A longitudinal outcome study of intrusive luxation injuries to permanent maxillary incisors of children and adolescents

by

Janice M. Humphrey

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

© Copyright by Janice Humphrey 1999
The author has granted a non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author’s permission.

0-612-45971-3
Abstract

A longitudinal outcome study was undertaken to identify variables that significantly influenced tooth survival and pulpal and periodontal outcomes of intruded permanent maxillary incisors of children and adolescents. All cases were treated between June 1988 and June 1998 at The Hospital for Sick Children. Clinical and radiographic data were collected for 26 patients (19 males, 7 females) and 31 permanent maxillary incisors. Mean age at the time of injury was 9.3 years (range 5.5-17.8 years). All pulpal and periodontal outcomes were observed at follow-up. Survival analysis was used to identify variables significantly related to the survival and pulpal necrosis for these incisors. Proportional hazards regression yielded significant prognostic models for survival and the diagnosis of pulpal necrosis ($p<0.05$ and $p<0.02$ respectively). Incisors intruded $>6$mm had a significantly decreased survival compared with incisors intruded $<3$mm (5 year survival 0.45 and 1.0 respectively; $p<0.01$). Complex crown fractures were significantly related to the development of pulpal necrosis ($p<0.01$). Logistic regression analysis showed that treatment method and severity of intrusion were related to the presence of RRR ($p<0.04$). This study represents the largest sample of intruded permanent incisors examined in an outcome study of children and adolescents exclusively. This information will be useful to clinicians and parents involved in emergency and follow-up care of these injuries.

**Key Words:** permanent incisor, intrusion, survival, pulpal necrosis, replacement resorption, prognosis
Acknowledgements

It will be impossible to name all family, friends and colleagues who shared in my excitement at the beginning, who provided encouragement, guidance and taught me to have patience throughout this project then shared in the celebration upon its successful completion. Thanks to all of you, your support will not be forgotten.

I will take this opportunity to acknowledge several of these individuals for their direct and valuable contributions.

Dr. David Kenny, my graduate supervisor, I am indebted to you for your support and guidance, for always being available to listen and to provide the necessary assistance. I extend my appreciation to you for sharing your wisdom, patience and enthusiasm during my graduate education.

Dr. E. Barrett, member of my supervisory committee, thank-you for your time spent editing, your assistance and expertise in the preparation of both poster and slide presentation materials.

Dr. D. Johnston and Dr. M. Sigal, members of my supervisory committee, thank-you for your time and recommendations that assisted me towards completion of the thesis.

Dr. M. Casas and Dr. P. Judd, thank-you for your assistance and valuable time spent evaluating radiographs for this research project.

Thank-you to the Dental Trauma Research Group at The Hospital for Sick Children who provided the necessary computer software, equipment and assistance for this project and for the quality presentation material.

Finally
To my parents, I cannot express in words how much your incredible understanding, care and support means to me. Thank-you.
Table of Contents

Abstract ii
Acknowledgements iii
Table of Contents iv
List of Tables vi
List of Figures vii
List of Appendices viii

Introduction 1

Literature Review 3
Classification and diagnosis 3

Pathophysiology of Intrusions 5
Periodontium and dental pulp 5
Root development 7
Animal studies 7
Summary 9

Management 9
Clinical guidelines 9
1994 Clinical protocol of Andreasen and Andreasen 13
1997 Clinical guidelines of the Royal College of Surgeons of England 19
Antibacterials 21
Splinting 22
Endodontic therapy 23
Follow-up 24
Summary 25

Prognosis- Healing and healing complications 25
Animal studies 26

Human studies 26

Survival 26

Dental pulp 28
Pulp canal obliteration 28
Pulpal necrosis 29
Summary 31

Periodontium 32
External root resorption 32
Inflammatory root resorption 33
Replacement root resorption 34
List of Tables

<table>
<thead>
<tr>
<th>Table</th>
<th>Name</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Table 1</td>
<td>Summary of clinical outcome studies in the English language dental literature on permanent incisor intrusions</td>
<td>12</td>
</tr>
<tr>
<td>Table 2</td>
<td>Comparison of available and unavailable samples</td>
<td>47</td>
</tr>
<tr>
<td>Table 3</td>
<td>Comparison of survived and failed samples</td>
<td>48</td>
</tr>
<tr>
<td>Table 4</td>
<td>Summary of the frequency of variables in the available sample (N=31)</td>
<td>49</td>
</tr>
<tr>
<td>Table 5</td>
<td>Summary of outcomes for the available sample (N=31)</td>
<td>49</td>
</tr>
<tr>
<td>Table 6</td>
<td>Summary of Kappa values and confidence intervals for inter-examiner reliability radiographic assessment of root development, external root resorption and pulp canal obliteration</td>
<td>50</td>
</tr>
<tr>
<td>Table 7</td>
<td>Proportional hazards regression for incisor survival; best=worst case sample</td>
<td>52</td>
</tr>
<tr>
<td>Table 8</td>
<td>Distribution of incisors with pulpal necrosis</td>
<td>54</td>
</tr>
<tr>
<td>Table 9</td>
<td>Proportional hazards regression for pulpal necrosis best=worst case sample</td>
<td>54</td>
</tr>
<tr>
<td>Table 10</td>
<td>Summary of clinical expectations for intrusions of the permanent maxillary incisors in children and adolescents</td>
<td>64</td>
</tr>
</tbody>
</table>
List of Figures

Figure 1 Kaplan-Meier survival curve for incisor survival of the available sample 52

Figure 2 Kaplan-Meier survival curves for incisor survival stratified by the RCSE classification of severity of intrusion for the best-worst case sample 53

Figure 3 Kaplan-Meier survival curves for the diagnosis of pulpal necrosis when stratified on the degree of crown fracture for the best-worst case sample 55

Figure 4 Kaplan-Meier survival curves for the diagnosis of pulpal necrosis stratified on treatment method for the best-worst case sample 55
## List of Appendices

| Appendix I | Standardized trauma data form-emergency visit | 75 |
| Appendix II | Protocol for management of permanent incisors-Intrusions | 77 |
| Appendix III | Patient follow-up data collection form | 78 |
| Appendix IV | Radiographic evaluation scoring sheet | 79 |
| Appendix V | Patient contact algorithm | 80 |
| Appendix VI | Data and outcomes for available and unavailable sample | 81 |
| Appendix VII | Kappa score interpretation | 82 |
| Appendix VIII | Algorithm used for the determination of best and worse case scenarios | 83 |
Introduction

The intrusion (intrusive luxation) of a maxillary incisor is the rarest and most complex luxation injury of the permanent dentition. The low incidence (<3.6% of all permanent incisor luxation injuries) suggests that clinicians will have little experience with diagnosis and treatment of this injury (Andreasen and Ravn, 1972; Meadow et al., 1984; Eriksson, 1993). Clinicians may be uncertain about management of intrusions due to variations in published treatment protocols by Andreasen and Andreasen (1994); Andreasen et al. (1999) and guidelines by the Royal College of Surgeons of England (1997) (RCSE). Current management strategies include waiting for re-eruption, surgical repositioning and repositioning with traction.

Clinical studies that report the prognosis of intrusions are rare, are not comparable and do not consider all variables (Andreasen, 1970a; Jacobsen, 1983; Andreasen and Vestergaard-Pedersen, 1985; Kinirons and Sutcliffe, 1991). Clinical outcomes following intrusions have been reported to include pulp canal obliteration (PCO), pulpal necrosis (PN), external root resorption (external RR), alveolar bone loss and tooth extraction (Skeiller, 1960; Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985; Kinirons and Sutcliffe, 1991; Eriksson, 1993). Relationships between clinical outcomes and variables such as severity of injury, concurrent crown fractures and treatment methods have not been studied with appropriate statistics. Data from different studies cannot be compared until there is uniform description of the injury and consistent use of treatment protocols.

Treatment protocols for intrusions must be based on the pathophysiology of the injury to both the periodontium and dental pulp (Barrett, 1995; Lee, 1996). Well-designed clinical outcome
studies will validate treatment protocols and contribute clinically useful prognostic data for the clinician, patient and parent.
LITERATURE REVIEW

Classification and diagnosis

Dental intrusions are catastrophic traumatic injuries due to the extensive damage sustained by the periodontium and dental pulp (Skeiller, 1960; Andreasen, 1970b; Turley et al., 1984). A direct impact injury to the incisal edge of an incisor causes axial displacement of an incisor into the alveolar bone and is always accompanied by concussion and usually by comminution or fracture of the alveolar socket (Andreasen, 1970a). The result is expansion of the alveolus to accommodate the new position of the root (Andreasen, 1970a; Andreasen and Andreasen, 1985; Andreasen and Vestergaard Pedersen, 1985). The rarity of intrusions in the permanent dentition (<3.6% of all luxation injuries) not only suggests that clinicians have little experience with the treatment of this injury but that it will take time to collect sufficient data for clinically meaningful outcome studies (Andreasen, 1970b; Eriksson, 1993).

The diagnosis of intrusions is based on both clinical and radiographic examinations. Visual inspection, palpation and percussion are essential to diagnosis. The most widely accepted standard for classification of luxation injuries by Andreasen and Andreasen (1994) was adopted and published by the World Health Organization (1995) in the Application of the International Classification of Diseases to Dentistry and Stomatology. Clinical measurement of axial displacement provides a rapid, reproducible reference to quantify the intrusion if there are well-documented landmarks such as fully erupted uninjured teeth (Andreasen and Vestergaard Pedersen, 1985). The procedure for measurement has not been defined in the literature. If crown fracture has occurred measurement will be difficult, therefore radiographs are a necessary adjunct to assess the severity of intrusion. The crown of severely intruded incisors may also be
completely covered by gingiva and alveolar bone and the root apex may perforate the floor of the nasal cavity. These severe intrusions may be incorrectly diagnosed as avulsed teeth or as partially erupted incisors. Percussion and mobility testing are the standard methods used to differentiate partially erupted incisors from intruded incisors. Intruded incisors that are impacted and wedged into alveolar bone are immobile and will elicit a distinct sound similar to that heard when ankylosed teeth are percussed (Andersson et al., 1984). Some intruded incisors will be accompanied by fracture of the alveolar bone (Andreasen, 1970b). In these cases, mobility can be expected.

Radiographs taken at initial presentation are required for accurate diagnosis, assessment of the severity of the injury and assessment of the stage of root development. Andreasen and Andreasen (1985) reported that retrospective radiographic diagnosis of intrusions was improved when both occlusal and periapical radiographs were taken. A missing or diminished periodontal ligament (PL) space is coincident with intrusions. The extent of intrusion is measured from the level of the interdental septum to the cemento-enamel junction (Bruszt, 1958; Andreasen and Andreasen, 1985). Radiographic techniques that use film holders are essential both at initial presentation and follow-up in order to produce accurate documentation (Andreasen and Andreasen, 1985). At follow-up appointments, radiographs serve as a reference to assess healing and the development of undesirable clinical outcomes that include PN, PCO, inflammatory root resorption (IRR), replacement root resorption (RRR) and alveolar bone loss.

The superior method for measuring the severity of intrusion has not been identified in the literature. Both methods have limitations. Clinically, it will be difficult to assess the severity of intrusion for incisors with crown fractures and for partially erupted incisors. Radiographically,
the severity of intrusion depends upon an accurate technique to avoid error in angulation that would result in inaccurate measurement. A combination of both clinical and radiographic measurement will result in the most accurate description for the severity of intrusion.

Pathophysiology of Intrusions

*Periodontium and dental pulp*

Intrusions are characterized by complex damage to the periodontium and dental pulp (Skeiller, 1960; Andreasen, 1970b; Turley et al., 1984). Since it is not possible to develop a human model to study intrusions, clinicians must base their knowledge on their understanding of the anatomy of the periodontium and pulpal neurovasculature and animal studies. The full spectrum of acute injury includes disrupted gingival epithelium, loss of epithelial attachment, crushed and torn cementum, crushed, sheared, stretched or torn PL, compression of alveolar bone and dental pulp vasculature, and the potential for crown and/or root fracture (Andreasen, 1970b; Andreasen and Andreasen, 1994; Cunha et al., 1995).

The periodontium consists of gingival tissues, PL, cementum, alveolar bone and a neurovascular system. The gingival tissues will be lacerated and may be torn from alveolar bone when an incisor is intruded (Andreasen, 1970b). The dentogingival junction, an apical connective tissue attachment and a coronal junctional epithelium (0.25 to 1.30 mm) wide band of stratified squamous epithelium (Schroeder, 1986) that produces and maintains an epithelial attachment will also be torn from the root. Bacteria and foreign material from the oral cavity may pass through the torn dentogingival junction and contribute to inflammation and infection at the injury site (Andreasen and Andreasen, 1994).
Variable amounts of crushing, compression, tearing, stretching and shearing of the PL will be produced by the intrusion. The PL space varies with age and location along the length of the root but is reported to have an average width of 0.21 mm during adolescence (Coolidge, 1937). Severe injury to all PL cells and fibers is expected when the crown of a fully erupted incisor is intruded below the margin of the gingiva. Incisors intruded to a lesser degree may have fibers in selected locations that were compressed, torn and/or stretched.

The cementum and associated cells may be crushed and/or sheared. This may create a root that has denuded areas of cementum that may not regenerate and so the PL cannot re-establish continuity. The loss of cementum will allow direct communication between the dental pulp and the outer surface of the root through the exposed dentinal tubules. Bacteria and/or necrotic debris may pass between the dental pulp and PL through the exposed dentinal tubules.

During intrusion, the PL space will be obliterated and the alveolar bone will be compressed by the root. In some cases, the cortical plate may also be fractured (Andreasen, 1970b). The alveolar bone can be expanded by the tooth crown and fractured.

The neurovasculature of both the periodontium and pulp will be damaged by the intrusion. The periodontal vasculature will be crushed or sheared between the alveolar bone and root surface. The pulpal neurovasculature enters the root canal through the apical foramen and lateral canals and will be compressed and/or torn. As a result, perfusion of both periodontal and pulpal tissue will be compromised and ischemic damage leading to necrosis is an expected outcome (Tronstad et al., 1986).
Root development

The population of intruded incisors consists of teeth in a number of stages of root development. Root length and apical development are dynamic components of root development. Root development is complete when the incisors are fully erupted with complete root length and mature apices (Moorrees, Fanning and Hunt, 1963). Anatomical differences between incisors with immature and mature apices include root length, width of the apical foramen and width of the PL. Erupting incisors have incomplete root length, thin dentinal walls and loosely packed, thick PL fibers that are synthesized and remodeled at an increased rate compared to incisors with full root length (Schroeder, 1986). Consistency in radiographic evaluation of the stage of root formation is required if it is to be used as a predictor of prognosis. Clinical studies have used the stages proposed by Moorrees, Fanning and Hunt (1963) or another system to assess root development (Andreasen, 1970a; Andreasen and Andreasen, 1985; Andreasen and Vestergaard Pedersen, 1985; Kinirons and Sutcliffe, 1991). Intruded incisors with immature apices may have an increased potential for perfusion of the pulp that may reduce the incidence of pulpal ischemia due to revascularization at the immature apices (Andreasen and Vestergaard Pedersen, 1985). This concept remains unproven. Another possibility is that a decreased compression of the apical vessels at the time of the injury is due to the width of the immature apex. The pathophysiology of the intruded incisor with incomplete root development requires additional investigation.

Animal studies

Appropriate animal models may be used to provide information about the acute injury and the subsequent chronology of healing provided anterior teeth are chosen for study. Variations in root and alveolar bone morphology as well as physiological variations between animals may
predispose the incisor to a different response when subjected to an intrusive force. Problems with existing animal studies include species differences, variation in teeth chosen for study and lack of quantitative and qualitative descriptions, mixing injury types, use of non-standardized techniques and variability in recall schedules. (Birkedal-Hasen, 1973; Turley et al., 1984; Turley et al., 1987; Miyashin et al. 1990, 1991; Cunha et al., 1995).

Only one animal study has attempted to assess the chronology of morphological and histological changes following simulated intrusions. Cunha et al. (1995) subjected 20 incisors of 10 dogs to a standardized technique and a force application of 4 kg intensity designed to produce approximately 3mm intrusions. All incisors were observed for passive repositioning. The pulpal and periodontal reactions were then examined histologically from the time of acute injury to day 60. The usefulness of the study is questionable. A 3mm intrusion in mongrel dog incisors appears to approximate a severe intrusion in humans. One would expect a variety of healing outcomes due to the severity of the intrusions and the 60 day length of this study. This is sufficient time for the development of outcomes that could include PN, PCO, IRR, RRR, alveolar bone loss. Cunha et al. (1995) reported that by day 7 healing was well underway with reorganization of PL fibers and by day 15 the PL and pulpal tissue showed normal histology. The number of animals chosen allowed a recall frequency for histological evidence from 4 teeth each at day 7, 15, 25 and 60. Root fracture is not a usual outcome of intrusions in humans yet the force applied to dogs produced root fractures in 'some roots' (Andreasen, 1970b; Andreasen and Ravn, 1972). The question remains whether the fractures are due to the force applicator or if they were an outcome of the intrusion. Outcomes that include disrupted growth in root length must be viewed with caution. Root fractures and displacement of Hertwig's epithelial root sheath reported at the time of the injury may have been a result of the experimental force applicator. The results of this
animal study are not consistent with the results of the animal outcome studies and human clinical outcome studies to be discussed later (Skeiller, 1960; Andreasen, 1970a; Turley et al., 1984; Andreasen and Vestergaard-Pedersen, 1985; Turley et al., 1987).

Summary

An intrusion causes a complex injury to the pulpal tissues and periodontium. The critical amount of intrusion that results in the most severe healing complications has not been defined in the literature. The exact severity of the damage sustained by an incisor and subsequent healing complications is unknown but anticipated to parallel the severity of intrusion. Differences are expected between the incisors with immature and mature root development. There is no histological evidence to describe the pathogenesis of healing for intrusions. This information is necessary for the clinician in order to provide prognosis and treatment options to the patient and parent.

Management

Clinical guidelines

In recent years, clinical guidelines have been under scrutiny due to lack of supporting scientific evidence of outcomes. Historically, clinical guidelines were developed on the basis of expert consensus. Evidence-based guideline development has evolved and aims to produce guidelines based on critical evaluation of the scientific evidence (Bader and Shugers, 1995; Hoyt, 1997). A major shortcoming of guideline development is that valid recommendations cannot be developed in the absence of acceptable evidence. Therefore, a methodology that combines an evidence-based approach with consensus of expert opinion to fill the voids is the current compromise in clinical guideline development (Hoyt, 1997).
Uniform guidelines for the management of intrusions do not exist. Clinicians usually follow the consensus-based protocol proposed by Andreasen and Andreasen (1994) (Tainter, 1979; Perez et al., 1982; Spalding et al., 1985; Shapira et al., 1986; Tronstad et al., 1986; Mamber, 1994; Jacobs, 1995, Oulis et al., 1996). Recently this protocol has been revised and published by Andreasen et al. (1999). The Royal College of Surgeons of England produced alternative evidence-based guidelines for management of intrusions (Kinirons, 1998). The protocol by Andreasen et al. (1999) and the clinical guidelines by the RCSE vary in their recommendations for repositioning intruded incisors, particularly those intruded to or below the gingival margin.

Barrett and Kenny (1997) assessed guidelines for management of avulsions and recommended that the merits and shortcomings of any guideline must be understood before it is used to make clinical decisions. They identified an assessment tool by Bader & Shugars (1995). This tool enables clinicians and investigators to compare guidelines against the same standard so their strengths and weaknesses can be identified. Using this strategy the major shortcoming of both sets of recommendations for intrusions is that neither has been tested by clinical trials. In contrast to Andreasen and Andreasen (1994) and Andreasen et al. (1999), the RCSE described the level of evidence for their recommendations. Finally, the protocol by Andreasen and Andreasen (1994) and the guidelines by the RCSE (1997) lack information on specific outcome information, economic cost, risk and benefits of treatment. This is important information that may influence treatment decisions by the parent and patient.

Recommended strategies for the management of intruded incisors are; waiting for re-eruption, immediate repositioning (partial or complete) or delayed repositioning with light splinting forces and traction. A single longitudinal outcome study by Kinirons and Sutcliffe (1991) studied the
relationship between treatment method, severity of intrusion, extent of root development and outcomes. The remaining evidence for clinical outcomes of intrusions is derived from retrospective outcome studies (Skeiller, 1960; Andreasen, 1970a; Jacobsen, 1983; Andreasen and Vestergaard Pedersen, 1985; Andreasen et al., 1986; Andreasen et al., 1987) and case reports (Bruszt, 1958, 1967; Tainter, 1979; Kinirons, 1982; Perez et al., 1982; Spalding et al., 1985; Shapira et al., 1986; Tronstad et al., 1986; Mamber, 1994; Oulis et al., 1996; Çaliskan, 1998; Çaliskan et al., 1998) that describe a variety of management strategies derived from anecdotal clinical evidence and indirect evidence from animal studies (Turley et al., 1984; Turley et al., 1987). Outcome studies vary in the level of evidence and report conflicting results for spontaneous re-eruption and surgical repositioning (Jacobsen, 1983; Andreasen and Vestergaard Pedersen, 1985; Kinirons and Sutcliffe, 1991). There is variability and lack of detail in study design, control of variables, support for the mechanism of repositioning and variability with respect to statistical treatment of the data (Table 1). It is difficult to extend the results of any study beyond its experimental population due to this variability.

Similarly, the terminology used to describe management is inconsistent and some terms such as spontaneous re-eruption may lead to false expectations. In order to clarify the terminology used to describe management strategies described in the literature, this author proposes a simplified terminology for post-intrusion management since previous descriptions of intrusions do not reflect the biology of intrusions. For example, it is inappropriate to describe waiting for intruded teeth to reappear or approximate their original state as spontaneous re-eruption since as mentioned reeruption is unpredictable and not physiological. Passive repositioning is a more accurate alternative to both spontaneous re-eruption and passive re-eruption that does not imply the presence of a physiological process (eruption). Active repositioning more accurately describes
management strategies previously described as orthodontic repositioning and orthodontic reduction. Since there is little or no PL, the traction used to reposition an intruded incisor is not orthodontic in either its nature or in the forces utilized. Surgical treatment of intrusions is better described as immediate reduction (compression injury) or if delayed, reduction. For the purpose of this review, the terminology used by previous authors has been changed to this terminology.

Table 1  Summary of clinical outcome studies in the English language dental literature on permanent incisor intrusions

<table>
<thead>
<tr>
<th>Authors</th>
<th>Total n (Incisors) (Patients)</th>
<th>Intrusions (Incisors)</th>
<th>Age range (Years)</th>
<th>Prospective/Retrospective</th>
<th>Follow-up Period (Years)</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skeiller, 1960</td>
<td>107 60</td>
<td>14</td>
<td>5-18</td>
<td>Retro</td>
<td>1-6</td>
<td>Descriptive</td>
</tr>
<tr>
<td>Andreasen, 1970</td>
<td>189 108</td>
<td>23</td>
<td>0-80</td>
<td>Retro</td>
<td>1-12</td>
<td>Multivariate analysis</td>
</tr>
<tr>
<td>Jacobsen, 1980</td>
<td>40 40</td>
<td>40</td>
<td>6-16</td>
<td>Abstract</td>
<td>5</td>
<td>Descriptive</td>
</tr>
<tr>
<td>Andreasen &amp; Vestergaard Pedersen, 1985</td>
<td>637 400</td>
<td>61</td>
<td>*NR</td>
<td>Retro</td>
<td>up to 10</td>
<td>$\chi^2$, univariate survival analysis $\chi^2$, multivariate survival analysis</td>
</tr>
<tr>
<td>Andreasen et al., 1986</td>
<td>637 400</td>
<td>61</td>
<td>*NR</td>
<td>Retro</td>
<td>up to 10</td>
<td>$\chi^2$, univariate survival analysis $\chi^2$, multivariate survival analysis</td>
</tr>
<tr>
<td>Andreasen et al., 1987</td>
<td>637 400</td>
<td>61</td>
<td>*NR</td>
<td>Retro</td>
<td>up to 10</td>
<td>$\chi^2$, univariate survival analysis $\chi^2$, multivariate survival analysis</td>
</tr>
<tr>
<td>Crona-Larsson et al., 1991</td>
<td>171 108</td>
<td>9</td>
<td>6-19</td>
<td>Retro</td>
<td>$\geq$1</td>
<td>Descriptive Fisher’s exact</td>
</tr>
<tr>
<td>Kinirons and Sutcliffe, 1991</td>
<td>29 20</td>
<td>29</td>
<td>7-12 years</td>
<td>Prospective</td>
<td>2</td>
<td>Fisher’s exact</td>
</tr>
<tr>
<td>Eriksson, 1993</td>
<td>3804 2542</td>
<td>11</td>
<td>5-18 years</td>
<td>Retro</td>
<td>5</td>
<td>Fisher’s exact</td>
</tr>
</tbody>
</table>
1994 Clinical Protocol of Andreasen and Andreasen

The 1994 and subsequent revision of this protocol published in 1999 recommended treatment according to stage of root development. Andreasen and Andreasen (1994) recommended two treatment options for incisors with incomplete root development. Passive repositioning may occur or active repositioning may be carried out over a period of 3-4 weeks. Andreasen and Andreasen (1990) recognized passive repositioning as unpredictable in intruded incisors with complete root development and recommended early active repositioning for these teeth. Andreasen and Andreasen (1994) recommended that intruded incisors might need to be luxated prior to initiation of active repositioning. They also recommended that completely intruded incisors may need to be partially reduced at the time of injury in order to facilitate bonding of an orthodontic bracket. In 1994, luxation and/or partial repositioning were at the discretion of the clinician. The 1999 protocol remains the same with the exception that Andreasen et al. advocated initial reduction and/or partial repositioning. No new evidence is referenced for these 1999 changes, therefore in the following sections evidence for the 1994 protocol will be reviewed.

Passive repositioning

All evidence to support passive repositioning in the 1994 protocol is descriptive and based upon case reports (Bruszt, 1958, 1967; Shapira et al., 1986; Tronstad et al., 1986) and a single abstract (Jacobsen, 1983). Bruszt (1958) was the first to document a management protocol for intruded permanent incisors with incomplete root development. Based upon his observation of passive repositioning of 4 incisors intruded to the level of the interseptal bone and the 10 year survival of 3 of these incisors reduction was not recommended (Bruszt, 1967). Clinicians continue to wait for passive repositioning despite an unknown biological mechanism and report that the occurrence of repositioning is unpredictable regardless of the stage of root development (Bruszt,
1958, 1967; Tainter, 1979; Jacobsen, 1983; Heling, 1984; Spalding et al., 1985; Shapira et al., 1986; Tronstad et al., 1986; Jacobs, 1995). Oulis et al. (1996) recognized that clinical studies and case reports on management of intrusions have not identified variables that include severity of intrusion that might influence predictability of treatment. Further clinical studies are required to examine this variable.

Endodontic access can be impeded while waiting for passive repositioning, and predictable, time dependent sequelae that include abscess formation and IRR can develop. Single case reports describe incisors intruded to the level of the gingival margin can take more than two weeks to become repositioned. They may require a gingivectomy to uncover the crown in order to gain access for pulpectomy before the incisors approximate their pre-injury positions (Heling, 1984; Spalding et al., 1985; Shapira et al., 1986; Tronstad et al., 1986). If an incisor has not become repositioned by two weeks, the 1994 protocol recommended active repositioning rather than reduction (Andreasen and Andreasen, 1994).

**Immediate reduction**

The protocol of Andreasen and Andreasen (1994) did not recommend immediate reduction based on the results of two clinical studies (Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985). Immediate reduction was postulated to inflict further damage to the PL and contribute to the development of external RR and alveolar bone loss (Andreasen, 1970a). The incidence of external RR was reported as 47% and 66% respectively and for alveolar bone loss as 43% and 26% respectively (Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985). Unfortunately, heterogeneity of root development and failure to report the severity of intrusion limits the utility of these two retrospective outcome studies.
The Andreasen (1970a) sample included 27 maxillary and mandibular incisors, no classification of the severity of intrusions and a non-randomized methodology. Andreasen recognized these weaknesses and suggested further investigations to examine treatment variables and outcomes before advocating a change from the usual treatment procedures (at the time) of immediate reduction of intruded incisors. In spite of this assertion, Andreasen published a contradictory management protocol in 1972 that no longer advocated immediate reduction.

In a retrospective outcome study, Andreasen and Vestergaard Pedersen (1985) examined 61 intruded incisors for the presence of PN. Variables that included methods of treatment were examined for associations with the development of PN using univariate, multivariate and survival analyses. The frequency of external RR and alveolar bone loss were reported but not statistically analyzed. The findings were consistent with the findings of Andreasen (1970a). It is possible, if not likely that the population reported by Andreasen and Vestergaard Pedersen (1985) included incisors used in Andreasen (1970a). Therefore the finding of similar amounts of root resorption should not be surprising. However, the level of evidence is low since these data are descriptive.

The 1999 (Andreasen et al., 1999) and 1994 protocols (Andreasen and Andreasen, 1994) have remained unchanged since Andreasen (1972) and the findings of both Andreasen (1970a) and Andreasen and Vestergaard Pedersen (1985) continue to be cited in recent case studies (Mamber, 1994; Jacobs, 1995; Skockledge et al., 1995; Oulis et al., 1996) as the reason to avoid immediate reduction. It does appear contradictory that Andreasen et al. (1999) recommended luxation and/or partial reduction prior to either passive repositioning and/or active repositioning yet indicate that immediate reduction should be avoided. This author feels that each procedure is comparable.
**Active repositioning**

Andreasen and Andreasen (1994) recommended active repositioning over a period of 3-4 weeks. This approach was designed to enable access to perform a pulpectomy in the second to third week and was first described in Andreasen (1981a) and subsequently modified in Andreasen and Andreasen (1990). Appliances that reposition intruded incisors are compared with those used for extrusion of crown-fractured teeth (Andreasen and Andreasen, 1994). This association is inaccurate since crown fractured teeth have an intact or minimally damaged PL compared to the severely damaged periodontium of an intruded incisor. Outcome studies have not examined the relationship between active repositioning techniques and clinical outcomes such as the development of PN, PCO, external RR or tooth survival (Andreasen and Vestergaard Pedersen, 1985). Furthermore, the variables of active repositioning (removable vs fixed appliance, timing of the initiation of traction, traction forces) have not been identified nor have their associations with clinical outcomes.

Two animal studies provide some support for Andreasen's (1981a) recommendation of active repositioning. Turley et al. (1984) subjected all four first premolars of three young adult dogs to a non-standardized acute intrusive force that produced intrusions of variable amounts ranging from 0.5-4.0mm. Active appliances were placed on two premolars in each dog. The appliances were activated with 250 to 300 gm forces 5-6 days after intrusion. Twelve to 13 weeks following intrusion, the premolars were clinically and radiographically examined and prepared for histological analysis. The authors reported that the effectiveness of 'orthodontic forces' to reposition traumatically intruded premolars of young adult dogs was dependent upon the severity of the original injury. Premolar teeth intruded greater than 0.5 mm did not move with
active repositioning. This might represent a 0-3mm intrusion in a human. Radiographic and histological examination reported the presence of RRR.

Turley et al. (1984) proposed that ankylosis could be present from day 5 or 6 since there was no movement from the time of the application of the forces. They proposed an alternative explanation that the intruded tooth may be so tightly secured to bone that the orthodontic forces could not overcome the mechanical resistance. This would provide favorable conditions for the development of ankylosis. The evidence from Turley et al. (1984) support Andreasen’s (1981a) recommendation to partially reduce a severely intruded incisor in order to facilitate the placement of the appliance. However, Turley et al. (1984) were the first to recognize that the PL may be so severely damaged that it may be unable to produce the cellular responses necessary for orthodontic tooth movement.

Turley et al. (1987) reported that the amount of intrusion and the mobility of a tooth must be considered in the management of intrusions. In 1987, Turley et al. used an improved but non-standardized intrusion technique on all first premolars of three young adult dogs. Active repositioning with 300 gm forces was initiated 2-14 days after the injury. Intruded teeth with no mobility were luxated with a small elevator to a 2+ mobility before applying the orthodontic force. Final clinical, photographic and radiographic examinations were completed 12-13 weeks after intrusion and the teeth were evaluated histologically. Immobile premolars did not respond to active repositioning forces. Results of this study suggest that the effectiveness of active repositioning appears to be dependent on the mobility of the premolars immediately following injury.
Unfortunately, both animal studies used non-standardized techniques that produced unpredictable amounts of intrusion and there were few subjects in each study. Premolars of dogs do not compare favorably with the incisors of humans. The concepts developed by Turley et al. (1984, 1987) that consider the pathophysiology of the intruded tooth and the pathogenesis of ankylosis and then relate these principles to the ineffectiveness of active repositioning are noteworthy. Further animal and human outcome studies are required to support their hypotheses.

Anecdotal evidence that supports active repositioning appears in numerous single case reports (Tainter et al., 1979; Morley and Bellizzi, 1981; Perez et al., 1982; Hayes and MacPherson, 1984; Spalding et al., 1985; Mamber, 1994; Jacobs, 1995; Skockledge et al., 1995; Oulis et al., 1996). In these case reports, the protocols by Andreasen (1972) and by Andreasen and Andreasen (1990, 1994) are cited as the rationale for treatment. A total of 14 intruded incisors that were actively repositioned have been documented in isolated case reports. Three intruded incisors received immediate partial reduction prior to active repositioning (Perez et al., 1982; Hayes and MacPherson, 1984; Oulis et al., 1996). Four intruded incisors were repositioned by orthodontic techniques alone (Perez et al., 1982; Hayes and MacPherson, 1984; Mamber, 1994). The remaining 7 incisors required conventional orthodontic techniques due to the failure of passive repositioning to occur. The intervention took place at varying time intervals (5 - 12 weeks) after initial injury (Tainter, 1979; Spalding et al., 1985; Morley and Bellizzi, 1981; Jacobs, 1995; Skockledge et al., 1995). The use of active repositioning returned all fourteen intruded incisors to acceptable positions. However, outcomes such as PN, PCO, external RR, alveolar bone loss and tooth survival cannot be assessed since documentation of the severity of the injury, classification of root development, type and time of active repositioning and follow-
up is inconsistent and incompletely documented. This review of the literature supports Çaliskan (1998) who was the first to note that these case reports clearly demonstrate that active repositioning requires optimal patient cooperation and can involve increased treatment time, long retention periods and additional costs. These variables influence clinical outcomes and need to be considered prior to choosing this treatment option.

1997 Clinical Guidelines of the Royal College of Surgeons of England

The RCSE guidelines were the first to direct management according to severity of intrusion and stage of root development. The RCSE classified intrusions into three categories (mild <3mm; moderate 3-6mm and severe >6mm). There was no explanation or support for the choice of these three arbitrary categories. The average crown height of a central incisor is 10.5 mm and for a lateral incisor is 9 mm (Kraus et al., 1969). Kinirons and Sutcliffe (1991) classified intrusions as moderate (<5mm) or severe (>5mm). Categories chosen by Kinirons and Sutcliffe suggest incisors are intruded either less or more than half of the clinical crown height. The RCSE’s three categories suggest dividing the incisor into thirds in order to assess the severity of intrusion. There are no studies to support either classification. The question that remains unanswered is how much intrusion is necessary to destroy by compression, shearing and/or tearing, all of the PL fibers and associated cells necessary for healing. If an incisor was intruded into a rigid material then the distance would be as little as the width of the PL (approximately 0.21mm). However, alveolar bone is plastic, particularly in growing children and adolescents. The force of the intrusion, stage of root and periodontal development will have considerable influence on the amount of expansion of the alveolar bone prior to complete compression, shearing and tearing of the PL and cementum. It is certain that the physics of the pathophysiology of an intrusion are complex. However, well-designed clinical outcome studies will likely support this type of classification for intrusions.
The RCSE guidelines are consistent with the recommendations of Andreasen and Andreasen (1994) for incisors that have been minimally or moderately intruded. Passive repositioning may be anticipated in incisors with incomplete root development that are intruded <3mm (Bruszt, 1958, 1967; Shapira et al., 1986). Either passive repositioning or active repositioning is recommended for incisors with complete root development and intruded <3mm (Spalding et al., 1985; Shapira et al., 1986; Tronstad et al., 1986; Turley et al., 1984, 1987). Similarly, passive or active repositioning is recommended for incisors with incomplete root development that are intruded 3-6 mm (Bruszt 1958, 1967; Shapira et al., 1986; Tronstad et al., 1986; Turley et al., 1984, 1987). Active repositioning is recommended for incisors with complete root development and 3-6 mm of intrusion (Andreasen 1970a, Turley et al., 1984, 1987).

The RCSE guidelines recommend immediate reduction and splinting for incisors intruded >6mm regardless of root development. This is contrary to Andreasen and Andreasen (1994) who recommend active repositioning or at most immediate partial reduction of the completely intruded (>6mm) incisor. The outcome study of Kinirons and Sutcliffe (1991) provides evidence contrary to Andreasen's (1970a) original report that immediate reduction increased the risk of external RR and alveolar bone loss. Kinirons and Sutcliffe (1991) treated 29 intrusions in their longitudinal outcome study. Nineteen incisors were immediately reduced and 10 incisors were left for passive repositioning. The severity of intrusion was documented as moderate (<5mm) or severe (>5mm) and the extent of apical development was classified as open, parallel or closed. Radiographs taken at the time of injury and again at 1, 3, 6, 12 and 29 months post injury were examined for evidence of PN, loss of alveolar bone, ankylosis, external RR and tooth loss. Treatment methods for moderate and severe intrusions were examined but Fisher's exact tests failed to show a significant difference ($p=0.60$) between the two groups. Immediate reduction was
not associated with an increased or decreased occurrence of root resorption compared to incisors left for passive repositioning ($p=0.54$). Alveolar bone loss was significantly more common in incisors observed for passive repositioning ($p=0.03$). Tooth survival was reported more frequently in incisors that were immediately reduced. Those incisors that had a delay before treatment also had decreased survival.

This study differs from others since it was the first to quantify severity of intrusion and to quantitatively analyze by univariate analysis the relationship between treatment methods, severity of the intrusion, root development and clinical outcomes. This study is also the first to investigate the survival of intrusions. Unfortunately, the statistical results were descriptive and interactions between variables with outcomes could not be shown with these statistics. Finally, it is the first study to provide evidence to support immediate repositioning as a treatment option.

**Antibacterials**

The protocol by Andreasen and Andreasen (1994) and the RCSE guidelines (Kinirons, 1998) agree that the benefits of antibiotic treatment are unproven and their use is governed by clinical judgement and preference. Antibiotic coverage is prudent since intrusion injuries produce torn epithelial attachments that allow saliva-borne bacteria to pass to the PL space, root and alveolar bone surfaces. The drug of choice, its dosage and duration is determined empirically. Penicillin is usually preferred and is prescribed at a dosage if 25-50 mg/kg in three or four daily divided doses for seven days (Lee, 1996). In addition to antibiotics, Lee (1996) recommended an antibacterial mouthrinse, 0.1 per cent chlorhexidine gluconate rinse, for the duration of splinting.
Splinting

Guidelines for splinting intruded incisors are not offered by the Andreasen and Andreasen protocol or the RCSE guidelines (Kinirons, 1998). The type, method and duration of splint placement for intrusions is unknown since studies that examine splinting for luxated incisors have not included intrusions. PL repair may benefit from a flexible splint to allow movement while repair takes place (Oikarinen, 1990). The influence of this flexibility on the socket/root is unknown and may not be relevant for intrusions. When PL cells are crushed and cementum is compressed against alveolar bone, it is inevitable that ankylosis will result if the tooth remains impacted (Andreasen and Hjørting-Hansen, 1966). It has been demonstrated that ankylosis is produced after prolonged rigid splinting of replanted incisors following avulsion but this is not a comparable injury (Andreasen, 1975). For injuries that involve alveolar bone fractures, a 6-8 week period of rigid fixation has been recommended and ankylosis is expected (Andreasen, 1981a; Andreasen and Andreasen 1994).

If the treatment of intrusions involves splinting, a flexible splint is recommended for 2-3 weeks (Kinirons, 1998). However, a fully developed incisor that is intruded to or below the gingiva with maximal damage to the PL and fractured alveolar bone may benefit from immediate reduction followed by ankylosis for retention of the incisor if growth is complete since re-establishment of the normal PL is not expected. Under these circumstances, a rigid splint for 6-8 weeks would be recommended. This has not been addressed in the literature for intrusions. Flexible splints are used for incisors that are minimally intruded (<3mm) and have PL with potential for repair.
Endodontic Therapy

The RCSE guidelines are consistent with the protocol by Andreasen and Andreasen (1994) on endodontic management of intruded incisors. Endodontic protocols are based on the stage of root development of the incisor (Skeiller, 1960; Andreasen and Vestergaard Pedersen, 1985). The goal of treatment is to control infection and prevent IRR. Early root canal treatment for severely intruded (>6mm) incisors with complete root development is based on clinical outcome studies that have reported the development of PN in incisors with complete root development (Skeiller, 1960; Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985; Kinirons and Sutcliffe, 1991). In a retrospective outcome study, Andreasen and Vestergaard Pedersen (1985) investigated intruded incisors for PN. Variables were examined for associations with the development of PN using univariate, multivariate and survival analysis. A significant finding between complete root development and diagnosis of PN was found ($p=0.0002$). However, problems of the study include a sample that included both children and adults and the study did not examine the interaction between severity of intrusion with the diagnosis of PN. There was no examination of the possible interaction between variables. These variables are present at the time of injury and must be considered.

Other studies reported 96-100% of intruded incisors with complete root development developed PN (Skeiller, 1960; Andreasen, 1970a; Kinirons and Sutcliffe, 1991). The findings support Andreasen and Vestergaard Pedersen (1985) but these studies did not take into account the severity of intrusion and in some studies the stage of root development.
Repair or regeneration of the pulp in incisors with incomplete root development is minimal and unpredictable (Andreasen and Vestergaard Pedersen, 1985). Outcome studies report 62-78% PN in incisors with incomplete root development (Skeiller, 1960; Andreasen and Vestergaard Pedersen, 1985) and PN may be diagnosed as late as two years post injury (Andreasen and Vestergaard Pedersen, 1985). Andreasen et al. (1986) reported that the apical diameter of the intruded incisor is related to the diagnosis of. However, this has not been verified statistically.

The optimum time to initiate root canal treatment is within two weeks of the injury (Andreasen and Vestergaard Pedersen, 1985). Therefore, an incisor with complete root development must be sufficiently repositioned to permit root canal access and pulpectomy within two weeks. Treatment of PN involves pulpectomy; interim medication of the root canal (usually with calcium hydroxide) and final obturation of the root canal with gutta percha and sealer.

Calcium hydroxide is placed in the canal for 3-12 months or until IRR has become RRR (Andreasen and Andreasen, 1994; Kinirons, 1998). In cases where intruded incisors have incomplete root development, calcium hydroxide is used to facilitate periodontal healing and/or stimulate formation of an apical hard tissue barrier, against which permanent obturation of the root canal can be performed (Cvek, 1972).

**Follow-up**

Scheduling of follow-up visits depends upon the reduction or repositioning method and the time taken to return an intruded tooth to its original position. Kinirons and Sutcliffe (1991) recalled patients at one, three, six, twelve and twenty-four months post injury for clinical and radiographic evaluation of PN, alveolar bone loss, ankylosis, RR and tooth loss. This protocol, as
described previously, may not accurately reflect the expected chronology of PL healing. Since PN occurs as early as two weeks, follow-up should commence before 2 weeks after the injury. A suggested protocol includes a 1-2 week, 3-6 week, 3 month, 6 month and 1 year re-evaluation depending upon the repositioning technique chosen (Andreasen and Vestergaard Pedersen, 1985; Barrett, 1995).

Summary

Two sets of recommendations exist for the management of intrusions (Andreasen and Andreasen 1994; Kinirons, 1998). Only the RCSE guidelines categorize the severity of intrusion. The protocol by Andreasen and Andreasen (1994) and the RCSE guidelines (Kinirons, 1998) are comparable for management of incisors intruded <6mm. However recommendations differ for incisors intruded >6mm. Minimal evidence is available from outcome studies to support either recommendation. Further outcome studies are necessary to evaluate management protocols for superiority.

Prognosis: Healing and Healing Complications

Prediction of prognosis for intrusions is impossible with the current state of knowledge and the use of treatment guidelines. Healing of the pulp tissue and periodontium may include tissue regeneration (restoration of anatomy and function) or repair (restoration of tissue continuity with tissue that differs in anatomy and function) (Andreasen and Andreasen, 1994). A requirement for PL regeneration is the presence of a progenitor cell population, conditions that allow migration of progenitor cells into the wound (decompression) and a manageable level of foreign bodies and bacteria (Andreasen and Andreasen, 1994).
Complications include PCO, PN, external RR, bone loss and tooth loss. Relationships between outcomes and variables such as severity of the intrusion, root development and/or treatment methods have not reported sufficient data to predict clinical outcomes. Variations exist in the level of evidence and results (Andreasen and Andreasen, 1994; Kinirons, 1998).

**Animal studies**

Animal studies have attempted to study the chronology of healing and outcomes after luxation injuries but variations in experimental design do not permit comparison of the results (Turley et al., 1984; Turley et al., 1987; Cunha et al., 1995).

**Human studies**

Healing of the pulp and periodontium after intrusions in humans has not been consistently reported. Many complications are to be expected and have been reported by eight retrospective and one prospective clinical outcome study (Table 1). The most frequently reported complications include PN, external RR and loss of alveolar bone support. Pulp canal obliteration is reported less frequently. Frequencies should be viewed with caution and not compared since sample demographics and stages for root/apex formation are inconsistently defined. The study designs differ and the results are descriptive.

**Survival**

Research into conditions that influence survival generally involve either a comparison or a prognostic study. Comparison studies assess whether improved survival is associated with a
particular treatment regime. Prognostic studies attempt to determine whether patients with a greater chance of survival can be distinguished from those with a lesser chance on the basis of measurable features of the disease (Brown and Swanson, 1994).

There is no research that provides quantitative prognostic information on survival of intruded incisors. The few clinical studies of intrusions have focused on the associations between clinical variables and the development of healing complications. Only one study has reported an incidence of 20% tooth extraction of intrusions within the 2 year study period (Kinirons and Sutcliffe, 1991). Kinirons and Sutcliffe (1991) report a higher retention for incisors that are minimally intruded, those that were immediately reduced or those with complete root development. The small sample size of this study prevented multivariate analysis of all variables. Summary of median survival duration or mean survival time as well as analysis between variables and survival outcome without considering the staggered entry into the study or censored data is inappropriate (Brown and Swanson, 1994). Further studies are required to examine the interaction of the variables on survival using appropriate analysis.

Survival analysis consists of a body of statistical techniques that can be used to quantify survival in a systematic way and to compare survival in different groups of patients (comparison study), or to assess the relationship between survival and other measurable variables (prognostic study) (Brown and Swanson, 1994). Survival analysis techniques are ideal for studies that investigate relatively rare conditions that need to accommodate the staggered entry of patients into a trial over time. The techniques are only useful if 1) there is an identifiable starting point 2) there is a dichotomous and well-defined outcome 3) patients lost to follow-up are not related to outcome and 4) there is no change in study design over time (Brown and Swanson, 1994). The goal of
survival analysis is to detect clinically worthwhile differences in survival experiences or to
construct a prognostic index that would contribute to a clinician’s judgement of survival
prospects. Survival analysis has not been reported in the literature on intrusions of children and
adolescents.

Dental Pulp

Pulp canal obliteration

Hard tissue apposition along root canal walls is a physiologic process that can be accelerated
following dental trauma. The actual mechanism of PCO remains unclear but radiographs are used
to confirm diagnosis. Incisors with PCO demonstrate partial or complete obliteration of the pulp
canal. Andreasen et al. (1986) report that PCO is first diagnosed approximately 4-6 months
following trauma. Total obliteration was reported between 2 and 5 years post trauma. Color is
not a significant diagnostic variable for PCO (Holcomb and Gregory, 1967; Jacobsen and
Kerkes, 1977; Andreasen et al., 1987; Robertson et al., 1996). However, the color of the crown of
an affected incisor may be darker than crowns of adjacent untraumatized teeth. The decreased
translucency is due to the greater thickness of dentine that produces a darker yellow color.

Incisors with PCO have a vital pulp but can have either a positive or negative response to vitality
tests depending upon the amount of canal obliteration. Pulpal necrosis can develop subsequently
to PCO and lead to a negative response to vitality tests. Another objective sign, such as
radiographic periapical radiolucencies, is necessary for confirmation of PN prior to the decision
to proceed with endodontic treatment. Clinical studies that used survival analysis confirm that
secondary PN is rare and occurs later than 5 years after trauma (Holcomb and Gregory, 1967;
Jacobsen and Kerekes, 1977; Andreasen et al. 1986; Robertson et al. 1996). Based on these results endodontic intervention is recommended only after diagnosis of PN.

Pulp canal obliteration has been reported to occur in 0-27% of intruded teeth with incomplete apex development at the time of injury (Andreasen, 1970a; Jacobsen, 1983; Andreasen and Vestergaard Pedersen 1985; Andreasen et al. 1987; Crona-Larsson et al., 1991). Pulp canal obliteration can occur in teeth with complete root development but the literature has not described the development of PCO in mature incisors. Studies report only descriptive data for PCO and vary in sample size and experimental design. Further studies are required to assess if the stage of root and/or apical development, severity of intrusion or treatment method influence the development of PCO.

Pulpal necrosis

Pulpal necrosis can occur as a result of reduced blood flow, inflammation or bacterial invasion of the pulpal tissue. Intrusion causes compression, crushing and tearing of the vasculature. Concomitant crown fractures and the disrupted dentogingival complex create direct routes for passage of bacteria into the dental pulp. It is expected that these features of an intrusion predispose pulpal tissue to the diagnosis of PN. Studies of intrusions have not examined the interaction between crown fracture, severity of the intrusion and PN.

The chronology and diagnosis of PN after an intrusion has been derived from clinical studies. Pulpal necrosis may occur weeks to months after intrusion. Pulpal necrosis is generally diagnosed within three (Andreasen and Vestergaard Pedersen, 1985) to four months of an intrusion (Jacobsen, 1980) but may be diagnosed as late as two years later (Andreasen and Vestergaard
Pedersen, 1985). Clinical diagnosis of PN depends upon the absence of vitality using indirect neural electrical or thermal tests and at least one other objective sign (Andreasen and Vestergaard Pedersen, 1985). Objective signs include grey color changes to the crown, tenderness to percussion, presence of a fistula, mobility, periapical radiolucency or radiographic appearance of external RR (Andreasen and Vestergaard Pedersen, 1985). The utility of neural conduction for vitality testing is questionable because of the potential for false negative results (Skeiller, 1960; Jacobsen, 1980). The crown may exhibit a pink hue within 1-8 days that will change to shades of grey after 6 days to 3 weeks (Jacobsen, 1980). The radiographic signs of PN including the presence of external RR (67-82%) (Jacobsen, 1980; Andreasen and Vestergaard Pedersen, 1985) and/or an apical radiolucency (67%) (Andreasen and Vestergaard Pedersen, 1985) are not apparent during the first three weeks following injury (Jacobsen, 1980; Andreasen and Vestergaard Pedersen, 1985). The chronology of the diagnosis of PN and associated clinical and radiographic symptoms are likely related more to the recall interval chosen by the investigators rather than progression of PN.

Pulpal necrosis has been reported more frequently than other outcomes of intruded incisors. Andreasen and Vestergaard Pedersen (1985) provide the only study that reported significant results relating the diagnosis of PN with complete root development. The results were subjected to statistical analysis and the relationship between intruded incisors with complete root development and PN was significant ($p=0.0002$). This study supports the findings by Skeiller (1960) who was the first to report 100 % PN in intruded incisors with complete root development but these results were empirically based from 4 incisors.
Andreasen and Vestergaard Pedersen (1985) reported that intruded incisors with incomplete root development demonstrated a lower incidence of PN than incisors with complete root formation. Twenty-four incisors had incomplete apex development. Fifteen of these incisors developed PN (62%). These results were not subjected to statistical analysis. Other studies also reported fewer cases of PN in incisors with incomplete root development compared with incisors with complete root development but also failed to provide statistical analysis (Skeiller, 1960; Jacobsen, 1983). Four other clinical studies of intruded permanent incisors reported the prevalence of PN to be 70-97% after intrusions (Andreasen, 1970a; Jacobsen, 1980, Kinirons and Sutcliffe, 1991). Variables that were not analyzed in the studies include stage of root development, severity of the injury or treatment method.

Andreasen (1970a) and Andreasen et al. (1986) speculated that if the size of the apical foramen was adequate to permit vascular in-growth and if infection or inflammation could be controlled, gradual revascularization and re-innervation of pulp tissue might occur. Andreasen (1988) histologically and bacteriologically examined extirpated pulp tissue from luxated incisor injuries to determine if the pulp tissue contained the cellular elements required for repair. The study was inconclusive regarding the potential for repair but found a significant relationship between total autolysis of pulp tissue at the time of extirpation and intrusions. It was found that longer observation periods were associated with total autolysis at the time of pulp extirpation.

**Summary**

An intrusion is a severe luxation injury. The pathophysiology of this injury predisposes the pulpal tissue to the diagnosis of PN. It has been concluded that PN is an expected outcome and that regeneration of pulpal tissue in incisors with complete root development does not occur. Re-
establishment of pulpal continuity in incisors with incomplete root development is rare and unpredictable (Andreasen and Vestergaard-Pedersen, 1985).

Periodontium

External root resorption

Root resorption is a common healing complication following intrusions. Information about the etiology and pathogenesis of external RR following an intrusion has been extrapolated from experimental and avulsion studies and critically reviewed Barrett and Kenny (1997). Within the root canal the dentin is lined with predentin and odontoblasts and on the root surface with precementum and cementoblasts (Tronstad, 1988). If the predentin or precementum is damaged during compression and fracture against the alveolar bone of the socket multinucleated osteoclasts may colonize the root surfaces and initiate external RR. Andreasen and Hjorting-Hansen (1966) were the first to classify external RR according to its histological and radiographic appearance. Inflammatory root resorption and RRR are classified as invasive or progressive RR (Tronstad, 1988; Barrett and Kenny, 1997). The slower RRR consists of ankylosis and replacement of the root by alveolar bone. Following intrusion, external RR (both types combined) has been reported and cited to be between 28% and 66% (Skeiller, 1960; Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985; Crona-Larsson et al., 1991; Kinirons and Sutcliffe, 1991; Eriksson, 1993). These frequencies are based on 5 outcome studies that have variability in design and statistical treatment of the data. The incidence of external RR is subjective due to variability in recall schedules and length of study periods. Each type of resorption needs to be studied and frequencies cannot be compared until standardized protocols have been utilized.
Inflammatory root resorption

The incidence of IRR has been reported descriptively in four clinical outcome studies. The frequency of IRR is 22-57% after intrusion of an incisor based on these four studies. The mechanism for the development of IRR was first proposed by Andreasen and Hjørting-Hansen (1966) and later by Andreasen (1981b). Andreasen and Hjørting-Hansen (1966) proposed that IRR is initiated with the resorption of cementum to such an extent that the underlying dentin is exposed. If the dentinal tubules communicate with an infected necrotic pulp then it is possible that toxic substances may pass into the periodontal space and initiate an inflammatory reaction that would lead to rapid resorption of the root of the tooth. The theory did not explain observations that included the occurrence of IRR in the absence of bacteria. Secondly, in some cases the completion of root canal therapy did not arrest the IRR (Andreasen and Hjørting-Hansen, 1966).

Andreasen (1981b) studied the relationship between the exposure of dentinal tubules and the contents of the root canal with IRR in replanted vervet monkey teeth under controlled conditions. Inflammatory root resorption occurred more often on the convex surfaces of the root (p = .04, Fisher’s exact test). Pulp tissue adjacent to areas of IRR was always found to be necrotic or to contain leukocyte infiltrate. Inflammatory replacement resorption is first observed two weeks after replantation but Andreasen (1981b) reported that at this early stage bacteria were not identified in the dentinal tubules. Thus the initiation of IRR may not require the presence of infected dentin. If an infection that originates in the pulp can cause IRR then it may be possible for an infection in the PL to initiate the process as well. The issue has not been addressed in the literature (Barrett, 1995). Damage to the PL or a cementum-denuded area of the root surface of an intruded incisor appears to initiate IRR. Inflammatory root resorption may be prolonged by
mechanical irritation of the root, ongoing compression of the tissue or infection from the dentin and pulpal tissue (Andreasen and Andreasen, 1992). Continuous stimulation of the osteoclasts by bacterial products and necrotic debris from an infected root canal or PL may excentuate the process (Andreasen, 1981b).

IRR can be recognized radiographically as periradicular bowl-shaped radiolucent defects that penetrate dentin and encompass the root and adjacent alveolar bone after three to four weeks (Andreasen and Hjørting-Hansen, 1966). Radiographically IRR can be visualized only on the proximal surfaces (mesial and distal) of affected teeth. It has been reported that IRR is initiated on the convex buccal and lingual areas of the root within the first two weeks due to the damage of the cementum sustained by the root on extraction (Andreasen, 1981b). These surfaces are not apparent on periapical radiographs, therefore when IRR is diagnosed radiographically it is late in the process.

*Replacement root resorption*

The incidence of RRR following an intrusion has been reported to be between 3-24% in the same four studies that reported IRR following an intrusion. The studies did not consider the etiology or pathogenesis of the RRR. Information regarding the pathogenesis of RRR following intrusions is derived from animal, experimental and replantation studies.

Extensive damage to the PL establishes a competitive wound healing process between the root PL and socket wall PL (Andreasen and Andreasen, 1992; Løe and Waerhaug, 1961 and Andreasen and Kristerson, 1981). When the PL fails to regenerate after an injury, osteogenic cells repopulate the PL space and dentoalveolar ankylosis results (Hammarström et al., 1989). Replacement root
resorption follows whereby the root becomes incorporated into the adjacent bone through remodeling. Physical attachment of the root to alveolar bone in a child with incomplete skeletal growth leads to progressive infraocclusion of the incisor (Andreasen and Hjørting-Hansen, 1966). Andersson et al. (1989, 1990) reported RRR progresses more rapidly in a growing individual. In most instances, it takes years, sometimes decades before a root is fully resorbed (Andersson et al., 1984). The ultimate outcome of RRR is destruction of the root.

Clinically, ankylosis is diagnosed by a lack of tooth mobility, cessation of eruption and a characteristic sound when percussed (Andersson et al., 1984). In the pre-adolescent, ankylosis may lead to infraocclusion and distortion of the gingival margin of the affected tooth secondary to growth of the adjacent alveolus (Andreasen and Andreasen, 1992; Andreasen and Hjørting-Hansen, 1966). Andersson et al. (1984) reported that correct diagnosis is only possible by mobility and percussion testing if 10% and 20% or more, respectively, of the incisor root is ankylosed. Despite this problem, Andersson et al. (1984) reported that mobility and percussion tests are more accurate for diagnosis than radiographic signs of ankylosis. Radiographic diagnosis of dentoalveolar ankylosis or RRR is complicated by the fact that radiographs are two dimensional representations of a three dimensional system. Radiographically, ankylosis presents as absence of PL space or as a moth-eaten appearance of the root due to replacement with bone (Andreasen and Hjørting Hansen, 1966). As a result, visualization of the affected areas is possible only if they occur on the mesial and distal radiographic margins of the root. Failure to identify areas of RRR include overlapping structures and bone marrow spaces that were interpreted as normal periodontal spaces. Diagnosis of ankylosis and RRR must always use combined clinical and radiographic measures (Andersson et al., 1984).
Alveolar bone loss

Andreasen (1970a) reported that alveolar bone loss was to be expected due to the forceful impact of the incisor into the alveolar bone. Later interpretations of his 1970 results relate alveolar bone loss to the method of repositioning (Andreasen, 1972). Variables surrounding bone loss have not been considered. The literature for intrusions has not precisely defined the measures for alveolar bone loss.

Summary

Healing complications of the periodontium that include IRR, ankylosis and RRR, bone loss and tooth extraction can be expected but their etiology, pathogenesis and frequencies after intrusions in children and adolescents needs to be examined in further experimental, animal and human clinical studies.
Statement of the Problem

Intrusions of the permanent maxillary incisors of children are a rare and complex injury. Severe damage is sustained by the dental pulp and periodontium. The uncertainty pertaining to management may affect outcomes of this injury. The literature focuses on descriptions of clinical outcomes and has not considered analysis of variables and outcomes. Results of the clinical studies are difficult to interpret due to the lack of standardization of study design, lack of consistent clinical classification of the injury and studies using statistical analysis are mainly descriptive. Predicting prognosis will remain impossible until there is proper design of clinical studies and statistical analysis of outcomes.

There are no data in the literature that pertain to the survival of intruded incisors and the variables that influence survival. These data would allow clinicians to provide parents with quantitative information to guide them in the decision-making process.

Hypotheses

1. Permanent maxillary incisors intruded >6mm will exhibit a lower 5 year survival compared with similar incisors that have been intruded between 3-6mm and those intruded <3mm.

2. Injury-related variables (severity of intrusion, stage of root development and crown fracture) and treatment variables (passive or active repositioning, or immediate reduction) will be significantly related to the survival of intruded permanent maxillary incisors.
3. Injury-related variables (severity of intrusion, stage of root development and crown fracture) and treatment variables (passive or active repositioning, or immediate reduction) will be significantly related to the diagnosis of PN of intruded permanent maxillary incisors.

4. Pulp canal obliteration will be significantly related to the stage of root development and the severity of intrusion.

5. The presence of IRR and RRR is significantly related to the injury-related variables that include severity of intrusion, stage of root development and crown fracture and treatment variables (passive or active repositioning, or immediate reduction).

6. Alveolar bone loss will be significantly related to severity of intrusion and treatment method.
Objectives

1. To quantitatively describe the survival of intruded permanent maxillary incisors and their pulpal vitality in children and adolescents treated at the Hospital for Sick Children between June 1988 and June 1998.

2. To determine which of the following variables influence the development of a range of common healing outcomes (PCO, PN, IRR, RR, bone loss and tooth extraction) in intruded permanent maxillary incisors.

<table>
<thead>
<tr>
<th>Injury-related variables</th>
<th>Treatment variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount of intrusion</td>
<td>Passive repositioning</td>
</tr>
<tr>
<td>Apical development</td>
<td>Active repositioning</td>
</tr>
<tr>
<td>Crown fracture</td>
<td>Immediate reduction</td>
</tr>
</tbody>
</table>
MATERIALS and METHODS

The entire sample consisted of 47 intruded permanent maxillary incisors (35 central and 12 lateral incisors) from 36 patients (26 males and 10 females) with a mean age of 9.5 years (range 5.5-17.8 years). Patients presented to the dental department at The Hospital for Sick Children, Toronto between June 1988 and June 1998 and the intruded incisors were treated following an established protocol.

Protocol-based clinical management for dentoalveolar trauma was introduced at The Hospital for Sick Children June 1988 and was derived from and based upon protocol that was consistent with the recommendations of Andreasen (1981a). Standard data-collection forms (Appendix I) were developed in order to obtain a database of sufficient size and integrity to allow the application of statistical methods.

Candidates eligible for this investigation were identified by defining search parameters that matched the inclusion criteria for the study. Intrusion injuries must have been classified according to Andreasen’s classification system (Andreasen, 1972) and treated by the dental clinic’s protocol (Appendix II). Only vital permanent maxillary incisors of healthy children and adolescents were selected for study. The limitations placed on the dates of entry coincided with the introduction of the treatment protocols and a closing date that allowed a minimum recall period of six months. Incisors extracted prior to six months were included in the study. The resulting list was refined after reviewing the dental chart of each patient to determine if there were any reasons for exclusion. A patient was excluded from the study if he/she was not in good general health and/or if there was a pre-existing medical condition that would affect healing (ie.
chronic systemic disease, diabetes, haematological disorder, chronic steroid use). Patients were also excluded if the intruded incisor had experienced previous trauma or had associated root fractures or fractures that involved multiple teeth, alveolar bone, alveolar process or the maxilla.

**Emergency, follow-up treatment and patient recall protocol**

Protocols for the emergency visit, follow-up examination as well as patient recall examinations have been described in previous studies of avulsions (Barrett, 1995) and extrusions (Lee, 1996) of the permanent maxillary incisors of children and adolescents. The emergency treatment protocol specific to intrusions (Appendix II) differs from other luxation protocols in that there are three management options for repositioning the intruded incisor depending upon the amount of intrusion and stage of root development compared to one treatment option for lateral luxations, extrusions, avulsions. The three options include passive repositioning, immediate reduction and/or active repositioning. If the intruded incisor was immediately reduced to its original position, a splint was placed using the technique previously described by Lee (1996). Treatment of the pulp was recommended within 7-10 days of the injury for severely intruded incisors with complete apex development (Andreasen and Vestergaard Pedersen, 1985).

Patients were appointed within two weeks of the emergency visit to assess the progression of passive repositioning as well as healing of the pulp and periodontium. Parents were given the option of returning to their private dentist or continuing at the hospital. If the patient chose to return to his/her private dentist, a letter that outlined the treatment rendered and duplicate radiographs were forwarded to the dentist. The timing of follow-up examinations for patients who chose to continue at the hospital followed a timetable specific to intrusion injuries. The
recommended timing of follow-up visits corresponded to intervals outlined in the protocol (Appendix II).

The healing of the pulp and periodontium was assessed by clinical and radiographic examinations at each follow-up visit. Data collected at the clinical examination included tooth color, mobility, periodontal sulcus depths, percussion sensitivity and tone, presence or absence of fistula and tooth vitality. Radiographs taken at follow-up were viewed for the presence or absence of periapical radiolucencies, IRR, RRR, PCO, and alveolar bone loss. The techniques for collecting clinical and radiographic data have been previously described by Barrett (1995) and Lee (1996). The diagnosis for tooth survival, presence of PN, PCO, periodontal status (healthy vs loss of alveolar bone), IRR and RRR was based on clinical and radiographic data collected at each recall examination.

Pulpal necrosis was diagnosed based on the absence of pulpal sensibility and at least one other clinical or radiographic sign (ie. grey color changes in the crown, periapical radiolucency or tenderness to percussion) (Jacobsen, 1980; Andreasen and Vestergaard Pedersen, 1985). Once PN was diagnosed endodontic therapy was initiated. If root development was incomplete, a calcium hydroxide paste (pulpdent, Pulpdent Corporation, U.S.A) was placed in the root canal with a spiral paste-filling instrument. Periapical radiographs were obtained after placement of calcium hydroxide paste to ensure satisfactory obturation. Calcium hydroxide was left in the teeth until apical constriction was complete and replaced if follow-up radiographs revealed apical loss of calcium hydroxide. If further signs of infection were present at follow-up examination, irrigation of the root canal, re-medication with calcium hydroxide and a course of antibiotics was prescribed. Calcium hydroxide was also placed in incisors with mature apices if there was IRR.
Final root canal obturation with gutta percha and sealer was completed only after the injured teeth showed no progressive signs of IRR and apical closure was present.

**Patient contact algorithm and recall protocol**

Patients were contacted following an algorithm adapted from Lee (1996) (Appendix V). Those who agreed to a recall examination at The Hospital for Sick Children had standard clinical, radiographic and photographic examinations performed by the same author (JH) after a review of the child's medical history. All data were documented on a follow-up data collection form adapted from Lee (1996) (Appendix IV).

**Radiographic Assessment**

Radiographs were used to evaluate the stage of apex and root development at the time of injury, to measure the quantity and type of external RR and to diagnose PCO. Radiographic assessment was used in addition to clinical assessment to diagnose PN and alveolar bone loss. The reliable interpretation of dental radiographs may be affected by a variety of biases including quality of the radiographs, the environment in which the interpretation occurs, education, training and experience of the examiner and multiple examiners (Valachovic et al., 1986). The subjective nature of radiographic assessment can lead to variation between individuals when assessing the same radiographs. Therefore, two calibrated paediatric dentists evaluated all radiographs.

Apex and root development at the time of trauma was scored according to the stages defined by Moorrees, Fanning and Hunt (1963). Root resorption was scored using the root resorption index developed by Andersson et al. (1984). These indices have been previously illustrated and described by Barrett (1995) and Lee (1996).
The two examiners were trained for assessment of the stage of root development and root resorption indices. In addition, the training session provided the examiners information necessary to provide radiographic evaluation of the other outcomes including PN, PCO, alveolar bone loss. The examiners were asked to apply all criteria to a series of radiographs drawn from a different sample representative of all possible outcomes for the present study.

Radiographs from each patient were mounted in series with an identification number and were kept in separate envelopes with the same number to prevent excessive handling. Radiographs were viewed and rated independently by the two examiners in rooms equipped with a lighted view-box with the overhead lights off. The assistant verified that the radiographs were placed correctly under a black screen on the view-box for the examiners following a random order and indicated the incisors to be evaluated using the FDI tooth code (ie. 2.1 or 1.1). The first radiograph in the series was either a periapical or an occlusal film taken at the time of the injury. It was used to score the stage of root development and to serve as a baseline reference for scoring external RR. The next films were the periapical and occlusal views obtained at the follow-up visit. Examiners were asked to use both films to identify healing complications and to score root resorption if necessary. Each examiner had a standardized radiographic scoring sheet (Appendix V) for evaluations of each intruded incisor.

A random sample of 12 of the same subjects was drawn for each examiner to score. The second sessions were conducted seven days after the first session under identical conditions.
All data were transferred to a spreadsheet program (Microsoft Excel) for analysis. The variance in amount of intrusion was made discrete by classifying the intrusions according to the scale recommended by the RCSE guidelines (Kinirons, 1998). This was possible since the amount of intrusion was originally recorded in millimeters on the standardized data collection form.

**Statistical methods**

The entire sample, available sample and unavailable sample were assessed using descriptive statistics determining the mean, median, range and standard deviation for age, amount of intrusion and time to follow-up. Student's t-tests were used to determine if there were significant differences between the samples. A critical $p$-value of 0.05 was used for all statistical tests in the study.

Kappa statistics (Fleiss and Chilton, 1983) were calculated to determine diagnostic consistency between the two examiners for their radiographic assessment of stage of apical development, IRR (present or absent), RRR (present or absent) and PCO (present or absent). Kappa values were interpreted according to the Kappa score interpretation described by Landis and Koch (1977) ( Appendix VII).

Because some patients may have had more than one intruded incisor repositioned, individual observations were not independent. Therefore, in addition to doing analysis using individual teeth, the data were sorted into best and worst case scenarios so each patient was represented by a single incisor. Best and worst cases were determined objectively using an algorithm similar to the one used by Barrett (1995) ( Appendix VIII).
Logistic regression analysis was used to study associations between clinical outcomes (PCO, IRR, RR, BL) and injury-related variables (crown fracture, severity of intrusion, stage of root development). Proportional hazards regression (Cox, 1972) was performed to determine if any injury-related variable (crown fracture, stage of root development, severity of intrusion) or treatment variable was significantly related to incisor survival or the diagnosis of PN. Teeth were censored if they had not been diagnosed with PN at the close of the study. Kaplan-Meier survival analysis was generated for the significant variables. All data were analyzed using JMP 3.2 (SAS Institute, Cary, N.C., USA).
RESULTS

An entire sample of 36 patients (26 males, 10 females) that represent 47 intruded permanent maxillary incisors (35 centrals, 12 laterals) that met inclusion criteria was identified from the database. The mean patient age at the time of trauma was 9.5 years (range 5.5-17.8). The available sample consisted of 26 patients (19 males, 7 females) with 31 intruded permanent maxillary incisors (22 centrals, 9 laterals) and a mean patient age of 9.3 (range 5.5-17.8). Ten patients were unavailable for follow-up after completing the algorithm for patient contact (Appendix III).

Demographic data at the time of the injury includes age, sex, tooth involved and severity of the intrusion. The available sample was compared to the unavailable sample (Table 2).

Table 2 Comparison of available and unavailable samples

<table>
<thead>
<tr>
<th>Demographic Information</th>
<th>Available sample</th>
<th>Unavailable sample</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>26</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Ratio (M:F)</td>
<td>2.7:1</td>
<td>2.3:1</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>9.3</td>
<td>9.7</td>
<td></td>
</tr>
<tr>
<td>range</td>
<td>5.5-17.8</td>
<td>6.6-14.4</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>2.7</td>
<td>2.6</td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td></td>
<td></td>
<td>0.50 (NS)</td>
</tr>
<tr>
<td>Severity of intrusion (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>5.5</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>range</td>
<td>0.5-12.0</td>
<td>0.5-7</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>3.3</td>
<td>1.86</td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td></td>
<td></td>
<td>0.002</td>
</tr>
</tbody>
</table>

*Student's t-test with p=0.05
Within the available sample, twenty-six incisors survived and five failed prior to follow-up appointment. Incisors either failed due to ankylosis or IRR. A comparison of these two groups is presented in Table 3.

Table 3 Comparison of survived and failed samples

<table>
<thead>
<tr>
<th>Demographic Information</th>
<th>Survived</th>
<th>Failed</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of incisors</td>
<td>26</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>9.1</td>
<td>10.8</td>
<td>p=0.25 (NS)</td>
</tr>
<tr>
<td>range</td>
<td>5.5-17.8</td>
<td>9.2-12.3</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>2.9</td>
<td>1.4</td>
<td></td>
</tr>
<tr>
<td>Severity of intrusion (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mean</td>
<td>5.3</td>
<td>6.6</td>
<td>p=0.42 (NS)</td>
</tr>
<tr>
<td>range</td>
<td>0.5-12</td>
<td>4.0-9.0</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>3.5</td>
<td>2.0</td>
<td></td>
</tr>
</tbody>
</table>

*Student's t-test with p=0.05

Clinical and radiographic data

Data collected at the emergency visit included stage of root development, severity of the intrusion, crown fracture and treatment method (Appendix VI). Data collected at follow-up appointments for the available sample were the basis for diagnosis of outcomes that included, PCO, IRR, RRR, alveolar bone loss and incisor survival. The time to failure was the day of extraction, documented in the subjects health record. The time to diagnosis of PN was determined by diagnosis made during treatment visits for repositioning, at recall examinations or due to reports of pain. Whereas the date of extraction was exact, the diagnosis of PN was a maximal value that was dependent upon time from the previous appointment (1-8 mos.)
A summary of the frequency of the variables and outcomes is presented in Table 4 and 5 respectively.

Table 4 Summary of the frequency of variables in the available sample (N=31)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCSE Classification (Severity of intrusion)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;3mm</td>
<td>8</td>
<td>26</td>
</tr>
<tr>
<td>3-6mm</td>
<td>12</td>
<td>39</td>
</tr>
<tr>
<td>&gt;6mm</td>
<td>11</td>
<td>35</td>
</tr>
<tr>
<td>Stage of root development (Moorrees, Fanning and Hunt, 1963)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mature apex</td>
<td>14</td>
<td>45</td>
</tr>
<tr>
<td>Immature apex</td>
<td>17</td>
<td>55</td>
</tr>
<tr>
<td>Type of crown fracture</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>17</td>
<td>55</td>
</tr>
<tr>
<td>Enamel only</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>Enamel and dentin</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>Enamel, dentin and pulp</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Treatment method</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Passive repositioning</td>
<td>11</td>
<td>35</td>
</tr>
<tr>
<td>Active repositioning</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>Immediate reduction</td>
<td>14</td>
<td>45</td>
</tr>
</tbody>
</table>

Table 5 Summary of outcomes for the available sample (N=31)

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Survived incisors</td>
<td>26</td>
<td>84</td>
</tr>
<tr>
<td>Failed incisors</td>
<td>5</td>
<td>16</td>
</tr>
<tr>
<td>Pulpal necrosis</td>
<td>14</td>
<td>45</td>
</tr>
<tr>
<td>Pulp canal obliteration</td>
<td>8</td>
<td>26</td>
</tr>
<tr>
<td>External root resorption</td>
<td>25</td>
<td>81</td>
</tr>
<tr>
<td>Inflammatory root resorption</td>
<td>24</td>
<td>77</td>
</tr>
<tr>
<td>Replacement root resorption</td>
<td>7</td>
<td>23</td>
</tr>
<tr>
<td>Alveolar bone loss</td>
<td>12</td>
<td>39</td>
</tr>
</tbody>
</table>
Radiographic Analysis

Kappa scores were calculated to determine inter-examiner diagnostic consistency of radiographic assessment for apical development, external RR, and PCO. All values are in the range of moderate to substantial agreement (K = between 0.50 and 0.80) (Landis and Koch, 1977; Appendix VII). A summary of the Kappa values and 95% confidence intervals is presented in Table 6.

Table 6 Summary of Kappa values and confidence intervals for inter-examiner reliability for the radiographic assessment of root development, external root resorption and pulp canal obliteration

<table>
<thead>
<tr>
<th></th>
<th>Root Development</th>
<th>External RR</th>
<th>PCO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kappa</td>
<td>0.76</td>
<td>0.65</td>
<td>0.56</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.65-0.87</td>
<td>0.33-0.97</td>
<td>0.41-0.65</td>
</tr>
</tbody>
</table>

Statistical Analysis

Proportional Hazards Regression/Kaplan-Meier Survival Analysis

Two outcomes were identified in the data, failure of an intruded incisor and the diagnosis of PN. Proportional hazards regression was performed to determine if an injury-related or treatment variable was significantly related to these outcomes. Kaplan-Meier survival curves were plotted for significant variables identified in the proportional hazards regression. Log-rank statistics were used to determine if the curves were significantly different from each other.
Prior to analysis the entire sample was subdivided into best and worst case samples to eliminate bias from possible dependence of incisors in the same patient following an algorithm adapted from Barrett (1995) (Appendix VIII). The results showed that the best case did not differ significantly from the worst case (best=worst case sample). If the patient had two intruded incisors both either failed or survived. Proportional hazards regression for incisor survival and diagnosis of PN was performed for both the entire sample and the best=worst case sample. Kaplan-Meier survival curves for each outcome were compared and were not significantly different from each other. Therefore, further testing involved only the best=worst case sample to ensure minimal bias.

*Incisor Survival*

Eighty-four percent (26/31) of the available incisors were present at follow-up. Figure 1 shows the Kaplan-Meier survival curve for the entire sample. The results of the proportional hazards regression for the best=worst case sample is presented in Table 7. The very large relative risk for each variable in the model may be explained clinically, but the confidence intervals for the risk ratios calculated by the proportional hazards regression analysis were too wide to conclude that they could not have occurred by chance, based on this sample size.
Fig. 1 Kaplan Meier survival curve for incisor survival of the available sample

Table 7 Proportional hazards regression for incisor survival; best=worst case sample

<table>
<thead>
<tr>
<th>Variable</th>
<th>Multivariate Analysis</th>
<th>Relative risk</th>
<th>Risk ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$p$-value</td>
<td>Injury-related and treatment variables</td>
<td></td>
</tr>
<tr>
<td>RCSE Classification</td>
<td>0.008</td>
<td>RCSE Classification $&gt;$6mm vs $&lt;$3mm</td>
<td>Approaching $\infty$</td>
</tr>
<tr>
<td>Type of Crown Fracture</td>
<td>0.10 (NS)</td>
<td>Crown fracture involving enamel/dentin/pulp vs none</td>
<td>1667</td>
</tr>
<tr>
<td>Treatment method</td>
<td>0.10 (NS)</td>
<td>Active vs passive repositioning</td>
<td>30534</td>
</tr>
<tr>
<td>Root development</td>
<td>1.0 (NS)</td>
<td>Immature vs mature root development</td>
<td>1123</td>
</tr>
<tr>
<td>Model</td>
<td>0.05</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Critical $p$-value = 0.05
Kaplan-Meier survival curves for the RCSE classification were plotted and compared (Figure 2). The log-rank test showed that the curves differed significantly ($p=0.04$).

Fig. 2 Kaplan-Meier survival curves for incisor survival stratified by the RCSE classification of severity of intrusion for the best-worst case sample

<table>
<thead>
<tr>
<th>RCSE Classification</th>
<th>Severity of Intrusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;3mm</td>
</tr>
<tr>
<td>2</td>
<td>3-6mm</td>
</tr>
<tr>
<td>3</td>
<td>&gt;6mm</td>
</tr>
</tbody>
</table>

Pulpal necrosis

Pulpal necrosis was diagnosed in 8 of the 31 intruded incisors. Six incisors had prophylactic root canal therapy initiated according to the protocol (Appendix II) rather than based upon diagnostic signs of PN. These six incisors were eliminated from analysis. The distribution of incisors with PN for the best-worst case sample as well as the entire sample is presented in Table 8. The results of the proportional hazards regression for the best-worst case sample are presented in Table 9. The relative risk of each variable in the model for the diagnosis of PN can be explained clinically, however the confidence intervals for the risk ratios calculated by the proportional
hazards regression analysis were too wide to conclude that they could not have occurred by chance, based on this sample size.

Table 8 Distribution of incisors with pulpal necrosis

<table>
<thead>
<tr>
<th>Sample</th>
<th>Total Incisors</th>
<th>PN</th>
<th>Censored</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entire</td>
<td>26</td>
<td>8</td>
<td>18</td>
</tr>
<tr>
<td>Best=Worst Case</td>
<td>22</td>
<td>7</td>
<td>15</td>
</tr>
</tbody>
</table>

Table 9 Proportional hazards regression for diagnosis of pulpal necrosis; best=worst case sample

<table>
<thead>
<tr>
<th>Variable</th>
<th>Multivariate Analysis p-value</th>
<th>Relative risk</th>
<th>Risk ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Injury-related and treatment variables</td>
<td></td>
</tr>
<tr>
<td>Type of Crown Fracture</td>
<td>0.008</td>
<td>Crown fracture involving enamel/dentin/pulp vs none</td>
<td>3.15</td>
</tr>
<tr>
<td>Treatment method</td>
<td>0.03</td>
<td>Active vs passive repositioning</td>
<td>3.6</td>
</tr>
<tr>
<td>RCSE Classification</td>
<td>0.11 (NS)</td>
<td>RCSE Classification &gt;6mm vs ≤3mm</td>
<td>11.89</td>
</tr>
<tr>
<td>Root development</td>
<td>0.20 (NS)</td>
<td>Immature vs mature root development</td>
<td>13.34</td>
</tr>
<tr>
<td>Model</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Critical p-value = 0.05

Kaplan-Meier survival curves (Fig.3) for type of crown fracture were significantly different for the diagnosis of PN (Log rank test, p=0.001). While it appeared that incisors that were actively repositioned experienced PN more frequently than passive or immediately repositioned incisors, no significant difference between Kaplan-Meier survival curves for treatment methods (Fig. 4) was found (Log rank test, p=0.08).
Fig. 3 Kaplan-Meier survival curves for the diagnosis of pulpal necrosis based on the degree of crown fracture for the best=worst case sample

Fig. 4 Kaplan-Meier survival curves for the diagnosis of pulpal necrosis stratified on treatment method for the best=worst case sample
Logistic regression analysis

A logistic regression analysis was used to analyze PCO, IRR, RRR and alveolar bone loss. Multivariate analysis failed to demonstrate significant associations between variables in the model and the following outcomes: PCO, IRR and alveolar bone loss. Treatment method and the RCSE classification were predictive for the presence of RRR ($p=0.03$). Active repositioning had the greater likelihood for developing RRR ($p=0.03$). There was a trend that incisors intruded greater than 6mm also had a greater likelihood for the presence of RRR ($p=0.09$). Since results failed to produce significant values for all parameter estimates, further statistical analysis at this time was abandoned.
DISCUSSION

An intrusion is the rarest luxation injury sustained by a permanent maxillary incisor. This makes it difficult to collect sufficient data for statistical analysis. The major shortcomings of current clinical outcome studies of intrusions (Table 1) are small sample size, heterogeneity of the sample, extended age range and lack of appropriate statistical analysis. Furthermore, there is a lack of knowledge of the influence of some injury-related variables (severity of the intrusion, crown fracture and stage of root development) and treatment variables (passive or active repositioning or immediate reduction) on pulpal and periodontal healing and tooth survival.

The sample population in this study was restricted to children and adolescents due to anatomical differences between this group of individuals and adults. Intrusions occur more often in children and adolescents than adults due to the elasticity of alveolar bone and periodontium. In addition, incisors in this age group often do not have mature root length or completion of apical constriction. These anatomical differences combined with the continuing alveolar bone development of the growing face affects healing differently in children and adolescents than adults.

Prior to this outcome study, the only investigation of survival of intruded incisors in children reported descriptive statistics and univariate chi-square analysis of overall survival (Kinirons and Sutcliffe, 1991). In this study, survival analysis statistics were used to provide increased strength to a case series similar to that of Kinirons and Sutcliffe (1991). Survival and outcome data provides the clinician with quantitative information about prognosis to support clinical decision making.
Survival analysis techniques are well suited to studies that investigate rare conditions by accommodating staggered entry of patients over time (Brown and Swanson, 1994). The ten year period (1988-1998) for data collection provided an extended time for recruitment and follow-up. Seventy-three percent of the available sample presented for follow-up appointments. Missed appointments can be due to the absence of a sense of urgency and/or financial problems. Transition from childhood to adolescence, transportation, distance and lack of a telephone are only a few of the factors that determine a lack of compliance.

The demographics of available and unavailable samples were compared to determine if the loss of patients would bias the results of this study. The differences for age, gender or type of incisors were not significant. However, the mean severity of intrusion in the unavailable sample was significantly less than the available sample ($p<0.003$). This finding biased the results of this study. However, this study showed that incisors intruded <3mm had a probability of 1.0 for survival. Therefore, the results would be strengthened if the unavailable sample was present for follow-up. The Hospital for Sick Children is a tertiary care center and it was expected that the available sample would not represent all intrusions in the population. Children and adolescents with more severe injuries seek dental attention in hospital and those with milder intrusions may have gone unnoticed or not been registered by patients or parents. Further investigation of the available sample validated its use since the range of severity of intrusion was evenly distributed at all levels of severity.
This study represents the largest outcome study of intruded permanent maxillary incisors of children and adolescents (26 patients, 31 incisors). It is the second clinical outcome study to specifically address intrusions.

**Radiographic analysis**

Measurement error of radiographs can influence statistical analysis (Fleiss and Shrout, 1977). The design of this study took into consideration sources of bias for radiographic assessment. The inter-rater reliability (Table 7) indicates moderate to substantial agreement beyond chance for assessment of stage of root/apical development, PCO and external RR and showed that the radiographic interpretations were reliable between raters.

**Survival**

There is a lack of data on incisor survival following intrusions. This investigation demonstrated that the severity of intrusion at the time of injury is significantly related to tooth survival. The categories for severity of intrusion used in this study were chosen according to the guidelines of the RCSE for intrusions, an arbitrary designation developed by consensus (Kinirons, 1998). This is the first study to statistically support the RCSE classification. Incisors intruded >6mm (RCSE 3) had significantly decreased survival than incisors intruded <3 mm (RCSE 1) at five years. These findings are consistent with Kinirons and Sutcliffe (1991) who reported a trend towards decreased survival for severely intruded incisors (>5mm) at a two year follow-up using descriptive statistical analysis.

An incisor intruded >6mm is intruded approximately two-thirds of the clinical crown (Kraus et al., 1969). The abbreviated survival of these incisors may be explained by extensive damage to
the pulp and periodontium (Turley et al., 1987). The severity of injury suggests that intruded incisors are wedged into alveolar bone to the point where the root surface is in intimate contact with the fractured alveolar bone. The existence of intact PL is unexpected and cementum would most likely be sheared or torn from the root surface as well. Ischemia would be the outcome of compression of the neurovasculature. These elements of an acute intrusion injury predispose the root to healing complications that include IRR and RRR if the root surface remains compressed. In this study, failure of an incisor was a clinical decision based on radiographic interpretation of external RR that either reduced root volume or produced ankylosis that interfered with alveolar development.

Method of treatment may be related to incisor survival but this sample size was insufficient to identify treatment choice as a predictive variable. The goal of treatment is to return the incisor to its original position and ultimately, its pre-injury state. Reduction of an incisor to its original position is not the only treatment option nor is it necessarily the first choice for treatment according to Andreasen and Andreasen (1994). The treatment protocol for intrusions in this study was based upon the literature of the 1980's and a protocol by Andreasen in 1981. Three treatment options were available; passive repositioning, active repositional and immediate reduction. The Hospital for Sick Children protocol differed from Andreasen (1981a) in three areas. First, the protocol of The Hospital for Sick Children recommended that the severity of intrusion be the basis for the choice of treatment. Andreasen (1981a) based his protocol on the stage of root formation. Second, immediate reduction was at the discretion of the clinician. Andreasen (1981a) did not advocate immediate reduction. Finally, if active repositioning was the treatment of choice, The Hospital for Sick Children protocol recommended a longer waiting period prior to initiation of traction. This was based upon the supposition that waiting 2-3 weeks
would allow sufficient time for reorganization of the PL and allow for some indication that passive repositioning might occur. Andreasen (1981a) recommended that traction begin before 2 weeks.

Proportional hazards regression showed no significant difference between the three treatment methods in affecting PN. Immediate reduction, generally not recommended in the literature could not be shown to be worse than the other two choices. On the other hand, active repositioning, described in numerous case reports as successful could not be shown to be better than either immediate reduction or passive repositioning.

This study and the study of Kinirons and Sutcliffe (1991) recognize that delayed repositioning (active or passive) maintains the root in intimate contact with alveolar bone. Compression of the root against alveolar bone and delay in mechanical repositioning facilitates RRR in the intruded location rather than reorganization and reattachment of the PL fibers in the normal location. This further complicates return of normal gingival architecture and accentuates the effects of continuing alveolar bone growth as ankylosis occurs.

**Outcomes**

Outcomes for pulpal and periodontal healing reported in this study included PN, PCO, IRR, RRR and alveolar bone loss (Table 6). This is consistent with the literature on dental intrusions (Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985; Kinirons and Sutcliffe, 1991). Logistic regression multivariate analysis failed to demonstrate a significant relationship between the injury-related variables (severity of intrusion, stage of root development, crown fracture) and
treatment methods (passive or active repositioning, or immediate reduction) with PCO, IRR and alveolar bone loss.

_Pulpal necrosis_

The incidence of PN (45%) was less than reported in previous studies (85-96%) (Andreasen, 1970; Andreasen and Vestergaard Pedersen, 1985; Kinirons and Sutcliffe, 1991). The development of PN after intrusions has been reported to be dependent upon the stage of root development at the time of injury (Skeiller, 1960; Andreasen, 1970a; Andreasen and Vestergaard Pedersen, 1985). In the current study, 42% of incisors that developed PN had complete root development. The results of this study cannot be compared to the results of Andreasen and Vestergaard Pedersen (1985) who found 100% PN with incisors with complete root development ($p=0.0002$) since their sample included both adults and children, did not report severity of the intrusion and did not include crown fractures involving dentin and/or pulp as possible prognostic factors in their multivariate analysis. Crown fractures and severity of injury are etiological factors for the diagnosis of PN and might also affect this outcome.

The time to diagnosis of PN described in this study is consistent with the literature (Jacobsen, 1980; Andreasen and Vestergaard Pedersen, 1985). Six of the seven cases in the current study developed PN within 2 years or less. The diagnosis of PN was significantly ($p=0.001$) related to severity of crown fracture. The findings are consistent with previously published results that the risk of PN in incisors with crown fractures increases with the complexity of fracture (no fracture, enamel fracture, enamel-dentin fracture, enamel-dentin-pulp fracture) and increases with the complexity of periodontal tissue damage (Zadik, 1979; Ravn, 1981; Robertson, 1998).
Active repositioning was implemented for incisors that either failed to passively reposition or for incisors intruded >6mm to facilitate pulpal access. An interaction between active repositioning, severity of intrusion and stage of root development with the diagnosis of PN is expected but an increased sample size is required to identify these interactions.

**Replacement root resorption**

Logistic regression analysis demonstrated a significant relationship between the development of RRR and two variables, active repositioning and severity of intrusion. These findings are consistent with an animal study by Turley et al. (1987) that suggested the severity of intrusion increases the mechanical resistance that must be overcome to reposition an incisor and encourages the development of RRR due to compression injury. Delayed active repositioning leaves the root in close contact with the alveolar bone. This facilitates RRR and associated ankylosis.

**Clinical decision making**

**Severity of Intrusion**

The present study is the first to use survival analysis to provide quantitative prognostic information on the five year survival of intruded permanent maxillary incisors in children and adolescents. The results of this study are consistent with those of Kinirons and Sutcliffe (1991) but the use of survival analysis in this study increases the statistical power of their results. Categorization by severity of intrusion based on the RCSE guidelines increases the clinical usefulness of the results. The quantitative information from this study will allow clinicians to provide patients and parents with 5 year survival expectations for intruded permanent maxillary incisors (Table 10).
**Treatment method**

All three treatment methods were used for the full range of severity of intrusion. A trend towards more frequent failure was noted if active repositioning was delayed beyond two weeks, but no treatment method was identified as superior in this study (Table 10). If clinicians propose active repositioning, this trend suggests they should begin early and inform patients and parents of the need for compliance with the additional observation and adjustment appointments that are required as failure may be no less likely. Costs of the active repositioning option include travel, parking and potential lost wages in addition to cost of the appliance.

**Table 10 Summary of clinical expectations for intrusions of the permanent maxillary incisors in children and adolescents**

<table>
<thead>
<tr>
<th>Severity of intrusion (mm)</th>
<th>5 year survival</th>
<th>Risk associated with injury-related and treatment variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3</td>
<td>1.0</td>
<td>- Survival is not dependent upon treatment method.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Survival is not dependent upon stage of root development or severity of crown fracture.</td>
</tr>
<tr>
<td>3-6</td>
<td>0.90</td>
<td>- Survival is not dependent upon treatment method.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Failure is associated with the development of external RR.</td>
</tr>
<tr>
<td>&gt;6</td>
<td>0.45</td>
<td>Incisors that failed, failed prior to 2 years</td>
</tr>
</tbody>
</table>

**Outcomes**

Parents and patients can be informed of the range of clinical outcomes associated with intrusions. Based on the results of the current study, PN was diagnosed more frequently with incisors intruded >6mm with associated complex crown fractures and immature root development. This study shows that the severity of injury was more influential than the stage of root formation for
diagnosis of PN. Based on this study and the study by Kinirons and Sutcliffe (1991), the incidence of PN following intrusion in children is between 45-96%.

Dentists can be informed that RRR will occur more often with incisors that were intruded >6mm and those that were actively repositioned. One quarter of the sample in this study showed RRR. This is serious as RRR is associated with ankylosis that can lead to infraocclusion in children as the alveolus continues to grow. Clinicians should be aware of the need for extraction prior to the development of discrepancies in gingival contour and inform parents of this expected outcome at the time of the accident.

The information provided by this study has immediate application for dentists who treat dental trauma and provides statistical support for the RCSE guidelines (Kinirons, 1998). Additional information will come from new cases at this centre and from proposed multicentre collaborations.
CONCLUSIONS

In a population of children and adolescents:

1. A significant difference in survival between permanent maxillary incisors intruded >6mm and those intruded <3mm was shown. The 5 year survival for incisors intruded >6mm is 0.45, those intruded 3-6mm is 0.90 and for incisors intruded <3mm is 1.0.

2. Severity of intrusion, stage of root development, crown fracture and treatment method were shown to be essential elements of a predictive model for survival of intruded permanent maxillary incisors.

3a. Severity of intrusion, stage of root development, crown fracture and treatment method were shown to be essential elements of a predictive model for the development of PN of intruded permanent maxillary incisors.

3b. Crown fractures that involve either the dentin or dentin and pulp are predictive for diagnosis of PN (within two years) of intruded permanent maxillary incisors.

4. Logistic regression multivariate analysis failed to demonstrate a significant relationship between the injury-related variables (severity of intrusion, stage of root development, crown fracture), treatment method (passive or active repositioning, or immediate reduction) and PCO of intruded permanent maxillary incisors.
5a. Logistic regression multivariate analysis failed to demonstrate a significant relationship between the injury-related variables (severity of intrusion, stage of root development, crown fracture), treatment method (passive or active repositioning, or immediate reduction) and IRR of intruded permanent maxillary incisors.

5b. A significant relationship was shown between the combined variables; severity of intrusion of permanent maxillary incisors and treatment method and the presence of RRR.

6. Logistic regression multivariate analysis failed to demonstrate a significant relationship between injury-related variables (severity of intrusion, stage of root development, crown fracture), treatment methods and alveolar bone loss of intruded permanent maxillary incisors.
REFERENCES


**APPENDIX I**

<table>
<thead>
<tr>
<th>Soft tissue assessment:</th>
<th>Gingiva/oral mucosa trauma: Yes ☐; No ☐</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>laceration ☐, contusion ☐, abrasion ☐</td>
</tr>
<tr>
<td></td>
<td>tooth fragment/foreign body</td>
</tr>
<tr>
<td>Extra-oral trauma: Yes ☐; No ☐</td>
<td>Gingiva/oral mucosa treatment: Yes ☐; No ☐</td>
</tr>
<tr>
<td></td>
<td>laceration ☐, contusion ☐, abrasion ☐</td>
</tr>
<tr>
<td></td>
<td>tooth fragment/foreign body</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TMJ assessment:</th>
<th>normal</th>
<th>restricted</th>
<th>painful</th>
</tr>
</thead>
<tbody>
<tr>
<td>open/close</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>protrusion</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>lateral r/lft</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

**Emergency treatment:**
Radiographs: Yes ☐; Number: ☐; No ☐

Photographs: Yes ☐; Number: ☐; No ☐

Splint: Yes ☐; No ☐
  rigid: ☐  nonrigid: ☐  teeth included in splint: ☐

Antibiotics: Yes ☐; No ☐
(type, dosage, duration)

Chlorhexidine: Yes ☐; No ☐
(dosage, duration)

**Emergency treatment notes:** (including previous treatment)

Schedule for further treatment at HSC: Yes ☐; No ☐
If no further treatment, schedule for followup at HSC: Yes ☐; No ☐

Signature: ___________________________  Date: ___________________________
## PERMANENT TOOTH TRAUMA INFORMATION SHEET

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

<table>
<thead>
<tr>
<th>Date of trauma:</th>
<th>Date of HSC emergency treatment:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time of trauma:</td>
<td>Time treatment commenced:</td>
</tr>
<tr>
<td>Previous treatment of currently injured tooth/teeth: Yes □ (describe on back); No □</td>
<td></td>
</tr>
<tr>
<td>Tooth discoloured prior to treatment: Yes □; tooth #(#): ______; colour: ______ □: No □</td>
<td></td>
</tr>
<tr>
<td>Angle Class: ______; N/A □; N/R □</td>
<td>Overjet: ______ mm; N/R □; Overbite: ______ %; N/R □</td>
</tr>
<tr>
<td>Epistemic classification: fall □; impact □; collision □</td>
<td></td>
</tr>
</tbody>
</table>

### 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 □
- Communion 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 □
- Communion 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: □
APPENDIX II

Protocol for management of permanent incisors - Intrusions

Division of Paediatric Dentistry
The Hospital for Sick Children
June 1988

a) Mild Intrusions

- Routine clinical and radiographic follow-up at 2 weeks, 6-8 weeks and then every 6 months
- If spontaneous re-eruption is not evident in 2-3 weeks, orthodontic traction should be instituted to produce alignment

b) Severe Intrusions

- Reposition the tooth slightly and compress the alveolar plates in order to facilitate bonding of orthodontic brackets, soft tissue closure and access to the crown of the tooth for initiation for endodontic therapy

- Delay orthodontic traction for 2-3 weeks to provide adequate time for reorganization and reattachment of periodontal ligament fibers

- Extrude the tooth orthodontically over a period of 4-8 months in cases of vital teeth or over a period of 3-5 weeks in cases of non-vital teeth

- For teeth with a mature apex, pulpal extirpation with Ca(OH)2 (Pulpdent TM) placement should be performed 7-10 days following the visit

- For teeth with an immature root, an apexification procedure may be necessary if the tooth becomes non-vital

- Administer a 1 week course of antibiotics (e.g. Penicillin, but if sensitive, use Erythromycin) accompanied by a 0.1 percent chlorhexidine mouthrinse (10-15ml) twice daily for 10 days commencing 48 hours after the emergency visit.
APPENDIX III

Patient follow-up data collection form

**Patient Information**

<table>
<thead>
<tr>
<th>Last Name:</th>
<th>First Name:</th>
<th>D.O.B.</th>
<th>Sex:</th>
<th>Address</th>
</tr>
</thead>
</table>

Phone#

Dentist:

Consent for Release of Information obtained  
[ ] Yes  [ ] No

<table>
<thead>
<tr>
<th>Date of Intrusion</th>
<th>Age:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.2</td>
<td></td>
</tr>
<tr>
<td>1.1</td>
<td></td>
</tr>
<tr>
<td>2.1</td>
<td></td>
</tr>
<tr>
<td>2.2</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Date of Endo</th>
<th>Date of Ortho</th>
<th>Finish Ortho</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Follow-up information

<table>
<thead>
<tr>
<th>Date</th>
<th>Days:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mobility</th>
<th>1.2</th>
<th>1.1</th>
<th>2.1</th>
<th>2.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periodontal probing</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tooth color</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percussion sens</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percussion tone</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fistula</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitality-electric</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitality-cold</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX IV

Radiographic Evaluation Scoring Sheet - December 7, 1998

Rater Identification #1 #2 Patient Identification

☐ A. Tooth # 1.2 1.1 2.1 2.2

Stage of Apical Development
Moorrees, Fanning, Hunt 1963
2 3 4 5 6 7

☐ B. Pulp Necrosis
Yes No

Pulp Canal Obliteration
Yes If yes No Partial Complete

Root Resorption
Yes No

Bone Loss
Yes No

Infraocclusion
Yes No

Other

☐ C. Root Resorption Andersson, 1989

<table>
<thead>
<tr>
<th>Score</th>
<th>Type</th>
<th>Score</th>
<th>Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2</td>
<td>I R</td>
<td>Apical</td>
<td>0 1 2</td>
</tr>
<tr>
<td>0 1 2</td>
<td>I R</td>
<td>Middle</td>
<td>0 1 2</td>
</tr>
<tr>
<td>0 1 2</td>
<td>I R</td>
<td>Cervical</td>
<td>0 1 2</td>
</tr>
</tbody>
</table>

For Office Use only

Inflammatory Root Resorption ____/12
Replacement Resorption ____/12
Patient contact algorithm (Adapted from Lee, 1996)

Name: ________________________  Phone Number: ________________________

☐ Chart found
☐ Phone patient  ☐ Book appointment  ☐ Appt. date

☐ Check Number with the operator

☐ Check Dental Chart for correct number

☐ Check main Chart for correct or New number

☐ Check with family M.D. for New Number

☐ Check parents places of employment

Sent letter with consents for release of information and map to parent  Yes ☐ No ☐
Consents signed and photocopied  Yes ☐ No ☐
Letter to dentist (copy) ________________________  Yes ☐ No ☐
Notification from dentist ________________________  Yes ☐ No ☐
Slides taken  Yes ☐ No ☐
### AVAILABLE SAMPLE - 31 INCISORS.

<table>
<thead>
<tr>
<th>Rows</th>
<th>HSC 1</th>
<th>Age</th>
<th>Tooth</th>
<th>Severity</th>
<th>RCS</th>
<th>Apex</th>
<th>Cr #</th>
<th>Ex</th>
<th>Stu</th>
<th>En Survi</th>
<th>Survi</th>
<th>PM Diagnosis</th>
<th>PM</th>
<th>PCO</th>
<th>IRR</th>
<th>RR</th>
<th>BL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1715</td>
<td>5.5</td>
<td>1.1</td>
<td>2.1</td>
<td>0</td>
<td>1</td>
<td>3394</td>
<td>3394</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>1290</td>
<td>8.8</td>
<td>2.1</td>
<td>5.2</td>
<td>0</td>
<td>1</td>
<td>PR</td>
<td>3200</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>1065</td>
<td>4.8</td>
<td>1.1</td>
<td>4.2</td>
<td>1</td>
<td>0</td>
<td>PR</td>
<td>1635</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>12394</td>
<td>7.2</td>
<td>2.1</td>
<td>2.1</td>
<td>1</td>
<td>0</td>
<td>PR</td>
<td>668</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>1280</td>
<td>6.3</td>
<td>2.1</td>
<td>2.1</td>
<td>0</td>
<td>0</td>
<td>PR</td>
<td>1277</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>1777</td>
<td>8.3</td>
<td>2.1</td>
<td>10.3</td>
<td>0</td>
<td>2</td>
<td>PR</td>
<td>2413</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>1778</td>
<td>8.6</td>
<td>1.1</td>
<td>6.2</td>
<td>0</td>
<td>2</td>
<td>AR</td>
<td>2533</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>1112</td>
<td>7.2</td>
<td>2.1</td>
<td>5.3</td>
<td>0</td>
<td>0</td>
<td>PR</td>
<td>1385</td>
<td></td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>1260</td>
<td>10.2</td>
<td>2.1</td>
<td>0.3</td>
<td>0</td>
<td>2</td>
<td>AR</td>
<td>1676</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>1820</td>
<td>5.1</td>
<td>2.1</td>
<td>4.2</td>
<td>2</td>
<td>0</td>
<td>PR</td>
<td>1676</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>5388</td>
<td>6.3</td>
<td>1.1</td>
<td>6.3</td>
<td>0</td>
<td>0</td>
<td>PR</td>
<td>2074</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>1421</td>
<td>10.1</td>
<td>1.2</td>
<td>8.3</td>
<td>1</td>
<td>0</td>
<td>AR</td>
<td>563</td>
<td></td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>1289</td>
<td>12.3</td>
<td>2.1</td>
<td>8.3</td>
<td>1</td>
<td>0</td>
<td>AR</td>
<td>932</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>1472</td>
<td>11.8</td>
<td>2.1</td>
<td>4.2</td>
<td>1</td>
<td>0</td>
<td>PR</td>
<td>655</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>1472</td>
<td>11.8</td>
<td>2.2</td>
<td>6.2</td>
<td>1</td>
<td>2</td>
<td>AR</td>
<td>1918</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>1368</td>
<td>7.6</td>
<td>1.1</td>
<td>1.1</td>
<td>0</td>
<td>1</td>
<td>PR</td>
<td>686</td>
<td></td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>32</td>
<td>1755</td>
<td>8.6</td>
<td>1.1</td>
<td>6.2</td>
<td>0</td>
<td>0</td>
<td>AR</td>
<td>1959</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>33</td>
<td>1866</td>
<td>6.2</td>
<td>1.1</td>
<td>2.5</td>
<td>1</td>
<td>0</td>
<td>IR</td>
<td>894</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>1572</td>
<td>13.5</td>
<td>2.2</td>
<td>1.1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1415</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>36</td>
<td>1373</td>
<td>12.1</td>
<td>1.1</td>
<td>12.3</td>
<td>1</td>
<td>0</td>
<td>IR</td>
<td>2829</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>37</td>
<td>1373</td>
<td>12.1</td>
<td>1.2</td>
<td>12.3</td>
<td>1</td>
<td>0</td>
<td>IR</td>
<td>2829</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>38</td>
<td>12024</td>
<td>11.8</td>
<td>2.2</td>
<td>1.1</td>
<td>1</td>
<td>0</td>
<td>IR</td>
<td>1899</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>39</td>
<td>2135</td>
<td>17.3</td>
<td>2.2</td>
<td>8.3</td>
<td>0</td>
<td>1</td>
<td>IR</td>
<td>629</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>1380</td>
<td>8.8</td>
<td>1.1</td>
<td>6.2</td>
<td>0</td>
<td>0</td>
<td>IR</td>
<td>1263</td>
<td></td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>11</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>41</td>
<td>1389</td>
<td>8.8</td>
<td>1.1</td>
<td>6.3</td>
<td>0</td>
<td>0</td>
<td>IR</td>
<td>1263</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>42</td>
<td>1587</td>
<td>10.2</td>
<td>2.1</td>
<td>7.3</td>
<td>0</td>
<td>0</td>
<td>IR</td>
<td>264</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>44</td>
<td>12580</td>
<td>10.1</td>
<td>2.1</td>
<td>7.3</td>
<td>0</td>
<td>0</td>
<td>IR</td>
<td>264</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>45</td>
<td>1054</td>
<td>10.1</td>
<td>2.1</td>
<td>4.2</td>
<td>1</td>
<td>3</td>
<td>IR</td>
<td>2779</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>46</td>
<td>1329</td>
<td>9.2</td>
<td>1.1</td>
<td>7.3</td>
<td>0</td>
<td>2</td>
<td>IR</td>
<td>684</td>
<td></td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

### UNAVAILABLE SAMPLE

<table>
<thead>
<tr>
<th>Rows</th>
<th>HSC 1</th>
<th>Age</th>
<th>Tooth</th>
<th>Severity</th>
<th>Withdrawn</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1798</td>
<td>8.4</td>
<td>2.1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>1065</td>
<td>8.3</td>
<td>1.1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>12394</td>
<td>7.2</td>
<td>2.1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>1744</td>
<td>7.8</td>
<td>1.1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>1537</td>
<td>8.3</td>
<td>1.1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>1153</td>
<td>14.4</td>
<td>1.1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>1151</td>
<td>14.4</td>
<td>2.1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>1151</td>
<td>14.4</td>
<td>2.1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>1509</td>
<td>11.7</td>
<td>2.2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>1437</td>
<td>10.8</td>
<td>2.1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>11</td>
<td>1533</td>
<td>10.6</td>
<td>2.1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>1231</td>
<td>13.4</td>
<td>1.1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>13</td>
<td>1231</td>
<td>13.4</td>
<td>1.1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>14</td>
<td>2131</td>
<td>8.8</td>
<td>1.1</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>
# APPENDIX VII

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

<table>
<thead>
<tr>
<th>Kappa Value</th>
<th>Descriptive Agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0</td>
<td>Poor</td>
</tr>
<tr>
<td>0.0-0.2</td>
<td>Slight</td>
</tr>
<tr>
<td>0.41-0.60</td>
<td>Moderate</td>
</tr>
<tr>
<td>0.61-0.80</td>
<td>Substantial</td>
</tr>
<tr>
<td>0.81-1.0</td>
<td>Perfect</td>
</tr>
</tbody>
</table>
Algorithm used for determination of best and worst case scenarios
(Adapted from Barrett, 1995)

Was > 1 tooth intruded

Yes

Have any of the teeth failed?

Yes

>1

Count the tooth lost first as the 'worst' case and the last as the 'best' case

1

Count the tooth as the 'worst' case

No

Best = Worst

No

Best Case
Assess outcomes