on the other side. Differences in damage to the PL associated with this type of injury appear to affect ankylosis and subsequent infraocclusion.

### **Clinical Decision Making**

This is one of a series of outcome studies on traumatic injuries that have so far investigated avulsions (Barrett, 1995) extrusions, (Lee, 1996) and intrusions (Humphrey, 1999).

These studies have been designed to provide outcome-based prognostic information on luxation injuries to the permanent maxillary incisors of children and adolescents. An objective of the current investigation was to provide clinicians and parents with prognostic information about the healing outcomes of laterally luxated incisors.

In this study, no incisors failed and thus, survival prospects for long term retention of laterally luxated incisors of children and adolescents are excellent. Healing complications observed were PN and PCO. This study supports the Andreasen *et al.* (1999) observation that external RR is less likely to occur compared with other luxation injuries. Consequently, ankylosis and infraocclusion are not features of this injury. Pulpal necrosis was usually diagnosed within the first year. Therefore, parents can be informed that PN is more likely to occur within the first year after injury. This information on the occurrence of PN is useful for parents who are concerned about future endodontic treatment.

Based upon the current investigation, parents can be informed that PCO is a common healing complication following lateral luxation injury. This may comfort parents because as long as the pulp remains vital, endodontic treatment will not be required. However, parents and adolescent patients should be guided on esthetic issues of coronal discolouration related to PCO.

## **Conclusions**

**This study has shown that:**

- 1. Pulpal necrosis (PN) is a common (40 per cent) complication following lateral luxation injuries. In this study, PN usually developed within the first year of injury. Pulp canal obliteration (PCO) is also a common outcome (40 per cent) following lateral luxation injuries. External RR is less likely to occur compared with other luxation injuries. Consequently, ankylosis and infraocclusion are not features of this injury. These findings differ from those reported in previous studies of mixed populations of children and adults.**
- 2. The stage of root development and extent of lateral luxation injury were not significantly related to the development of healing complications (PN and PCO) following lateral luxation injuries in children and adolescents.**
- 3. In this population, no incisors (n=58) failed. Therefore, survival prospects for laterally luxated incisors in children and adolescents are excellent.**

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## Appendix I

### WHO codes for Dentistry and Stomatology

#### 1. Injuries to the Hard Dental Tissues and Pulp

- N502.50 - enamel infraction
- enamel fracture, uncomplicated
- N502.51 - enamel-dentin fracture, uncomplicated
- N502.52 - complicated crown fracture
- N502.54 - uncomplicated crown-root fracture
- complicated crown-root fracture
- N502.53 - root fracture

#### 2. Injuries to the Periodontal Tissues

- N503.20 - concussion
- N503.20 - subluxation
- N503.20 - extrusive luxation (peripheral dislocation, partial avulsion)
- N503.20 - lateral luxation
- N503.21 - intrusive luxation (central dislocation)
- N503.22 - avulsion (exarticulation)

#### 3. Injuries to the Supporting Bone

- N502.60 - comminution of the mandibular alveolar socket
- N502.60 - fracture of mandibular alveolar socket wall
- N502.60 - fracture of mandibular alveolar process
- N502.61 - fracture of mandible
- N502.40 - comminution of the maxillary alveolar socket
- N502.40 - fracture of maxillary alveolar socket wall
- N502.40 - fracture of maxillary alveolar process
- N502.42 - fracture of maxilla

#### 4. Injuries to the Gingiva or Oral Mucosa

- S01.50 - laceration of the gingiva or oral mucosa
- S00.50 - contusion of the gingiva or oral mucosa
- S01.50 - abrasion of the gingiva or oral mucosa

## Appendix II

### PERMANENT TOOTH TRAUMA INFORMATION SHEET

Department of Dentistry  
Division of Paediatric Dentistry  
The Hospital for Sick Children  
555 University Avenue  
Toronto, Ontario, M5G 1X8

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Date of trauma: \_\_\_\_\_ Date of HSC emergency treatment: \_\_\_\_\_

Time of trauma: \_\_\_\_\_ Time treatment commenced: \_\_\_\_\_

Previous treatment of currently injured tooth/teeth: Yes  (describe on back); No

Tooth discoloured prior to treatment: Yes ; tooth #(s): \_\_\_\_\_; colour: \_\_\_\_\_; No

Angle Class: \_\_\_\_\_; N/A ; N/R  Overjet: \_\_\_\_\_ mm; N/R  Overbite: \_\_\_\_\_%; N/R

Etiologic classification: fall  impact  collision

---

Tooth #: \_\_\_\_\_

**Periodontal trauma:** Yes  No

Concussion:  (tender to percussion)

Subluxation: M1 , M2 , M3

Intrusion:  (\_\_\_\_mm)

Extrusion (partial avulsion):  (\_\_\_\_mm)

Lateral luxation:  
lab , ling , mes , dist   
estimate (mm) luxation \_\_\_\_\_

Complete avulsion:

tooth lost:

storage media: \_\_\_\_\_

---

\*time of insertion: \_\_\_\_\_

\*length of time out of mouth: \_\_\_\_\_

**Radiographic diagnosis:**

Root (apex) development:  
immature:   
mature:

Root trauma: Yes ; No   
root fracture:  separated segments:

Bone trauma: Yes ; No   
comminution of socket:   
socket wall fracture:   
alveolar process fracture:   
jaw fracture: Mx ; Mand

**Crown/pulp trauma:** Yes ; No

crazing:

enamel fracture only:

enamel and dentin fracture:

pulp exposed:  (\_\_\_\_mm)

crown-root fracture/no pulp exposure:

crown-root fracture/pulp exposure:

Tooth #: \_\_\_\_\_

**Periodontal trauma:** Yes  No

Concussion:  (tender to percussion)

Subluxation: M1 , M2 , M3

Intrusion:  (\_\_\_\_mm)

Extrusion (partial avulsion):  (\_\_\_\_mm)

Lateral luxation:  
lab , ling , mes , dist   
estimate (mm) luxation \_\_\_\_\_

Complete avulsion:

tooth lost:

storage media: \_\_\_\_\_

---

\*time of insertion: \_\_\_\_\_

\*length of time out of mouth: \_\_\_\_\_

**Radiographic diagnosis:**

Root (apex) development:  
immature:   
mature:

Root trauma: Yes ; No   
root fracture:  separated segments:

Bone trauma: Yes ; No   
comminution of socket:   
socket wall fracture:   
alveolar process fracture:   
jaw fracture: Mx ; Mand

**Crown/pulp trauma:** Yes ; No

crazing:

enamel only fracture:

enamel and dentin fracture:

pulp exposed:  (\_\_\_\_mm)

crown-root fracture/ no pulp exposure:

crown-root fracture/pulp exposure:



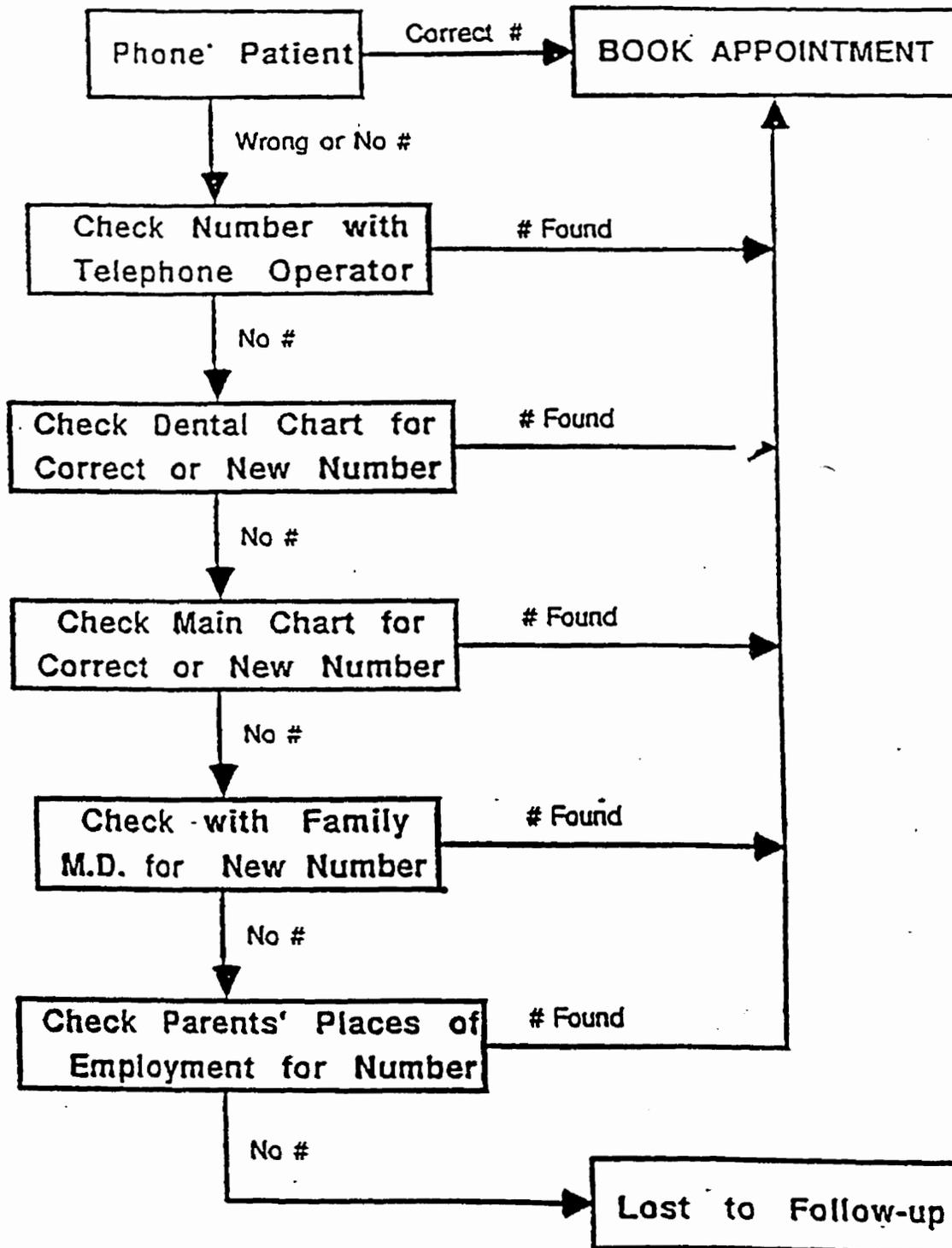
## **Appendix III**

### **HSC protocol for the management of laterally luxated permanent maxillary incisors (June 1988)**

1. Reposition the incisor to normal functional position.
2. Splint the tooth with a light-wire/acid-etch splint if the tooth is excessively mobile.
3. Penicillin is the drug of choice at a dosage of 25 to 50 mg/kg in three or four daily divided doses for seven days. Erythromycin at a dosage of 20 to 40 mg/kg is the suggested drug for patient allergic to Penicillin. In addition, a 0.1 per cent chlorhexidine mouthrinse (10 to 15 ml twice daily for 10 days) is recommended following 48 hours after the emergency.
4. Clinical and radiographic assessment within two weeks of the initial visit. Parents or adolescents were then given the option of either returning to their private dentist or continuing at HSC dental clinic.
5. Recall at 2 and 6 weeks for clinical and radiographic assessment and splint removal time optional between 3 and 6 weeks.
6. Continuing clinical and radiographic assessment at 6 months and 1 year.

## Appendix IV

## Patient Recall Scheduling Protocol



## Appendix V



## Authorization for Release of Information

I hereby authorize Dr. \_\_\_\_\_ to release to Dr. Mandana Nikoui, Paediatric Dentist, and Dr. David J. Kenny, Director Dental Research and Graduate Studies, The Hospital for Sick Children, 555 University Avenue, Toronto, Ontario, M5G 1X8, information in the form of **radiographs ("x-rays") and clinical information** from the file of \_\_\_\_\_

I understand that this information will be used for research purposes only and will be kept strictly confidential.

\_\_\_\_\_  
Signature of Parent

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature of Witness

\_\_\_\_\_  
Date

Please provide the dentist's full name and mailing address below.

Name \_\_\_\_\_

Address \_\_\_\_\_

Telephone \_\_\_\_\_

Thank you.

## Appendix VI

### LATERAL LUXATION FOLLOW-UP INFORMATION SHEET

<b><i>PATIENT INFORMATION</i></b>
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Chart # D.O.B.

Name: D.O.T.

<b><i>POST-TRAUMA EVALUATION</i></b>	<i>Date:</i>
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1.2

1.1

2.1

2.2

Crown Colour:

Percussion tone:

Percussion sensitivity:

Periodontal probing:

Mobility\*:

Pulp Vitality\*\*:

*\*(M0=Normal, A= Ankylosed)*

*\*\* (Electric pulp tester=EPT, Cold ice= C)*

<b><i>POST-TRAUMA TREATMENT</i></b>	<i>Date:</i>
-------------------------------------	--------------

RCT :

Extraction :

*Other affected tooth/teeth at the time of trauma:*

## Appendix VII

### Kappa Score Interpretation (Landis and Koch, 1977)

<b>Kappa value</b>	<b>Descriptive Agreement</b>
<0	poor
0.0 - 0.20	slight
0.41 - 0.60	moderate
0.61 - 0.80	substantial
0.81 - 1.0	perfect