A Case Series Study on Pulp Calcification Following Orthodontic Treatment of Ectopic Maxillary Canine Teeth

Abstract

Introduction: Impaction of maxillary canines is an identifiable anomaly that is encountered in orthodontics. Extrusive orthodontic forces on ectopic teeth may have a correlation with the cause of altered pulpal conditions such as pulpal necrosis, pulp obliteration, and pulp calcifications. **Objective:** The aim of this study was to describe the pulp status and recognize the incidence of pulp calcification on a series of orthodontically treated, ectopic maxillary canine cases. **Materials and Methods:** Sixteen patients that had undergone orthodontic extrusion of their maxillary canines were evaluated. These patients had either unilateral or bilateral ectopic maxillary canines at or above the CEJ of adjacent teeth. A total of 22 canines were examined clinically and radiographically. **Results:** From the sample of 22 maxillary canines which had undergone orthodontic extrusion, 7 canines (31.8%) displayed evidence of having non-vital pulps and 4 of the canines (18.2%) were determined to show an asymptomatic pulpal response due to pulp calcification. **Conclusion:** Essential information correlating the effects of maxillary extrusion to pulpal calcification may be utilized with further studies to determine definitive correlations of any cause-and-effect relationship that may exist.

Introduction

Maxillary canine impaction is a recognizable dental anomaly of clinical concern.[1] The approximate incidence of ectopic maxillary canines was found to range from 0.8% to 5.0%.[2,3] The frequency of impacted canines in female patients is roughly double that as seen in male patients. Furthermore, palatal impactions occur more frequently than buccal impactions, with a ratio varying from 2:1 to 6:1.[1,4] If maxillary primary canines are not mobile by the age of 10

years and observation or palpation of a canine bulge is absent, then it should be suspected that there is ectopic eruption of permanent maxillary canines.[5]

Once ectopic permanent maxillary canines are diagnosed, appropriate treatment modalities in terms of surgery and orthodontics are considered. Becker and Chaushu[6] mention that there are several appropriate surgical procedures available which are classified into open procedures and closed procedures that allow orthodontics to properly resolve impactions. They go on to describe that a canine impacted in a palatal position may be accessed by using the open exposure technique, leaving the canine exposed to the oral environment. Furthermore, a canine situated deeply in the palatal position may benefit from the closed exposure technique by preventing mucosal deficiency in the palate and preserving the hard and soft tissues. Labially positioned canines can be accessed by performing a window technique, a closed exposure technique, or utilizing an apically repositioned flap. The window technique consists of exposing the tooth and opening a semilunar window in the oral mucosa over it. The closed exposure technique creates a small aperture on the dental follicle for a bonded attachment while leaving most of the follicle intact. Lastly, the apically repositioned flap technique involves raising a flap from the keratinized gingiva at the ridge crest or gingival margin of the retained primary canine to reveal the dental follicle of the permanent canine. Only the labial surface of the follicle is opened and the flap is sutured firmly to the cervical half of the crown, exposing the coronal half.

Once tooth access has been obtained and the bonding of attachments is completed, traction forces are applied to the ectopic teeth.[6] Orthodontic extrusive forces on ectopic teeth

have been shown to cause pulpal conditions such as internal root resorption, pulpal necrosis, pulp obliteration, pulp microcirculation, hypoxia, or pulp calcifications.[7–9] Pulp calcifications are abnormalities that may be found in healthy, diseased, or unerupted teeth.[10] When calcifications occur, they may have variations in radiographic presentations varying from pulp stones to larger diffuse calcifications within the pulp.[11] Understanding the implications of extrusive forces on the pulp may lead to the prevention and reduction of pulpal abnormalities.

There is a general consensus that the incidence of calcifications within the pulp increases with the age of an individual and higher incidences of calcified bodies were identified in the pulps of carious and/or operated teeth.[12] The study of Sayegh and Reed utilized 591 human teeth and compared pulp calcification in caries-free teeth in various age categories. They determined that the incidence of pulpal calcification in carious teeth was approximately five times that in non-carious teeth within the same age group. The incidence of pulp calcification in the teeth of children and young adults was found to increase with the presence of dental caries.[13] This study also revealed that the incidence is much greater in teeth from older patients (individuals older than 45 years) than in teeth from younger ones. Older patients were found to have approximately ten times the occurrence rate of pulpal calcifications when compared to younger patients.[13]

Counter to this, other literature have revealed that no significant correlation exists between the condition of the crowns of teeth and the calcification in the pulps.[12] Sundell et al[14] hypothesize that trauma may cause capillary thrombosis and/or damage to the vascular-wall. If mineralization of the lesions occurs, then pulp stones may form. However, their research

suggests that no significant correlation appeared to exist between formation of pulp stones and the patient's age, sex, remaining dentin thickness under the cavity preparation, preparation time, or the trauma endured during operative procedures.[14] Furthermore, Tamse et al[15] radiographically classified the condition of 1380 crowns within their study as intact, carious, or restored. Their study was also unable to identify any significant difference among intact teeth, teeth with caries, or restored teeth in relation to the susceptibility of developing pulp stones.

There is conflicting literature regarding the etiology of pulpal calcification. The origin of some pulp calcifications are unknown.[13,14] Furthermore, it is unclear whether pulpal calcifications are biological variations or the result of pathology.[10,16] However, it is widely accepted that pulpal calcifications are not often the cause of disease to the dental pulp or the cause of subjective symptoms, and the prevalence of pulpal calcifications are variable.

In the orthodontic literature, it has been mentioned that the duration, type, and magnitude of orthodontic force as well as physiological tissue tolerance could affect the pulp tissue in a reversible or irreversible manner.[17] The intensity and duration of orthodontic appliances generate cellular responses to tooth movement, producing localized stress and strain within pulpal tissue. This may trigger changes in blood flow and activate metabolic responses.[18] Poor control of orthodontic forces, heavy continuous force, and a sequence of abrupt movements might also be sources of pulpal damage. This may lead to loss of pulp vitality or the severing of blood vessels to the root apex as they enter the pulp canal.[5]

Previous studies have identified deleterious effects in dental pulp and periodontal ligament due to extrusive orthodontic forces, including vascular stasis and altered pulp metabolism.[8,19,20]

It has been stated that an increased metabolic rate within the pulp may be harmful as increased pulpal activity may be associated with the appearance of premature aging of the pulp and narrowing of the pulp chamber itself.[21]

Histologically, it has been demonstrated by Ramazanzadeh et. al[22] that odontoblastic layer disruption and vacuolization occurred in teeth that were subject to extrusive or intrusive forces. There were significant differences between these test teeth and control teeth, but there were insignificant differences between extruded and intrude teeth. It has been concluded that histologically, there were no differences between three days versus three weeks of extrusive forces. The 6.35mm (¼-inch), 127.6g (4.5-oz) elastics only exception to this is that after three weeks or more of extrusive forces, fibrous tissue formation was found.[22,23] Differentiating from intrusive forces, extrusive forces had no effect on the pulpal blood flow.[24]

According to Sübay et. al[25] and their study on clinical response of pulpal tissues to extrusive forces, none of the teeth in the study demonstrated any inflammatory reaction. The study involved two groups: Group 1 was comprised of 20 teeth from 10 patients who had treatment with a fixed sectional orthodontic appliance group 2 was comprised of 20 teeth from 5 patients that had treatment done with 0.635cm (¼-inch), 127.6g (4.5oz) elastics. Two of the 20 teeth in the fixed sectional appliance group demonstrated several large pulp stones. The study showed that initial elastic forces of 75g (2.6oz) and 127.6g (4.5oz) for 10 and 40 days do not cause any major pathological pulpal changes.[25] However, this study made no mention of the total distance that the teeth moved during the extrusive procedures.

Although, the study above demonstrated that a small number of the test teeth resulted with pulpal calcifications due to extrusive orthodontic forces, scientific evidence to determine a concrete relationship between orthodontic force level and dental pulp tissue reactions are inconclusive. There is an insufficient amount of long-term results that are capable of describing pulpal injury as a result of orthodontic treatment alone.[26] Javed et. al[27] described that pulpal response to the magnitude and duration of orthodontic forces vary significantly. They believe that lower orthodontic forces exerted on teeth for a few minutes, in comparison to longer durations, can cause minor or no change in pulpal blood flow. However, a standardized assessment tool and randomized controlled trials are lacking.[26] The literature evaluated in this systematic review was limited by a lack of standardization in study protocols.

The purpose of this study was to report the pulp status and prevalence of pulp calcification on a series of cases of orthodontically treated ectopic maxillary canines, whose clinical endodontic assessment showed some form of pulp alteration which required further evaluation by means of periapical radiography. Although far from the top of the evidence pyramid, the follow-up of patients whose ectopic canines were moved orthodontically can be helpful in providing further information on the relationship between pulp status/calcification and extrusion, during orthodontic treatment.

Materials & Methods

All participants underwent a pulp sensitivity evaluation that included thermal, electrical, palpation and percussion tests. The thermal test consisted of a heat test and a cold test. For the heat test, a heated gutta percha stick was placed on the tested tooth until a sensation of

heat was felt. The duration of lingering heat sensation was observed once the gutta percha was removed. The cold test consisted in applying Coltene Hygenic[R] Endo-Ice spray to a cotton pellet and observing if the patient had a sensation of cold to the tooth and also noting the duration of lingering sensation once the cotton pellet was removed. Thermal stimuli which evoked a sharp, brief pain implied that the pulp was vital, but not necessarily normal. However, if an intense and lingering pain was observed after the removal of thermal stimuli, then this indicated irreversible pulpitis, where the pulp is vital but inflamed and incapable of healing. Periapical radiographs were immediately obtained in cases where endodontic testing indicated non-vital or altered pulp status, such as having no response to heat or cold, or an Electrical pulp vitality test (EPT) of 80/80. EPT was completed with the Kerr Vitality Scanner 2006. Percussion tests and palpation of the apical cortical plates aided in the diagnosis of pulpal and periodontal conditions.

It is possible that false-negative responses to thermal stimuli may be observed when cold is applied to teeth with calcified canals, which can therefore be mistaken for necrotic pulps.[28] EPT scores of 80/80 assisted in the decision to obtain periapical radiographs for further analysis of the subject tooth. The probability of obtaining a sensitive reaction that indicates a vital pulp is 90% with the cold test, 84% with the electrical test, and 83% with the heat test with the cold test being the most accurate.[29,30]

Sample selection

At the University of Manitoba, there are 3 different types of exposure techniques for labially impacted canines: gingivectomy (open exposure), apically positioned flap, and closed

exposure. The selection of the best method depends on the mesio-distal and labio-palatal location of the tooth, the amount of attached gingival as well as the position in relation to the mucogingival junction. When located in the palate, the protocol includes open exposure and a 6 month follow up if the impaction is mild and the diagnosis is made at the end of the mixed dentition. Otherwise, forced eruption is recommended.[31] Forced eruption is normally performed by means of TMA wire cantilevers, overlay NiTi wires, NiTi coils, power chain elastic or supercable wires. A combination of these is more a rule than an exception. When the impacted canine is brought into alignment, treatment proceeds as in any other conventional orthodontic treatment.

As of June 2014, the orthodontic records of a patient pool of 3200 individuals from the graduate orthodontic clinic archives at the University of Manitoba were examined. Intraoral photographs and radiographs were evaluated. The inclusion criteria required individuals to have either unilateral or bilateral ectopic maxillary canines at the initial examination with the cusp tip at the level of, or above the cement-enamel junction (CEJ) of the adjacent teeth prior to any orthodontic treatment. The exclusion criteria were: 1. Presence of systemic conditions that could alter bone metabolism such as prescribed medications, use of bisphosphonates, bone diseases, Paget's disease, radiotherapy and hypothyroidism, and 2. Occlusal factors such as signs of severe abfraction and/or abrasion.

Out of 3200 patient files, 31 individuals had qualified for our study. Of these 31 patients, 16 patients (10 females and 6 males) agreed and were available to participate in this study. These participants had either unilateral (10 individuals) or bilateral (6 individuals) ectopic

maxillary canines at or above the CEJ of adjacent teeth (Table 1). The patients varied in ages between 11 to 17 years old with a mean age of 14.19 years when their orthodontic treatment had started. Upon completion of orthodontic treatment, the ages of the patients ranged between 15 to 21 years with a mean of 17.69 years. At the time of their research appointment, their ages ranged from 18 to 29 with a mean age of 22.69 years. Of the 15 individuals that did not participate in this study, 2 were unable to contact, 8 had moved away from the city, 1 could not make an appointment due to transportation issues, 1 no longer qualified due to canine being extracted, 1 had deceased, 1 patient evaluation still required completion, and 1 patient was unavailable during hours of operation of the University clinic.

As a result, a total of 22 maxillary permanent canines (10 unilateral and 12 bilateral) fulfilled the inclusion criteria. The non-ectopic contralateral tooth (10 maxillary canines) served as a control to aid in identification of an abnormality. The prevalence of buccally and palatally ectopic canines in the present sample was 50% each. The majority of the canines had undergone open exposure, one had already partially erupted, and two were accessed by the closed technique. Table 1 summarizes the ages of each patient within this study, each maxillary canine that was ectopic, and those of which had calcified or undergone necrosis.

Table 1: Patients with ectopic canines treated with orthodontic extrusion at the University of Manitoba Orthodontic Clinic. In unilateral cases, the non-ectopic contralateral tooth (10 maxillary canines) served as a control.

Patient	Age Extrusion Commenced (Years)	Age Extrusion Terminated (Years)	Age At Examination (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, 2.3
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
Mean	14.19	17.69	22.69	

Results

After a review of the data obtained from each of the patient assessments, this study determined that of 22 maxillary canines which had undergone orthodontic extrusion, 11 canines (50.0%) remained vital, 7 canines (31.8%) displayed evidence of having non-vital pulps, and an asymptomatic pulpal response due to pulp calcification was identified in 4 of the canines (18.2%). All things considered, 4 of the 16 patients examined (25.0%) displayed evidence of pulp calcification after orthodontic extrusion. These patients were absent of any systemic conditions that could alter bone status and their maxillary canines were absent of any abfractions or abrasions.

Patient 1 originally had bilateral ectopic maxillary canines. Calcification was present in their upper left canine after corrective orthodontic treatment. Patient 2 also had bilateral ectopic maxillary canines. Calcification occurred in their upper right canine. In both cases, the pulp status was not possible to determine due to the calcified canal interfering with clinical diagnosis and patient assessment. Additionally, patient 3 previously had a unilateral ectopic maxillary left canine and patient 4 had a unilateral ectopic maxillary right canine prior to orthodontic treatment. After extrusion, it was determined that both patient 3 and patient 4 had both resulted in pulp calcification of their ectopic canines. The pulp statuses for these canines were not possible to determine due to the calcified canals interfering with the clinical assessments. There was no clinical or radiographic evidence for lesions of endodontic origin or periodontal origin from the canines with pulp calcification from patients 1, 2, 3, and 4. Additionally, Patient 4 had also shown signs of having a non-vital maxillary canine on the

contra-lateral side after orthodontic extrusion on tooth 23. This tooth was one of the 10 canines used as a control to compare with the pulp status of those that had calcified.

The following briefly describes the patients that were identified with non-vital maxillary canines without indications of pulp calcifications after orthodontic treatment: Patient 5 had a unilateral ectopic maxillary canine 13 prior to orthodontic treatment. Follow up of tooth 13 after extrusion, was recorded to be non-vital. Tooth 23 was not originally ectopic and was used as a control tooth which remained vital after orthodontic treatment. Patient 6 had bilateral maxillary ectopic canines prior to orthodontic treatment, of which both were found to be non-vital after orthodontic extrusion. Patient 12 had a unilateral ectopic maxillary canine 23 prior to orthodontic treatment. Tooth 13 was not originally ectopic and was used as a control tooth. Interestingly, both maxillary canines of patient 12 were determined to be non-vital after orthodontic treatment. Patient 13 initially had an ectopic maxillary canine 13 prior to extrusion which tested as non-vital during the follow up after orthodontic treatment. The contralateral canine 23 was not ectopic and used as a control tooth for comparison. Tooth 23 was recorded as vital after orthodontic treatment. Both patient 12 and 13 were unable to recall whether their canines were subject to previous trauma. Lastly, patient 16 had bilateral maxillary ectopic canines prior to orthodontic treatment. Both canines were identified as non-vital after orthodontic extrusion. Table 2 summarizes these findings.

Table 2: Patients with pulp calcification and pulp diagnosis

Patient	Ectopic Canine	Hx of Trauma	Hx of Bleaching	Hx of Bruxism/ Clenching	Suspected Endodotic Lesion	Suspected Periodontic Lesion	Exprosure Type	Pulp Calcification	Pulp Status
1	1.3 Buccal	No	No	No	No	No	Open	No	1.3 Vital.
	2.3 Buccal	No	No	No	No	No	Open	Yes	2.3 Asymptomatic. Status Indeterminable due to pulp/radicular calcification.
2	1.3 Buccal	No	Yes	Bruxing & Clenching	No	No	Open	Yes	1.3 Asymptomatic. Status Indeterminable due to pulp/radicular calcification.
	2.3 Buccal	No	Yes		No	No	Open	No	2.3 Vital
3	1.3 Control	No	Yes	Patient unsure	No	No	Open	No	1.3 Vital
	2.3 Palatal	No	Yes		No	No	Open	Yes	2.3 Asymptomatic. Status Indeterminable due to pulp/radicular calcification.
4	1.3 Palatal	Yes	No	Bruxing & Clenching	No	No	Open	Yes	1.3 Asymptomatic. Status Indeterminable due to pulp/radicular calcification.
	2.3 Control	Yes	No		No	No	Open	No	2.3 Non-Vital
5	1.3 Palatal	No	No	No	No	No	Open	No	1.3 Non-Vital
	2.3 Control	No	No	No	No	No	Open	No	2.3 Vital
6	1.3 Buccal	No	No	Patient unsure	No	No	Open	No	1.3 Non-Vital
	2.3 Buccal	No	No		No	No	Open	No	2.3 Non-Vital
7	1.3 Palatal	No	Yes	No	No	No	Open	No	1.3 Vital
	2.3 Control	No	Yes	No	No	No	Open	No	2.3 Vital
8	1.3 Control	No	Yes	No	No	No	Open	No	1.3 Vital
	2.3 Palatal	No	Yes	No	No	No	Open	No	2.3 Vital
9	1.3 Buccal	No	No	Bruxing & Clenching	No	No	Open	No	1.3 Vital
	2.3 Control	No	No		No	No	Open	No	2.3 Vital
10	1.3 Buccal	No	No	Bruxing	No	No	Open	No	1.3 Vital
	2.3 Buccal	No	No		No	No	Open	No	2.3 Vital
11	1.3 Buccal	No	No	No	No	No	Open	No	1.3 Vital
	2.3 Buccal	No	No	No	No	No	Exposed	No	2.3 Vital
12	1.3 Control	Patient unsure	No	No	Yes	No	Open	No	1.3 Non-Vital
	2.3 Palatal		No	No	No	No	Open	No	2.3 Non-Vital
13	1.3 Palatal	Patient unsure	Yes	No	No	No	Open	No	1.3 Non-Vital
	2.3 Control		Yes	No	No	No	Open	No	2.3 Vital
14	1.3 Palatal	No	No	Patient unsure	No	No	Open	No	1.3 Vital
	2.3 Control	No	No		No	No	Open	No	2.3 Vital
15	1.3 Palatal	No	No	Clenching	No	No	Open	No	1.3 Vital
	2.3 Control	No	No		No	No	Open	No	2.3 Vital
16	1.3 Palatal	No	Yes	No	No	No	Closed	No	1.3 Non-Vital
	2.3 Palatal	No	Yes	No	No	No	Closed	No	2.3 Non-Vital

Case Reports

The case reports for each participant who displayed evidence of pulp calcification in this case series is provided below. For our study, initiation of extrusion was determined to be at the time of button placement onto the ectopic canine. Termination of extrusion was defined as the time at which brackets on the maxillary canines were debonded.

Patient 1

Patient 1 was an 18-year-old Indian female who had orthodontic extrusion of ectopic canines 13 and 23 (fig 1). Both canines had erupted and the cusp tips were above the CEJ of the fully erupted adjacent lateral incisors and premolars. A frontal view shows that both canines horizontally overlapped the distal lateral incisors less than 1/3 of the facial surface (fig. 1). Both canines were in a buccal position relative to the rest of the arch. Orthodontic extrusion of her maxillary canines began at the age of 13 years 1 month. The orthodontic treatment was completed when she was 15 years 3 months (fig 2). Extrusion of both canines required open exposure technique with NiTi arch wires and 0.022" self-ligating brackets. She claims that she has been wearing her maxillary retainer for approximately 2 years since the completion of her orthodontic treatment. She also stated that she does not clench or grind her teeth and that neither 13 nor 23 had suffered any previous trauma. She does not bite on foreign objects such as pencils, pens, or fingernails. She has never bleached her teeth and neither 13 nor 23 had required extensive dental work, such as a crown, bridge, veneer, or root canal therapy (RCT). She has not experienced any tooth sensitivity during brushing, biting, chewing, or when drinking and eating hot or cold food. Intraoral examination revealed no craze lines on tooth 13

but craze lines were present on tooth 23. Neither 13 nor 23 had any fractures present. Patient has canine guidance on her right side and group function on her left side. Maximum habitual intercuspation (MHI) contacts were normal. Endodontic evaluation revealed that tooth 13 had a delayed response to heat testing, a normal response to cold testing, and an electrical pulp test (EPT) of 80/80. Tooth 23 had a delayed response to both heat and cold testing, and an electrical pulp test of 60/80. Periapical radiographs were taken of each tooth (fig 3). Radiographic analysis aided in the conclusion that tooth 13 was normal and vital, and tooth 23 had undergone pulp/radicular calcification. There was no radiographic evidence displaying lesions of endodontic origin or periodontal origin.

Fig. 1

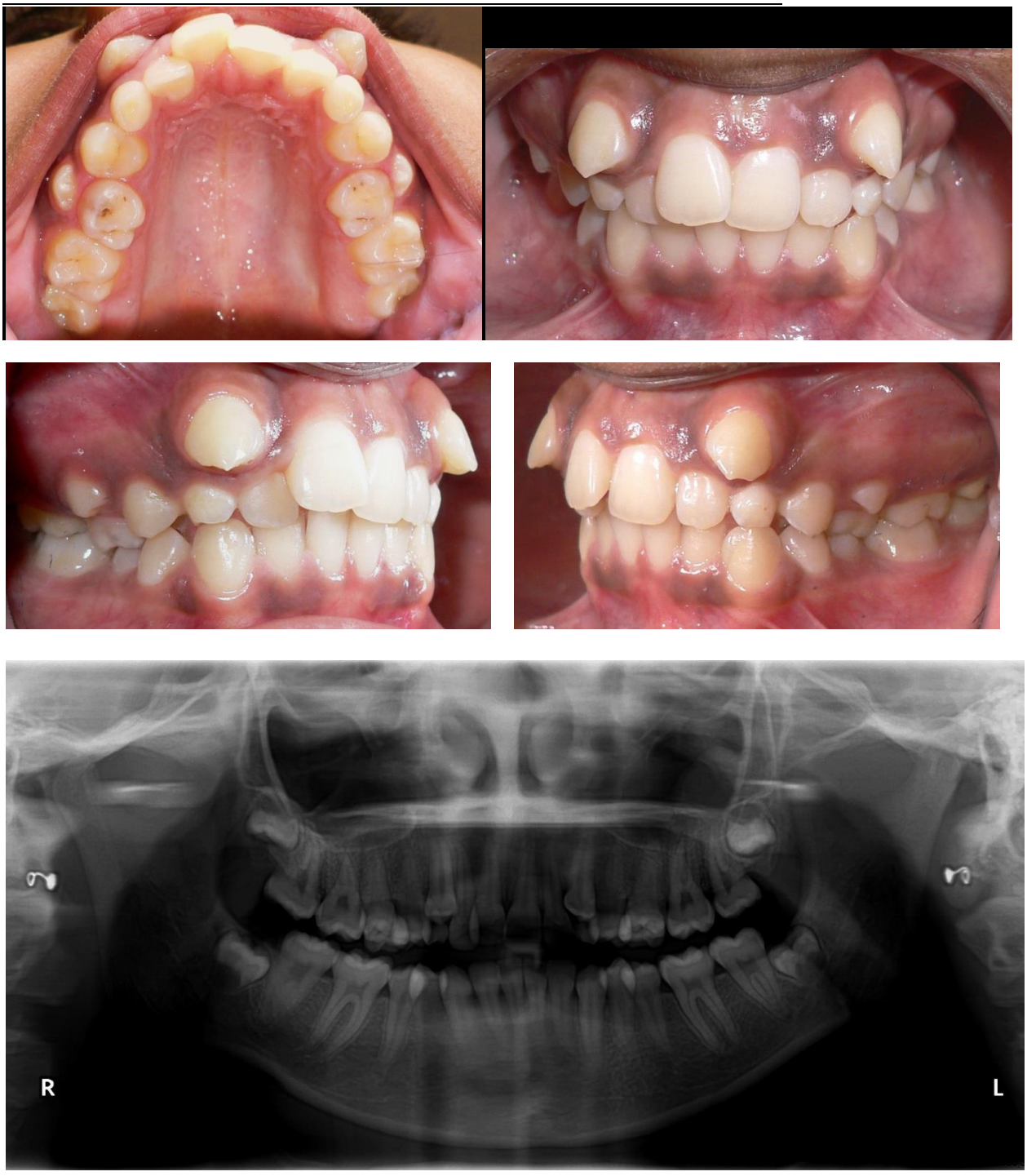


Fig. 2

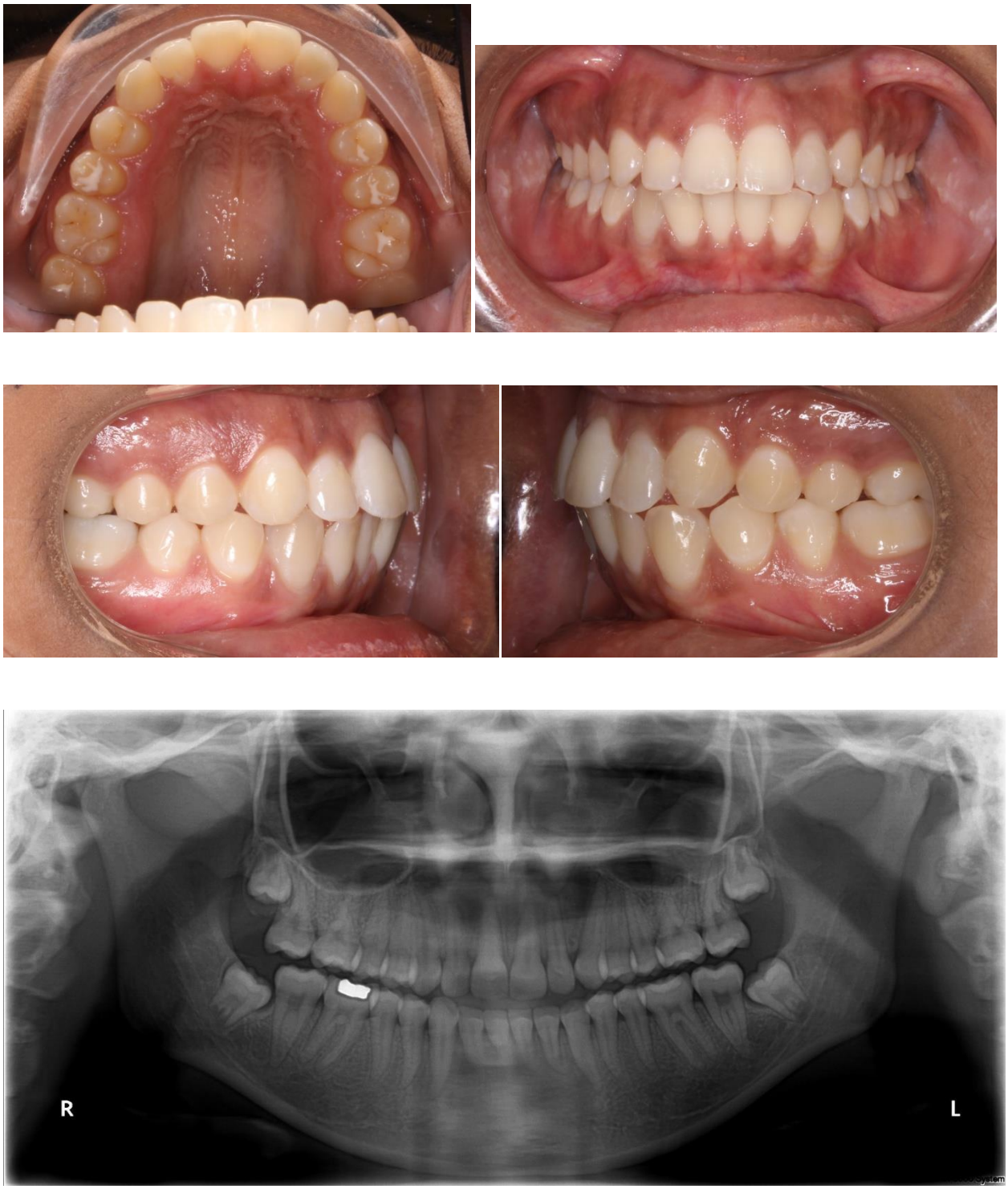
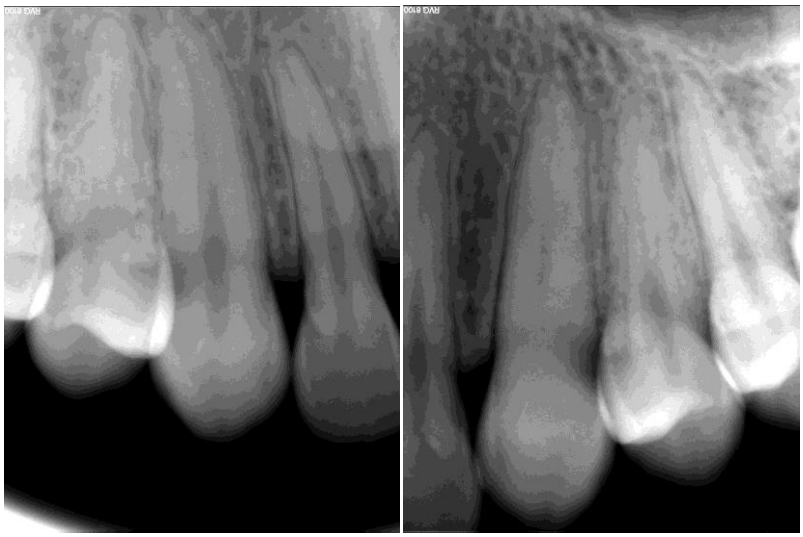


Fig. 3



Patient 2

Patient 2 was a 21-year-old Northern European/Caucasian male who had orthodontic extrusion of ectopic canines 13 and 23 (fig 4). Both canines had not erupted and the cusp tip of each canine was initially at the level of the CEJ of the fully erupted adjacent lateral incisors and premolars. A panoramic radiograph indicated that both canines horizontally overlapped the lateral incisor facially beyond the mesial 1/3 (fig. 4). Both canines were in a buccal position relative to the rest of the arch. Orthodontic extrusion of his maxillary canines began at the age of 14 years 4 months. The orthodontic treatment was completed when he was 16 years 4 months (fig 5). It is unknown whether the technique used was a closed exposure or an open exposure. However, extrusion of both canines required NiTi arch wires and 0.022" self-ligating brackets. He had been taking a prescribe medication (Acutane) during his orthodontic treatment. He claims that he has been wearing his maxillary retainer for approximately 6 months since the completion of his orthodontic treatment and that neither 13 nor 23 had suffered any previous trauma. Patient 2 also stated that he has a history of clenching and grinding his teeth for long periods, and that he does have a history of biting on foreign objects such as pens. He has also bleached his teeth previously at home with no professional oversight. Neither teeth 13 nor 23 had required extensive dental work, such as a crown, bridge, veneer, or RCT. He has not experienced any tooth sensitivity during brushing, biting, chewing, but experiences sensitivity when drinking hot or cold beverages. Intraoral examination revealed no craze lines on tooth 13 but craze lines were present on the buccal surface of tooth 23. Neither 13 nor 23 had any fractures present. Patient has canine guidance on his right side and group function on his left side. MHI contacts were normal. Endodontic evaluation

revealed that neither 13 nor 23 had any apparent colour change in comparison to adjacent teeth. Neither 13 nor 23 had any fistulas. Tooth 13 had no response to heat testing, a delayed response to cold testing, and an EPT score of 72/80. Tooth 23 responded normally to the heat test and to the cold test, with an EPT score of 66/80. Both 13 and 23 had normal responses to percussion tests and normal responses to palpation of the apical cortical plates. A periapical radiograph was taken of tooth 13 (fig 6). Radiographic analysis determined that the pulp status of tooth 13 was not possible to determine and that the pulp chamber had undergone pulp/radicular calcification. There was no radiographic evidence displaying lesions of endodontic origin or periodontal origin.

Fig. 4

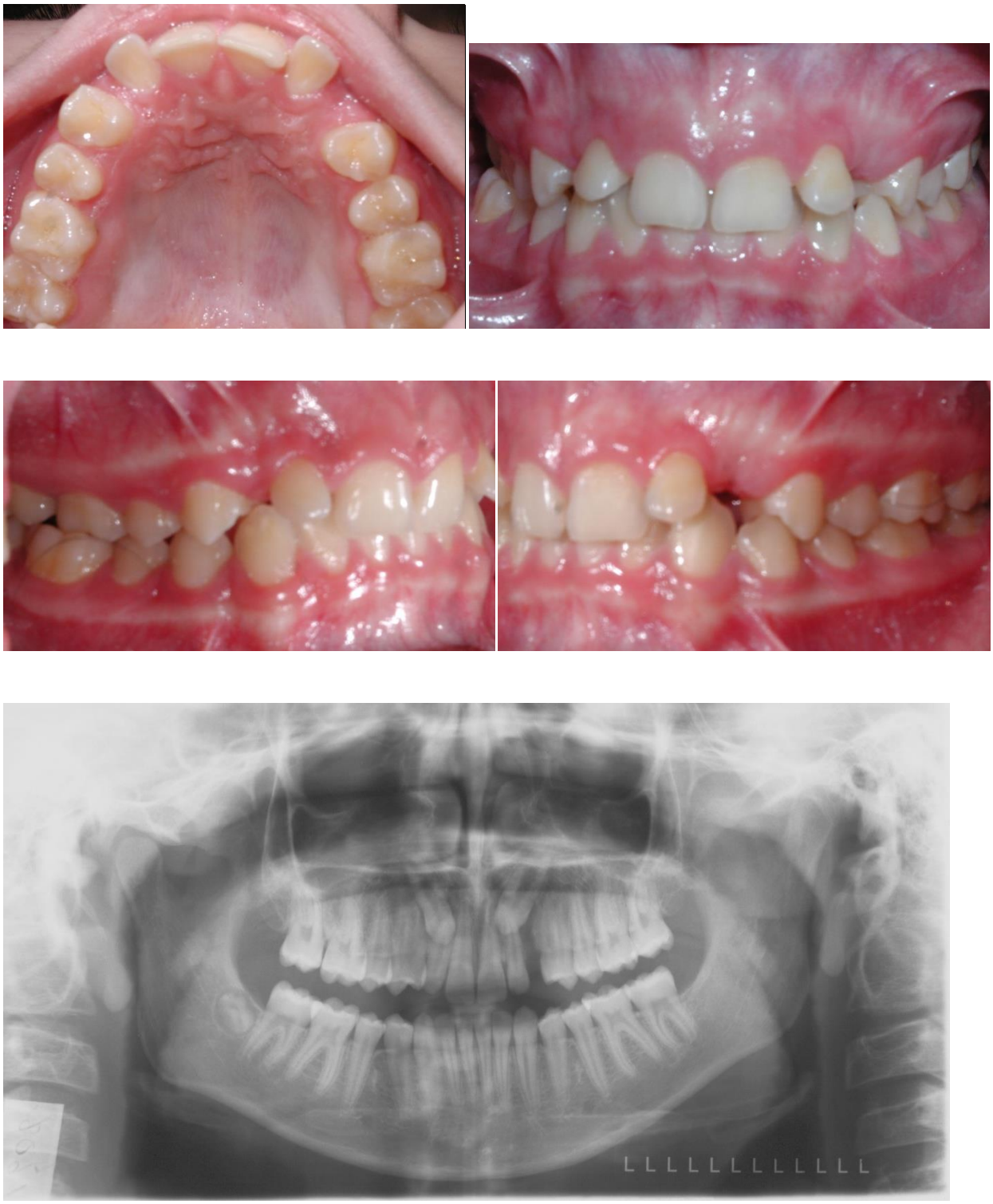


Fig. 5

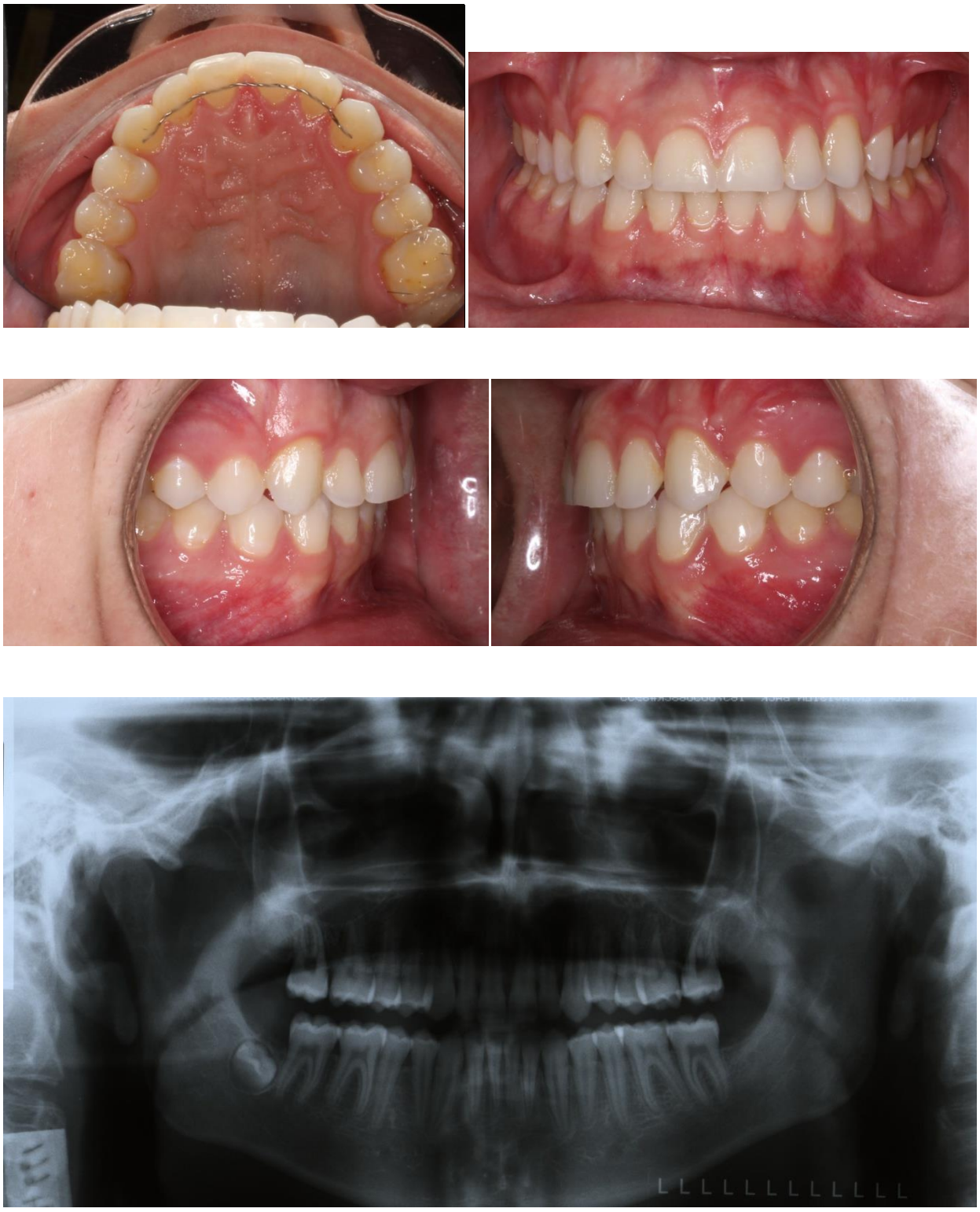
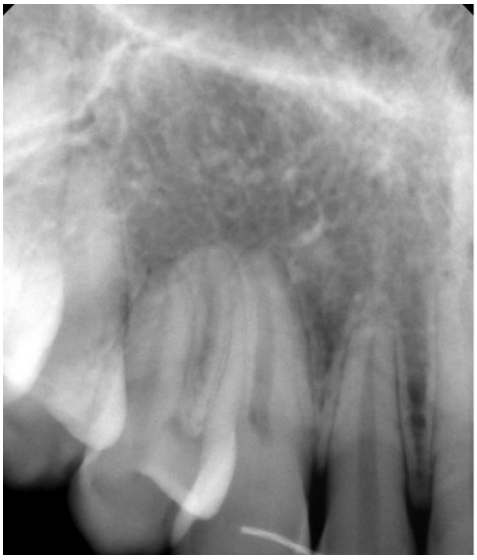


Fig. 6



Patient 3

Patient 3 was a 26-year-old Northern European/Caucasian female who had orthodontic extrusion of ectopic canine tooth 23 (fig 7). The cusp tip of the ectopic canine was initially above the CEJ of the fully erupted adjacent lateral incisor and premolar. A panoramic radiograph indicated that the canine horizontally overlapped the distal lateral incisor with the canine cusp tip reaching the middle 1/3 (fig. 7). Tooth 23 was displaced in a palatal position relative to the rest of the arch. Orthodontic extrusion of her maxillary canine began at the age of 15 years 3 months. Her treatment was completed when she was 18 years 7 months (fig 8). Extrusion of the canine required open exposure technique with a power-chain and conventional brackets. She claims that she has not been wearing her maxillary retainer well and had used it for approximately 6 months after the completion of her orthodontic treatment. Patient 3 also stated that neither 13 nor 23 had suffered any previous trauma but is unsure of whether she has a history of clenching or grinding her teeth for long periods. Patient 3 does not have a history of biting on foreign objects such as pencils, pens, or fingernails. She has bleached her teeth previously at home as per her dental professional's advice. Neither teeth 13 nor 23 had required extensive dental work, such as a crown, bridge, veneer, or RCT. She has not experienced any tooth sensitivity during brushing, biting, chewing, or when drinking and eating hot or cold food. Intraoral examination revealed the presence of craze lines on the buccal surfaces of both 13 and 23. Neither 13 nor 23 had any fractures present. Patient has canine guidance bilaterally. MHI contacts were normal. Endodontic evaluation revealed tooth 23 had

a slight colour discrepancy in comparison to adjacent teeth. Neither 13 nor 23 had any fistulas. Tooth 13 had a normal response to heat testing and cold testing, and a delayed EPT score of 78/80. Tooth 23 had no response to the heat test or to the cold test, and had an EPT score of 80/80. Both 13 and 23 had normal responses to percussion and palpation tests. A periapical radiograph was taken of tooth 23 (fig 9). Radiographic analysis determined that the pulp status of tooth 23 was not possible to determine and that the pulp chamber had undergone pulp/radicular calcification. There was no radiographic evidence displaying lesions of endodontic origin or periodontal origin.

Fig. 7

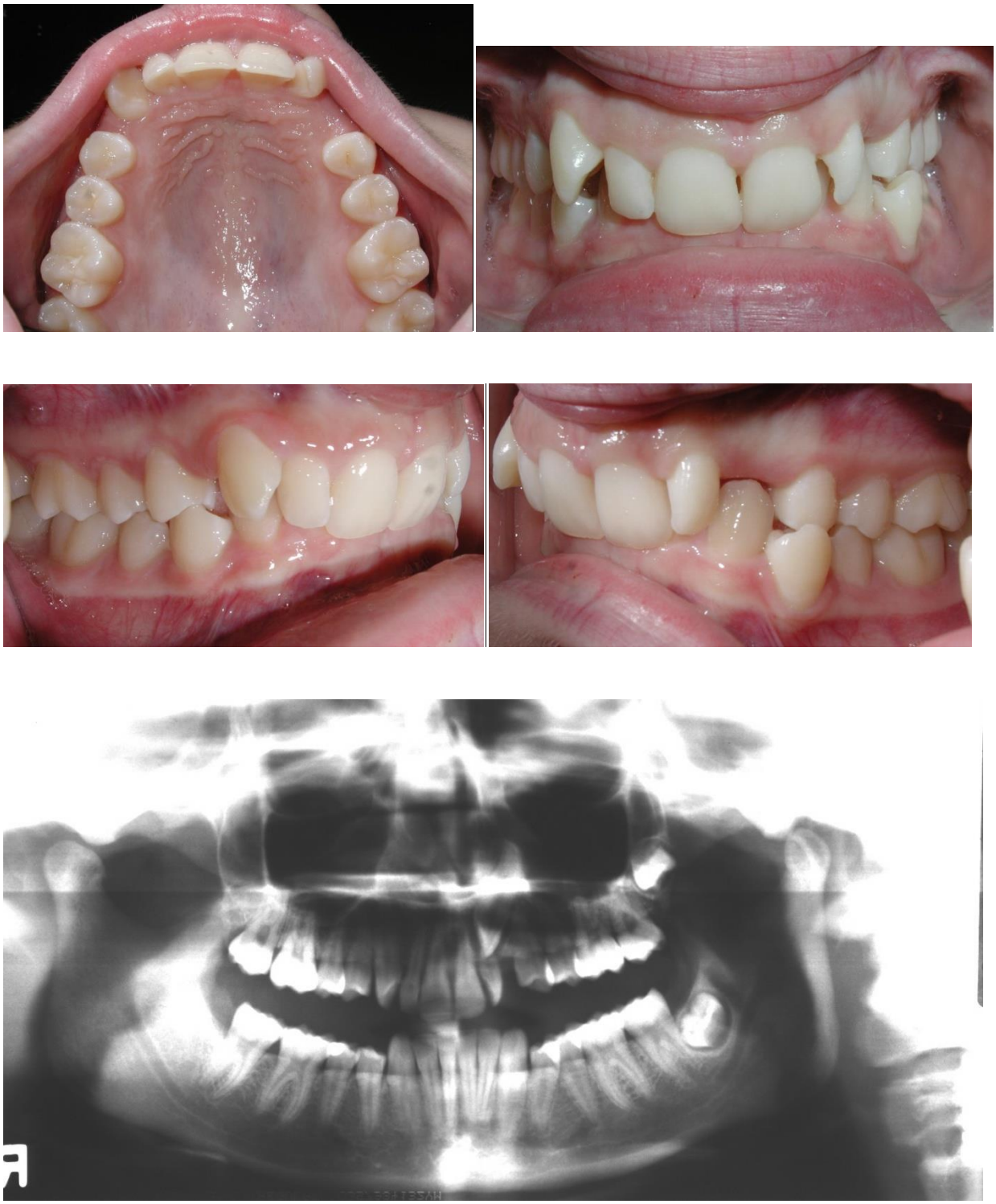


Fig. 8

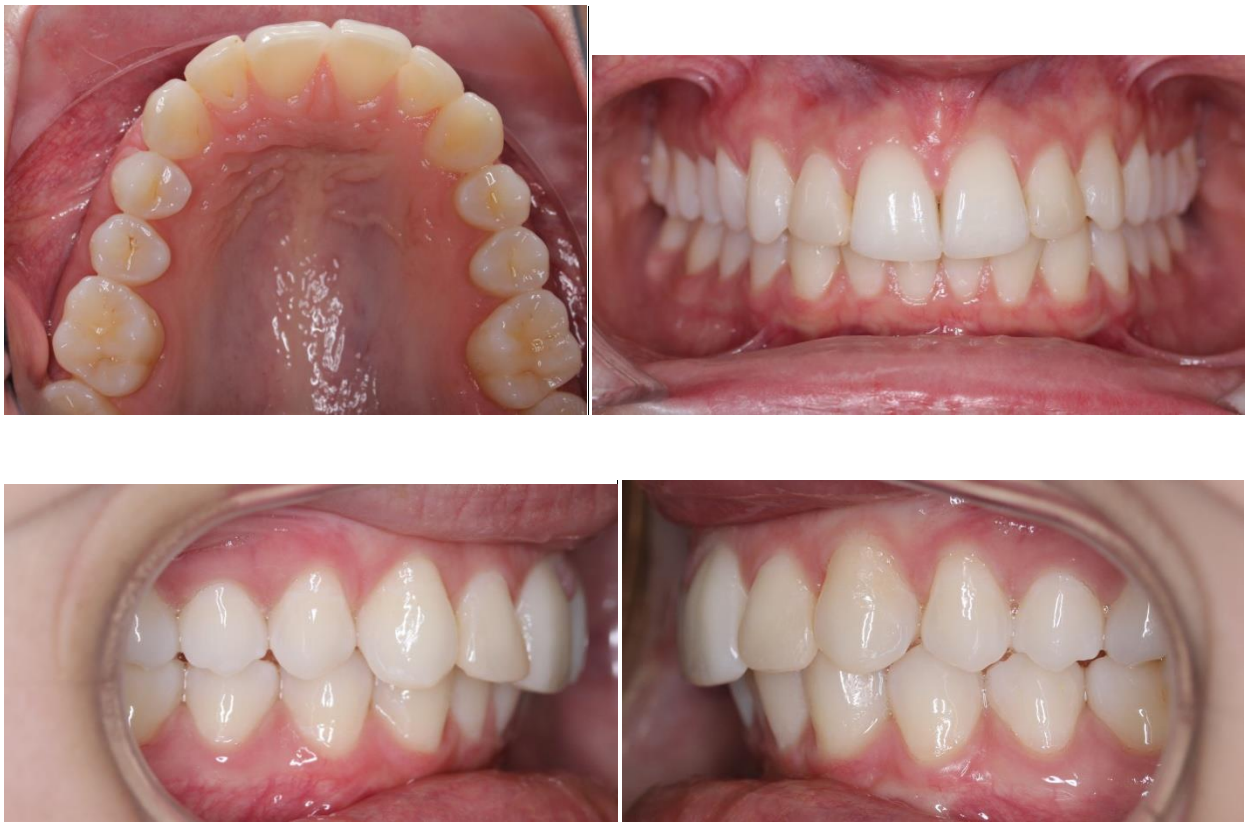
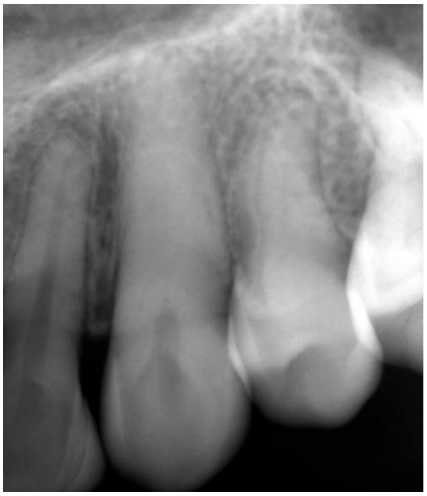


Fig. 9



Patient 4

Patient 4 was a 23-year-old Northern European/Caucasian female who had orthodontic extrusion of ectopic canine tooth 13 (fig 10). The cusp tip of the ectopic canine was initially above the CEJ of the fully erupted adjacent lateral incisor and premolar. A panoramic radiograph indicated that the canine horizontally overlapped the distal lateral incisor with the cusp tip reaching the distal 1/3 (fig. 10). Tooth 23 was displaced in a palatal position relative to the rest of the arch. Orthodontic extrusion of her maxillary canine began at the age of 15 years 9 months and had completed her treatment when she was 20 years 9 months (fig 11). Extrusion of both canines required open exposure technique with NiTi arch wires and 0.018" self-ligating brackets. She had been taking prescribed medications (YAZ - Drospirenone/Ethinyl Estradiol and Simvacor - Simvastatin). She claims that she has been wearing her maxillary retainer well for approximately 2 years and 9 months since the completion of her orthodontic treatment. Patient 4 had recalled that both 13 and 23 had suffered previous trauma. She stated that she has a history of both clenching and grinding her teeth for long periods. Patient 4 does not have a history of biting on foreign objects such as pencils, pens, or fingernails. She has never bleached her teeth for whitening. Neither teeth 13 nor 23 had required extensive dental work, such as a crown, bridge, veneer, or RCT. She has not experienced any tooth sensitivity during brushing, biting, chewing, or when drinking and eating hot or cold food. Intraoral examination revealed the presence of craze lines on the buccal surfaces of both 13 and 23. Neither 13 nor 23 had any fractures present. Patient has group function on her right side and canine guidance on her left side. MHI contacts were normal. Endodontic evaluation revealed that neither 13 nor 23 had any apparent colour change in comparison to

her adjacent teeth. Neither 13 nor 23 had any fistulas. Tooth 13 had a delayed response to heat testing and to cold testing, with an EPT score of 60/80. Tooth 23 had a delayed response to the heat test and a normal response to the cold test. The EPT score was 45/80 for tooth 23. Both 13 and 23 had normal responses to percussion tests and normal responses to palpation of the apical cortical plates. A periapical radiograph was taken of 13 (fig 12). Radiographic analysis determined that the pulp status of tooth 13 was not possible to determine and that the pulp chamber had undergone pulp/radicular calcification. There was no radiographic evidence displaying lesions of endodontic origin or periodontal origin.

Fig. 10

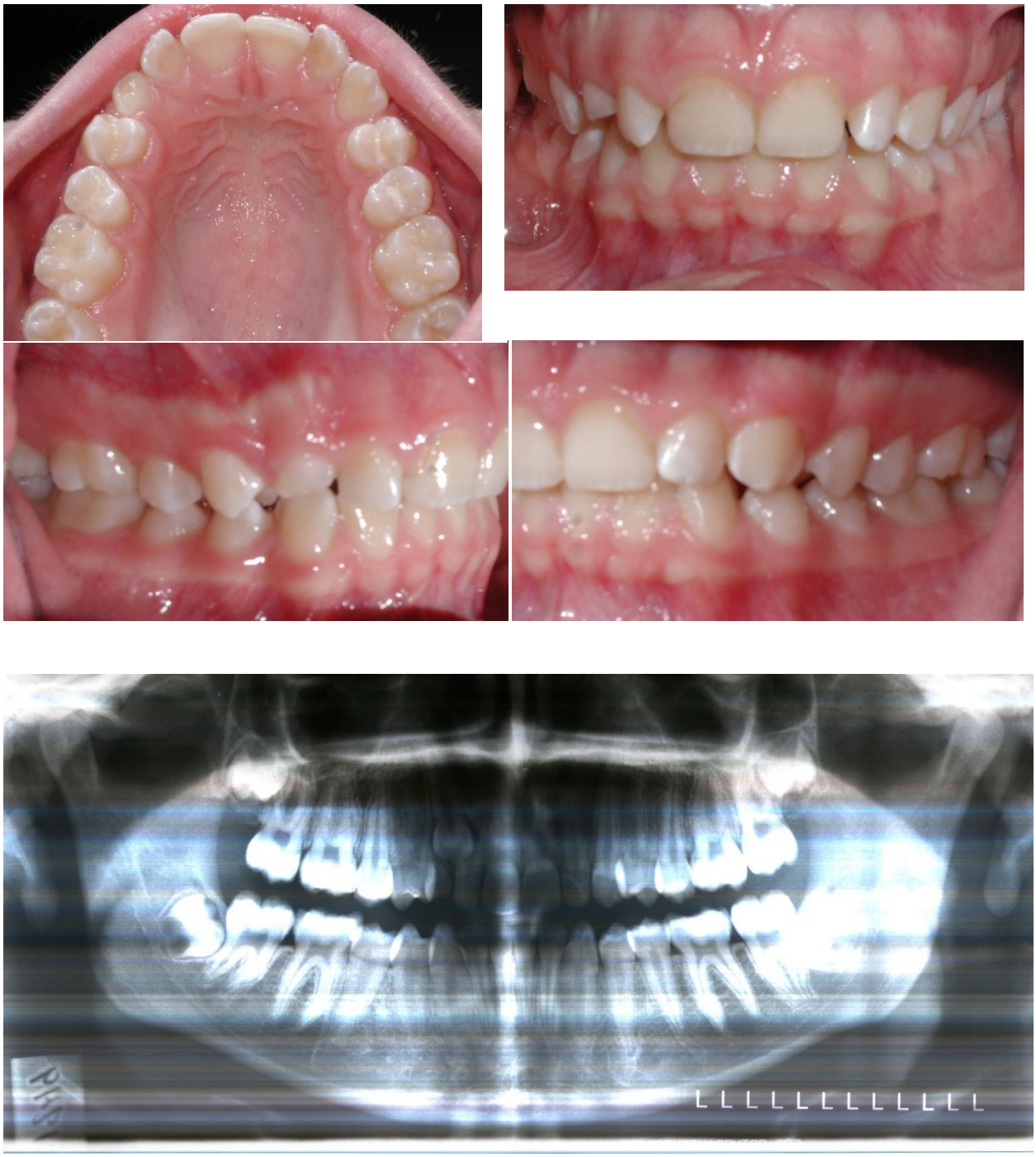


Fig. 11

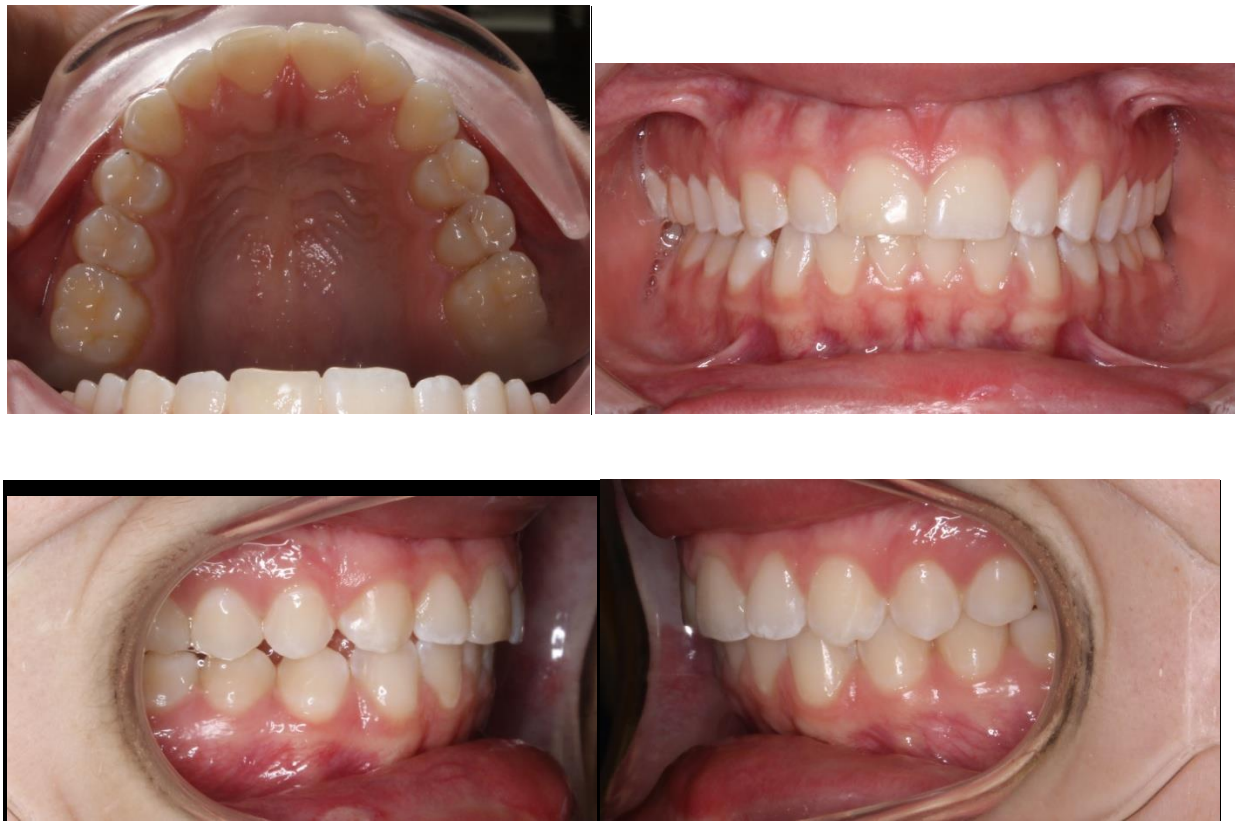
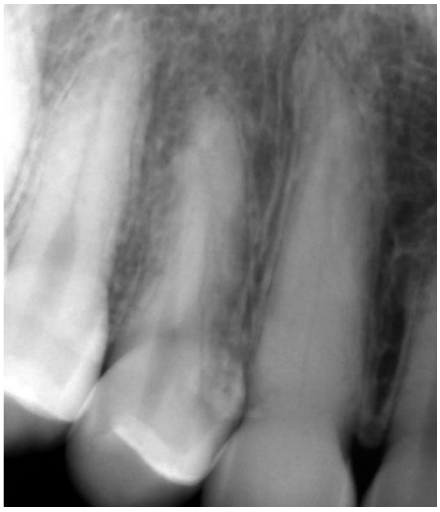


Fig. 12



Discussion

Several studies in orthodontic literature have mentioned that heavy orthodontic forces are capable of generating pulp and periodontal ligament damage, or even cause loss of tooth vitality.[5,7,8,14,17,18] Studies to investigate the pulp reaction following orthodontic extrusion are rare. After completing a PubMed database search using the key words "pulp calcification", "extrusion", and "orthodont", 8 articles were retrieved, but none of them addressed the influence of extrusive orthodontic forces. There is a lack of long-term studies and randomized control trials that can definitively correlate pulpal injury due to orthodontic treatment alone. Furthermore, there is a lack of studies that have been directed at demonstrating the possible effects of orthodontic tooth movement on the pulp.[32] Current literature is limited by a lack of standardization in study protocols and a standardized assessment tool.[26,27,34,35]

Interestingly, the retrospective clinical study conducted by Ferreira et al[32] compared 32 maxillary canines submitted to orthodontic traction to maxillary canines that had never been subjected to any orthodontic treatment. Patients with a history of dental trauma, having an open apex, or having endodontic treatment done on their maxillary canine were not included in their study. The type of traction used was not specified. Their results indicated that within their experimental group of those individuals submitted to orthodontic traction, 14 out of 32 teeth (43.8%) were lacking sensitivity to a cold test with Endo-Frost refrigerating gas. Additionally, their control group only had 1 tooth out of 32 control teeth (3.1%) that displayed a negative response to the cold sensitivity test. They concluded that maxillary canine teeth

submitted to orthodontic traction presented with a higher incidence of negative sensitivity when compared to teeth that have not been treated orthodontically.

Our study obtained a similar result in regard to a lack of tooth sensitivity of ectopic canines treated with orthodontic traction. After utilizing Coltene Hygenic^{R} Endo-Ice spray, lack of sensitivity occurred in 11 out of 22 (50%) maxillary canines. Four of these canines which lacked sensitivity were due to radicular calcification and the other 7 were due to pulp necrosis. This finding is comparable to the results obtained by Ferreia et al.

The exact cause of calcification remains uncertain in the literature.[33] It is suggested that there is often a history of previous tooth trauma prior to orthodontic treatment which may lead to pulp calcification.[5,7,8,14,17,18] The greater the obliteration of the pulp results in a greater risk of pulp vitality loss during orthodontic treatment.[5] In our study, the canines with necrosis did not have a calcified pulp.

We determined that 4 out of 22 (18.2%) ectopic maxillary canines displayed pulp/radicular calcification and 7 out of 22 (31.8%) were identified as non-vital. Two out of 10 (20%) maxillary canines that served as a control were determined to be non-vital. Of the 4 individuals with pulp calcification, only 1 had previous history of trauma, 2 of the patients reported having a history of bruxism, and 3 out of the 4 patients had bleached their teeth. Although bleaching and clenching were common findings among the calcified canines, it was also seen among the non-vital canines and the control canines. Furthermore, non-vital ectopic canines were recorded for 2 patients who were unable to recall whether they had previously experienced trauma to their canines. Although there may be a relationship between previous

trauma and pulp alteration, the present methodology does not allow for an accurate determination of the exact role of trauma (Table 2). The unilateral pulpal alteration seems to indicate that those non-orthodontic factors may not be the main role players.

A limitation of this retrospective study was due to the lack of periapical radiographs prior to orthodontic treatment of the maxillary canine teeth. This negatively impacts the determination that calcification had occurred in relation to extrusive orthodontic forces. The panoramic radiographs lack the ability to display the fine anatomic detail available on intraoral periapical radiographs, therefore, are limited in detecting dental or pulpal issues.[36] Another limiting factor was the lack of information on the pulp statuses of the canines prior to orthodontic treatment. This limited our ability to definitively compare the current pulp status of our samples with their status prior to treatment.

Consequently, the findings herein presented, although intriguing, should be taken with caution. The cases presented in this study have strengthened the importance of understanding consequences of orthodontic extrusion leading to dental complications, such as pulp calcification or loss of pulp vitality. In orthodontics, the clinician must become aware of abnormalities that may occur during treatment, take diagnostic radiographs for accurate monitoring, and follow up with radiographs after the completion of treatment to ensure overall health of their patient's teeth.

Conclusion

Pulp Calcification was found in 4 maxillary canines (18.2% of the sample) that underwent orthodontic treatment to correct their ectopic position. Seven canines (31.8% of

the sample) were reported to have non-vital pulps. No possible cause could be determined due to the type of study design. Further studies are necessary to determine definitive correlations and any cause-and-effect relationship that may exist between orthodontic extrusion and altered pulp status.

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Bibliography

1. Bishara SE. Impacted maxillary canines: a review. *Am J Orthod Dentofacial Orthop*. 1992;101(2):159-171. doi:10.1016/0889-5406(92)70008-X.

2. Aydin U, Yilmaz HH, Yildirim D. Incidence of canine impaction and transmigration in a patient population. *Dentomaxillofacial Radiol*. 2004;33(3):164-169. doi:10.1259/dmfr/15470658.

3. Dalessandri D, Migliorati M, Visconti L, Contardo L, Kau CH, Martin C. KPG index versus OPG measurements: a comparison between 3D and 2D methods in predicting treatment duration and difficulty level for patients with impacted maxillary canines. *Biomed Res Int*. 2014;2014:537620. doi:10.1155/2014/537620.

4. Torres-Lagares D, Hita-Iglesias P, Azcárate-Velázquez F, et al. What Are the Histologic Effects of Surgical and Orthodontic Treatment on the Gingiva of Palatal Impacted Canines? *J Oral Maxillofac Surg*. 2014:2273-2281. doi:10.1016/j.joms.2015.07.002.

5. Proffit WR, Fields HW, Sarver DM. *Contemporary Orthodontics*. 5th ed. St Louis: Mosby; 2013.

6. Becker A, Chaushu S. Surgical Treatment of Impacted Canines: What the Orthodontist Would Like the Surgeon to Know. *Oral Maxillofac Surg Clin North Am*. 2015;27(3):449-458. doi:10.1016/j.coms.2015.04.007.

7. Barwick PJ, Ramsay DS. Effect of brief intrusive force on human pulpal blood flow. *Am J Orthod Dentofacial Orthop*. 1996;110(3):273-279. doi:10.1016/S0889-5406(96)80011-4.

8. Caviedes-Bucheli J, Moreno JO, Ardila-Pinto J, et al. The effect of orthodontic forces on calcitonin gene-related peptide expression in human dental pulp. *J Endod*. 2011;37(7):934-937. doi:10.1016/j.joen.2011.03.035.

9. Woloshyn H, Artun J, Kennedy DB, Joondeph DR. Pulpal and periodontal reactions to orthodontic alignment of palatally impacted canines. *Angle Orthod*. 1994;64(4):257-264. doi:10.1043/0003-3219(1994)064<0257:PAPRTO>2.0.CO;2.

10. Şener S, Cobankara FK, Akgünlü F. Calcifications of the pulp chamber: Prevalence and implicated factors. *Clin Oral Investig*. 2009;13(2):209-215. doi:10.1007/s00784-008-0212-x.

11. Khojastepour L, Bronoosh P, Khosropanah S, Rahimi E. Can dental pulp calcification predict the risk of ischemic cardiovascular disease? *J Dent (Tehran)*. 2013;10(5):456-460.

12. Moss-Salentijn L, Hendricks-Klyvert M. Calcified structures in human dental pulps. *J Endod*. 1988;14(4):184-189. doi:10.1016/S0099-2399(88)80262-0.

13. Sayegh PS, Sayegh FS, Reed AJ. Calcification in the dental pulp. *Oral Surg Oral Med Oral Pathol*. 1968;25(6):873-882. doi:10.1016/0030-4220(68)90165-5.

14. Sundell JR, Stanley HR, White CL. The relationship of coronal pulp stone formation to experimental operative procedures. *Oral Surg Oral Med Oral Pathol*. 1968;25(4):579-589. http://www.ncbi.nlm.nih.gov/pubmed/4868444. Accessed March 10, 2016.

15. Tamse A, Kaffe I, Littner MM, Shani R. Statistical evaluation of radiologic survey of pulp stones. *J Endod*. 1982;8(10):455-458. doi:10.1016/S0099-2399(82)80150-7.

16. Herman NG. Radiographic oddities: Unusual calcifications in the dental pulp. *N Y State Dent J*. 2004;70(4):28-29. http://www.ncbi.nlm.nih.gov/pubmed/15270311. Accessed March 10, 2016.

17. Vandevska-Radunovic V, Kvinnsland S, Kvinnsland IH. Effect of experimental tooth movement on nerve fibres immunoreactive to calcitonin gene-related peptide, protein gene product 9.5, and blood vessel density and distribution in rats. *Eur J Orthod*. 1997;19(5):517-529.

18. Quinn RS, Ken Yoshikawa D. A reassessment of force magnitude in orthodontics. *Am J Orthod*. 1985;88(3):252-260. doi:10.1016/S0002-9416(85)90220-9.

19. Kanzaki H, Chiba M, Sato A, et al. Cyclical tensile force on periodontal ligament cells inhibits osteoclastogenesis through OPG induction. *J Dent Res*. 2006;85(5):457-462. http://www.ncbi.nlm.nih.gov/pubmed/16632761. Accessed March 9, 2016.

20. Hamersky PA, Weimer AD, Taintor JF. The effect of orthodontic force application on the pulpal tissue respiration rate in the human premolar. *Am J Orthod*. 1980;77(4):368-378. doi:10.1016/0002-9416(80)90103-7.

21. Anstendig, H., Kronman J. A histologic Study os pulpal reacion to orthodontic tooth movement in dogs. *Angle Orthod*. 1972;Vol. 42(No. 1):pp. 50-55.

22. Ramazanzadeh B a, Sahhafian A a, Mohtasham N, Hassanzadeh N, Jahanbin A, Shakeri MT. Histological changes in human dental pulp following application of intrusive and extrusive orthodontic forces. *J Oral Sci*. 2009;51(1):109-115. doi:10.2334/josnusd.51.109.

23. Mostafa YA, Iskander KG, El-Mangoury NH. Iatrogenic pulpal reactions to orthodontic extrusion. *Am J Orthod Dentofacial Orthop*. 1991;99(1):30-34. doi:10.1016/S0889-5406(05)81677-4.

24. Brodin P, Linge L, Aars H. Instant assessment of pulpal blood flow after orthodontic force application. *J Orofac Orthop = Fortschritte der Kieferorthopädie Organ/official J Dtsch Gesellschaft für Kieferorthopädie*. 1996;57(5):306-309. http://www.ncbi.nlm.nih.gov/pubmed/8874175. Accessed March 10, 2016.

25. Sübay RK, Kaya H, Tarim B, Sübay a, Cox CF. Response of human pulpal tissue to orthodontic extrusive applications. *J Endod*. 2001;27(8):508-511. doi:10.1097/00004770-200108000-00003.

26. Von Böhl M, Ren Y, Fudalej PS, Kuijpers-Jagtman AM. Pulpal reactions to orthodontic force application in humans: A systematic review. *J Endod*. 2012;38(11):1463-1469. doi:10.1016/j.joen.2012.07.001.

27. Javed F, Al-Kheraif AA, Romanos EB, Romanos GE. Influence of orthodontic forces on human dental pulp: A systematic review. *Arch Oral Biol*. 2015;60(2):347-356. doi:10.1016/j.archoralbio.2014.11.011.

28. Torabinejad M, Walton RE. *Endodontics: Principles and Practice*. St. Louis: Saunders; 2009.

29. Petersson K, Söderström C, Kiani-Anaraki M, Lévy G. Evaluation of the ability of thermal and electrical tests to register pulp vitality. *Endod Dent Traumatol*. 1999;15(3):127-131.

http://www.ncbi.nlm.nih.gov/pubmed/10530156. Accessed March 10, 2016.

30. Villa-Chávez CE, Patiño-Marín N, Loyola-Rodríguez JP, Zavala-Alonso N V, Martínez-Castañón GA, Medina-Solís CE. Predictive values of thermal and electrical dental pulp tests: a clinical study. *J Endod*. 2013;39(8):965-969. doi:10.1016/j.joen.2013.04.019.

31. Kokich VG. Surgical and orthodontic management of impacted maxillary canines. *Am J Orthod Dentofacial Orthop*. 2004;126(3):278-283. doi:10.1016/S0889540604005268.

32. Ferreira LL, Macluf Filho E, Rodrigues CL, Bueno CE da S, Cunha RS. Lack of pulp sensitivity in maxillary canines submitted to orthodontic traction: a retrospective clinical study. *RSBO*. 2013;10(1):29-33. http://revodonto.bvsalud.org/scielo.php?script=sci_arttext&pid=S1984-56852013000100006. Accessed March 10, 2016.

33. Popp TW, ??rtun J, Linge L. Pulpal response to orthodontic tooth movement in adolescents: A radiographic study. *Am J Orthod Dentofac Orthop*. 1992;101(3):228-233. doi:10.1016/0889-5406(92)70091-N.

34. Katrak P, Bialocerkowski AE, Massy-Westropp N, Kumar S, Grimmer K a. A systematic review of the content of critical appraisal tools. *BMC Med Res Methodol*. 2004;4:22. doi:10.1186/1471-2288-4-22.

35. Sanderson S, Tatt ID, Higgins JPT. Tools for assessing quality and susceptibility to bias in observational studies in epidemiology: A systematic review and annotated bibliography. *Int J Epidemiol*. 2007;36(3):666-676. doi:10.1093/ije/dym018.

36. White SC. *Oral Radiology: Principles and Interpretation*. 6th editio. St. Louis: Mosby; 2009.