Lack of Pulpal Sensitivity in Ectopic Maxillary Canines After Orthodontic Treatment: A Clinical Study

by

Dr. Alvaro Wagner Rodrigues Salles

A thesis submitted to Faculty of Graduated Studies of the University of Manitoba in partial fulfillment of the requirements for the degree of
MASTER OF SCIENCE

Department of Preventive Dental Science

College of Dentistry
Division of Orthodontics
University of Manitoba
Winnipeg, Manitoba

Abstract

Introduction: Although pulpal necrosis can be associated with orthodontically treated ectopic maxillary canine, the literature is scarce on this topic. **Objective**: This retrospective study aimed to investigate if ectopic maxillary canines were more predisposed to lack of sensitivity due to orthodontic treatment. **Materials and Methods**: The sample was comprised of 20 patients (13 females and 7 males) with a mean age of 21.7 years. Forty maxillary canines were divided into two groups: ectopic (n=26) and non-ectopic (n=14). The post-treatment pulpal assessment included thermal, electrical, and percussion tests. Initial panoramic radiographs were used to assess the axial angulation of the ectopic canines and their linear distance to the occlusal plane. A clinical examination and a questionnaire were used to collect data on 14 background variables capable of influencing on pulp status. **Results**: There was no influence from axial angulation (p=0.0661) and linear distance (p=0.4840), nevertheless rather from duration of traction (p=0.0437). The mixed-effects logistic regression showed no statistically significant difference between ectopic and non-ectopic canines with regard to pulpal sensitivity (p=0.0744). From a clinical standpoint, 12 teeth (46.15%) presented with lack of sensitivity in the ectopic group whereas only two (14.29%) had the same outcome in the control group. Of the 14 background variables, only four would be worth pursuing further research: initial location, presence of gingival recession, bracket slot size, and history of spontaneous pain. **Conclusion**: Although further studies are still necessary, lack of pulpal sensitivity can be a side effect when treating ectopic maxillary canines.

Acknowledgments

I would like to express my deeply appreciation to Dr. Fabio Pinheiro for your support since my first day at University of Manitoba. Thank you for all the moments reading and correcting my research.

I also would like to be grateful to Dr. Robert Drummond and Dr. Eraldo Batista, members of my thesis committee, who had a great contribution in my project. I know how difficult was to manage all your duties and my research.

Dr. Rodrigo Cunha thank you for your help in the data collection, for your clinical expertise and friendship.

Lastly, I would like to state my gratitude to Dr. William Wiltshire for been accepted into the Orthodontic program. It was a dreaming that becomes reality in July 2014.

Dedication

I would like to dedicate this research to my wife Adriana, my son Alvaro and my daughter Laura.

Thank you for your love and support during this long journey.

Contents

List of Figures and Tables

Chapter 1

Introduction

A tooth can be considered ectopic when it fails to erupt in its normal pattern and is prevented from doing this by mechanical obstruction or the position of the adjacent teeth (Moskowitz and Garcia 2014). Failure to erupt without any obstruction has also been reported in the literature (Moskowitz and Garcia 2014). The prevalence of maxillary canine impaction ranges from 0.27% to 3% of the population (Baccetti et al. 2011; Ericson and Kurol 1986; Naoumova et al. 2011), and affects females more than males (Ericson and Kurol 2000; Fleming et al. 2009; Moskowitz and Garcia 2014) with a higher incidence on the palate (Evren et al. 2014; Fleming et al. 2009; Moskowitz and Garcia 2014). There are several theories to explain the etiology of maxillary palatally displaced canines such as genetic factors (Peck et al. 1994), displaced or peg shape lateral (Becker et al. 1981; Moskowitz and Garcia 2014), endocrine deficiency, irradiation, alveolar cleft, cystic, neoplastic formation and idiopathic conditions (Becker et al. 1981; Bishara 1992). On the other hand, maxillary arch crowding has been more associated with labially displaced maxillary canines (Jacoby 1983; Peck et al. 1994).

An interdisciplinary team comprised of orthodontists, oral surgeons, periodontists and prosthodontists is frequently required for the final treatment plan (Fleming 2015). Management of ectopic and unerupted maxillary canines can involve different procedures: interception, space recreation, auto-transplantation and orthodontically forced eruption. Ultimately, the previously ectopic canine will still need to be submitted to orthodontic forces in order to be positioned correctly in the line of occlusion.

A frequent reaction to orthodontic forces is an inflammatory process in the periodontal tissues. This is a necessary condition for the teeth to move, but it can affect the dental pulp (von Bohl et al. 2012).

Historically, vascular alterations to the pulp blood flow (PBF) have been reported in response to orthodontic movements (Oppennhein 1942; Reitan 1951). Studies on blood supply during orthodontic movement have been the object of investigation for more than sixty years (von Bohl et al. 2012).

Previous studies on PBF changes during tooth movement made use of qualitative techniques, for example, histological or cellular change (Anstending 1972) as well as quantitative techniques such as radioactive or fluorescent microspheres (Vandevska-Radunovic et al. 1994). Other techniques such as radiospirometry, in vivo observation by microscope (Guevara 1980) and pulpal respiration rate (Hamersky et al. 1980; Unsterseher 1987) have also been reported. All those studies are invasive and destructive, not to mention that they do not allow for the assessment of dynamic PBF in real time for long periods.

Laser Doppler Flowmetry (LDF) has been introduced as a new technique for the evaluation of PBF (Evans 1999; Roeykens 2000), but some technical limitations have also been observed (Jafarzadeh 2009; Salles et al. 2013). Its use in Orthodontics has been reported on intrusive intermittent forces (Ikawa et al. 2001), continuous intrusive forces for a short period of time (Barwick and Ramsay 1996), intrusive continuous forces for six days (Sano et al. 2002), tipping continuous forces for three days (McDonald F.; Pitt Ford 1994), continuous intrusive forces by mini implants for 21 days (Sabuncuoglu and Ersahan 2014), rapid palatal expansion (Babacan et al. 2010) and leveling and alignment with superelastic wires for 30 days (Salles et al. 2013). Despite their technical limitations, those studies have brought a better understanding of the PBF alterations to orthodontic forces applied in a way similar to that of a clinical setting.

Cellular alterations in the pulp due to orthodontic movement have been extensively documented. Cell damage (Mostafa et al. 1991; Nixon et al. 1993; Stenvik and Mjor 1970), fibrosis (Lazzaretti et al. 2014), depression of the tissue respiration rate (Hamersky et al. 1980), an increase in the pulpal release of calcitonin gene-related peptide (Caviedes-Bucheli et al. 2011), apoptosis (Veberiene R 2009), vacuolization of odontoblasts and angiogenesis (Derringer et al. 1996), in addition to vascular changes ranging from stasis to necrosis (Bauss et al. 2008a; Bauss et al. 2008b; Gopikrishna et al. 2009; von Bohl et al. 2012; Woloshyn et al. 1994) are some of the side effects associated with the orthodontic movement.

Pulp necrosis has been reported as a result of different types of tooth movement such as retraction, intrusion, extrusion and jiggling. The prevalence of pulp injuries related to orthodontic treatment ranges from 2% to

17% for root canal obliteration and from 1% to 14% for pulp necrosis (von Bohl et al. 2012).

There is scarce information in regards to ectopic maxillary canines submitted to orthodontic traction (Javed et al. 2015; von Bohl et al. 2012). So far, only a few authors (Blair et al. 1998; D'Amico et al. 2003; Evren et al. 2014; Ferreira et al. 2013; Woloshyn et al. 1994) have evaluated the relationship between ectopic maxillary canines and pulp sensitivity.

Further understanding on the prognosis of the pulp status following the orthodontic treatment of ectopic maxillary canines is necessary to achieve better standards of care. The primary aim of this study was to investigate if ectopic maxillary canines are more susceptible to present lack of pulp sensitivity at the end of orthodontic treatment.

Chapter 2

Literature Review

This literature review encompasses a series of relevant topics that can help to understand the diagnosis and treatment of ectopic maxillary canines. It also provides the reader with information relating to the pulp status of teeth submitted to orthodontic traction.

2.1. Ectopic maxillary canines

This section covers several aspects related to the diagnosis and treatment of ectopic maxillary canines. A comparison of the reported treatment outcomes is also presented in the end.

2.1.1. Diagnosis and location techniques for ectopic maxillary canines

The maxillary canine usually erupts at a mean age of 10.5 years in girls and 11.5 years in boys (Naoumova et al. 2011; Shapira and Kuftinec 2001).

The maxillary canine has been reported to have the longest development time and the most tortuous pathway of eruption from its point of formation lateral to the piriform fossa until its final occlusal position (Bishara 1992). This is a tooth that remains quite high, just above the root of the maxillary lateral incisor, until the crown is fully calcified when it finally descends along the distal surface of the lateral incisor, what helps to close the physiological diastema present in the so-called “ugly duckling stage” (Kumar et al. 2015).

In the absence of radiographs, the most efficient way to evaluate the eruption and position of the maxillary canine during the mixed dentition is to perform bilateral digital palpation of the maxillary canine areas. It has been found that a positive palpation (a canine bulge) projected a good eruption prognosis (Ericson and Kurol 1986; 1988). After 10 years or older, findings such as the absence of the canine bulge, delayed or ectopic eruption of the maxillary canine, palatal bulge and/or distal root tipping of the adjacent maxillary lateral incisor should serve as an alert to request supplementary radiographic examination.

Correct identification of an unerupted maxillary canine can prevent subsequent impaction, facilitate surgical access and aid in defining the most appropriate force vector to move the maxillary canine toward the line of occlusion (Kumar et al. 2015).

There are different radiographic techniques to determine the position of the maxillary canine including panoramic views, periapical views, occlusal films, postero-anterior views and the lateral cephalogram. These techniques are limited to two dimensions. Another approach is the 3-dimension radiograph, which includes Computed Tomography (CT), Spiral CT and Cone Beam Computed Tomography (CBCT) (Kumar et al. 2015).

For a long time, different authors have utilized the panoramic radiographic view to determine the position of unerupted maxillary canines. Ericson and Kurol (Ericson and Kurol 1986; 1988) described three different predictive anatomic sectors or scenarios: a) sector 1 - the cusp tip of the canine lies between the inter-incisal midline and the long axis of the central incisor; b) sector 2 - the cusp tip of the canine lies between the major axes of the maxillary lateral and central incisors; c) sector 3 - the cusp tip of the canine lies between the major axes of the maxillary lateral and first premolars. Those sectors represented the possible positions of an ectopic maxillary canine. In addition, the authors used the alpha angle to represent the angle formed between the inter-incisal midline and the long axis of the canine. A line perpendicular to the occlusal plane also was traced from the cusp of the ectopic canine and was denominated as “distance” (D). They reported that the risk of resorption of the lateral incisor increased by 50% if the cusp of the canine was inside of sectors 1 and 2 and whenever the alpha angle was greater than 25 degrees. As a matter of fact, as the alpha angle increased the necessity for and difficulty of treatment also increased.

Wardorf et al. (2003) (Warford et al. 2003) evaluated ectopic maxillary canines using four sectors and angular measurements between the bicondylar area and the long axis of the canines. The authors concluded that the chance for canine impaction was greater if such angle was reduced and the sector number increased.

Katsnelson et al. (2010) (Katsnelson et al. 2010) reported that panoramic radiographs have high sensitivity to anticipate ectopic maxillary canines when the angle between the canine and a horizontal line passing at the mesiobuccal cusps of the maxillary molars is greater than sixty-five degrees.

Sajnani and King (Sajnani and King 2012) described a method for early diagnosis of unilateral ectopic maxillary canines. The authors observed that, at the age of 8 years and beyond, a clinical difference of 4 mm between the tips of the maxillary canines was already an indication of ectopic eruption. In addition, they found, at the age of 9 years and beyond, a statistically significant difference between the two groups in regards to the location in different sectors and according to the mean angle formed with the midline. They concluded that the diagnosis of ectopic maxillary canines is feasible since 8 years of age by using geometric measurements on panoramic radiographs.

The periapical radiograph is a simple technique with less exposure radiation and provides information regarding the state of development, the presence of a follicle and the extent of resorption of deciduous teeth (Kumar et al. 2015).

The postero-anterior radiograph allows for a medio-lateral position evaluation of the maxillary canines with respect to a line connecting the inferior border of the orbits. This was done by measuring the angle formed between the long axis of the canine and the aforementioned transorbital line. However, this technique is more indicated to assess prognosis rather than aiding in diagnosis (Kumar et al. 2015).

According to Orton in 1995, (Orton HS 1995) the lateral cephalogram is useful in establishing the height of the ectopic maxillary canine with respect to the anteroposterior position of the apices of the maxillary incisors, thereby indicating if the canine is in a buccal or palatal position. However, this information could be misleading if both maxillary canines are ectopic. This radiographic technique also provides another information such as the angle formed between the long axis of the canine and a line perpendicular to the Frankfurt plane. If the angle is less than 10 degrees, the tooth is considered to be in a normal position. If between 15 and 25 degrees, the necessity of treatment increases. If between 25 and 45 degrees, spontaneous eruption is an exception and the complexity of treatment also increases. If over 45 degrees, doubts as to the possibilities of a successful treatment arise.

In order to establish whether the position of the maxillary ectopic canine is labial or palatal, an occlusal radiographic can be used where the beam perpendicular to the film passes through glabella. If the cusp of the canine is positioned in front of the line connecting the apices of the lateral incisors, the position is labial (Kumar et al. 2015).

Another technique is Clark's rule or parallax method or the tube shift method (Bedoya and Park 2009). Two radiographic images of the same object are taken. For the first, proper technique and angulation are used. For the second image, all parameters are constant except for the direction of the central ray which can be either horizontal or vertical. Regardless, the SLOB principle (same lingual opposite buccal) applies, so that if the tooth shifts in the same direction as the tube then the tooth is lingual. If it moves in the opposite direction, it is buccal (Bedoya and Park 2009).

The Right Angle technique is another approach to establish the position of ectopic canines. In this technique, two radiographs (lateral cephalometric and postero-anterior) are taken at right angles to each other (Bedoya and Park 2009). The problem found with this technique is the superimposition of structures and frequently an additional intraoral film is required to show more details of the ectopic tooth (Bedoya and Park 2009).

Three-dimensional images have been frequently in the recent years. They provide more accurate images when compared to two-dimensional radiographic views. Their main advantage is that they make it possible to visualize the exact position of the crown, the root apex and the long axis of the ectopic tooth. They also allow to quantify the distance from the ectopic tooth to the roots of the adjacent teeth and detect the presence of any pathologic lesion and its relationship with the tooth of interest. The presence of any adverse conditions affecting the adjacent teeth, including root resorption, can also be disclosed. Both CT and CBCT are gold standard techniques to locate and examine ectopic canines radiographically. However, cost, radiation levels, risk/benefit, access and expertise required in reading the images have been listed as limitations of these techniques (Bedoya and Park 2009).

2.1.2. Clinical characteristics of ectopic maxillary canines that determine the type of surgical exposure procedure

The isolated extraction of primary maxillary canines has been proposed as an interceptive, predictable and relatively conservative solution for the correction of ectopic canines. It has a success rate ranging from 62% (Baccetti et al. 2008) to 78% (Ericson and Kurol 1988). However, two recent systematic reviews (Naoumova et al. 2011; Parkin et al. 2012) concluded that there was no evidence to support this clinical procedure. Approaches for the management of ectopic canines can vary from surgical exposure combined with orthodontic traction or extraction with or without transplantation to no treatment (observation only) (Husain et al. 2012).

Two surgical techniques have been developed to expose the labially and palatally displaced canines: 1. the open and 2. the closed techniques. In the open, the canine moves or is moved above the palatal mucosa whereas in the closed technique it is moved beneath the palatal mucosa. A systematic review reported that there is no evidence to support one technique over the other in terms of dental health (Parkin et al. 2008).

According to Kokich (Kokich 2004), the most common methods for the management of ectopic canines is to surgically expose the tooth and allow its natural eruption during the early or late mixed dentitions. The author also has described three methods for the uncovering of labially ectopic canines and four criteria to determine the correct approach for palatally ectopic canines.

A more specific description of surgical techniques for labial and palatal impacted canines follows below (Bishara 1992 ; Cooke and Wang 2007; Crescini et al. 2007a; Crescini et al. 2007c; Kokich 2004; Ngan et al. 2005; Proffit et al. 2007; Quiryen M.; Op Heij DG 2000; Schmidt and Kokich 2007; Vermette et al. 1995; Zasciurinskiene et al. 2008).

2.1.2.1. Exposure techniques for labially ectopic canines

Labially ectopic canines can be exposed in three different ways as follows:

2.1.2.1.1. Gingivectomy

This technique is indicated in cases where the canine cusp lies coronal to the mucogingival junction (MGJ), there is an adequate band of keratinized gingival tissue and the tooth is not covered by bone. Orthodontic traction usually is not necessary because the tooth will erupt spontaneously. This is a simple procedure to perform. Loss of attached gingiva, damage to the periodontum and possible gingival overgrowth at the surgical site are the main disadvantages of this technique (Vermette et al. 1995).

2.1.2.1.2. Apically positioned flap

This technique is indicated when the canine crown is located apically to the MGJ. This is a common procedure used for the conservation of keratinized gingiva. An increased risk of gingival height discrepancies and orthodontic relapse are the main disadvantages of this technique. Orthodontic treatment can start between two or three weeks after this type of surgery (Vermette et al. 1995).

2.1.2.1.3. Closed eruption

This technique is recommended in cases where the canine is in the center of the alveolus and the crown is apical to the MGJ. This procedure allows for great esthetics and the mechanics to move the tooth is straightforward. The possibility of a second surgery, patient discomfort and mucogingival problems are the main disadvantages related to this technique. Orthodontic treatment can start two weeks after this type of surgery (Quiryen M.; Op Heij DG 2000; Vermette et al. 1995).

2.1.2.2 Exposure of palatally ectopic canines

Palatally ectopic canines can be exposed in three different ways.

3.1.2.2.1. Closed eruption

To recommend this technique, the maxillary ectopic canine must be located near the lateral and central incisors, be horizontally positioned and higher in the roof of the mouth. Bone necrosis, root resorption, repeated surgeries, bond failure due to the presence of saliva and/or blood and fracture of the wire ligature are the main disadvantages of this technique. Orthodontic traction can start one or two weeks after this surgery (Cooke and Wang 2007; Kokich 2004) .

2.1.2.2.2. Open eruption

This technique is preferred during the late mixed dentition and the orthodontic treatment can start when the canine cusp tip lies at the level of the occlusal plane. The main advantages are improved bone levels, low risk of root resorption, fewer re-exposures, short treatment time and improved oral hygiene. However, there have been reports of eruption failure and unchanged path of eruption (Kokich 2004).

2.1.2.2.3. Tunnel traction

This is indicated when the primary canine is present. The advantage of this approach is that it allows the permanent canine to be guided through the primary canine socket, reducing the amount of bone around the ectopic tooth. The presence of the primary canine is the main disadvantage of this technique. The suture is removed 10 days after surgery and orthodontic traction can be started immediately afterwards (Crescini et al. 1994; Crescini et al. 2007c).

2.1.3. Types of orthodontic mechanics used to extrude ectopic maxillary canines

The orthodontic management of ectopic canines requires complex orthodontic therapeutic techniques where the natural eruption force and orthodontic biomechanics can both work together. The main goal is to move the ectopic tooth toward the centre of the alveolar ridge to avoid bone dehiscence, facilitate the orthodontic movement and achieve improved esthetics (Nieri et al. 2010). Different orthodontic techniques and appliances have been designed.

Jacob described the Ballista spring made of round wire, which accumulates energy by being twisted on its long axis (Jacob 1979).

McBride proposed a wire lasso or snare after the surgical exposure of the ectopic tooth (McBride 1979).

Taylor used a transpalatal arch for anchorage, welded oval buccal tubes to the maxillary first molar in which a sectional archwire with a helix was inserted while its other end was attached to a small hook bonded on the ectopic canine crown (Taylor 1979).

Tan proposed a canine extrusion auxiliary archwire that resembled a reverse torquing spring with power arms (Tan 1983).

Crescini et al. (1994) used a handmade wire chain of rings (1.5 mm in diameter) prepared with a 0.011-inch ligature wire passing through the osseous tunnel. During surgery, the chain was attached to a button or bracket bonded on the ectopic canine, passing through the bone onto the oral cavity (Crescini et al. 1994).

Bedoya et al. (Bedoya and Park 2009) described several other orthodontic techniques for extrusion of ectopic maxillary canines as follows:

2.1.3.1. Cantilever system

This is an orthodontic procedure with the advantage of generating predictable tooth movements by very light force. It requires less periodic activation and the anchor teeth must be closely monitored to avoid cantilever-related side effects.

2.1.3.2. Temporary anchorage devices (TAD's)

These devices produce absolute anchorage and the brackets can be bonded after the canine is brought into a better position. The main disadvantages are the extra cost and the need for surgical insertion and removal.

2.1.3.3. Double archwire

The double archwire minimizes maxillary lateral incisor root resorption and allows for horizontal tooth movement.

2.1.3.4. Auxiliary arm from a transpalatal arch

This system is characterized by a simple design and is very easy to activate. However, it tends to break easily and a laboratory procedure is necessary for its fabrication.

2.1.3.5. Auxiliary spring

No laboratory assistance is needed for this system. It allows complete control of the direction of eruption in addition to the amount of force delivered and avoids damage to adjacent teeth. Its main disadvantage is extra chair time to bend the spring.

Other less intricate but efficient methods have been described and include the use of removable appliances with a fingespring, magnets, elastic threads, nickel-titanium coils and elastomeric chains. The main disadvantages from removable appliances include the necessity of patient cooperation, limited control of tooth movement and an inability to treat complex problems (Bishara 1992).

Last but not least, the direction and the force applied to move an ectopic canine are crucial aspects that have been extensively under study in the literature. The use of minimal force (no more than 2 ounces or 60 grams) is recommended to move an ectopic canine. Its direction should be strategic to help to move the tooth away from the adjacent teeth. Other recommendations included an appraisal of the space available and progression to a stiff base archwire capable of resisting deformation by the forces applied to extrude the canine (Bishara 1992).

2.1.4. Methods of canine attachment

Crowns, wire ligatures, chain links, bands and bonded brackets are the most common methods of attachment reported in the literature (Bishara 1992). The use of a circumferential, dead soft, ligature wire (lasso) as an attachment around the cervical area of the tooth is another common procedure. However, with the use of this technique, an excessive amount of bone needs to be removed so the wire can be placed around the tooth cervical circumference. In addition, such a location increases the risk of damage to the adjacent teeth, root resorption (8% to 14%) and ankylosis. According to Bishara (Bishara 1992), this approach should be discouraged and a bonded bracket or button used.

2.1.5. Clinical outcomes and stability following orthodontic treatment of ectopic maxillary canines

Becker et al. (1983) evaluated the posttreatment alignment of ectopic canines and observed an increase in rotation and spacing on the affected side in 17.4% of the cases compared with 8.7% on the non-affected side (Becker et al. 1983).

D'Amico et al.(2003) evaluated the esthetics and function as well as the periodontal condition in 61 children. Only four were not satisfied with the final esthetic result whilst Orthodontists judged 44% of the results as esthetically compromised. In 11 cases, lateral incisors had to be extracted because of extensive root resorption (D'Amico et al. 2003).

Crescini et al. (2007) studied the posttreatment outcome of a large sample of ectopic canines and reported a lack of significant association between age, sex, site of impaction and duration of treatment. In addition, they also evaluated the periodontal status in the form of pocket depth and width of keratinized tissue following surgical flap and orthodontic traction. The authors concluded that the periodontium remained healthy after treatment. There were no prognostic indicators of the final periodontal status of repositioned canines (Crescini et al. 2007b). However, conflicting results from other authors showed greater pocket depths and a significantly lower crestal bone height around treated ectopic canines when compared to their counterparts (Woloshyn et al. 1994). A reduced width of attached gingiva also was reported when compared to their contralateral canine (Vermette et al. 1995).

Despite the development of techniques and appliances to bring the impacted canine into position, treatment failure has been reported in the literature(Becker et al. 2010). The multiple reasons for failure could be divided into three groups: a) the patient: lack of compliance, abnormal morphology of impacted teeth, root resorption of adjacent teeth; b) the orthodontist: diagnosis location, inadequate anchorage, appliance, torque and ankylosis; and c) the surgeon: diagnosis location, injury of the impacted canine and injury of the adjacent teeth surgery without orthodontic planning. An inaccurate 3-dimensional diagnosis location and failure to deliver proper anchorage have been reported as the major reasons for treatment failure (Becker et al. 2010).

2.2. The dental pulp and its alterations in response to orthodontic treatment

This section aims at providing the reader with a general knowledge on the dental pulp and its alterations in connection with orthodontic tooth movement, therein including extrusion of ectopic canines.

2.2.1. Histological characteristics of the dental pulp

Confined to a reduced space, the pulp histology basically consists of cells, nerves and vessels.

2.2.1.1. Pulpal cells

The dental pulp is a soft tissue of mesenchymal origin composed by several tissue elements such as: axons, vascular tissue, connective tissue fibers, interstitial fluid, odontoblasts, fibroblasts, immunocompetent cells (Torabinejad et al. 2014).

2.2.1.2. Pulpal innervation

The teeth are innervated by the fifth cranial nerve (trigeminal nerve), which is divided into three branches. The maxillary (V2) branch innervates the upper jaw whereas the mandibular (V3) branch innervates the lower jaw. The dental pulp is a highly innervated tissue and contains sensory trigeminal afferent axons. The axons enter the pulp through the apical foramen ramifying and following the distribution of the blood supply. The nerve bundles reach the coronal dentin where they form the nerve plexus of Raschkow. There, the sensory fibers anastomose and terminate as free nerve endings (Gopikrishna et al. 2009). Free sensory nerve endings in mature teeth are found in the peripheral plexus of Raschkow, the odontoblastic layer, the predentin, and the dentin. Free nerve endings are the most numerous in those regions near the tip of the pulp horn, where more than 40% of the dentinal tubules can be innervated (Abd-Elmeguid and Yu 2009a; Gopikrishna et al. 2009).

Teeth are innervated by unmyelinated sympathetic axons (C-fiber), and by small myelinated sensory axons A-fibers (A-delta, and A-beta) (Abd-Elmeguid and Yu 2009a). The A-delta fibers are located at the pulp-dentin border in the coronal portion and pulp horns. They represent 90% of the A fibers. The A-delta fibers have lower electrical thresholds than the C-fibers. They respond to a different stimulus that does not activate the C-fibers, but rather mediate acute pain (Olgart 1996). The C-fibers have a high threshold and can be activated by heating or cooling of the tooth crown. In addition, they mediate a dull, burning, poorly located pain and are activated by a stimulus that reaches the pulp. These fibers are related with tissue injury and are modulated by inflammatory mediators, vascular changes in blood flow and volume increase in pressure (Heyeraas and Berggreen 1999).

The dental pulp is regulated by sympathetic and parasympathetic fibers. The influence of parasympathetic fibers seems to be small (Olgart 1996) and there is no consensus about their role. The sympathetic nerves regulate the blood flow by vasoconstriction under the influence of noradrenaline and neuropeptide Y (Olgart 1996).

2.2.1.3. Pulpal vascularization

The largest vessels to enter the apical foramen are arterioles that are branches of the inferior alveolar artery, the superior posterior alveolar artery, or the infraorbital artery. Within the radicular pulp, the arterioles travel in the direction of the crown where they become tight and lose their muscle sheath before forming a capillary bed. Efferent blood vessels are formed by venules, which are somewhat larger than the analogous arterioles. Venules are formed from the junction of venous capillaries. The venules and the arterioles exit at the apical foramen to drain posteriorly into the maxillary vein through the pterygoid plexus or anteriorly into the facial vein. The lymphatic vessels arise as small thin-walled vessels in the margin of the pulp. They pass through the pulp to exit as one or two larger vessels through the apical foramen. There are spaces in the walls of these vessels and in their basement membranes. This porosity permits the passage of interstitial tissue fluid and lymphocytes. The lymphatic system helps in the elimination of inflammatory exudates and cellular debris. After exiting from the pulp, some vessels connect with analogous vessels from the PDL and drain into regional lymph glands (Torabinejad et al. 2014).

2.2.2 Pulpal alterations at the cellular and biochemical levels

The initial vascular reactions during the inflammation are vasodilatation and increased vessel permeability. Both reactions cause increased pulpal volume (Olgart 1996). In the dental pulp, the inflammatory vascular reactions take place in a rigid enclosed dentin chamber. Due the lack of distensibility, any gain in pulpal volume will increase the pulpal tissue pressure. There are two conditions which may increase the fluid volume in any tissue: 1. an increase in blood volume due to increase blood flow or venous statis and 2. an increase in interstitial fluid volume caused by raised capillary filtration or fall in lymph flow (Heyeraas and Berggreen 1999).

The increase in pulpal tissue pressure may compress blood vessels leading to pulp ischemia and necrosis. However, elevations in tissue fluid pressure remain localized to the injured area. A short distance from the injury, tissue fluid pressure is maintained within normal limits. As interstitial fluid pressure rises, the intraluminal (internal) pressure of the local capillaries increases to maintain the vessels patent. The gradients by which nutrients and wastes leave and enter the capillaries change to allow greater exchange. At the same time these changes occur in the capillaries, and lymphatic vessels become more active in removing the excess of tissue fluid and debris (Heyeraas and Berggreen 1999). In addition, anastomoses in the microvascular bed allow blood to be shunted around an area of injury, so that the oxygenation and nutrition of nearby uninjured tissue are not compromised. If the cause of the injury is removed, these processes gradually revert and repair or regeneration can take place. If the injury persists and increases in size, the tissue undergoes necrosis (Torabinejad et al. 2014).

It has been reported that stimulation of neurons results in peripheral vasodilatation. In 1927, Lewis (Lundy and Linden 2004) described the classic "triple response" where a mechanical stimulus on the skin resulted in a sequence of events as follows: 1. capillary vasoconstriction as a result of the mechanical stimulus, 2. vasodilatation around the injury as a consequence of arteriolar dilatation and 3. edema due to local arteriolar permeability resulting in healing.

Euler and Gaddum in 1930 (Lundy and Linden 2004) were the first to describe a substance able to decrease blood pressure named substance P (SP). Later research concluded that substance P was a neurotransmitter released from axon-reflex with a direct action on the inflammatory process. Since then, the term “neurogenic inflammation” has been used to describe this association of events (Lundy and Linden 2004). In 2004, Lundy and Linden (Lundy and Linden 2004) summarized the main neuropeptide family of neurotransmitters as follows: a) Substance P causes vasodilatation by acting directly on smooth muscle cells, b) neurokinin A increases microvascular permeability and edema formation, resulting in plasma protein extravasation, c) calcitonin gene-related peptide (CGRP) is a powerful vasodilator and has immunosuppressive properties acting on macrophage and leucocytes, d) vasoactive intestinal peptide (VIP) has immunosuppressive properties, stimulates the production of anti-inflammatory cytokine IL 10 and suppresses T-cell proliferation, and e) neuropeptide Y is a strong vasoconstrictor and amplifies other vasoconstrictors such as noradrenaline and promotes vessel-sprouting and endothelial cell proliferation (Lundy and Linden 2004). In 1988, it was reported for the first time that neurogenic inflammation could act on periodontal ligament as result of orthodontic forces (Vandevska-Radunovic 1999). Fristad et al. in 1997(Fristad et al. 1997) observed that the stimulation of nervous fibers in the dental pulp could release substance P and CGRP resulting in vasodilatation and an increase in blood flow. Those neuropeptides also increased vascular permeability leading to an increase in vascular filtration.

2.2.3 Pulpal alterations in response to orthodontic forces

Pulpal cellular and histological changes have been reported in the literature due to different forces (0.3N to 4.4N) and movement directions (intrusion, extrusion and tipping) applied on teeth during orthodontic treatment (von Bohl et al. 2012).

Intrusion movements can cause vascular alterations, increase the presence of fibrosis and pulp calcifications (Lazzaretti et al. 2014). In addition, they can lead to a vacuolization and disruption of osteoblasts (Han et al. 2013; Ramazanzadeh et al. 2009; Stenvik and Mjor 1970).

An increased expression of CGRP was found during orthodontic extrusion and tipping with forces ranging from 0.56N to 2.24N (Caviedes-Bucheli et al. 2011). In addition, it has been reported that CGRP can cause discomfort during orthodontic treatment (Chavarria-Bolanos et al. 2014). Likewise, Parris et al. also found an increase in substance P in patients undergoing orthodontic treatment (Parris et al. 1989). GCRP and substance P are potent vasodilators and mediators of neurogenic inflammation (Lundy and Linden 2004) and are related to vascular changes.

Derringer and Linden (Derringer et al. 1996) have reported an increase in microvessels and angiogenic growth factors on teeth undergoing orthodontic movement when compared to a control group. The possible explanation for their result could be a reduction in blood flow leading to hypoxia. According to

Niklas et al. (2013), areas of compression were formed on the periodontal ligament with a total or partial occlusion of blood vessels reducing the blood flow (hypoxia). Subsequently, hypoxia induced the formation of the active transcription factor HIF-1 and activated the vascular endothelial growth factor (VEGF) known as one of the most important mitogen to induce angiogenesis (Niklas et al. 2013). It has been reported that the periodontal ligament, the alveolar bone and the dental pulp act as a unit during orthodontic movement and any change to the periodontal ligament can affect the pulp (Vandevska-Radunovic 1999; Vandevska-Radunovic et al. 1994).

Moreover, cell damage, inflammation, vascular stasis, depression of the respiration rate, reduced alkaline phosphatase activity, apoptosis, vacuolization of odontoblasts and tissue damage have been reported as pulpal reactions to the application of orthodontic forces (von Bohl et al. 2012).

It has been reported in the literature that the application of orthodontic forces to a tooth can cause alterations in cells of the periodontal ligament, the alveolar bone and the dental pulp (Vandevska-Radunovic 1999). Overall, the research in this field has primarily focused on histological alterations and modifications in pulpal blood flow.

Clinically, not much data is available in the literature on the prevalence of pulp damage due to orthodontic movement. Some authors have observed a lack of vitality from poorly controlled orthodontic forces (Hamilton and Gutmann 1999; von Bohl et al. 2012), incisor retraction in monkeys (Butcher and Taylor 1952;

von Bohl et al. 2012), extrusive orthodontic movements (Mostafa et al. 1991) (Yamaguchi and Kasai 2007) and intrusion followed by extrusion in dogs (Turley et al. 1984). In a systematic review, Javed et al. (2015) concluded that there is insufficient information about the influence of orthodontic force on the human dental pulp (Javed et al. 2015).

2.2.4 Clinical methods to diagnose pulpal alterations

The ideal pulp test should be a simple, objective, standardized, reproducible, non-painful, non-injurious, accurate and inexpensive way to assess the health of the pulp tissue. Different methods have been used to evaluate pulp sensitivity.

2.2.4.1. Sensitivity tests

Thermal and electric pulp tests (EPT) are used on a daily basis in dental practice as they can assess the response to a given stimulus. To evaluate pulpal blood flow, the use of Laser Doppler Flowmetry and pulse oximetry have been described in the literature (Abd-Elmeguid and Yu 2009b; Jafarzadeh 2009).

The sensitivity tests assess the integrity of A-delta nerve fibers, which are located inside the pulp, by applying a stimulus on the tooth surface. If the fibers are stimulated, the patient will respond with short, sharp sensation/tingling from the tooth and it means that the nerve fibers are functioning. However, they do not provide any information on the blood flow. If there is no vascular supply to the pulp, the A-delta fibers will cease to function and no response will occur when the patient is stimulated. It should be noted that following a dental trauma the A-delta nerve fibers will not respond to any stimulus despite a blood flow being present (Abd-Elmeguid and Yu 2009b).

2.2.4.1.1. Thermal tests

To determine sensitivity by means of thermal changes cold and heat are applied to the tooth surface.

The cold test causes contraction of fluid within the dentinal tubules resulting in a rapid outward flow of fluid within the dental tubules. The rapid movement of dentinal tubes fluid results in hydrodynamic forces acting on A-delta fibers thereby producing a sharp sensation lasting for the entire time of the test (Oskui et al. 2014). Different methods of applying cold to a dental surface have been employed such as ice sticks (0^{o}C), sticks (-78^{o}C), ethyl chloride (-5^{o}C) and dichlorodifluoromethane (DDM, -50^{o} C). It has been reported that CO2 and DDM are more effective than ethyl chloride and ice sticks (Abd-Elmeguid and Yu 2009b), although the latter is much more commonly used. The cold test is a very common test used by practitioners and should be utilized in association with the EPT. If a mature and non-traumatized tooth does not respond either to cold or to the EPT, it shall be considered non-vital (Peters et al. 1994).

The heat test can be carried out using a stick of heated gutta-percha or hot water. The gutta-percha is heated and applied to the tooth surface. Prolonged heat application could result in a bi-phasic stimulation of A-delta nerves fibers in the first moment followed by the activation of C fibers. A lingering pain could result from the activation of the C fibers. Therefore, the heat test should not be applied for more than five seconds. It is important to use heat with caution to avoid damage to the pulp tissue (Abd-Elmeguid and Yu 2009b).

2.2.4.1.2. Electric pulp test (EPT)

The main objective of the EPT is stimulating myelinated A-delta nerve fibers in the pulp-dentin complex by using an electric current on the tooth surface. A positive response to the EPT represents an ionic shift in the dentin fluid within the tubules causing local depolarization and stimulation of A-delta fibers (Pantera et al. 1993). C fibers are not stimulated because of their higher threshold (Abd-Elmeguid and Yu 2009b). The EPT normality represents the neural stimulation and presence of vital nerve fibers, but it does not measure pulpal blood flow and its integrity. Although this technique is sensitive, it has some limitations such as the necessity for adequate stimulus, an appropriate application method and careful interpretation of the results. False positive results could be obtained when teeth that are partially necrotic give a positive response even if there is lack of blood flow. A false negative result can also be found when teeth are traumatized and they lose their sensory function temporarily, but show no response to the device even though their vascularity is intact (Lin and Chandler 2008).

2.2.4.2. Laser Doppler Flowmetry (LDF)

This technique is a non-invasive method to evaluate blood flow in the microvascular system. It was first described by Gazelius et al. (Gazelius et al. 1986). The LDF utilizes a beam of infrared light produced by a laser which is delivered by a probe in contact with the enamel surface that directs the laser beam toward the dental pulp. The light passes through the enamel, dentin and dental pulp tissue and is scattered and absorbed by moving red blood cells and stationary elements. Photons then interact with moving cells and shift the frequency according to the Doppler principle. A portion of the light is returned to the photon detector and a signal is produced. Gazilleus et al. (1996) have reported that the LDF is a reliable tool for differentiating between healthy and non-vital teeth (Gazelius et al. 1986).

The LDF has been used successfully to evaluate pulp vitality in adults and children, making a differential diagnosis of apical radiolucencies (on the basis of pulp vitality). It has also been employed to examine the pulp reaction to pharmacological agents or electrical and thermal stimulation, and monitor pulpal responses to orthodontic procedures and traumatic injuries (Ikawa et al. 2001; Jafarzadeh 2009; Salles et al. 2013; Sano et al. 2002).

The need for a LDF probe stabilization and contamination of the pulpal blood flow by gingival and periodontal signals (Soo-ampon et al. 2003) are some limitations that have been reported in the literature regarding this methodology (Soo-ampon et al. 2003).

2.2.4.3 Presence of fistula and/or radiographic lucency around the tooth

Needless to say, the presence of fistula clinically represents that the associated tooth underwent necrosis. The radiographic image of a periapical lesion, so many times detected as an incidental finding, also indicate that the involved tooth is non-vital. Furthermore, it can be used to confirm a suspicion of necrosis.

According to Chapman et at. (2013)(Chapman et al. 2013), 78% of all periapical lesions came from infections or inflammatory processes derived from pulpal or apical periodontal disease. Peterson et al. (2012) (Petersson et al. 2012), in a systematic review, reported that periapical bone lesions could be either of endodontic or nonendodontic origin. The authors debated if the characteristics of bone damage could provide information about the nature of the inflammatory process, so that the radiographic image could determine the appropriated treatment (surgery or root canal therapy). They stated that no conclusions could be drawn in this regard.

2.2.4.5 Alterations in blood flow

Laser Doppler Flowmetry has been used in different orthodontic movements to evaluate pulpal blood flow (PBF). The literature has focus on tooth movements such as intrusive with intermittent force(Barwick and Ramsay 1996; Brodin et al. 1996; Ikawa et al. 2001; Sano et al. 2002), extrusive with intermittent force(Brodin et al. 1996), tipping with continuous forces for three days(McDonald F.; Pitt Ford 1994), rapid palatal expansion(Babacan et al. 2010; Ozturk et al. 2003) and leveling and alignment for 30 days (Salles et al. 2013).

Different effects on PBF have been reported. Authors have shown an initial decrease of PBF during orthodontic movement(Brodin et al. 1996; Ikawa et al. 2001; Ozturk et al. 2003; Sabuncuoglu and Ersahan 2014; Salles et al. 2013; Sano et al. 2002). However, two authors have reported different results with an increase in PBF followed by recovery to normal PBF (Babacan et al. 2010; McDonald F.; Pitt Ford 1994) and one author has indicated that there was no change in PBF with orthodontic tooth movement(Barwick and Ramsay 1996).

An initial decrease in PBF can be explained by two hypotheses: 1- The decrease in blood flow immediately after the application of forces can be explained by the constriction of vessels that enter and leave the apical foramen through the action of dental dislocation. When afferent vessels are strangled, arteriolar resistance increases and the flow decrease (ischemia). When efferent vessels are constricted, the venular resistance is increased (passive hyperemia). 2- in an inflammatory process, vasodilatation and increased vascular permeability are initial reactions. In the pulpal chamber, both these reactions cause an increase in the interstitial pressure in response to the increased volume of pulpal fluid. Due to the low compliance of the pulpal environment, it has been speculated that the increased interstitial pressure could result in a vascular compression, thereby reducing blood flow (Olgart 1996; Vandevska-Radunovic 1999).

McDonald and Pitt Ford (1994)(McDonald F.; Pitt Ford 1994) explained that the increase in blood flow could be the result of a reactive hyperemia whereas Babacan et al. (2010)(Babacan et al. 2010) attributed this increase to an inflammatory process.

The LDF is a sensitive technique that can be influenced by probe design and position, tooth isolation and gingival contamination. The non-pulpal signal capture especially by the gingival area could lead to a misinterpretation of the flux and final blood flow and the results from LDF should be interpreted with caution(Jafarzadeh 2009; Sano et al. 2002).

2.2.5. Studies linking the orthodontic treatment of ectopic maxillary canines to alterations in pulp sensitivity and/or necrosis.

There are only a few orthodontic studies that link ectopic maxillary canines to pulp sensitivity and/or necrosis.

Woloshyn et al. (1994) evaluated 32 patients with unilateral impacted canines and reported that eight canines responded negatively to the EPT whereas all other teeth responded positively (Woloshyn et al. 1994). One canine was diagnosed with a necrotic pulp for which endodontic treatment was necessary. Three canines with partial pulpal obliterations and other three canines with total pulp obliteration also were observed. No pulp obliteration was found in the control group.

Blair et al. (1998) evaluated 30 canines with unilateral or bilateral impactions. The unilateral maxillary impacted group had the contra lateral canine as control and for the bilateral maxillary impacted group the upper central incisors served as control. Tooth vitality was assessed with the EPT grading from 1 to 10, being 10 marked as non-vital. Periapical radiographs were taken to evaluate the lamina dura, the pulp chamber, root canals, and the root structure. The results showed one tooth with abnormal pulp chamber and root structure in the impacted group. The unilateral or bilateral impacted canines had higher scores on the EPT when compared to the control group (Blair et al. 1998).

D'Amico et al. (2003) studied 83 impacted canines where 31 were bucally displaced, 41 palatally displaced and 11 centrally displaced. They used 39 normally erupted canines as control. The vitality test was performed with the EPT from the maxillary right canine to the maxillary left canine. They found that four maxillary impacted canines did not respond properly to the EPT. Those teeth belonged to the group where the canines were impacted bilaterally and had been transplanted. In addition, two maxillary incisors showed lowered vital responses (D'Amico et al. 2003).

Ferreira et al. (2013) evaluated 32 maxillary impacted canines submitted to orthodontic traction and compared them to a control group with 32 maxillary canines that had never been submitted to orthodontic treatment. They used the cold test (Endo-Frost) to estimate canine sensitivity in association with the periapical radiographic exam for the teeth showing no response to cold. They reported that 14 (43.8%) teeth from the experimental group had lack of pulp sensitivity in comparison to only one (3.1%) tooth in the control group (Ferreira et al. 2013).

Evren et al. (2014) evaluated the tooth vitality of 30 maxillary impacted canines that were palatally (15) and bucally (15) displaced and used the contralateral canine as control. Tooth vitality was measured by the EPT grading 1 to 10, being 10 non-vital. They found that palatally displaced canines had a higher EPT score when compared to the bucally displaced canines (Evren et al. 2014).

In general, it was noted a lack of standardization in terms of the methodology used to evaluate the pulp response of maxillary ectopic canines submitted to orthodontic treatment, and that is why conclusive results cannot be presented, thereby instigating the need for further investigation.

No literature was found correlating lack of pulp sensitive in ectopic maxillary canines with gingival recession, type of bracket/slot, bleaching, grinding, oral habits and lateral excursions.

Chapter 3

Objectives

The primary aim of this retrospective study was to evaluate if orthodontically treated ectopic maxillary canines would be more predisposed to lack of pulp sensitivity.

The secondary aims focused on assessing the influence of the initial position of orthodontically treated ectopic maxillary canines and their treatment duration as well as reporting the frequency of external clinical and radiographic variables that could also influence on the final pulpal status.

Chapter 4

Null Hypotheses

This research aimed at testing the following null hypotheses:

1-That there is no statistically significant difference between ectopic and non-ectopic the maxillary canines in regard to pulp sensitivity.

2-That there is no statistically significant difference between sensitive and non-sensitive ectopic maxillary canines in regard to their alpha angle.

3-That there is no statistically significant difference between sensitive and non-sensitive ectopic maxillary canines in regard to their initial distance from the tip to the occlusal plane.

4-That there is no statistically significant difference between sensitive and non-sensitive ectopic maxillary canines in regard to duration of traction (in months).

In order to better understand the characteristics of the sample in this study, it was also aimed at describing the frequency of clinical and radiographic variables capable of influencing on pulpal status.

A list containing the variables of interest follows below:

1) Ectopic canine initial position (buccal or palatal)

2) Presence of gingival buccal recession on ectopic canines

3) Type of surgery to expose the ectopic canines

4) Type of brackets used during orthodontic treatment and their slot size

5) History of dental trauma

6) History of dental bleaching

7) Report of dental clenching during daytime

8) Report of dental grinding at night

9) Report of spontaneous dental pain on the ectopic canines

10) Report of oral habits such as opening bottles, cans or biting on any other object using the maxillary canines

11) Presence of gingival pockets greater than 4 millimeters

12) Type of lateral excursion

13) Mesiodistal relationship of the tip of the maxillary ectopic canines and the long axis of their adjacent maxillary lateral incisor: before, at and beyond the long axis

14) Presence of pulp calcification

Chapter 5

Material and Methods

5.1Ethics approval

This research was approved by The Bannatyne Campus Research Ethics Board (ethics file number H2013:311).

5.2 Sample size calculation

The sample size calculation was based on a previous research published by Ferreira et al. (2013) where the authors found a statistically significant difference in the incidence of lack of sensitivity cases in the group of ectopic canines (43.8%) in comparison to the group of non-ectopic canines (3.1%). The power of the test was set at 80% at a significance level of 5% for alpha. The sample size calculator suggested a number of 16 subjects for each group: experimental and control. This sample size calculation method was previously described elsewhere (Rosner 2011)

A 9.0 statistical package (SPSS for Windows; SPSS, Chicago,Ill) was used to store and analyze the data.

5.3 Sample selection

5.3.1 Inclusion and exclusion criteria

All patients enrolled in the University of Manitoba Orthodontic Graduate Clinic and from one orthodontic private practice in Winnipeg, Manitoba, were screened for unilateral or bilateral ectopic maxillary canines situated at or above the cement-enamel junction of their adjacent maxillary lateral incisor. As part of the inclusion criteria, orthodontic extrusion should have been carried out to move the tooth down to its correct position. Pre-treatment panoramic radiographs and intra-oral photographs were used to determine if the maxillary canines fulfilled the aforementioned inclusion criteria. Patient charts were utilized to determine the type of surgical exposure and which orthodontic brackets and techniques had been used.

The exclusion criteria were based on the presence of systemic conditions that could alter bone status such as prescribed medications, the use of bisphosphanates, bone diseases, Paget's disease, radiotherapy and hypothyroidism. Occlusal factors such as signs of abfraction and/or abrasion also served as exclusion criteria.

5.3.2 Patient recruitment

Out of 3200 patients enrolled in the Division of Orthodontics at the University of Manitoba, 31 individuals fulfilled the inclusion criteria and qualified for study. Of these, 16 (10 females and 6 males) agreed to participate. Of the 15 individuals that were excluded, two were unable to locate, eight had moved away, one was unable to book an appointment due to transportation issues, one no longer qualified due to canine extraction, one had deceased, one had incomplete records, and one patient was unavailable during the hours of operation of the university clinic.

It was possible to recruit and recall four patients from a private practice in Winnipeg. In total 20 patients participated in this study: 16 from the University of Manitoba and four from the private practice that agreed to collaborate.

5.4 Data collection

5.4.1 Pretreatment radiographic measurements

The measurements on the pretreatment panoramic radiographs followed previously established protocols (Alessandri Bonetti et al. 2011; Baccetti et al. 2011; Crescini et al. 1994; Crescini et al. 2007a; Ericson and Kurol 1988; Fleming et al. 2009). They aimed at assessing the following variables: 1. the mesial inclination of the crown of the maxillary ectopic canines to the midline, also known as the alpha angle (α) and 2. the linear distance (D) from the cusp tip of the maxillary ectopic canine to the occlusal plane. A line drawn from the mesial cusps of the permanent maxillary first molars to the incisor edge of the permanent maxillary central incisors on the same side represented the occlusal plane. The panoramic X-Ray magnification factor at the University of

Manitoba had already been previously measured and found to be of 30%. Whenever the magnification factor was unknown, such as in those patients recruited from private practice, the equation proposed by Blum and Smith (2002)(Blum IR 2002) was used to make the necessary adjustments.

5.4.2 Recall appointment logistics

All participants were invited to revisit the University of Manitoba Orthodontic clinic or the collaborating orthodontic private practice after completion of their orthodontic treatment in order to undergo a pulp sensitivity test on their maxillary canines. Protocol questionnaires and charts were developed (See appendix) to document patient medical history, collect orthodontic data, and store radiographic information as well as make an endodontic diagnosis.

The data collection was divided into parts and followed a specific sequence. At first, a research assistant (C.K.) collected personal information in a private area. After this, a dental student (M.R.) directed the patient to a dental chair where all questionnaires were filled. The dental student started with questions one to fifteen in Questionnaire 1 (See appendix). In the sequence, an orthodontic resident (O.K.) inquired on questions one to thirteen in Questionnaire 2 (see appendix). A certified endodontist (R.C.) performed the thermal and the EPT tests as well as filled the Endodontic Questionnaire (see appendix). After all such procedures, the patient was dismissed. An orthodontic resident (A.S.) filled Questionnaire 3 (see Appendix) and measured all linear and angular variables related to the initial position of the maxillary ectopic canines. 5.4.3 Endodontic examination

An experienced certified endodontist assessed pulpal sensitivity by thermal, electrical and percussion tests. A periapical radiograph was obtained in cases with an inconclusive diagnosis. The homologous non-ectopic tooth that served as control also was evaluated. There was no control in cases of bilateral maxillary ectopic canines.

5.4.3.1. Thermal and percussion tests

For the thermal test, the maxillary hemi-arch was isolated with cotton rolls. Endo-Frost (Coltene-Roeko, Germany) refrigerated gas was then applied with a cotton pellet, firstly on the canine, then on the lateral incisor, and finally on the first premolar. The patient's sensory response to the contact between the tooth and the refrigerated cotton was recorded as either positive or negative.
The percussion test was performed using a clinical mirror handle by tapping on the incisal edge of the maxillary canine. The response was recorded as either positive or negative for discomfort pain during percussion.

5.4.3.2 Electric pulp test

Once thermal and percussion tests were completed, the same aforementioned methodology to isolate the hemi-arch was used to perform the electric pulp test by using an EPT equipment manufactured by Analytic

Technology Corporation, Redmond, WA. The teeth were dried, and the tip of the EPT placed on the incisal third of the crown using toothpaste as conducting medium. A positive response was recorded when the patient's left hand was raised. If no sensation was observed when the reading instrument reached the level 80, the tooth was recorded as necrotic until proved otherwise by means of radiographic analysis (Abd-Elmeguid A 2009).

5.4.3.3. Radiographic examination

Patients with no pulp sensitivity were referred for a periapical radiograph using periapical films (Kodak – New York, USA) and film positioners according to the paralleling technique. A radiographic analysis was then pursued to determine the occurrence of any periapical lesion, periodontal ligament thickening or pulp calcification (Ferreira LL 2013).

5.5 Statistical Analysis

In most occasions, the descriptive statistics was comprised of the following parameters: number of teeth evaluated, mean, standard deviation, median, minimum, maximum, interquartile range and confidence interval.

Instead of a chi-square test, a mixed-effects logistic regression model was preferred to assess the relationship between tooth status (ectopic and non-ectopic) and pulp diagnosis (sensitive and non-sensitive), with pulp diagnosis as the dependent variable. This modeling approach accounts for the correlation between teeth nested within the same patient, a statistical feature of the data that arises from the nearly “split mouth” design, something quite usual in Dentistry.

The Shapiro-Wilk test detected lack of a normal distribution for the alpha angle, Distance (D) and duration of traction in months. It was possible to appraise the error of the method using the Spearman`s rank correlation coefficient test for two of these variables (alpha angle and distance (D) by re-measuring them on all patients (100% of the sample) two weeks apart. This was accomplished by two independent calibrated examiners (A.S. and Y.H.).

Despite falling short of the recommended sample size when compared to the sample size reported elsewhere, Crescini et al. (2007)(Crescini et al. 2007b), the Kruskal-Wallis test was used to analyze the influence of all three quantitative variables (alpha angle, distance (D) and duration of traction).

All aforementioned analyses were performed by SAS version 9.3 (SAS Institute, Cary NC) with a level of significance of 5%.

The frequency of all other variables was tabulated and presented in the form of observational data only.

Chapter 6

Results

6.1 Error of the method

The intra-examiner agreement varied from high to perfect for the linear ($\rho=0.994$) and angular ($\rho=1.00$) measurements, respectively. The inter-examiner agreement was high for both linear ($\rho=0.971$) and angular ($\rho=0.999$) measurements. All correlations were statistically significant with $P < 0.01$.

6.2 General Descriptive statistics

6.2.1 Sample Characteristics

The sample was comprised of twenty subjects and consisted of seven males (35%) and 13 females (65%) with the majority being of a Northern European background (Table 3). The mean age of the patients at the beginning of the treatment was 16.7 ± 7.8 years. The mean age at the end was 20.1 ± 7.8 years and 24 ±7.2 at the recall. On average, the orthodontic treatment lasted 3.4 ±1.14 years. Six patients presented with bilateral ectopic canines and 14 patients with unilateral ectopic canines. According to Angle classification, 21 canines were in Class I, 18 in Class II (1/4 cusp), and only one in Class III. Canine guidance was the type of lateral excursion on 12 teeth whereas group function was observed on 14 teeth.

Of the forty teeth evaluated, 26 maxillary canines were ectopic and 14 were non-ectopic, thereby serving as control (Table 7). The prevalence of ectopic canines that were displaced bucally was 53.84% (14 teeth) and palatally 46.16% (12 teeth). No ectopic canine had a physical obstruction on its eruption pathway. All of them were located above the cementoenamel junction (CEJ) of the fully erupted adjacent lateral incisor. They presented all with normal anatomy and no endodontic or periodontal lesions at the beginning of the treatment. At the end of the treatment, only one patient presented with root resorption on a maxillary lateral incisor, but this was not considered a reason for exclusion.

Of the forty maxillary canines evaluated, 38 were at the same level of the incisal edges of the maxillary central incisors at the end of treatment. No decay was diagnosed. There were no incisal edge fractures, but 19 teeth had craze lines. Eleven teeth showed gingival recession and none palatal recession. Eight presented with periodontal pockets greater than 4 mm and four relapsed when compared to photographs taken immediately after debonding.

Out of the 40 maxillary canines evaluated, two had history of trauma. None of them needed root canal therapy. Four teeth had some composite restorations done after the orthodontic treatment. Three teeth presented sensitivity during tooth brushing, seven when exposed to cold or hot food, and other seven teeth were reported to have short spontaneous pain from no specific cause.

Forty-five percent of the subjects had bleached their teeth in a dental office or at home under a dentist's supervision. Twenty-five percent reported clenching their teeth while awake and 25% informed that they used to grind their teeth while sleeping.

6.2.2 Orthodontic mechanotherapy

Different mechanotherapies were used for traction of the 26 maxillary ectopic canines. The two major approaches were: 1. power chain on eight teeth (30.76%), 2. NiTi archwire on other eight teeth (30.76%), 3. stainless Steel ligatures on five teeth (19.23%) and 4. NiTi piggy-back on two teeth (7.69%) (Table 1). A cantilever was used on one tooth (3.85%), a NiTi coil-spring on another tooth (3.85%) and one more tooth (3.85%) was corrected by means of a supercable archwire (Table 1).

Table 1: Frequency of types of traction methods used to extrude the ectopic maxillary canines

Method	Frequency n (%)
Power-chain	8 (30.76))
NiTi archwire	8 (30.76)
Stainless Steel ligature	5 (19.23)
NiTi piggy-back	2 (7.6)
Cantilever	1 (3.85
Supercable archwire	1 (3.85)
NiTi coil-spring	1 (3.85)

6.2.3 Maxillary arch retention protocol

A Hawley retainer was prescribed for eighteen patients (90%) whereas a bonded retainer for two patients (10%) (Table 2).

Table 2: Frequency of the type of retainer

Appliance	Frequency n (%)
Hawley	18 (90)
Bonded	2 (10)

6.2.4 Endodontic outcome

No fistula was observed on any of the teeth evaluated. All teeth were considered normal according to the percussion test and palpation of the apical cortical plates.

With regard to the thermal test, 14 teeth showed delayed response to heat (Table 3), other 14 to cold (Table 4), and 12 to the EPT test (Table 5). A periapical radiograph was needed on 18 teeth to confirm the diagnosis (Table 6). Of these, 14 were diagnosed as non-sensitive (Table 7).

Table 3: Outcome of the heat test

Heat test	Ectopic n (%)	Non-ectopic n (%)	Frequency n (%)
Normal	14 (56)	11 (44)	25 (62.5)
Exacerbated	1 (100)	0 (0)	1 (2.5)
Delayed of sensitivity	11 (78.5)	3 (21.5)	14 (35)

Table 4: Outcome of the cold test

Cold test	Ectopic n (%)	Non-ectopic n (%)	Frequency n (%)
Normal	13 (56.5)	10 (43.5)	23 (57.5)
Exacerbated	2 (66.6)	1 (33.4)	3 (7.5)
Delayed of sensitivity	11 (78.5)	3 (21.5)	14 (35)

Table 5: Outcome of the Electric pulp test (EPT)

EPT test	Ectopic n (%)	Non-ectopic n (%)	Frequency n (%)
Normal	14 (56)	11 (44)	25 (62.5)
Exacerbated	2 (66.6)	1 (33.4)	3 (7.5)
Delayed of sensitivity	10 (83.3)	2 (16.7)	12 (30)

Table 6: Frequency of periapical radiographs (PA) requested to complete the endodontic evaluation when the clinical diagnosis was deemed indeterminate

PA	Ectopic n (%)	Non-ectopic n (%)	Frequency n (%)
Yes	14 (77.7)	4 (22.3)	18 (45)
No	12 (54.5)	10 (45.5)	22 (55)

6.3 Intergroup comparisons

The ectopic and the non-ectopic groups were compared statistically in terms of sensitivity, alpha angle, distance (D) and duration of traction. All other comparisons involving clinical and radiographic characteristics, which were collected either through the questionnaires or during clinical examination, were presented as observational background data.

6.3.1 Comparison between ectopic and non-ectopic maxillary canines in regards to tooth sensitivity

In order to test the null hypothesis that orthodontically treated ectopic maxillary canines would not be predisposed to lack of pulpal sensitivity, the frequency of cases with lack of pulpal sensitivity was compared between the ectopic and the non-ectopic contra-lateral teeth (control group).

No statistically significant association was found between ectopic maxillary canines and lack of tooth sensitivity (p= 0.0744). Of the 26 ectopic teeth evaluated, 14 (53.84%) presented as sensitive and twelve (46.16%) as non-sensitive. Of the 14 teeth evaluated in the control group, 12 (85.71%) were sensitive and only two (14.29%) were non-sensitive (Table 7).

Table 7: Comparison between ectopic and non-ectopic maxillary canines in regards to tooth sensitivity

Tooth status	Sensitive n (%)	Non-sensitive n (%)	Total n (%)	OR/ Pv
Ectopic	14 (53.84)	12 (46.16)	26 (65)	5.919/ 0.0744
Non- ectopic	12 (85.71)	2 (14.29)	14 (35)	
Total	26	14	40	

Level of significance = p< 0.05; Test = Mixed-effects logistic regression model; OR = Odds Ratio; Pv =p-value

6.3.2 Comparison between sensitive and non-sensitive ectopic maxillary canines in regards to their alpha angle (α)

In order to test the null hypotheses that there is no statistically significant difference between sensitive and non-sensitive ectopic maxillary canines in regards to the alpha angle, the medians of these two groups were compared.

No statistically significant difference was found between sensitive and non-sensitive ectopic maxillary canines in regards to their alpha angle (p=0.0661). Of the 26 ectopic teeth evaluated, 14 (53.84%) were sensitive with a median angulation of 24.95 degrees whereas 12 (46.16%) were non-sensitive with a median angulation of 23.7 (Table 8, Figure 1).

Table 8: Comparison between sensitive and non-sensitive ectopic maxillary canines in regards to their alpha angle

	n (%)	Mean	SD	Median	Min	Max	IR	95%CI	Pv
Sensitive	14 (53.84)	26.79	17.31	24.95	1.8	56.4	13.2-39.4	16.79-36.79	0.0661
Non-sensitive	12 (46.16)	24.48	20.08	23.70	2.1	60	6.3-35.9	11.72-37.24	

Test = Kruskal Wallis (p= 0.0661); n= number of teeth; ST= Standard deviation; Min= Minimum; Max= Maximum; IR = Interquartile range; CI= Confidence interval; Pv =p-value

6.3.3 Comparison between sensitive and non-sensitive ectopic maxillary canines in regards to their initial linear distance (D) from the tip to the occlusal plane

In order to test the null hypotheses that there is no statistically significant difference between sensitive and non-sensitive ectopic maxillary canines in regards to distance (D), the medians of these two groups were compared.

No statistically significant difference was found between sensitive and non-sensitive ectopic maxillary canines in regards to the distance within which they moved until the occlusal plane (p=0.4840). Of the 26 ectopic teeth evaluated, 14 (53.84%) were sensitive with a median distance of 10.80

millimeters, and 12 (46.16%) were non-sensitive with a mean distance of 11.30 millimeters (Table 9, Figure 1).

Table 9: Comparison between sensitive and non-sensitive ectopic maxillary canines in regards to their initial linear distance from the tip to the occlusal plane

	n (%)	Mean	SD	Median	Min	Max	IR	95%CI	Pv
sensitive	14 (53.84)	12.90	5.27	10.80	6.15	25	10.2-14.2	9.72-16.09	0.4840
Non-sensitive	12 (46.16)	11.07	3.63	11.30	6.70	17.5	7.47-13.3	8.64-13.52	

Test = Kruskal Wallis (p= 0.4840); n= number of teeth; ST= Standard deviation; Min= Minimum; Max= Maximum; IR = Interquartile range; CI= Confidence interval; Pv =p-value

6.3.4 Comparison between sensitive and non-sensitive ectopic maxillary canines in regards to duration of traction (in months)

In order to test the null hypotheses that there is no statistically significant difference between sensitive and non-sensitive ectopic maxillary canines in regards to the time taken to extrude them until the line of occlusion, the medians of these two groups were compared.

Statistically significant difference was found between sensitive and non-sensitive ectopic maxillary canines in regards to duration of traction in months (p=0.0437).

Of the 26 ectopic teeth evaluated, 14 (53.84%) were sensitive with a median duration of 5.01 months and 12 (46.16%) were non-sensitive with the median duration of 5.66 months (Table10).

Table 10: Comparison of the duration of traction (in months) between sensitive and non-sensitive ectopic maxillary canines

	N (%)	Mean	SD	Median	Min	Max	IR	95%CI	Pv
sensitive	14 (53.84)	5.01	2.04	5.0	1.2	8.0	2.2-3.4	2.13-3.36	0.0437*
Non-sensitive	12 (46.16)	5.66	2.96	5.0	2.0	11.0	3.0-4.1	2.50-4.30	

Test = Kruskal Wallis (p= 0.0437); n= number of teeth; ST= Standard deviation; Min= Minimum; Max= Maximum; IR = Interquartile range; CI= Confidence interval; Pv =p-value

6.3.5 Frequency of clinical and radiographic variables capable of affecting the pulp status (sensitive or non-sensitive) of ectopic maxillary canines

The information herein presented is a report containing observational background data only and was presented in the form of frequency distribution (%).

6.3.5.1 Pulpal status of the ectopic maxillary canines in regards to their initial location (buccal or palatal)

Of the 26 ectopic teeth evaluated, 14 (53.84%) teeth, nine sensitive and five non-sensitive, were bucally displaced whereas 12 (46.15%) teeth, five sensitive and seven non-sensitive, were palatally displaced (Table11).

Table 11: Frequency distribution of pulp status of the ectopic maxillary canines according to their initial location (buccal or palatal)

	Buccal n (%)	Palatal n (%)	Total n (%)
Sensitive	9 (64.29)	5 (35.71)	14 (53.84)
Non-sensitive	5 (41.67)	7 (58.33)	12 (46.16)
Total	14	12	26

6.3.5.2 Pulpal status of the ectopic maxillary canines in regards to the presence of posttreatment gingival buccal recession

Of the 14 ectopic teeth with normal sensitivity, seven (50%) presented with gingival buccal recession whereas the other seven (50%) did not. Of the 12 ectopic teeth lacking sensitivity, two teeth (16.67%) presented with gingival buccal recession whereas the other ten (83.33%) did not (Table 12).

Table 12: Frequency distribution of pulp status according to the presence of posttreatment gingival buccal recessions on the ectopic maxillary canines

	Gingival buccal recession n (%)	Non gingival buccal recession n (%)	Total n (%)
Sensitive	7 (50)	7 (50)	14 (53.84)
Non-sensitive	2 (16.67)	10 (83.33)	12 (46.16)
Total	9	17	26

6.3.5.3 Pulpal status of the ectopic maxillary canines in regards to the type of surgical exposure (open or closed) performed

Of the 26 ectopic teeth evaluated, no sensitive tooth had been exposed by means of the closed technique. Open exposure had been performed on 12 sensitive teeth (85.71%). Two canines (14.29%) exposed themselves spontaneously. Two non-sensitive teeth (16.67%) had been exposed by the closed technique, eight (66.76%) by the open exposure technique and two (16.67%) exposed themselves spontaneously (Table 13).

Table 13: Frequency distribution of the pulpal status according to the type of surgical exposure (open, closed or spontaneous) of the maxillary ectopic canines

	Closed exposure n (%)	Open exposure n (%)	Spontaneous exposure n (%)	Total n (%)
Sensitive	0 (0)	12 (85.71)	2 (14.29)	14 (53.84)
Non-sensitive	2 (16.67)	8 (66.67)	2 (16.67)	12 (46.16)
Total	2	20	4	26

6.3.5.4 Pulpal status of the ectopic maxillary canines in regards to the type of orthodontic brackets used

Of the 26 ectopic teeth evaluated, one sensitive tooth (7.14%) had been treated with .022” slot conventional brackets, six (42.86%) with .018” slot conventional brackets, and seven (50%) with .022” slot self-ligating brackets. Five non-sensitive teeth (41.67%) were treated with .022” slot conventional brackets, other five (41.67%) with .022” slot self-ligating brackets, and two (16.66%) with .018” slot self-ligating brackets (Table 14).

Table 14: Frequency distribution of pulpal status according to the type of orthodontic brackets used

	.022” Conventional brackets n (%)	.018” Conventional brackets n (%)	.022” Self-ligating brackets n (%)	.018” Self-ligating brackets n (%)	Total n (%)
Sensitive	1 (7.14)	6 (42.86)	7 (50)	0 (0)	14 (53.84)
Non-sensitive	5 (41.67)	0 (0)	5 (41.67)	2 (16.67)	12 (46.16)
Total	6	6	12	2	26

6.3.5.5 Pulpal status of the ectopic maxillary canines in regards to history of dental trauma

Of the 26 ectopic teeth evaluated, no trauma was reported on 14 sensitive teeth (100%) whereas there was a report of trauma in two (16.67%) non-sensitive teeth. On two non-sensitive teeth (16.67%), the patients were unsure whether trauma had occurred (Table 15).

Table 15: Frequency distribution of pulpal status according to history of dental trauma to the ectopic maxillary canines

	No trauma n (%)	Trauma n (%)	Unsure n (%)	Total n (%)
Sensitive	14 (100)	0 (0)	0 (0)	14 (53.84)
Non-sensitive	8 (66.66)	2 (16.67)	2 (16.67)	12 (46.16)
Total	22	2	2	26

6.3.5.6 Pulpal status of the ectopic maxillary canines in regards to history of dental bleaching

Of the 26 ectopic teeth evaluated, seven sensitive teeth (35.71%) had been bleached whereas nine (64.29%) had not. Six non-sensitive teeth (50%) had been bleached whereas six (50%) had not (Table 16).

Table 16: Frequency distribution of pulpal status according to history of dental bleaching

	Bleached n (%)	Non-bleached n (%)	Total n (%)
Sensitive	5 (35.71)	9 (64.29)	14 (53.84)
Non-sensitive	6 (50.0)	6 (50.0)	12 (46.16)
Total	11	15	26

6.3.5.7 Pulpal status of the ectopic maxillary canines in regards to history of daytime dental clenching

Of the 26 ectopic teeth evaluated, four sensitive teeth (28.57%) belonged to patients who reported clenching while awake whereas nine (64.29%) did not. Two non-sensitive teeth (16.67%) belonged to patients who reported clenching while awake whereas seven (50%) did not (Table 17).

Table 17: Frequency distribution of pulpal status according to history of daytime dental clenching

	Clenching during daytime n (%)	Not Clenching during daytime n (%)	Unsure n (%)	Total n (%)
Sensitive	4 (28.57)	9 (64.29)	1 (7.14)	14 (53.84)
Non-sensitive	2 (16.67)	7 (58.33)	3 (25.0)	12 (46.16)
Total	6	16	4	26

6.3.5.8 Pulpal status of the ectopic maxillary canines in regards to history of nighttime dental grinding

Of the 26 ectopic teeth evaluated, five sensitive teeth (35.71%) belonged to patients who reported grinding while sleeping whereas nine (64.29%) did not. Two non-sensitive teeth (16.67%) were of patients who reported grinding at night whereas eight (66.67%) were not (Table 18).

Table 18: Frequency distribution of pulpal status according to nighttime dental grinding

	Grinding at night n (%)	Not Grinding at night n (%)	Unsure n (%)	Total n (%)
Sensitive	5 (35.71)	9 (64.29)	0 (0)	14 (53.84)
Non-sensitive	2 (16.67)	8 (66.67)	2 (16.67)	12 (46.16)
Total	7	17	2	26

6.3.5.9 Pulpal status of the ectopic maxillary canines in regards to history of spontaneous dental pain

Of the 26 ectopic teeth evaluated, one sensitive tooth (7.14%) was reported to have spontaneous pain whereas 13 (92.86%) were not. Four non-sensitive teeth (33.33%) reported spontaneous pain whereas eight (66.67%) did not (Table 19).

Table 19: Frequency distribution of pulpal status according to history of spontaneous dental pain

	Reported pain n (%)	Did not report pain n (%)	Total n (%)
Sensitive	1 (7.14)	13 (92.86)	14 (53.84)
Non-sensitive	4 (33.33)	8 (66.67)	12 (46.16)
Total	5	21	26

6.3.5.10 Pulpal status of the ectopic maxillary canines in regards to history of opening bottles, cans or biting on any other object using any of the maxillary canines

Of the 26 ectopic teeth evaluated, two sensitive teeth (14.29%) belonged to patients who reported such habits whereas 12 (85.71%) did not. Four non-sensitive teeth (33.33%) belonged to patients who reported these habits whereas eight (66.67%) did not (Table 20).

Table 20: Frequency distribution of pulpal status according to history of opening bottles, cans or biting on any other object using the maxillary canines

	Reported habit n (%)	No reported habit n (%)	Total n (%)
Sensitive	2 (14.29)	12 (85.71)	14 (53.84)
Non-sensitive	4 (33.33)	8 (66.67)	12 (46.16)
Total	6	20	26

6.3.5.11 Pulpal status of the ectopic maxillary canines in regards to the presence of gingival pockets greater than 4 millimeters

Of the 26 ectopic teeth evaluated, nine sensitive teeth (64.29%) had no pocket greater than 4 millimeters whereas five (35.71%) did. Nine non-sensitive teeth (75%) had no pocket greater than 4 millimeters whereas three (25%) did (Table 21).

Table 21: Frequency distribution of pulpal status according to presence of gingival pockets greater than 4 millimeters

	No Gingival pocket n (%)	Gingival pocket n (%)	Total n (%)
Sensitive	9 (64.29)	5 (35.71)	14 (53.84)
Non-sensitive	9 (75.00)	3 (25)	12 (46.16)
Total	18	8	26

6.3.5.12 Pulpal status of the ectopic maxillary canines in regards to the type of lateral excursion

Of the 26 ectopic teeth evaluated, seven sensitive teeth (50%) participated in canine guidance whereas the other seven (50%) participated in group function. Five non-sensitive teeth (41.67%) were found to participate in canine guidance whereas seven (58.33%) participated in group function (Table 22).

Table 22: Frequency distribution of pulpal status according to the type of lateral excursion

	Canine guidance n (%)	Group function n (%)	Total n (%)
Sensitive	7 (50.0)	7 (50.0)	14 (53.84)
Non-sensitive	5 (41.67)	7 (58.33)	12 (46.16)
Total	12	14	26

6.3.5.13 Pulpal status of the ectopic maxillary canines in regards to the type of mesiodistal relationship of the tip of the ectopic maxillary canines with the long axis of their adjacent maxillary lateral incisor

Of the 26 ectopic teeth evaluated, seven sensitive teeth (50%) had the canine tip beyond the long axis of the adjacent maxillary lateral incisor, four (28.57%) at the long axis and three (21.43%) before the long axis. Six non-sensitive teeth (50%) had the canine tip beyond the long axis of the adjacent maxillary lateral incisor, two (16.67%) at the long axis and four (33.33%) before the long axis (Table 23).

Table 23: Frequency distribution of pulpal status according to the mesiodistal relationship of the tip of the ectopic maxillary canines with the long axis of their adjacent maxillary lateral incisor

	Beyond the long axis n (%)	In the long axis n (%)	Before the long axis n (%)	Total n (%)
Sensitive	7 (50.0)	4 (28.57)	3 (21.43)	14 (53.84)
Non-sensitive	6 (50.0)	2 (16.67)	4 (33.33)	12 (46.16)
Total	13	6	7	26

6.3.5.14 Frequency distribution of pulp calcification on ectopic and non-ectopic non-sensitive maxillary canines

Of the 14 teeth diagnosed as non-sensitive, three ectopic teeth and one non-ectopic tooth presented with pulp calcification according to the radiographic examination (Table 24).

Table 24: Frequency distribution of pulp calcification on ectopic and non-ectopic non-sensitive canines

	Without calcification n (%)	Calcified n (%)	Total n (%)
Ectopic non-sensitive	9 (75)	3 (25)	12 (85.72)
Non-ectopic non-sensitive	1 (50)	1 (50)	2 (14.28)
	10	4	14

The table below (table 25) presents a summary of the aforementioned comparisons between ectopic sensitive and non-sensitive maxillary canines submitted to orthodontic treatment.

Table 25: Summary of all observational comparisons between sensitive and non-sensitive ectopic maxillary canines in regards to the clinical and radiographic variables of interest

Variables	**Ectopic Sensitive n (%)**	**Ectopic Non-sensitive n (%)**	**Pv**
Alpha angle (mean)	26.79	24.48	(p= 0.0661)+
Linear distance (D) in mm (mean)	12.9	11.07	(p= 0.4840)+
Duration of traction (in months) (mean)	5.0	5.66	(p= 0.0437)*
Initial position			
Bucally displaced	9 (64.29%)	5(41.67%)	
Palatally displaced	5(35.71%)	7(58.33%)	
Recession			
Buccal recession	7(50%)	2(16.67%)	
No buccal recession	7(50%)	10(83.33%)	
Surgical exposure			
Close exposure	0(0%)	2(16.67%)	
Open exposure	12(85.71%)	8(66.67%)	
Spontaneous eruption	2(14.29%)	2(16.67%)	
Type of bracket			
Conventional bracket, .022" slot	1(7.14%)	5(41.67%)	
Conventional bracket, .018" slot	6(42.86%)	0(0%)	
Self-ligating bracket, .022" slot	7(50%)	5(41.67%)	
Self-ligating bracket, .018" slot	0(0%)	2(16.67%)	
Dental trauma			
Reported dental trauma	0(0%)	2(16.67%)	
No dental trauma	14(100%)	8(66.66%)	
Unsure about dental trauma	0(0%)	2(16.67%)	
Dental bleaching			
Reported bleaching	5(35.71%)	6(50%)	
No bleaching	9(64.29%)	6(50%)	
Daytime clenching			
Reported clenching	4(28.57%)	2(16.67%)	

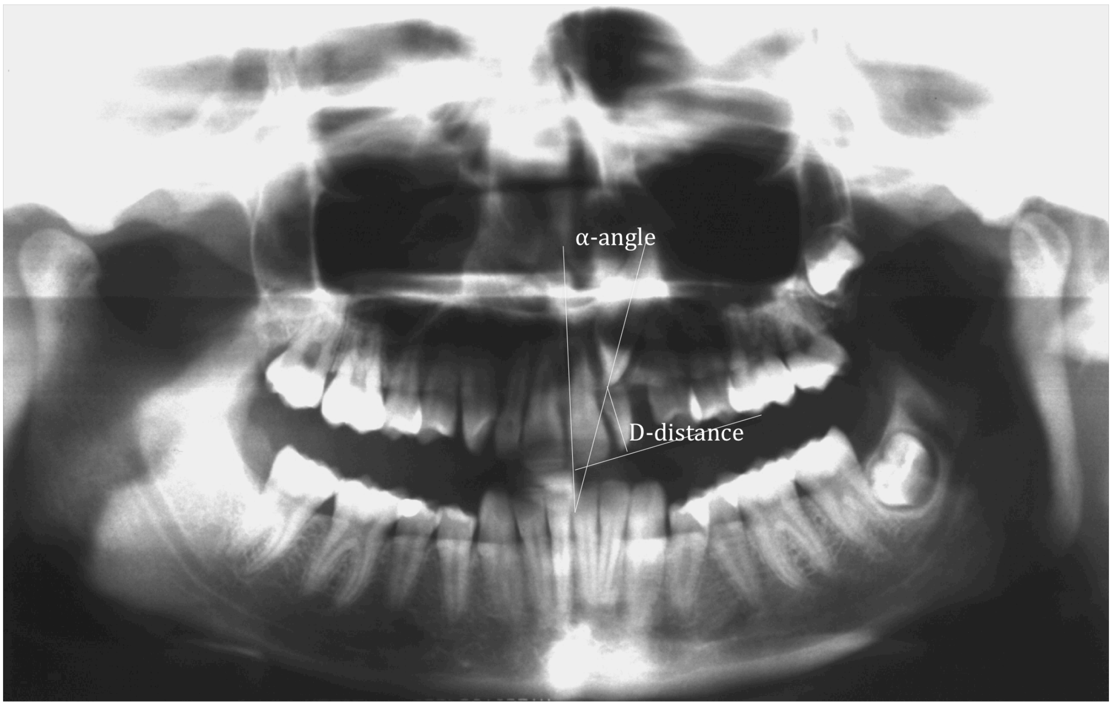
No clenching	9(64.29%)	7(58.33%)	
Unsure about clenching	1(7.14%)	3(25%)	
Nighttime grinding			
Reported grinding	5(35.71%)	2(16.67%)	
No grinding	9(64.29%)	8(66.67%)	
Unsure about grinding	0(0%)	2(16.67%)	
Spontaneous dental pain			
Reported pain	1(7.14%)	4(33.33%)	
Reported no pain	13(92.86%)	8(66.67%)	
Habit to bite on canines			
Reported habit	2(14.29%)	4(33.33%)	
No habit	12(85.71%)	8(66.67%)	
Gingival pocket			
Greater than 4 mm	9(64.29%)	9(75%)	
No pocket	5(35.71%)	3(25%)	
Lateral excursion			
Canine guidance	7(50%)	5(41.67%)	
Group function	7(50%)	7(58.33%)	
Relationship to the adjacent incisor			
Tip beyond the long axis	7(50%)	6(50%)	
Tip at the long axis	4(28.57%)	2(16.67%)	
Tip before the long axis	3(21.43%)	4(33.33%)	

Hx= History; Pv = p-value; Significant if p< 0.05*; test = Kruskal Wallis+

Table 26: Summary of the main characteristics of the sample

Characteristics	n (%)
Gender	
Male	35%
Female	65%
Ethnicity	
Caucasian	80%
Hispanic	10%
Indian	5%
Asian	5%
Age (years)	
Start of treatment	16.7
End of treatment	20.1
Mean treatment time (years)	3.4
Ectopic unilateral	70%
Ectopic bilateral	30%
Angle classification	
Class I	52.5%
Class II	45%
Class III	2.5%

Figure 1: Panoramic radiograph showing the alpha angle and the D-distance in one of the patients included in the study



Chapter 7

Discussion

7.1. Clinical relevance

Patients now participate actively in selecting treatment options and the process in obtaining informed consent relies on the premise that evidence-based information was provided to them in a concise and clear way. When weighting the pros and cons to either extract or force the eruption of an ectopic maxillary canine, it seems reasonable not only to question the periodontal prognosis but also the endodontic prognosis. Loss of pulpal vitality incur in additional financial expenses and colour change that might compromise esthetics, not to mention the associated discomfort, a burden that must be added to the inconvenience related to the use of anchorage devices and complex mechanics during the orthodontic treatment.

Five previous studies (Blair et al. 1998; D'Amico et al. 2003; Evren et al. 2014; Ferreira et al. 2013; Woloshyn et al. 1994) investigated an association between orthodontic traction of ectopic maxillary canines and lack of pulpal sensitivity. Ferreira et al.(2013) analyzed 32 ectopic maxillary canines submitted to orthodontic treatment, but used untreated maxillary canines as control. Although their sample size seemed adequate, the lack of control within the same individual did not allow to isolate the effect of being ectopic from the effect of being treated orthodontically. D’Amico et al. (2003) (D'Amico et al. 2003) worked with a much larger sample of 83 ectopic maxillary canines submitted to orthodontic treatment, but they focused on esthetic, functional and periodontal conditions rather than on pulpal response. The same occurred to the study published by Evren et al. (2014) (Evren et al. 2014), which was centered on periodontal parameters, being the electric pulp testing the only endodontic evaluation that was performed.

Two studies (Blair et al. 1998; Woloshyn et al. 1994) were outstanding in terms of methodology. Although Blair et al. (1998) used the split-mouth design and a sample size of 15 teeth, their patients were treated for a period of one year, which is substantially shorter than most treatments of this kind. It has been reported that the average treatment time for ectopic maxillary canines ranges from 18-30 months and some factors can delay the treatment such as the number of missed appointments, the number of replaced brackets and bands, the number of treatment phases, the number of negative chart entries regarding oral hygiene, headgear cooperation, the number of extracted premolars, the pretreatment mandibular plane angle, pretreatment ANB angle and the age at the start of treatment (Stewart et al. 2001). The mean for orthodontic treatment time was 39 months in this study, with a mean of 5.6 months for the traction of the impacted canines.

Woloshyn et al. (1994) (Woloshyn et al. 1994) perhaps presented the best study design so far with a split-mouth analysis of 32 canines. However, the authors relied solely on the electrical test (EPT) and the mean age of the sample was of 22 years, apparently much higher than what is commonly seen in North America when most cases are diagnosed in the early permanent dentition (Richardson and Russell 2000).

It has been reported that thermal tests were more reliable (Alomari et al. 2011; Cave et al. 2002) than the EPT test to evaluate pulp sensitivity. Alomari *et al.* (2011) compared EPT and cold test results in 47 subjects before, during orthodontic treatment and in the retention period for 12 months with Hawley appliances. They concluded that the EPT threshold increases during the application of orthodontic forces and returns to pre-initial values at the end of the retention phase. In our research, similarly to what was described by Woloshyn et al. (1994) (Woloshyn et al. 1994) and D’Amico et al.(2003) (D'Amico et al. 2003), all data was collected in average 3 years after the end of treatment, so no bias on EPT results should be expected in our sample. In addition, the diagnosis of lack of sensitivity was a combination of the result from EPT, thermal test and periapical radiograph such as recommended by Cave *et al.* (2002) (Cave et al. 2002), Alomari *et al.*(2011) (Alomari et al. 2011), Lin & Chandler (2008) (Lin and Chandler 2008) and Torabinejad (2014) (Torabinejad et al. 2014). According to Torabinejad (2014), the cold test has a sensitivity (ability to detect pulp necrosis that is verified clinically) of 75% and specificity (ability to identify a normal pulp) of 92%. The sensitivity of EPT was reported to be of 92%, and the specificity 75%.

In all the aforementioned studies (Blair et al. 1998; D'Amico et al. 2003; Evren et al. 2014; Ferreira et al. 2013; Woloshyn et al. 1994), sample selection was not so stringent, being difficult to reduce the number of variables. Except for the study done by Woloshyn et al. (1994) (Woloshyn et al. 1994), the influence of anatomical conditions such as the mesiodistal tipping of the tooth (alpha angle) and the distance within which it was moved until its final position were not taken into account. In most cases, potential lurking factors like type of exposure surgery, presence of deep periodontal pockets, severe gingival recession, history of grinding and/or bleaching were not described. Only Woloshyn et al. (1994) (Woloshyn et al. 1994) reported the presence of pulp calcification as a possible adverse effect from correcting ectopic maxillary canines.

The present study differs from the previous ones in the more stringent way how patients were selected and for having collected information on clinical and radiographic variables of interest, thereby describing the sample in a more comprehensive and transparent way. It is far from having a perfect methodology and lacks sufficient sample size in the control group, but can indeed confirm some of the previous findings and stimulate further research on this topic.

7.2. Sample, study design and measured outcome

Although the null hypotheses would be better tested by means of a randomized controlled clinical trial with a split-mouth design, such type of study also would fall short in terms of controlling operator bias. In the present study, the limitations of a retrospective study certainly apply due to the difficulty in collecting accurate clinical data from archived records, relying on information provided by patients and not having control over the type of orthodontic mechanics used, not to mention that all patients received treatment from different providers, most of them in a university setting under the supervision of multiple clinical instructors.

Despite not being a perfect split-mouth model, given that 12 patients presented with bilateral ectopic maxillary canines, the aforementioned limitations were partially compensated by having the majority of patients a contralateral homologous non-ectopic maxillary canine as control. Additional non-ectopic orthodontically treated canines could have been added to the control group, but this could introduce new types of bias.

Even though the calculated sample size of at least 16 patients was met for the group of ectopic canines, the non-ectopic group was short of only two teeth. This is another limitation of this study, which may have affected statistical power. On the other hand, given the scarcity of this type of sample due to stringent inclusion criteria and need of recall appointments, the opportunity to describe this data could not be taken for granted as this type of exercise can sometimes reveal information of clinical relevance or instigate future studies with a “per protocol” sample size.

Lurking variables certainly are an issue when conducting a retrospective study. Past history of dental trauma, clenching, dental bleaching and others, besides being variables that are impossible to control, can potentially influence on the final outcome. Even if statistical tests had been utilized to detect their influence, the shortcomings resulting from a reduced sample size would have rendered such tests invalid. The same would be true if constructing a model by logistic regression to evaluate the combined effect of such variables.

Notwithstanding the difficulty in addressing all issues related to the presence of multiple variables, descriptive frequency data was tabulated and the outstanding findings discussed. It is important to remind that the sample size of 16 teeth per group was calculated based on the data presented by Ferreira et al. (2013) where the authors compared two groups: ectopic and non-ectopic orthodontically treated canines. As theirs and the majority of the previous studies (Blair et al. 1998; D'Amico et al. 2003; Evren et al. 2014; Ferreira et al. 2013; Woloshyn et al. 1994) did not evaluate the influence of anatomical variables, let alone clinical variables either reported by the patients or collected during clinical examination, it was not possible to define a sample size appropriate for an inter-subgroup analytical statistical test within the ectopic group. This is the main reason why this type of data was presented in the form of frequency and can now serve as a parameter to calculate the sample size in future studies with a similar design.

It is important to point out that our study aimed at investigating what really happens in the clinical setting when deciding on the necessity of a root canal treatment, something that usually is based on methods such as periapical radiographs, thermal and electrical tests. For this reason, use of Laser Doppler Flowmetry was not considered in the current methodology, hence we cannot claim that our study evaluated pulpal vitality as a dependent variable. Instead, lack of pulpal sensitivity was the assessed outcome. The term pulpal vitality refers to the direct assessment of blood supply, and Laser Doppler Flowmetry is the technique used to evaluate the presence or absence of blood flow (Alghaithy and Qualtrough 2016; Gazelius et al. 1986; Jafarzadeh 2009).

There were also cases of obliterated pulp canals in which thermal and electrical methods are not appropriate to evaluate pulpal vitality. In teeth that have undergone different degrees of pulp calcification, no response to EPT may be found (Hamilton and Gutmann 1999). This could explain the lack of sensitivity on four calcified impacted maxillary canines found in this research (subjects 1, 2, 3 and 4, Appendix 2) as well as on six impacted teeth and two controls reported by Woloshyn *et al.* (1994).

7.3. Analysis of the hypotheses

All the hypotheses in this study were analyzed below by making a specific scientific question.

7.3.1. Are ectopic maxillary canines prone to pulpal sensitivity?

The first hypothesis aimed at testing if ectopic maxillary canines would be more susceptible to showing lack of pulpal sensitivity at the posttreatment follow-up. Although the mixed-effects logistic regression model did not detect a statistically significant difference between ectopic and non-ectopic canines in regards to sensitivity, the frequency of non-sensitive ectopic canines (46.15%) was remarkably higher in comparison to non-sensitive non-ectopic ones (14.29%). Considering that the p-value was 0.07, a value approximate to the statistically significant cut-off of 0.05, and that the odds ratio was quite high (5.92), it is correct to assume that orthodontically treated ectopic maxillary canines seems to be at greater risk of losing their pulpal sensitivity. Although the observational data already provides us with clinically significant information, it is tempting to assume that a larger sample size in both groups would likely show the presence of a statistically significant difference.

Our findings were very similar to the ones reported by Ferreira et al.(2013) (Ferreira et al. 2013) who evaluated 32 maxillary impacted canines using the cold test in addition to a periapical radiograph and found that 14 teeth (43.8%) did not respond to a cold stimulus in the ectopic group in comparison to one tooth (3.1%) in the non-ectopic group. In the same line, D'Amico et al. (2103)(D'Amico et al. 2003), Woloshyn et al. (1994) (Woloshyn et al. 1994), and Evren et al. (2014) (Evren et al. 2014) also observed pulpal alterations including necrosis following orthodontic treatment of ectopic maxillary canines. Woloshyn et al. (1994) (Woloshyn et al. 1994) found 8 (25%) maxillary impacted canines that did not respond to the EPT. Blair et al. (1998) (Blair et al. 1998) also noticed a tendency for treated ectopic canines to undergo pulpal changes, but could not demonstrate this statistically perhaps due to the small sample size.

Despite lack of a statistically significant difference, the clinical relevance of the observational data points in the direction that this null hypothesis should be rejected.

7.3.2. Do anatomical characteristics and treatment time influence on the endodontic prognosis?

The second, third and fourth hypotheses in chapter 5 focused on anatomical parameters that could influence on the complexity of the orthodontic treatment, thereby increasing the chances of inducing pulpal alterations. These factors were the distance until full eruption, the alpha angle, and the duration of traction.

In our study, a statistically significant difference was observed for duration of traction ($p=0.0437$) when comparing between sensitive and non-sensitive ectopic canines. The period of active traction was of five months, similar to the one reported by Crescini et al. (2007) of eight months. It is well known that treatment time tends to increase with lack of compliance (Albino et al. 1991), poor biomechanics (Crescini et al. 2007b; Fleming 2015), and/or degree of treatment complexity (Crescini et al. 2007b; Fleming 2015). The first two factors were not within the scope of the present study whereas the latter was assessed by measuring the alpha angle (α) and the distance (D).

The alpha angle was studied by Woloshyn et al. (1994), but their sample size of 35 patients may not have been large enough to detect a statistically significant difference. In fact, when using the statistical parameters published by these authors (standard deviation of approximately 12° and mean difference of 6.3°) at 5% level of significance and power of 80%, a sample size of 58 patients would be necessary. Considering that our sample size does not meet this criterium, the results presented below concerning the second and third hypotheses, as described in chapter 5, demand cautious interpretation.

It has been reported that the initial position of an ectopic canine can be associated with treatment complexity and its duration (Crescini et al. 2007b; Fleming 2015). According to Crescini et al. (2007) (Crescini et al. 2007b), each 1 mm of the D-distance of the cusp of the ectopic canine from the occlusal plane requires roughly one more week of traction and every 5^o of opening of the alpha angle requires approximately one more week of traction. Stewart *et al.* (2001) (Stewart et al. 2001) reported that canines farther than 14mm from the occlusal plane take even more time to be corrected.

Statistically, there was no difference between sensitive and non-sensitive ectopic canines when it comes to such variable. As mentioned above, a small sample size can be the cuprit for such lack of statistical significance. This commonly happens when analyzing subgroups of a sample, what might have weakened the power of the test. However, when comparing the medians to the means (Tables 8, 9, and10), their values were found to be very similar, implying that the distribution would tend to become more and more normal with a larger sample. This, together with the fact that, except for the alpha angle, the standard deviations were generally low, it is tempting to assume that this result would probably not change with a larger sample size. Further investigation with a larger sample is therefore advisable.

7.3.3. Are background variables capable of influencing on the pulpal status of ectopic maxillary canines submitted to orthodontic force?

Due to sample size limitations, analytical statistics was not performed for the fourteen background variables for which data was collected. The main goal in collecting this type of data was not only to characterize the sample, but to observe visible discrepancies that could foster discussion and stimulate further research. Most of these variables, even in a prospective type of study, would be difficult to isolate, if not impossible. Given the retrospective characteristic of this data, any assumption or allusion shall be made with caution. Overall, such variables served to remind us that the topic under study -“lack of sensitivity in orthodontically treated ectopic canines” - encompasses multiple variables that can either magnify or reduce the effect of one another.

In order to appreciate the differences between such variables in each frequency table (Tables 25), one can focus on the frequency values that stand out either within or between the subgroups (sensitive and non-sensitive). For obvious reasons, this was easier with dichotomous variables, but not unfeasible when more than two conditions were present.

The observational data for each background variable will be discussed in the following lines.

7.3.3.1. Are palatally located canines more prone to pulpal alterations?

In our sample, it was possible to observe that the majority of sensitive canines were located bucally (64.29%) (Table 11). This finding is in agreement with the literature that has indicated that a higher level of treatment complexity normally is expected from cases were the canine is located in the palate. Evren *et al.* (2014) spent 38 months treating bucally displaced impacted canines and 42 months treating palatally displaced canines. These authors also reported that palatally displaced maxillary canines had a higher EPT score (non-sensitivity) when compared to bucally maxillary impacted canines. Therefore, given that their treatment mechanics tends to be less complex, one can hypothesize that buccally ectopic maxillary canines may be less prone to irreversible pulpal alterations than their palatally displaced counterparts.

7.3.3.2. Are canines with buccal recessions or deep pockets more prone to pulpal alterations?

Considering that the etiology of endo-perio lesions already is well established in the literature (Crescini et al. 2007b; Parkin et al. 2008; Vermette et al. 1995; Woloshyn et al. 1994), one would expect a higher frequency of lack of sensitivity on canines presenting with buccal gingival recession. Given that the great majority (83.33%) of non-sensitive canines had no buccal gingival recession (Table 12), our data could not corroborate this. Either there was a very small number of cases with buccal gingival recession or this is a low weighting factor that can be affected by a high weighting confounder such as initial canine location (buccal or palatal). A similar situation occurred in relation to the frequency of gingival pockets greater than four millimeters (Table 21). In comparison to the sensitive subgroup (64.29%), a higher frequency of absent pockets was observed in the non-sensitive subgroup (75.0%). Although the same aforementioned justifications may apply, the difference here was relatively small.

Similar to what we observed, Woloshyn *et al.* (1994), Blair *et al.* (1998) and D'Amico *et al.* (2003) did not find any correlation between the pocket depth, the type of surgery and EPT results when studying on maxillary ectopic canines.

Based on our observation, although it seems more logical to expect pulpal issues from periodontally compromised teeth, we cannot state, at this stage, that this is the case with ectopic maxillary canines.

7.3.3.3. Does the type of surgical exposure influence on pulpal sensitivity?

The proportion of canines exposed by the open exposure technique (Table 13) dropped from 85.71% in the sensitive subgroup to 66.67% in the non-sensitive subgroup. This finding seems to be the first to indicate that the utilization of this technique can lead to less episodes of lack of pulpal sensitivity. In the literature, there is some evidence showing little difference between an open versus closed surgical approach in respect to surgical time, treatment outcome and periodontal status (Fleming 2015; Parkin et al. 2013), but nothing has been published in regards to pulpal health.

From a clinical perspective, it seems relatively easy to accept the assumption that the open exposure technique may lead to less pulpal alterations than the closed technique given that the canines are left to erupt spontaneously for a period of six months or more. Spontaneous eruption is known to cause less or no damage in comparison to forced eruption (Kokich 2004; Schmidt and Kokich 2007).

7.3.3.4. Would different types of bracket systems also differ in terms of pulpal sensitivity when treating ectopic maxillary canines?

Here, an interesting observation can be made. There was an increase in the frequency of .022” slot conventional brackets in the non-sensitive subgroup (41.67%) in comparison to the sensitive subgroup (7.14%). The frequency of .018” slot conventional brackets dropped in the non-sensitive subgroup (0%) in comparison to the sensitive subgroup (42.86%). There was no appreciable difference in relation to the frequency of self-ligating brackets in both subgroups.

This data leads to reason in favor of .018” slot conventional brackets in detriment of .022” slot conventional brackets when it comes to preserving the status quo of the pulp. Conversely, the latter type of bracket usually is recommended in order to avoid wire distortion when forcing the eruption of ectopic canines(Bishara 1992).

The orthodontic literature has compared the effect of bracket slot on dental health when it comes to parameters such as root resorption, periodontal destruction, and loss of crestal bone height (El-Angbawi et al. 2014; Mavragani et al. 2000; Sameshima and Sinclair 2001). Sameshima et al. (2001) (Sameshima and Sinclair 2001) observed no association between bracket slot size (.018"and .022") and root resorption. Based on our clinical experience, an .022" slot seems to render the appliance with more biomechanical stability when correcting ectopic maxillary canines. However, our data indicated that the reaction elicited on the pulp seems to be milder with .018" slot brackets.

7.3.3.5. Would ectopic maxillary canines submitted to trauma be more predisposed to lack of pulpal sensitivity?

One of the topics that appear to be well consolidated in the literature is the association between trauma and lack of pulpal sensitivity (Abd-Elmeguid and Yu 2009a; 2009b; Olgart 1996). It is not uncommon for traumatized teeth to require endodontic intervention either shortly after the trauma or in the long-term (Saoud et al. 2016) This was supported by our data as the frequency of teeth with a history of trauma increased from 0% in the sensitive subgroup to 16.67% in the non-sensitive subgroup (Table 15). The cases with no previous history of trauma dropped from 100% in the sensitive subgroup to 66.66% in the non-sensitive subgroup.

However, 16% corresponds to one patient diagnosed with two ectopic canines who reported history of dental trauma, having both teeth lack of sensitivity. Being this a retrospective study, it was difficult to associate this finding with the origin, the severity and the specific site of the trauma.

Although it seems reasonable to assume that orthodontically corrected ectopic canines submitted to trauma would more likely show lack of sensitivity, our data was collected in a way that does not allow a definite conclusion.

7.3.3.6. Would ectopic maxillary canines submitted to dental bleaching be more predisposed to lack of pulpal sensitivity?

When comparing the subgroups in terms of dental bleaching, it was apparent that the frequency of cases of bleaching was higher in the non-sensitive group (50.0%) than in the sensitive group (35.71%). It is well known that the bleaching products commonly used in Dentistry can cause pulpal alterations that can vary from mild to severe (Soares et al. 2015). Bearing in mind that both subgroups (sensitive and non-sensitive) derived from the same group of ectopic canines, a combination of forced eruption and dental bleaching could certainly increase the frequency of insult to the pulp up to a point where lack of sensitivity could ensue. This hypothesis, although impossible to prove in a multivariable setting like this one, cannot be ignored.

7.3.3.7. Would ectopic maxillary canines submitted to daytime (clenching) and/or nighttime (bruxism) grinding be more predisposed to lack of pulpal sensitivity?

We are not aware of any clear-cut evidence supporting that daytime and/or nighttime bruxism can lead to lack of pulp sensitivity, especially on the canines. This perhaps tends to be true for posterior teeth susceptible to fracture like the highly restored ones. However, together with the fact that the ectopic canines underwent orthodontic traction, it is possible that being under frequent pressure from grinding could increase the odds of undergoing irreversible pulpal changes. For both daytime and nighttime grinding (Tables 17, 18), our data could not make a decisive distinction between the two subgroups as the frequencies were not outstandingly different. There are three possible explanations for this result: 1.a larger sample is required to prove an association, 2. an association simply does not exist, or 3. the data collection was grounded on such a subjective information that, in combination with a small sample, ended up losing its robustness. Considering that either the first and the third justifications or a combination of both might have occurred, our data in this regards is to be considered inconclusive.

7.3.3.8. Would ectopic maxillary canines present more often with spontaneous pain before losing their pulp sensitivity?

Spontaneous dental pain was nearly five times more frequent in the non-sensitive subgroup (33.33%) than in the sensitive subgroup (7.14%) (Table 19). Conversely, no history of spontaneous pain was more frequent in the sensitive (92.86%) than in the non-sensitive cases (66.67%). This makes us aware to the fact that orthodontically treated ectopic maxillary canines will likely experience pain or discomfort at some point along or after the treatment and symptoms can subside with no guarantee that normal sensitivity was re-established. We all know, not only based on research but also on clinical experience, that pulpal pain can sometimes resolve spontaneously and be followed by a painless process of chronic necrosis that can remain dormant for quite a long time(Torabinejad et al. 2014).

7.3.3.9. Would ectopic maxillary canines participating in lateral excursion be more predisposed to lack of pulpal sensitivity?

So far, only one study has partially investigated this topic. D'Amico *et al.* (2003) reported that canine protection occurred more often on normally erupted canines when compared to the impacted side and the main reason could be the higher inclination of bucally or palatally impacted canines. However, the authors did not associate the type of lateral excursion guidance with lack of sensitivity. In our study, either canine guidance or group function did not appear to play a role in the type of pulpal response elicited on the ectopic canines. The frequency of either type of excursion was equally represented in each subgroup (Table 22). This supposedly occurred because the anatomy of the maxillary canines is supposed to withstand higher forces, hence not making much difference in what type of lateral excursion they participate.

7.3.3.10. Would the degree of mesial tipping of the ectopic maxillary canines play any role in their pulpal sensitivity during or after orthodontic treatment?

Some articles have suggested that the greater the mesio-distal tipping of the ectopic maxillary canine in relation to the long axis of the adjacent lateral incisor, the lower are the chances for self-correction and the more complex the biomechanics becomes to correct them, not to mention the increased risk of damage to the adjacent teeth (Crescini et al. 2007a; Nieri et al. 2010).

Yet empirical, it is not unrealistic to theorize that more complex cases can indeed become more susceptible to either excessive compression or overstretching of the pulpal neurovascular bundle thereby increasing the chances of pulpal irreversible alterations such as pulpitis, necrosis or calcifications. Surprisingly, the frequency of cases whose tip was located beyond the long axis of the adjacent lateral incisor was very similar in both subgroups (sensitive and non-sensitive) (Table 23). The same happened to the frequencies for the cases located at or before the long axis, what implies that, at least in terms of sensitivity, this variable seems to be of less importance. Again, due to the co-existence of other relevant variables and the small sample in each subgroup, this finding must be interpreted with caution.

7.3.3.11. Would ectopic maxillary canines be more susceptible to pulp calcification?

Our study also looked into the occurrence of pulp calcification following orthodontic treatment of maxillary ectopic canines (Table 24). This usually is a topic that orthodontists tend to overlook. Firstly, we do not have the habit of looking at the pulp on radiographic images. Secondly, calcified teeth are nearly always asymptomatic. Being this the case, why would we care about this type of finding? Whenever an orthodontic treatment leads to pulp calcification, a big problem is then created for the endodontist. It can sometimes be quite difficult for endodontists to determine whether a calcified tooth is non-vital in the absence of unconventional diagnostic methods and lack of periodontal ligament alterations at the periapical level. This may require long-term endodontic follow-ups and even when treatment is recommended, it may become very complicated or even impossible without a retrograde obturation of the apical aspect of the canal.

There was only one case, in our study, of calcified non-ectopic non-sensitive maxillary canine (Table 24). Among the non-sensitive ectopic cases, three teeth were calcified. This corresponds to approximately twenty five percent of the sample of non-sensitive ectopic canines, what indicates that orthodontically treated ectopic canines may become predisposed to pulp calcification.

The specific cause of pulpal calcification in the studied population is impossible to determine due to the co-existence of all the aforementioned variables.

7.4. Summary of the main findings and their clinical significance

In general terms, this study suggests that orthodontically treated ectopic maxillary canines are more predisposed to lack of sensitivity. The best prognosis appears to be in cases of buccally ectopic canines, open exposure technique and when an .018" slot bracket system was used. In addition, one should expect that approximately twenty-five percent of ectopic maxillary canines can undergo pulpal calcification following orthodontic treatment. This is the second time after the publication by Woloshyn et al. (1994) (Woloshyn et al. 1994) that pulp calcifications are found to be more prevalent in orthodontically treated ectopic maxillary canines.

The initial mesio-distal relationship, alpha angle and distance to the occlusal plane did not seem to play a role whereas traction duration does, but the small sample size reminds us that this is not a definite conclusion.

It is never too much to emphasize that the findings derived from the 14 background variables studied were based on observational data, thereby requiring further investigation. Caution is therefore recommended when using this data in a clinical decision making process.

In general, this data serves as an alert for orthodontists treating ectopic maxillary canines. It not only suggests that an endodontic follow up shall be recommended at the end of this type of orthodontic treatment, but also implies that patients should be aware of all such risks before consenting to this type of treatment.

7.5. Suggestion for future studies

It would be ideal to conduct a randomized clinical controlled trial with a split-mouth design to confirm our findings. The intricate logistics involved and the difficulty to obtain a large sample are certainly the main reasons why such type of study has not yet been carried out.

Further recruitment of patients who fulfill the inclusion criteria for this study will proceed until September 2017 and can be extended depending on the renewal of the ethics board authorization at the University of Manitoba, Canada.

Chapter 8

Conclusions

Despite the limitations of the study methodology, it was possible to draw the following conclusions:

1- No statically significance difference was found between sensitive and non-sensitive maxillary canines in regards to parameters such as initial angulation (alpha angle), height (D distance). Canines submitted to a longer treatment time appeared to be more predisposed to lack of pulp sensitivity, and this was statistically significant. Due to the slightly small sample size, caution is recommended when interpreting this result.

2- Despite lack of a statistically significant difference, the proportion of non-sensitive ectopic canines (46.15%) was visibly higher than the proportion of non-sensitive non-ectopic canines (14.29%), suggesting that clinicians should be aware of potential pulpal side-effects when treating cases with ectopic maxillary canines.

3. Although based on observational data, buccally ectopic canines, open exposure technique and an .018" slot bracket system seemed to favor pulpal health.

4. Twenty-five percent of ectopic non-sensitive maxillary canines were diagnosed with pulpal calcification at the posttreatment follow-up.

Apendix

Appendix 1: Ages at different time points and the number of the maxillary canine(s) that was (were) ectopic

Patient Number	Age when extrusion started (Years)	Age when extrusion ended (Years)	Age at end of Treatment (Years)	Ectopic Canine
1	13	15	18	1.3, 2.3
2	14	16	21	1.3, 2.3
3	15	18	26	2.3
4	15	20	23	1.3
5	11	15	22	1.3
6	17	20	26	1.3, 2.3
7	15	20	24	1.3
8	16	20	21	2.3
9	13	16	20	1.3
10	14	17	20	1.3, 2.3
11	11	15	23	1.3
12	13	17	23	2.3
13	15	21	24	1.3
14	14	16	22	1.3
15	17	20	29	1.3
16	14	17	21	1.3, 2.3
17	48	51	51	1.3
18	22	26	26	1.3
19	18	19	20	1.3, 2.3
20	13	15	16	2.3
Mean	16.7	20.1	24.0	

Appendix 2: Overview of variables and pulp diagnoses (sensitive, non-sensitive)

Patient	Ectopic Canine/location	Hx of Trauma	Hx of Bleaching	Hx of Bruxism/ Clenching	Suspected Endodotic Lesion	Suspected Periodontic Lesion	Exposure Type	Pulp Calc.	Pulp Status
1	1.3/Buccal 2.3/Buccal	No No	No No	No No	No No	No No	Open Open	No Yes	1.3 Sensitive 2.3 Indeterminate due to pulp calcification
2	1.3/Buccal 2.3/Buccal	No No	Yes Yes	Bruxing & Clenching	No No	No No	Open Open	Yes No	1.3 Indeterminate due to pulp calcification 2.3 Sensitive
3	1.3/Control 2.3/Palatal	No No	Yes Yes	Patient unsure	No No	No No	NA Open	No Yes	1.3 Sensitive 2.3 Indeterminate due to pulp calcification
4	1.3/Palatal 2.3/Control	Yes Yes	No No	Bruxing & Clenching	No No	No No	Open NA	Yes No	1.3 Indeterminate due to pulp calcification 2.3 Non-Sensitive
5	1.3/Palatal 2.3/Control	No No	No No	No No	No No	No No	Open NA	No No	1.3 Non-Sensitive 2.3 Sensitive
6	1.3/Buccal 2.3/Buccal	No No	No No	Patient Unsure	No No	No No	Open Open	No No	1.3 Non-Sensitive 2.3 Non-Sensitive
7	1.3/Palatal 2.3/Control	No No	Yes Yes	No No	No No	No No	Open NA	No No	1.3 Sensitive 2.3 Sensitive
8	1.3/Control 2.3/Palatal	No No	Yes Yes	No No	No No	No No	NA Open	No No	1.3 Sensitive 2.3 Sensitive
9	1.3/Buccal 2.3/Control	No No	No No	Bruxing & Clenching	No No	No No	Open NA	No No	1.3 Sensitive 2.3 Sensitive

10	1.3/Buccal 2.3/Buccal	No No	No No	Bruxing	No No	No No	Open Open	No No	1.3 Sensitive 2.3 Sensitive
11	1.3/Buccal 2.3/Control	No No	No No	No No	No No	No No	Open NA	No No	1.3 Sensitive 2.3 Sensitive
12	1.3/Control 2.3/Palatal	Unsure	No No	No No	Yes No	No No	NA Open	No No	1.3 Non-Sensitive 2.3 Non-Sensitive
13	1.3 Palatal 2.3 Control	Unsure	Yes Yes	No No	No No	No No	Open NA	No No	1.3 Non-Sensitive 2.3 Sensitive
14	1.3 Palatal 2.3 Control	No No	No No	Patient Unsure	No No	No No	Open NA	No No	1.3 Sensitive 2.3 Sensitive
15	1.3 Palatal 2.3 Control	No No	No No	Clenching	No No	No No	Open NA	No No	1.3 Sensitive 2.3 Sensitive
16	1.3 Palatal 2.3 Palatal	No No	Yes Yes	No No	No No	No No	Closed Closed	No No	1.3 Non-Sensitive 2.3 Non-Sensitive
17	13 Palatal 23 Control	No No	Yes Yes	Bruxing & Clenching	No No	No No	Open NA	No No	13 Sensitive 23 Sensitive
18	13 Buccal 23 Control	No No	Yes Yes	No No	No No	No No	Open NA	No No	13 Non-Sensitive 23 Sensitive
19	13 Buccal 23 Buccal	No No	No No	No No	No No	No No	Open Open	No No	13 Sensitive 23 Sensitive

20	13 Control 2.3 Buccal	No No	Yes Yes	No No	No No	No No	NA Open	No No	1.3 Sensitive 2.3 Sensitive

Hx=History
NA=Non-applicable
Pulp calc= pulp calcification

Appendix 3: Questionnaire to assess eligibility and form for orthodontic data collection

Protocol Title: Tooth Sensitivity of Ectopic Canines Submitted to Orthodontic Traction
(This sheet must be separated from the data collection sheet upon completion)

Demographic data and identifiers
Contact information:
Name: ____
Address: ____
Postal code: ___________
Phone: (___) _______________
E-mail: ____
Gender: ( ) M ( ) F
Ethnicity: ( ) 1st Nation ( ) Northern European/Caucasian ( ) Hispanic ( ) Black ( ) Black/Mixed ( ) Indian ( ) Asian ( ) South European/Mediterranean ( ) Eastern European ( ) Middle East ( ) Other __________
Date of Birth: ____/____/_____ (month/day/year)
Participant's unique code number: ____________________

Sample Data Collection from orthodontic charts, photographs, radiographs/capture sheet and patient history

Participant's unique code number: __________
Assessment of inclusion/exclusion criteria (See chart)

Before or during Orthodontic Treatment:
( ) Any form of bone diseases? ____________________________
( ) Thyroid or parathyroid-related disease? ____________________________
( ) Prescribed medications? ____________________________
( ) Radiotherapy? ____________________________
( ) Data not available ( ) Fully healthy ( ) Other: ______________________
( ) Ectopic canine erupted spontaneously with no orthodontic force whatsoever
( ) Important records missing? ____________________________

( ) Should be excluded
( ) Should be included
Age when orthodontic treatment started: ____ years ____months
Age when orthodontic treatment ended: ____ years ____months

Duration of orthodontic treatment in days: ______

<u>Is patient still eligible?</u>

( ) Should be excluded

( ) Should be included

*<u>**When contacting patient**</u>:

( ) Patient expressed interest in participating

( ) Patient refused to participate

If patient is willing to participate, proceed with the collection of the following data (see chart, photos and x-rays):

Questionnaire 3

Tooth #__ was ectopic

Status before orthodontic treatment

1. <u>Severity of ectopy</u>:
( ) No obstruction in its pathway, but lack of space in the arch
( ) No obstruction in its pathway and enough space in the arch
( ) Obstruction in its pathway and lack of space in the arch
( ) Obstruction in its pathway, but enough space in the arch

2. <u>Height before initiating traction</u>:
( ) At the CEJ of the fully erupted adjacent central incisor or premolar
( ) Above the CEJ of the fully erupted adjacent central incisor or premolar

3. <u>Vertical relationship to the adjacent lateral/central</u>:
( ) Coronal 1/3
( ) Middle 1/3
( ) Apical 1/3
( ) Above the apex

4. <u>Horizontal relationship to the adjacent lateral/central incisors</u>: ( ) Lateral ( ) Central
( ) Beyond mesial 1/3
( ) Mesial 1/3
( ) Middle 1/3
( ) Distal 1/3
( ) Before distal 1/3

5. <u>Endo lesion</u>: ( ) Yes ( ) No ( ) Not sure

6. <u>Perio lesion</u>: ( ) Yes ( ) No ( ) Not sure

7. <u>Buccal/lingual location</u>:
( ) Buccal
( ) Palatal
( ) Centre of alveolar ridge

8. <u>Root resorption on final PAN:</u>
( ) Yes: ( ) 1/3 ( ) 2/3 ( ) 1 ( ) No

9. <u>Anatomy at the end of orthodontic treatment</u>:
( ) Normal
( ) Incisal wear
( ) Horizontal fracture
( ) Vertical fracture
( ) Abfraction
( ) Interproximal restoration
( ) Buccal/Palatal restoration
( ) Incisal restoration
( ) Decayed

10. <u>Angle between the long axis of the canine and the long axis of the adjacent lateral</u>: _______

11. <u>Buccal gingival recession</u>: ( ) Yes ( ) No ( ) Not sure

12. <u>Lingual gingival recession</u>: ( ) Yes ( ) No ( ) Not sure

13. <u>Type of movement</u>:
( ) Canine moved orthodontically all the way
( ) Canine moved orthodontically partially
( ) Not enough information

14. <u>Approximate duration of traction</u>: _____ months

15. <u>Approximate distance of orthodontic/spontaneous movement until reaching final position</u>: _______ mm

16. <u>Technique</u>: ( ) Closed exposure ( ) Open exposure ( ) infraocclusion ( ) Surgical luxation ( ) Surgical repositioning

17. <u>Type of mechanics</u>: ( ) Cantilever ( ) Power-chain ( ) NiTi coil-spring
( ) NiTi piggy-back ( ) NiTi AW ( ) Supercable AW ( ) SS ligature

18. <u>Type of brackets</u>: ( ) Conventional ( ) Self-ligating ( ) .022” ( ) .018” ( ) Not enough info on _________

19. <u>Type of upper retainer</u>: ( ) Hawley ( ) None ( ) Bonded ( ) Essix ( ) Splint

Questionnaire 3

Tooth #__ ( ) Ectopic ( ) Control

If control, only questions # 5, 6, 8, 9, 11 and 12 have to be answered

Status before orthodontic treatment

1. Severity of ectopy:
( ) No obstruction in its pathway, but lack of space in the arch
( ) No obstruction in its pathway and enough space in the arch
( ) Obstruction in its pathway and lack of space in the arch
( ) Obstruction in its pathway, but enough space in the arch

2. Height before initiating traction:
( ) At the CEJ of the fully erupted adjacent central incisor or premolar
( ) Above the CEJ of the fully erupted adjacent central incisor or premolar

3.*Vertical relationship to the adjacent lateral/central:
( ) Coronal 1/3
( ) Middle 1/3
( ) Apical 1/3
( ) Above the apex

4.*Horizontal relationship to the adjacent lateral/central incisors: ( ) Lateral ( ) Central
( ) Beyond mesial 1/3
( ) Mesial 1/3
( ) Middle 1/3
( ) Distal 1/3
( ) Before distal 1/3

5. Endo lesion: ( ) Yes ( ) No ( ) Not sure
6. Perio lesion: ( ) Yes ( ) No ( ) Not sure

7. Buccal/lingual location:
( ) Buccal
( ) Palatal
( ) Centre of alveolar ridge

8. Root resorption on final PAN:
( ) Yes: ( ) 1/3 ( ) 2/3 ( ) 1
( ) No

9. Anatomy at the end of orthodontic treatment:
( ) Normal
( ) Incisal wear
( ) Horizontal fracture
( ) Vertical fracture
( ) Abfraction
( ) Interproximal restoration
( ) Buccal/Palatal restoration
( ) Incisal restoration
( ) Decayed

10. Angle between the long axis of the canine and the long axis of the adjacent lateral: _______

11. Buccal gingival recession: ( ) Yes ( ) No ( ) Not sure
12. Lingual gingival recession: ( ) Yes ( ) No ( ) Not sure

13. Type of movement:
( ) Canine moved orthodontically all the way
( ) Canine moved orthodontically partially
( ) Not enough information

14. Approximate duration of traction: _____ months

15. Approximate distance of orthodontic/spontaneous movement until reaching final position: _______ mm

16. Technique: ( ) Closed exposure ( ) Open exposure ( ) Infraocclusion ( ) Surgical luxation ( ) Surgical repositioning

17. Type of mechanics: ( ) Cantilever ( ) Power-chain ( ) NiTi coil-spring ( ) NiTi piggy-back ( ) NiTi AW ( ) Supercable AW ( ) SS ligature

18. Type of brackets: ( ) Conventional ( ) Self-ligating ( ) .022" ( ) .018" ( ) Not enough info on _________

19. Type of upper retainer: ( ) Hawley ( ) None ( ) Bonded ( ) Essix ( ) Splint

***After collecting data retrospectively, patient is**:
( ) Still eligible
( ) Not eligible

If eligible, book follow-up:
*Patient showed up for the recall. Please follow this check list:
1. Confirm patient's identity and write his/her unique code number here: _______________
2. Write down which canine is being evaluated: ( ) 13 ( ) 23
2. Explain the procedures and the time ( )
3. Obtain written consent ( )
4. Collect the following information below:
   - Today's date (day/month/year): _____/____/_____

- Duration of this follow-up since debonding: ________months _______ days

Appendix 4: Data collection questionnaires

<u>Ask directly the following questions</u>:

Questionnaire 1

	Regarding right side canine (Tooth #13)	**Regarding left side canine (Tooth #23)**
1. Do you remember if you ever suffered a trauma or a blow against these teeth?	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
2. Did you ever needed a root canal for these teeth?	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
3. Do you remember if you ever needed extensive dental work on these teeth such as a crown, bridge, veneer, large composite fillings, etc?	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
4. Have you ever bleached your teeth? ( ) Yes ( ) No If **<u>yes</u>**, how: ( ) In-office ( ) Home as per dentist's advice ( ) Home with no professional oversight		
5. Do you clench or have ever clenched your teeth while awake for a long period of your life?	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
6. Do you grind or have you ever ground your teeth at night for a long period of your life?	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
7. Have you ever felt pain on tooth # _______?	( ) Yes: Type: ( ) Prolonged ( ) Short duration ( ) No ( ) Yes ( ) No ( ) Not sure	( ) Yes: Type: ( ) Prolonged ( ) Short duration ( ) No ( ) Yes ( ) No ( ) Not sure

8. Has tooth #___ been sensitive when brushing, biting or chewing? ( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
9. Has tooth #_______ ever been sensitive when drinking or eating cold or hot food?	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
10. Have you ever received a gingival graft? Tooth # ___?	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
11. Have you ever noticed that the gum around tooth# _______ is moving upward with time?	( ) Yes ( ) No ( ) Not sure	( ) Yes ( ) No ( ) Not sure
12. How and for how long have you worn your upper retainer? ( ) Very well for __years __months ( ) Not well for ___ years ___ months		
13. Do you or have you ever held or bitten objects with tooth # _______, such as paper clips, pen, finger, hair clips, etc, for a long period of your life?	( ) Yes ( ) No ( ) Can`t remember	( ) Yes ( ) No ( ) Can`t remember
14. Have you ever opened bottles or cans or any other object with tooth# __?	( ) Yes ( ) No ( ) Can`t remember	( ) Yes ( ) No ( ) Can`t remember
15. Did you receive a second ortho treatment?	( ) Yes ( ) No	( ) Yes ( ) No

To be completed by an ortho resident:

Questionnaire 2	Regarding right side canine (Tooth #13)	Regarding left side canine (Tooth #23)
1. Are these teeth at approximately the same level or 0.5-1.0mm below the incisal edges of the central incisors?	( ) Yes ( ) No If "No": ( ) It is more than 1.0mm below than the central incisors ( ) It is___ mm above the incisal edge of the central incisors ( ) just like it was finished ( ) different than when it was finished ( ) due to some relapse	( ) Yes ( ) No If "No": ( ) It is more than 1.0mm below than the central incisors ( ) It is ____ mm above the incisal edge of the central incisors ( ) just like it was finished ( ) different than when it was finished ( ) due to some relapse
2. Do you see gingival recession on tooth # __?	( ) No ( ) Yes If " Yes": Amount of recession: ____ Is there root exposure? ( ) Yes ( ) No	( ) No ( ) Yes If "Yes": Amount of recession: ____ Is there root exposure? ( ) Yes ( ) No
3. Any restoration on tooth # __?	( ) Crown ( ) Bridge ( ) Veneer ( ) Composite ____ ( ) Other ___	( ) Crown ( ) Bridge ( ) Veneer ( ) Composite ____ ( ) Other ___
4. Do you see caries:	( ) No ( ) Yes on the: ( ) Palatal ( ) Mesial ( ) Distal ( ) Buccal	( ) No ( ) Yes on the: ( ) Palatal ( ) Mesial ( ) Distal ( ) Buccal
5. Any pocket greater than 4mm?	( ) No ( ) Yes on the: ( ) Palatal ( ) Mesial ( ) Distal ( ) Buccal	( ) No ( ) Yes on the: ( ) Palatal ( ) Mesial ( ) Distal ( ) Buccal
6. Any relapse in tooth position in comparison with B records?	( ) No ( ) Yes: ( ) Buccal tipping ( ) Lingual tipping ( ) Rotation ( ) Intrusion ( ) Extrusion ( ) Mesial root tip ( ) Distal root tip ( ) Mesial ( ) Distal	( ) No ( ) Yes: ( ) Buccal tipping ( ) Lingual tipping ( ) Rotation ( ) Intrusion ( ) Extrusion ( ) Mesial root tip ( ) Distal root tip ( ) Mesial ( ) Distal
7. Any craze lines:	( ) No ( ) Yes on the: ( ) Palatal ( ) Buccal	( ) No ( ) Yes on the: ( ) Palatal ( ) Buccal
8. Any incisal fracture?	( ) No ( ) Yes	( ) No ( ) Yes
9. Wear Index score: _______		
10. MHI contact(s):	( ) Premature leading to shifting ( ) Overcontact ( ) No contact ( ) Normal	( ) Premature leading to shifting ( ) Overcontact ( ) No contact ( ) Normal
11. Type of excursion:	( ) Canine guidance ( ) Group function ( ) Participates in protrusion	( ) Canine guidance ( ) Group function ( ) Participates in protrusion
12. Type of A-P dental arch relationship:	( ) I ( ) 1/4 Cl.II ( ) 1/2Cl.II ( ) 3/4 Cl.II ( ) 1/4 Cl.III ( ) 1/2Cl.III ( ) 3/4 Cl.III	( ) I ( ) 1/4 Cl.II ( ) 1/2Cl.II ( ) 3/4 Cl.II ( ) 1/4 Cl.III ( ) 1/2Cl.III ( ) 3/4 Cl.III

13. Is tooth # _______ mobile?	( ) No ( ) Yes If "Yes": mobility type ______.	( ) No ( ) Yes If "Yes": mobility type ______.

After this, please refer the patient for the endodontic assessment by Dr. Cunha.

Appendix 5: Endodontic data collection form

Endodontic questionnaire Patient's chart #: ________ Tooth 13 Visual analysis: Colour change? ( ) No ( ) Yes Fistula? ( ) No ( ) Yes Percussion test: ( ) Normal ( ) Exacerbated ( ) Ankylosed Palpation of the apical cortical plates: ( ) Normal ( ) Exacerbated Thermal tests: Heat ( )+normal ( ) +Exacerbated( ) +Delayed ( ) Cold ( )+normal ( ) +Exacerbated( ) +Delayed ( ) EPT ( )+normal ( ) +Exacerbated( )+Delayed ( ) Need to take a PA: ( ) Yes ( ) No If yes, outcome of the PA: ____ _____________ Conclusion: of current pulpal status: ( ) Vital (normal) ( ) Vital (hypersensitive) ( ) Non-vital (no apical lesion) ( ) Non-vital (with apical lesion) Comments: ____ _______	Tooth 23 Visual analysis: Colour change? ( ) No ( ) Yes Fistula? ( ) No ( ) Yes Percussion test: ( ) Normal ( ) Exacerbated ( ) Ankylosed Palpation of the apical cortical plates: ( ) Normal ( ) Exacerbated Thermal tests: Heat ( )+normal ( ) +Exacerbated ( ) +Delayed ( ) Cold ( ) +normal ( ) +Exacerbated ( )+Delayed ( ) EPT ( )+normal ( )+Exacerbated ( ) +Delayed ( ) Need to take a PA: ( ) Yes ( ) No If yes, outcome of the PA: ____ _____________ Conclusion: of current pulpal status: ( ) Vital (normal) ( ) Vital (hypersensitive) ( ) Non-vital (no apical lesion) ( ) Non-vital (with apical lesion) Comments: ____ _______

References

Abd-Elmeguid A, Yu DC. 2009a. Dental pulp neurophysiology: Part 1. Clinical and diagnostic implications. J Can Dent Assoc. 75(1):55-59.

Abd-Elmeguid A, Yu DC. 2009b. Dental pulp neurophysiology: Part 2. Current diagnostic tests to assess pulp vitality. J Can Dent Assoc. 75(2):139-143.

Albino J, Lawrence S, Lopes C, Nash L, Tedesco L. 1991. Cooperation of adolescents in orthodontic treatment. J Behav Med. 14(1):53-70.

Alessandri Bonetti G, Zanarini M, Incerti Parenti S, Marini I, Gatto MR. 2011. Preventive treatment of ectopically erupting maxillary permanent canines by extraction of deciduous canines and first molars: A randomized clinical trial. Am J Orthod Dentofacial Orthop. 139(3):316-323.

Alghaithy RA, Qualtrough AJ. 2016. Pulp sensibility and vitality tests for diagnosing pulpal health in permanent teeth: A critical review. Int Endod J.1-8.

Alomari FA, Al-Habahbeh R, Alsakarna BK. 2011. Responses of pulp sensibility tests during orthodontic treatment and retention. Int Endod J. 44(7):635-643.

Anstending HSK, J.H. 1972. A histologic study of pulpal reaction to orthodontic tooth movement in dogs. Angle Orthod. 42(1):50-55.

Babacan H, Doruk C, Bicakci AA. 2010. Pulpal blood flow changes due to rapid maxillary expansion. Angle Orthod. 80(6):1136-1140.

Baccetti T, Leonardi M, Armi P. 2008. A randomized clinical study of two interceptive approaches to palatally displaced canines. Eur J Orthod. 30(4):381-385.

Baccetti T, Sigler LM, McNamara JA, Jr. 2011. An rct on treatment of palatally displaced canines with rme and/or a transpalatal arch. Eur J Orthod. 33(6):601-607.

Barwick PJ, Ramsay DS. 1996. Effect of brief intrusive force on human pulpal blood flow. Am J Orthod Dentofacial Orthop. 110(3):273-279.

Bauss O, Rohling J, Rahman A, Kiliaridis S. 2008a. The effect of pulp obliteration on pulpal vitality of orthodontically intruded traumatized teeth. J Endod. 34(4):417-420.

Bauss O, Rohling J, Sadat-Khonsari R, Kiliaridis S. 2008b. Influence of orthodontic intrusion on pulpal vitality of previously traumatized maxillary permanent incisors. Am J Orthod Dentofacial Orthop. 134(1):12-17.

Becker A, Chaushu G, Chaushu S. 2010. Analysis of failure in the treatment of impacted maxillary canines. Am J Orthod Dentofacial Orthop. 137(6):743-754.

Becker A, Kohavi D, Zilberman Y. 1983. Periodontal status following the alignment of palatally impacted canine teeth. Am J Orthod. 84(4):332-336.

Becker A, Smith P, Behar R. 1981. The incidence of anomalous maxillary lateral incisors in relation to palatally-displaced cuspids. Angle Orthod. 51(1):24-29.

Bedoya MM, Park JH. 2009. A review of the diagnosis and management of impacted maxillary canines. J Am Dent Assoc. 140(12):1485-1493.

Bishara S. 1992 Impacted maxitlary canines- a review. Am J Orthod Dentofacial Orthop. 101(2):159-171.
Blair GS, Hobson RS, Leggat TG. 1998. Posttreatment assessment of surgically exposed and orthodontically aligned impacted maxillary canines. Am J Orthod Dentofacial Orthop. 113(3):329-332.
Brodin P, Linge L, Aars H. 1996. Instant assessment of pulpal blood flow after orthodontic force application. J Orofac Orthop. 57(5):306-309.
Butcher EO, Taylor AC. 1952. The vascularity of the incisor pulp of the monkey and its alteration by tooth retraction. J Dent Res. 31(2):239-247.
Cave SG, Freer TJ, Podlich HM. 2002. Pulp-test responses in orthodontic patients. Aust Orthod J. 18(1):27-34.
Caviedes-Bucheli J, Moreno JO, Ardila-Pinto J, Del Toro-Carreno HR, Saltarin-Quintero H, Sierra-Tapias CL, Macias-Gomez F, Ulate E, Lombana-Sanchez N, Munoz HR. 2011. The effect of orthodontic forces on calcitonin gene-related peptide expression in human dental pulp. J Endod. 37(7):934-937.
Chapman MN, Nadgir RN, Akman AS, Saito N, Sekiya K, Kaneda T, Sakai O. 2013. Periapical lucency around the tooth: Radiologic evaluation and differential diagnosis. Radiographics. 33(1):E15-32.
Chavarria-Bolanos D, Martinez-Zumaran A, Lombana N, Flores-Reyes H, Pozos-Guillen A. 2014. Expression of substance p, calcitonin gene-related peptide, beta-endorphin and methionine-enkephalin in human dental pulp tissue after orthodontic intrusion: A pilot study. Angle Orthod. 84(3):521-526.
Cooke J, Wang HL. 2007. Canine impaction incidence and management. Int J Periodontics Restorative Dent. 26(5):483-491.
Crescini A, Clauser C, Giorgetti R, Cortellini P, Pini Prato GP. 1994. Tunnel traction of infraosseous impacted maxillary canines. A three-year periodontal follow-up. Am J Orthod Dentofacial Orthop. 105(1):61-72.
Crescini A, Nieri M, Buti J, Baccetti T, Mauro S, Prato GP. 2007a. Short- and long-term periodontal evaluation of impacted canines treated with a closed surgical-orthodontic approach. J Clin Periodontol. 34(3):232-242.
Crescini A, Nieri M, Buti J, Baccetti T, Pini Prato GP. 2007b. Orthodontic and periodontal outcomes of treated impacted maxillary canines. Angle Orthod. 77(4):571-577.
Crescini A, Nieri M, Rotundo R, Baccetti T, Cortellini P, Prato GP. 2007c. Combined surgical and orthodontic approach to reproduce the physiologic eruption pattern in impacted canines: Report of 25 patients. Int J Periodontics Restorative Dent. 27(6):529-537.
D'Amico RM, Bjerklin K, Kurol J, Falahat B. 2003. Long-term results of orthodontic treatment of impacted maxillary canines. Angle Orthod. 73(3):231-238.
Derringer KA, Jaggers DC, Linden RW. 1996. Angiogenesis in human dental pulp following orthodontic tooth movement. J Dent Res. 75(10):1761-1766.
El-Angbawi AM, Bearn DR, McIntyre GT. 2014. Comparing the effectiveness of the 0.018-inch versus the 0.022-inch bracket slot system in

orthodontic treatment: Study protocol for a randomized controlled trial. Trials. 15(15):389.
Ericson S, Kurol J. 1986. Radiographic assessment of maxillary canine eruption in children with clinical signs of eruption disturbance. Eur J Orthod. 8(3):133-140.
Ericson S, Kurol J. 1988. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. Eur J Orthod. 10(4):283-295.
Ericson S, Kurol PJ. 2000. Resorption of incisors after ectopic eruption of maxillary canines: A ct study. Angle Orthod. 70(6):415-423.
Evans DR, J.; Strang, G.; Stirrups, D.A. 1999. A compariosn of laser doppler flowmetry with other methods of assessing the vitality of traumatized anterior teeth. Endod Dent Traumatol. 15:284-290.
Evren AD, Nevzatoglu S, Arun T, Acar A. 2014. Periodontal status of ectopic canines after orthodontic treatment. Angle Orthod. 84(1):18-23.
Ferreira LL, Filho EM, Rodrigues CL, Bueno CES, Cunha RS. 2013. Lack of pulp sensitivity in maxillary canines traction. RSBO. 10(1):29-33.
Fleming PS. 2015. Multi-disciplinary management to align ectopic or impacted teeth. Seminars in Orthodontics. 21(1):38-45.
Fleming PS, Scott P, Heidari N, Dibiase AT. 2009. Influence of radiographic position of ectopic canines on the duration of orthodontic treatment. Angle Orthod. 79(3):442-446.
Fristad I, Kvinnsland IH, Jonsson R, Heyeraas KJ. 1997. Effect of intermittent long-lasting electrical tooth stimulation on pulpal blood flow and immunocompetent cells: A hemodynamic and immunohistochemical study in young rat molars. Exp Neurol. 146(1):230-239.
Gazelius B, Olgart L, Edwall B, Edwall L. 1986. Non-invasive recording of blood flow in human dental pulp. Endod Dent Traumatol. 2(5):219-221.
Gopikrishna V, Pradeep G, Venkateshbabu N. 2009. Assessment of pulp vitality: A review. Int J Paediatr Dent. 19(1):3-15.
Guevara MJM, S. G. Jr. 1980. Effects of intrusive forces upon the microvasculature of the dental pulp. Angle Orthod. 77(4):129-134.
Hamersky PA, Weimer AD, Taintor JF. 1980. The effect of orthodontic force application on the pulpal tissue respiration rate in the human premolar. Am J Orthod. 77(4):368-378.
Hamilton RS, Gutmann JL. 1999. Endodontic-orthodontic relationships: A review of integrated treatment planning challenges. Int Endod J. 32(5):343-360.
Han G, Hu M, Zhang Y, Jiang H. 2013. Pulp vitality and histologic changes in human dental pulp after the application of moderate and severe intrusive orthodontic forces. Am J Orthod Dentofacial Orthop. 144(4):518-522.
Heyeraas KJ, Berggreen E. 1999. Interstitial fluid pressure in normal and inflamed pulp. Crit Rev Oral Biol Med. 10(3):328-336.
Husain J, Burden D, McSherry P, Morris D, Allen M, Clinical Standards Committee of the Faculty of Dental Surgery RCoSoE. 2012. National clinical guidelines for management of the palatally ectopic maxillary canine. Br Dent J. 213(4):171-176.

Ikawa M, Fujiwara M, Horiuchi H, Shimauchi H. 2001. The effect of short-term tooth intrusion on human pulpal blood flow measured by laser doppler flowmetry. Arch Oral Biol. 46(9):781-787.
Jacob H. 1979. The “ballista spring” system for impacted teeth. Am J Orthod. 75(2):143-151.
Jacoby H. 1983. The etiology of maxillary canine impactions. Am J Orthod. 84(2):125-132.
Jafarzadeh H. 2009. Laser doppler flowmetry in endodontics: A review. Int Endod J. 42(6):476-490.
Javed F, Al-Kheraif AA, Romanos EB, Romanos GE. 2015. Influence of orthodontic forces on human dental pulp: A systematic review. Arch Oral Biol. 60(2):347-356.
Katsnelson A, Flick WG, Susarla S, Tartakovsky JV, Miloro M. 2010. Use of panoramic x-ray to determine position of impacted maxillary canines. J Oral Maxillofac Surg. 68(5):996-1000.
Kokich VG. 2004. Surgical and orthodontic management of impacted maxillary canines. Am J Orthod Dentofacial Orthop. 126(3):278-283.
Kumar S, Mehrotra P, Bhagchandani J, Singh A, Garg A, Kumar S, Sharma A, Yadav H. 2015. Localization of impacted canines. J Clin Diagn Res. 9(1):ZE11-14.
Lazzaretti DN, Bortoluzzi GS, Torres Fernandes LF, Rodriguez R, Grehs RA, Martins Hartmann MS. 2014. Histologic evaluation of human pulp tissue after orthodontic intrusion. J Endod. 40(10):1537-1540.
Lin J, Chandler NP. 2008. Electric pulp testing: A review. Int Endod J. 41(5):365-374.
Lundy FT, Linden GJ. 2004. Neuropeptides and neurogenic mechanisms in oral and periodontal inflammation. Crit Rev Oral Biol Med. 15(2):82-98.
Mavragani M, Vergari A, Selliseth NJ, Boe OE, Wisth PL. 2000. A radiographic comparison of apical root resorption after orthodontic treatment with a standard edgewise and a straight-wire edgewise technique. Eur J Orthod. 22(6):665-674.
McBride LJ. 1979. Traction-a surgical :Orthodontic procedure. Am J Orthod. 76(3):287-299.
McDonald F.; Pitt Ford T. 1994. Blood flow changes in permanent maxillary canines during retraction. Eur J Orthod. 16(1):1-9.
Moskowitz EM, Garcia RC. 2014. The management of palatally displaced maxillary canines: Considerations and challenges. Seminars in Orthodontics. 20(1):46-58.
Mostafa YA, Iskander KG, El-Mangoury NH. 1991. Iatrogenic pulpal reactions to orthodontic extrusion. Am J Orthod Dentofacial Orthop. 99(1):30-34.
Naoumova J, Kurol J, Kjellberg H. 2011. A systematic review of the interceptive treatment of palatally displaced maxillary canines. Eur J Orthod. 33(2):143-149.
Ngan P, Hornbrook R, Weaver B. 2005. Early timely management of ectopically erupting maxillary canines. Seminars in Orthodontics. 11(3):152-163.
Nieri M, Crescini A, Rotundo R, Baccetti T, Cortellini P, Pini Prato GP. 2010. Factors affecting the clinical approach to impacted maxillary canines: A bayesian network analysis. Am J Orthod Dentofacial Orthop. 137(6):755-762.

Niklas A, Proff P, Gosau M, Romer P. 2013. The role of hypoxia in orthodontic tooth movement. Int J Dent. 2013:841840.
Nixon CE, Saviano JA, King GJ, Keeling SD. 1993. Histomorphometric study of dental pulp during orthodontic tooth movement. J Endod. 19(1):13-16.
Olgart L. 1996. Neural control of pulpal blood flow. Crit Rev Oral Biol Med. 7(2):159-171.
Oppennhein A. 1942. A human tissue response to orthodontic intervention of short and long duration. Am J Orthod. 28:263-301.
Orton HS GM, Pearson MH. 1995. Extrusion of the ectopic maxillary canine using a lower removable appliance. Am J Orthod Dentofacial Orthop. Apr(107(4)):349-359.
Oskui IZ, Ashtiani MN, Hashemi A, Jafarzadeh H. 2014. Effect of thermal stresses on the mechanism of tooth pain. J Endod. 40(11):1835-1839.
Ozturk M, Doruk C, Ozec I, Polat S, Babacan H, Bicakci AA. 2003. Pulpal blood flow: Effects of corticotomy and midline osteotomy in surgically assisted rapid palatal expansion. J Craniomaxillofac Surg. 31(2):97-100.
Pantera EA, Jr., Anderson RW, Pantera CT. 1993. Reliability of electric pulp testing after pulpal testing with dichlorodifluoromethane. J Endod. 19(6):312-314.
Parkin N, Benson PE, Thind B, Shah A. 2008. Open versus closed surgical exposure of canine teeth that are displaced in the roof of the mouth. Cochrane Database Syst Rev. 8(4):CD006966.
Parkin N, Furness S, Shah A, Thind B, Marshman Z, Glenroy G, Dyer F, Benson PE. 2012. Extraction of primary (baby) teeth for unerupted palatally displaced permanent canine teeth in children. Cochrane Database Syst Rev. 12(12):CD004621.
Parkin NA, Milner RS, Deery C, Tinsley D, Smith AM, Germain P, Freeman JV, Bell SJ, Benson PE. 2013. Periodontal health of palatally displaced canines treated with open or closed surgical technique: A multicenter, randomized controlled trial. Am J Orthod Dentofacial Orthop. 144(2):176-184.
Parris WG, Tanzer FS, Fridland GH, Harris EF, Killmar J, Desiderio DM. 1989. Effect's of orthodontic force on methionine enkephalin and substance p concentrations in human pulpal tissue. Am J Orthod Dentofacial Orthop. Jun 95(6):479-489.
Peck S, Peck L, Kataja M. 1994. The palatally displaced canine as a dental anomaly of genetic origin. Angle Orthod. 64(4):249-256.
Peters DD, Baumgartner JC, Lorton L. 1994. Adult pulpal diagnosis. I. Evaluation of the positive and negative responses to cold and electrical pulp tests. J Endod. 20(10):506-511.
Petersson A, Axelsson S, Davidson T, Frisk F, Hakeberg M, Kvist T, Norlund A, Mejare I, Portenier I, Sandberg H et al. 2012. Radiological diagnosis of periapical bone tissue lesions in endodontics: A systematic review. Int Endod J. 45(9):783-801.
Proffit WR, Fields HW, Sarver DM. 2007. Contemporary orthodontics. 4th ed. St. Louis: Mosby:234-267.

Quiryen M.; Op Heij DG AA, ; Opdebeeck HM; van Steenberghe. 2000. Periodontal health of orthodontically extruded impacted teeth- a split-mouth, long-term clinical evaluation. J Periodontol. 71(11):1708-1714.
Ramazanzadeh BA, Sahhafian AA, Mohtasham N, Hassanzadeh N, Jahanbin A, Shakeri MT. 2009. Histological changes in human dental pulp following application of intrusive and extrusive orthodontic forces. J Oral Sci. 51(1):109-115.
Reitan K. 1951. The initial tissue reaction incident to orthodontic tooth movement as related to the influence of function. Acta Odontol Scand. 6(1-24).
Richardson G, Russell KA. 2000. A review of impacted permanent maxillary cuspids--diagnosis and prevention. J Can Dent Assoc. 66(9):497-501.
Roeykens HVM, G.; De Moor, R.; Martens, L. 2000. Reliability of laser doppler flowmetry in a 2-probe assessment of pulpal blood flow. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 87(6):742-748.
Rosner B. 2011. Fundamentals of biostatistics. MA;Brooks/Cole. (7th ed. Boston).
Sabuncuoglu FA, Ersahan S. 2014. Changes in maxillary incisor dental pulp blood flow during intrusion by mini-implants. Acta Odontol Scand. 72(7):489-496.
Sajnani AK, King NM. 2012. Early prediction of maxillary canine impaction from panoramic radiographs. Am J Orthod Dentofacial Orthop. 142(1):45-51.
Salles AW, Salles AM, Nogueira GE. 2013. Laser doppler blood-flow signals from human teeth during an alignment and leveling movement using a superelastic archwire. ISRN Dent. 2013:102816.
Sameshima GT, Sinclair PM. 2001. Predicting and preventing root resorption: Part ii. Treatment factors. Am J Orthod Dentofacial Orthop. 119(5):511-515.
Sano Y, Ikawa M, Sugawara J, Horiuchi H, Mitani H. 2002. The effect of continuous intrusive force on human pulpal blood flow. Eur J Orthod. 24(2):159-166.
Saoud TM, Mistry S, Kahler B, Sigurdsson A, Lin LM. 2016. Regenerative endodontic procedures for traumatized teeth after horizontal root fracture, avulsion, and perforating root resorption. J Endod. 42(10):1476-1482.
Schmidt AD, Kokich VG. 2007. Periodontal response to early uncovering, autonomous eruption, and orthodontic alignment of palatally impacted maxillary canines. Am J Orthod Dentofacial Orthop. 131(4):449-455.
Shapira Y, Kuftinec MM. 2001. Maxillary tooth transpositions: Characteristic features and accompanying dental anomalies. Am J Orthod Dentofacial Orthop. 119(2):127-134.
Soares DG, Basso FG, Scheffel DS, Hebling J, de Souza Costa CA. 2015. Responses of human dental pulp cells after application of a low-concentration bleaching gel to enamel. Arch Oral Biol. 60(9):1428-1436.
Soo-ampon S, Vongsavan N, Soo-ampon M, Chuckpaiwong S, Matthews B. 2003. The sources of laser doppler blood-flow signals recorded from human teeth. Arch Oral Biol. 48(5):353-360.

Stenvik A, Mjor IA. 1970. Pulp and dentine reactions to experimental tooth intrusion. A histologic study of the initial changes. Am J Orthod. 57(4):370-385.
Stewart JA, Heo G, Glover KE, Williamson PC, Lam EW, Major PW. 2001. Factors that relate to treatment duration for patients with palatally impacted maxillary canines. Am J Orthod Dentofacial Orthop. 119(3):216-225.
Tan SS. 1983. Canine extrusion auxiliary. J Clin Orthod. 17(2):130-131.
Taylor GS. 1979. Simplified sectional arch to align palatally displaced maxillary canines. J Clin Orthod. 13(12):847.
Torabinejad M, Fouad A, Walton R. 2014. Endodontics: Principles and practice. 5th Edition:12-15.
Turley PK, Joiner MW, Hellstrom S. 1984. The effect of orthodontic extrusion on traumatically intruded teeth. Am J Orthod. 85(1):47-56.
Unsterseher REN, L.G.; Weimer, A.D.; Dyer, J.K. 1987. The response of human pulpal tissue after orthodontic treatment Am J Orthod Dentofacial Orthop. 92(3):220-224.
Vandevska-Radunovic V. 1999. Neural modulation of inflammatory reactions in dental tissues incident to orthodontic tooth movement. A review of the literature. Eur J Orthod. 21(3):231-247.
Vandevska-Radunovic V, Kristiansen AB, Heyeraas KJ, Kvinnsland S. 1994. Changes in blood circulation in teeth and supporting tissues incident to experimental tooth movement. Eur J Orthod. 16(5):361-369.
Vermette ME, Kokich VG, Kennedy DB. 1995. Uncovering labially impacted teeth: Apically positioned flap and closed-eruption techniques. Angle Orthod. 65(1):23-32; discussion 33.
von Bohl M, Ren Y, Fudalej PS, Kuijpers-Jagtman AM. 2012. Pulpal reactions to orthodontic force application in humans: A systematic review. J Endod. 38(11):1463-1469.
Warford JH, Grandhi RK, Tira DE. 2003. Prediction of maxillary canine impaction using sectors and angular measurement. American Journal of Orthodontics and Dentofacial Orthopedics. 124(6):651-655.
Woloshyn H, Artun J, Kennedy DB, Joondeph DR. 1994. Pulpal and periodontal reactions to orthodontic alignment of palatally impacted canines. Angle Orthod. 64(4):257-264.
Yamaguchi M, Kasai K. 2007. The effects of orthodontic mechanics on the dental pulp. Seminars in Orthodontics. 13(4):272-280.
Zasciurinskiene E, Bjerklin K, Smailiene D, Sidlauskas A, Puisys A. 2008. Initial vertical and horizontal position of palatally impacted maxillary canine and effect on periodontal status following surgical-orthodontic treatment. Angle Orthod. 78(2):275-280.